Ministry for the
Environment
Manatū Mō Te Taiao

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Air Quality

Preliminary Assessment of Potential Human Health Indicators of Air Quality

Prepared for the Environmental Performance Indicators Programme of the Ministry for the Environment by:

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Signposts for sustainability

Prepared for:

The Ministry for the Environment
Environmental Performance Indicators Programmeme

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Comments by the Ministry for the Environment

The Ministry is pleased to release this report – *Preliminary Assessment of Human Health Indicators of Air Quality,* by Prof Tord Kjellstrom. Prof Kjellstrom was commissioned to prepare this report in response to submissions[1] requesting the Ministry to develop human health effects indicators of air quality.

The Ministry thanks Prof Kjellstrom for his work. He provides a comprehensive review of the current international and New Zealand research into human health indicators of air quality and studies linking health effects to air pollution. Prof Kjellstrom makes some very useful recommendations for further investigations needed to develop human health indicators.

Having reviewed the report and recommendations, the Ministry considers that the fundamental research needed to develop a particular national human health indicator of air quality and appropriate monitoring method is beyond the scope of the EPI Programme. Therefore no human health indicator of air quality is proposed at this time.

The Ministry will use Prof Kjellstrom's report and recommended investigations to develop research priorities into the development of health indicators and for the current Review of the Ambient Air Quality Guidelines. The report has also been forwarded to the Ministry of Research Science and Technology to assist their task of prioritising research needs.

The Ministry will assess of the state of the air environment and its potential to affect human health using the Stage 1 "State" Indicators - nitrogen dioxide, carbon monoxide, particles (PM10), ozone and sulphur dioxide and Stage 2 Indicators (benzene, PM2.5) as they become available. As further research on human health indicators and monitoring methods is carried out, the Ministry will reconsider whether human health indicators can be included.

Please provide any comments on this report to the Ministry for the Environment by 1 September 1999:
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[1] Submissions made on *Environmental Performance Indicators - Proposals for air, freshwater and land* (MfE, 1997)

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Abbreviations

COHb	Carboxy-haemoglobin in blood
ESR	Institute of Environmental Science and Research
FEV1	Forced expiratory volume in one minute
MfE	Ministry for the Environment
NIWA	National Institute for Water and Air research
PEFR	Peak expiratory flow rate
PM	Particulate matter
PM10	Particulate matter in air with aerodynamic diameter < 10 um
TSP	Total suspended particulates in air
WHO	World Health Organisation

1 Introduction

1.1 Report Objectives

The objectives of this synoptic report are:

- to consider the potential for the development and application of human health indicators that can provide information on the effects of air pollution in New Zealand,
- to identify the indicators most likely to be useful in sustained monitoring
- to recommend further research and development in this field.

The report is based on an initial scoping exercise, primarily focussing on the issues that need further analysis rather than drawing definite conclusions. The exercise arose as a result of submissions made on the Air strand of the Ministry for the Environment's (MfE) Environmental Performance Indicators Programme (EPI Programme). Relevant MfE reports and submissions to the Ministry were made available to the author. In addition, published reports on air pollution health effects in New Zealand and selected overseas reports were consulted.

1.2 Air quality in New Zealand

The air pollution situation in New Zealand has recently been reviewed in detail in the Ministry for the Environment State of the Environment report (MfE, 1997). It provides details of the trends over time and the most extreme pollution situations for the following key pollutants:

- particulate matter (TSP and PM10)
- sulphur dioxide
- carbon monoxide
- nitrogen dioxide (and other oxides of nitrogen)
- ozone (photochemical smog)

In addition, it contains information on other important air pollutants:

- lead (currently mainly a problem of lead in paint on houses)
- hydrogen sulphide (local issue in thermal areas)
- benzene (emerging problem since lead-free petrol was introduced)

These are also the most likely pollutants to be of significance to human health. Generally, air pollution concerns are confined to the larger urban areas where the concentration of population leads to high emissions and elevated air concentrations of pollutants from motorcars, home heating and industry. The actual concentrations will be very much dependent on weather variables, such as wind speed, temperature inversions and rainfall. In certain localities there are significant pollution of other type, such as the natural hydrogen sulphide pollution in Rotorua (Wegmuller and Petersen, 1998), which on some days may be considered an odour nuisance.

Knowledge about the air pollution situation in New Zealand is constrained by the limited extent of air quality monitoring that has been carried out. In Auckland and Christchurch monitoring for TSP has been carried out at a small number of fixed sites since the 1960s (MfE, 1997). Sulphur dioxide and lead has been monitored at selected sites in the same cities since the 1970s. Other pollutants have only started to be monitored in the last decade. The Resource Management Act (RMA) which came into force in 1991 has been instrumental in encouraging a number of regional councils to start air quality monitoring activities. Currently, all of the 16 regions are involved in some type of air monitoring (MfE, 1997), mainly of particulate matter and NO_2. Air monitoring is carried out at fixed sites and few studies have attempted to establish the geographic distribution of air pollution levels within the regions or cities monitored.

In spite of these shortcomings a number of recent risk assessments of air pollution effects in New Zealand have produced estimates of the numbers of people affected. Such estimates can be used to set priorities for which pollutants and health effects to focus on. For instance, the particular problems of air pollution in Christchurch have been the focus of a number of studies and risk assessments (CRC, 1997). Because of the type of home heating systems used and the frequent occurrence of temperature inversion conditions during cold and still winter nights, the air pollution problems became a concern of community groups already in the 1930s.

Particulate air pollution monitoring started in Christchurch in the 1960s, when 24-hour average TSP levels up to several hundred ug/m^3 were frequently measured in the winter. Background 24-hour average levels in urban areas are now usually in the range 20 – 50 ug/m^3 (MfE, 1997) but during winter days in Christchurch the 24-hour levels typically range from 40 to 80 ug/m^3 with occasional excursions above 100 ug/m^3. In Auckland the levels are generally lower. They have decreased by about 50 % since 1970 (MfE, 1997; ARC, 1997). In central Christchurch, during each winter in the 1990s, the 24-hour PM10 guideline of 50 ug/m^3 adopted by the Canterbury Regional Council was exceeded during 25 – 35 days (CRC, 1997).

The Canterbury Regional Council’s emissions inventory has shown that home heating with coal and wood is the main contributor to particulate air pollution during the winter nights when the pollution is at its’ peak. There is a strong correlation between levels of PM10 and carbon monoxide, which emerges from the same home heating sources. The situation is improving slightly over time, but health concerns remain. The Canterbury Regional Council has proposed a regional policy to further reduce the air pollution problem by phasing out the use of open fires burning wood or coal for home heating (CRC, 1997). The potential health effects of PM and CO may occur after short-term exposure (over a few hours) as well as after long-term exposure. The monitoring of these air pollutants would therefore need to include both short- and long-term human health indicators.

In Auckland, the PM air pollution levels are also decreasing (ARC, 1997) and a similar pattern is seen for sulphur dioxide (MfE, 1997). Only occasionally do the PM10 levels exceed 50 ug/m^3, even in the winter in Penrose, the monitoring site with the highest levels (ARC, 1997). The greatest improvement in air quality over the last 10 years has been seen for lead (MfE, 1997). The decrease of air lead is parallel to the decrease in the total consumption of lead in petrol since petrol with reduced lead level was introduced in 1987 (MfE, 1997). In 1996 the addition of lead to petrol was made illegal. The most dramatic reductions were seen in the data from Queen Street, Auckland, where the average annual air lead concentration in 1985 was 1.4 ug/m^3 and in 1996 it was below 0.2 ug/m^3 (MfE, 1997, based on data from ESR).

Since lead addition to petrol was reduced, the chemical composition of car exhausts has changed and the emissions of aromatic hydrocarbons have increased. Because of a concern for increasing exposures a first study of emissions and air concentrations in different New Zealand cities was carried out in 1994-1995 (Narsey and Stevenson, 1997). An assessment of the toxicity of the different compounds and the measurements of concentrations in air concluded that Benzene was the aromatic pollutant of greatest concern as it is associated with an increased leukemia risk after long-term exposure (WHO, 1993). This risk develops over long-term exposure only, so the most useful air monitoring indicators are long-term averages, e.g. annual average concentrations.

The first study (Narsey and Stevenson, 1997) estimated benzene levels from emissions and carried out a number of short-term measurements, which indicated that there was potentially an increase of benzene levels over time and that annual averages could be in the range 2 - 12 ug/m^3 in different cities. A subsequent study (Stevenson and Narsey, 1998) carried out in 1996-1997 used longer-term monitoring methods (passive samplers) and estimated the equivalent annual exposure levels for different population groups. The estimated levels ranged from 1 - 11 ug/m^3 for typical residents, working or non-working, with the lowest exposures among non-smoking people with very short daily periods spent in motorcars. This is due to the high exposures caused by active or passive smoking and the high benzene levels found in motorcars while driving (Stevenson and Narsey, 1998).

Driving as an occupation created the highest estimated annual exposures levels (10 - 49 ug/m^3). A detailed study of the side of a busy Auckland road (Kuschel et al., 1998) showed an estimated annual average of 65 ug/m^3, indicating the type of exposures potentially occurring at sidewalks. This study also highlighted the strong influence of weather (the number of hours of calm wind) on benzene levels and the good correlation between air concentrations of different pollutants. This could be utilised to identify "sentinel" pollutants that could be measured instead of benzene to indicate benzene levels.

By comparing the results of air quality monitoring with the New Zealand air quality guidelines one can get a rough idea of which pollutants may be of greatest importance for health indicators development. The New Zealand guidelines have generally been based on WHO air quality guidelines (WHO, 1987). Such comparison also has to bear in mind that WHO is in the process of revising the guidelines (WHO, 1995). Regional councils are in a position to develop their own air quality management targets (such as the one developed by Canterbury Regional Council in 1997; 50 ug PM10/m^3 per 24 hours instead of the MfE national guideline of 120 ug/m^3). For PM10 the WHO expert group recommended no specific guideline value (WHO, 1995) as it was considered that there was no threshold for the effects on morbidity and mortality from PM. WHO recommends that decisions about air quality management for PM should be based on a health risk evaluation by the implementing authority (WHO, 1995).

Based on the data available (MfE, 1997) it appears that PM air pollution would be the greatest concern in terms of health effects, as existing guidelines are regularly exceeded in certain locations during the worst season. Excursions above the air quality guidelines occur only occasionally for carbon monoxide, nitrogen dioxide and ozone. The current monitoring results for aromatic hydrocarbons in air (particularly benzene) is indicating a new health concern. No air quality standard for benzene has been established in New Zealand, and the international trend has been to base risk management on health risk calculations as the key health concern is cancer (leukemia). However, in certain countries annual average benzene concentration expressed as an "air quality goal" or "recommended target level" have been established: e.g. UK 16 ug/m^3, USA 3.2 ug/m^3, Sweden 1.2 ug/m^3 (Stevenson and Narsey, 1998). The measured and estimated levels in New Zealand urban areas are, in some places, higher than these values.

It is pointed out in the State of New Zealand's Environment report (MfE, 1997) that the monitoring results for PM pollution depend on the methods used for monitoring and that not all of the figures presented in different reports are directly comparable. One notable observation is that the equipment used for TSP measurements in New Zealand gives results 25-50 % lower than the standard equipment used overseas (Graham and Narsey, 1994). The implication of this may be that the health risks associated with a measured TSP level of 100 ug/m^3 here would be the same as the risk at 125 – 150 ug/m^3 measured in other countries.

The monitoring data can be complemented by modeling based on emission inventories and meteorological variables. An example of this approach is the recent estimation of PM10 levels in Christchurch based on a "box model" with a certain volume of air into which the emissions are introduced (Fisher et al., 1998). There was a remarkable agreement between modelled and observed data. Validation of this approach at other times in the same area or in other locations would be of great interest. This study was used to analyse temporal variations. Other models can create better spatial variation analysis. Such spatial models have been used for CO and NOx in Auckland (Wegmuller et al., 1998) and for Hydrogen Sulphide in Rotorua (Wegmuller and Petersen, 1998). In future air quality indicators work as well as in future epidemiological studies these temporal and spatial models are likely to become very useful tools.

1.3 The concept of environmental health indicators for air quality

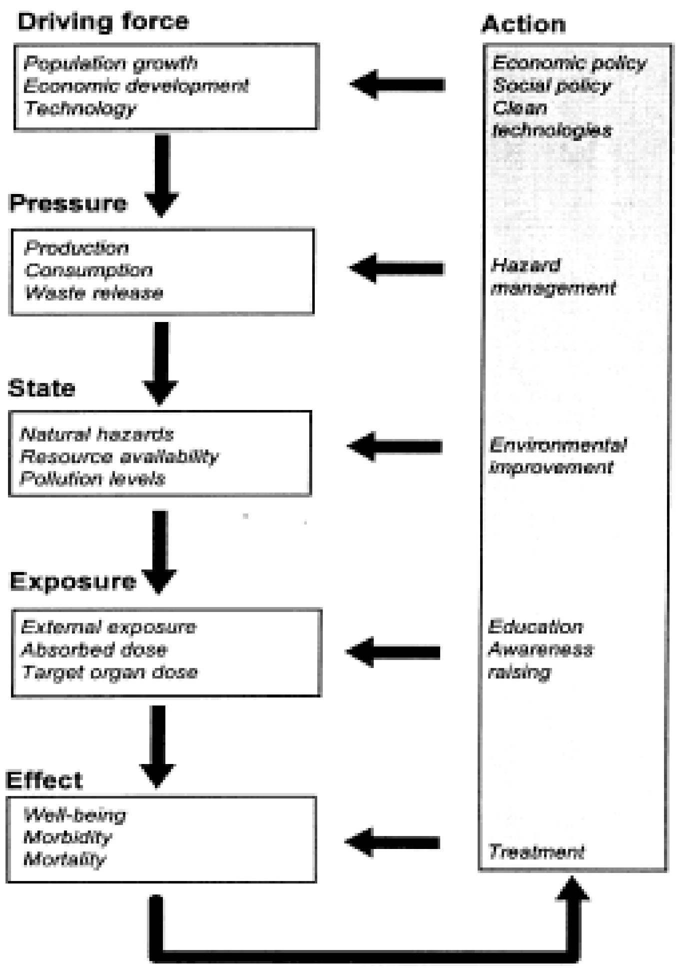
The EPI Programme (MfE, 1997b) was established based on the principles of the OECD *Pressure-State-Response* model (PSR). *Pressures* represent the emissions of pollutants into the environment and the activities that create these emissions. The *State* represents the condition of the environment and how the environment is changed by the Pressures. In the OECD model this includes the whole ecosystem, including human beings living in the environment under study. The *Response* represents the actions taken by humans (or society) to control the Pressures in order that the State of the environment is protected or restored. The OECD model was developed based on an environmental economics paradigm and was not intended to describe the links between 'cause' and 'effect' in detail.

This model has the drawback that it makes no clear distinction between the concentrations in the environment of a pollutant, the exposures to humans and other biota it leads to, and the actual damage caused to humans and other biota by the pollution[2]. In order to better analyse the human health risks of environmental conditions, a framework that better represented the 'cause-effect' relationships between human activities, environmental change and human health was developed (Corvalan et al., 1995). This framework includes *Driving Forces* that lead to *Pressures*. The *State* is divided into the environmental quality (*State*), the *Exposures* (to humans) and the *Effects* (in humans), and the Response has been labelled *Actions* to highlight an active role of society rather than a passive response. The framework outlined in Figure 1 was termed the DPSEEA model (Kjellstrom and Corva1an , 1995; Corvalan et al., 1995). It has retained the basic elements of the OECD PSR-model, but it is more advanced in that it facilitates a

[2] Comments by MfE: *"Although we use the Pressure-State-Response model as a basis for selecting indicators, we recognise the need to develop and link indicators from other groups outlined in the DPSEEA model. This why we commissioned this report on health "effects" and "exposure" indicators and are developing "driving force" indicators in the Transport Strand".*

more intuitive understanding of the links between steps in a causal 'chain' and the potential actions to break the chain. For a number of driving forces several different health effects may occur and these effects may be associated with several different exposures. Thus, the framework would take the shape of an inter-linking 'web' rather than a straight 'chain' for certain environmental health problems.

Figure 1. The DPSEEA framework to define environmental health indicators



The framework makes it possible to better identify the distinction between indicators of different proximity to the health effects of concern. For example, blood lead is an excellent *Exposure* indicator, which gives additional information to just using air lead concentration (*State*) as an indicator of potential health risk. The emissions of lead from motor vehicles (when lead is added to petrol - it is not in New Zealand) is a *Pressure* indicator more indirectly linked to health risk, and the policy of allowing or disallowing lead addition to petrol is a *Driving force* indicator most remote from the actual health effect of lead. The DPSEEA framework was adopted by WHO for use as the basic method of presenting its' progress report "Health and Environment in Sustainable Development" (WHO, 1997) for the special session of the General Assembly of the United Nations five years after the Rio Earth Summit.

The proposed environmental performance indicators for air from the MfE (Table 1) are defined mainly as environmental concentrations (*State*) of specific air pollutants, but visibility is a derived *State* indicator of air concentrations of several pollutants and lichen diversity/coverage is an ecological *effect* variable.

Table 1. Confirmed environmental performance indicators for Air

Stage 1 – ready to implement	**Stage 2 – further development required**
Particulate matter (PM10) Carbon Monoxide (CO) Nitrogen dioxide (NO_2) Sulphur dioxide (SO_2) Ground level ozone (O_3)	Benzene Particulate matter (PM2.5) Lichen diversity/coverage Visibility

Source: MfE (1998, in press)

Other examples of indicators of relevance to air pollution effects include those developed by the United Nations Commission on Sustainable Development (UN, 1996), which used the PSR framework (after renaming Pressures to Driving Forces). In the section dealing with 'the atmosphere' an undefined set of *State* indicators ('ambient concentrations of pollutants in urban areas') was proposed and one *Response* indicator ('expenditure on air pollution abatement') was proposed. Under 'health indicators' no specific indicator related to air pollution was proposed. Some of the *Driving force* indicators are of importance to air pollution and its' health effects; e.g. population growth rate, per capita consumption of fossil fuel by motor vehicle transport, and emissions of sulphur oxides and nitrogen oxides.

WHO has established key health indicators to be used in monitoring of progress towards "Health for All" (WHO, 1996). Unfortunately, none of these have specifically targeted air pollution effects, even though more than 1000 million people are exposed to severe urban air pollution (WHO, 1997) and an additional several hundred million people are exposed to high indoor air pollution from cooking fires and stoves.

Attempts by WHO to establish a list of Environmental Health Indicators for environmental health management programmes at local and national level failed to reach agreement on a standard set of indicators (WHO, 1998, in press) because each country has its' own priorities. The DPSEEA framework was seen as a useful way of structuring the types of indicators. It was also concluded that a menu of potential indicators from which each country chooses its' preferred indicators was the best approach (WHO, 1998, in press). The menu of direct relevance to air pollution is presented in Table 2.

Table 2. Examples of indicators related to air pollution

Pressure	* Emissions of specific air pollutants * Energy consumption from coal or wood * Traffic volume
State	* Average concentrations of specific pollutants in air * Peak concentrations (severe pollution episodes)
Exposure	* Proportion of population exposed in excess of guideline * Number of people exposed in excess of guideline * Number of people exposed within a certain concentration range * Product of exposure concentration and duration in specific period * Biological monitoring data (e.g. blood lead; blood COHb)
Effects	* Mortality and morbidity in lung disease and heart disease * Respiratory symptoms (particularly in asthmatics) * Eye irritation

Source: WHO (1998, in press)

2 Links between air quality and human health

2.1 International reports linking health effects and air quality

A recent international expert group review (WHO/NILU, 1996) quantified health effects in humans (Table 3). In order to establish a suitable health indicator, it is of importance that the indicator variable has a quantitative relationship to the air pollution exposure. Quantitative relationships were found for particulate air pollution (PM10 and black smoke) SO_2 and ozone (Table 3), whereas for NO_2 no agreed quantitative relationships were established. The type of effects reported in some of the studies of NO_2 exposure were "nuisance effects" and symptoms reported in diaries, pulmonary function change or hospitalisations for respiratory diseases (WHO/NILU, 1996). NO_2 often occurs together with other pollutants (e.g. particulate matter) making it very difficult to draw conclusions about which pollutant had the major causative role. In addition, many of the epidemiological studies of nitrogen dioxide provided conflicting results (WHO/NILU, 1996). Controlled human experiments have shown a 5-10 % decrease in lung function (PEFR) of asthmatics exercising in air with 560 ug NO_2/m^3, and increased airway responsiveness in asthmatics may occur at 190 ug NO_2/m^3 (Follinsbee, 1992).

Table 3. Percent change of risk of health effect associated with an increased exposure to selected air pollutants (ranges in different studies)

Health Effect	**PM_{10} 1.2)**	**BSmoke 1)**	**SO_2 1)**	**0_3 3)**
Total Mortality	0.6 (0.3 – 1.5)	0.6 (0.4 – 0.8)	0.6 (0.3 – 1.5)	(1-4)
Respiratory Mortality	1.2 (0.8 – 3.7)	0.9	1.2 (0.3 – 3.3)	
Cardiovascular Mortality	0.8 (0.8 – 1.8)	0.7	0.6 (0.3 – 1.5)	
Hospital/Emerg. Room Adm. (respiratory dis.)	0.5		0.5 5) (0.1-0.9)	6 (2-10)
Bronchodilator use 6)	2			

Upper Resp. Symptoms	1.2			
Lower Resp. Symptoms	1.3			
Cough	1.3			
Symptom Exacerbation among asthmatics	5			
Pulmonary Function change (% of FEV1)	-0.1 (0- -0.25)			-1.5 (-1- -2)

1) Per 10 $\mu g/m^3$ 24-hour mean PM_{10}, black smoke (BSmoke) or $S0_2$
2) Conversion used: $PM_{10} = 0.55$ TSP
3) Per 100 $\mu g/m^3$ of maximum daily 1-hour mean
4) No quantification available for NO2
5) In age of 65^+ years
6) In asthmatics

Source: adapted from WHO/NILU, 1996

In addition to the pollutants listed in Table 3, carbon monoxide was assessed for the new WHO air quality guidelines (WHO, 1995) and quantitative relationships have been established between the COHb level in blood and different health effects. At COHb levels in the range of 2-7 % exercise capacity decreases and ischaemic electrocardiogram (ECG) changes may occur in people with coronary artery disease. At levels of COHb above 5 % increased arteriosclerotic heart disease mortality occurred in tunnel workers and at this level neuro-behavioural effects, such as impaired coordination and driving ability, was reported (WHO, 1995). After 8 hours exposure to 11.5 mg/m^3 CO a person doing sedentary work would reach a COHb level of 1.5 % (WHO, 1987), and a person doing heavy work would reach a level of 1.7 %. The potential for a quantitative relationship between air levels and health effects for CO is there.

One recent study (Burnett et al., 1998) reported an association between CO in air and daily mortality in Toronto. A 1.4 ppm (1.5 mg/m^3) increase of CO between different days was associated with an increase of total mortality of 7 % (95% conf. Int. 5.3 - 8.7 %). However, similar to most studies of this type, the population was exposed to a mixture of PM, CO and other pollutants. CO alone may not be the cause of the increased mortality.

During recent years an increasing number of epidemiological studies, mainly carried out in the USA, have shown associations between relatively low PM air pollution levels and increased mortality and morbidity (Schwartz, 1994; WHO/NILU, 1996; USEPA, 1998). Different reports show increases in total mortality, respiratory mortality, cardiovascular mortality, total hospital admissions, asthma admissions, chronic obstructive pulmonary disease (COPD) admissions, and emergency room visits, as well as increased prevalence of respiratory symptoms and school absences.

These findings are in accordance with the findings of the detailed study of the first scientifically reported major air pollution public health crisis in London in 1952 (HMPHS, 1954). The PM air pollution level exceeded 1000 ug/m^3 for a week and the excess number of deaths was approximately 4000. The greatest mortality rate increase occurred among the elderly (over age 65) and infants. However, the increase of infant mortality was not commented upon in the conclusions, probably because modern methods of epidemiological analysis were not available. The air pollution levels of current concern are much lower. The measurement of air pollution levels in the early years was often in the form of Black Smoke or TSP, but increasingly the respirable fraction (less than 10 um, PM10) is being monitored and a number of studies use PM10 data to establish the associations (WHO/NILU, 1996). A conversion factor of PM10 = 0.55 x TSP has been used to compare results of different studies (WHO/NILU, 1996).

It appears that some of these mortality and morbidity effects start occurring at levels as low as 20 - 30 ug/m^3 (WHO/NILU, 1996), but a clear threshold of effects has not been established. The WHO expert group revising the WHO air quality guidelines made the same conclusion and proposed no specific maximum level for PM in air (WHO, 1995). Instead quantitative risk coefficients based on the dose-response relationships for the different effects were proposed and each jurisdiction was advised to establish its' own guidelines based on the 'acceptable risk' at the local level. For example, the estimated risk coefficient for increase of total mortality is a 1 % increase of deaths for each 10 ug/m^3 increase of PM10 (WHO, 1995) and for increased respiratory hospital morbidity it is 2 % for each 10 ug/m^3 of PM10. These numbers are somewhat higher than those given in Table 3. Another issue of importance is the impact of combined exposures, particularly PM and CO (Burnett et al., 1998).

The risk coefficients were derived mainly from short-term time series studies of associations between daily changes in pollution level and the occurrence of health effects in the same geographic area. The drawback with this type of analysis is that it may not reflect the longer term impact of air pollution on health, and the short-term increase in mortality is offset by a decrease in the next few days (McMichael, 1997). A few studies compared areas with different pollution levels over longer time periods (e.g. Dockery et al, 1993) finding larger mortality increases than the time-series studies. Short-term and long-term mortality effects may be induced by different mechanisms, or they may affect different population groups with different vulnerability.

In order to assess the statistical limitations of the use of the risk coefficients in Table 3 for air pollution health monitoring, it is of importance to consider the size of the studies that were the basis for Table 3 (cf. Section 4.3). Time-series studies and multiple regression cross-sectional studies in more than 10 US cities and some European cities showed an increase in total daily mortality (Schwartz, 1994) and hospital morbidity for respiratory disease (Schwartz, 1995). These studies invariably covered multiyear periods and large populations. For instance, in the study by Pope et al (1995) individual risk factor and mortality data from 552,138 adults in 151 US metropolitan areas was collected over a 7-year period.

Another study investigated daily deaths and air pollution in one county with 260,000 people over 4 years (Pope et al, 1992) and a third study of Philadelphia (Schwartz and Dockery, 1992) included all deaths in 1.7 million people during a 7-year period. On the other hand, a comparison of the 16-year mortality in 6 cities was carried out including only 8111 adults (Dockery et al. 1993). Mortality rate ratios between the cities with higher air pollution and the city with the lowest pollution (annual average TSP = 34 ug/m^3) were calculated, adjusting the rates for smoking habits and other individual risk factors. In the city with the highest pollution level (annual average TSP = 90 ug/m^3) the rate ratio was 1.26 (confidence interval = 1.06 – 1.50). Thus, if the observation period is sufficiently long, effects can be measured at these relatively low pollution levels even in relatively small study groups.

Evidence of the health effects of different air pollutants has emerged from specific epidemiological analysis based on routinely collected health monitoring data on mortality and morbidity. At the air pollution levels currently occurring in typical developed countries, it is likely that data from long time periods have to be used to establish with statistical significance a change in population health status associated with changes in air pollution. A system that monitors seasonal changes in health and air pollution may be feasible in certain locations, but the monitoring of daily or weekly changes is likely to work only for acute symptoms in sensitive people (e.g. asthmatics) and in relatively severe pollution situations.

Routine air pollution health effects monitoring systems based on short-term observations have not been established, as far as the author can ascertain. In France, there are advanced plans for a national multi-center project to assess the feasibility of an epidemiological surveillance system for air pollution effects (Quenel et al., 1997). The assessment will be based on retrospective analysis of air pollution and health data in nine large French cities with observation periods between 2 and 8 years. At the end of 1998 it is planned to evaluate whether it is feasible to establish an ongoing prospective monitoring system.

2.2 NZ reports linking air quality and health effects

A risk assessment, based on the dose-response relationships referred to above and the current air pollution levels in Christchurch (Foster, 1996) concluded that each year the days of high air pollution possibly causes 29 extra deaths and 40 extra hospital admissions. In addition, it was estimated that air pollution cause 82 000 days of 'restricted activity', such as absence from school or work due to respiratory symptoms (CRC, 1997). The method used was similar to that used by the British Columbia Ministry of Environment, Lands and Parks (BCMELP, 1995) to calculate the health impact of particulate air pollution in the province. For each 10 ug/m^3 "increment" of 24-hour particulate air pollution above 20 ug/m^3 a certain percentage increase of mortality or morbidity is assumed to occur. For instance, in Christchurch a 1% increase of total daily mortality was assumed to occur for each "increment". These calculations have been widely debated in Christchurch and some critics believe that the lack of local data supporting this risk assessment puts in question the regional air quality management policy.

However, a very recent study (Hales et al., submitted) indicates that an increased total and respiratory mortality can indeed be measured. Further research on mortality and morbidity in relation to Christchurch air pollution is in progress. It will make an important contribution to the management of the local air quality concern, as well as provide input into the New Zealand environmental indicators programme. It should be pointed out that 29 extra deaths may seem small, as it is only 1 % of all deaths in Christchurch during a year. However, these deaths are related to conditions during the 30 worst polluted days. Thus, 29 deaths is about 10 % of the deaths during those days. In addition, not all deaths are truly preventable. People still die of 'old age' and many of the deaths during the worst polluted days have nothing to do with air pollution. The 29 extra deaths may actually be a much larger proportion of the 'preventable' deaths during these days.

Another risk assessment of the health effects of air pollution has been produced for the Land transport pricing study of the Ministry of Transport (MoT, 1996). The aim was to estimate the cost of health damage due to air pollution and other environmental impacts from motor vehicles on roads. Based on a review of a number of epidemiological studies it was concluded that lifetime exposure to 10 ug/m^3 particulate air pollution would increase total mortality by 1.6 % and that lifetime exposure to 1 ug/m^3 benzene would increase cancer mortality by 4 per million. The estimates were eventually expressed as the estimated cost in dollars per km of road and the cost of particulate air pollution health damage was about 20 times greater than the cost of benzene health damage. These calculations are likely to be very approximate, but they indicate the importance of particulate air pollution when indicators are established to monitor health effects of air pollution.

No epidemiological studies of the association between air pollution and mortality have been carried out in New Zealand, except the recent study by Hales et al., (submitted). However, Dawson et al. (1983) studied the relationship between hospital attendance for acute asthma attacks and air pollution levels in Christchurch during the winter of 1981 and found a negative correlation. No explanation for this unexpected result was found, but the relatively small study size would have limited the statistical power of the study.

Another study of asthma in Christchurch children (Wilkie et al., 1995) focussed on potential air pollution during the summer of 1993 around a fertiliser plant. No increase of asthma was found compared to a control group of children from the whole of Christchurch. The pollution situation was quite different from the winter smoke of major concern.

The only other study is a panel study of 40 subjects with COPD (Harre et al., 1997), in which their reported prevalence of night time chest symptoms was increased during the day after a 24-hour period when the PM10 levels increased by 35 ug/m^3 or more. Again, the small study size makes it difficult to draw definite conclusions.

A small number of studies have investigated exposures to lead in New Zealand. One of these (Kennedy et al., 1988) studied the potential for exposure from air pollution close to Auckland motorways and documented in detail the increased dust and soil lead levels caused by lead emissions from motor vehicles. However, the contribution of air lead to children's blood lead could not be quantitatively estimated. Groups of children living in new houses along motorways appeared to have on average slightly higher blood lead levels than children in areas remote from major roads (not statistically significant). However, when the same children living along motorways were re-tested after one year, generally their blood levels decreased as an effect of age.

Another study of almost 3,000 blood leads analysed at Christchurch hospital during 1978- 1985 showed a steady decrease of the levels in spite of the fact that the use of lead in petrol had not changed (Hinton et al. 1986). Reduced exposure to other sources of lead, such as house paint and soldering in food cans, was considered the likely explanation of the time trends. An update of this study (Walmsley et al., 1993) did not show any reduction of the average lead levels that could be related to reductions in petrol lead. Other studies in New Zealand have focussed specifically on the issue of exposure to lead in house paint, which is an important remaining public health concern.

3 Potential indicators of human exposure and health effects for New Zealand

This section discusses potential human health indicators in two groups; indicators of human exposure and indicators of actual health effects. It then goes onto outline issues with the size of the population in many urban areas, which is often considered to be too small to carry out health effects studies.

3.1 Indicators of human exposure

A few potential exposure variables are mentioned in Table 2. The manner of expressing exposure can be on the population level (e.g. percent of population exposed above a certain level, or the average exposure level in a population) or on the individual level (e.g. daily average ug/m^3 for a person, or the total dose in ug/day). As mentioned above, some of the risk assessments have expressed exposure in the form of particulate air pollution increments of $10ug/m^3$.

For **PM air pollution, SO2, NO, NO2, NOx and O3** actual exposure can only be measured feasibly through measurements of the air the person breaths. There is no convenient biological monitoring method. Exposure is therefore often estimated directly from the ambient air concentrations, but this is clearly a very approximate method.

Variations occur within geographic areas, which could be described by GIS (geographic information system) methods. This method has recently been introduced into the exposure assessment field and has the appeal of expressing the variations in the form of maps (NIWA has recently carried out studies of the spatial distribution of NO_2 and CO levels in Auckland using GIS). Variations in ambient air pollution concentrations also occur during the day and the pollution level indoors may be quite different from the levels outdoors. For each individual ideally a time-activity pattern should be established to properly assess air pollution exposure. Personal air sampling equipment can also be used to measure what an individual is likely to inhale. No New Zealand studies of personal exposure using these approaches have been published. This is an area of useful future research.

It is proposed that for these air pollutants the current approach of using ambient levels, in combination with time-activity patterns, is the best approximate measure of human exposure. In geographic areas where elevated exposures occur and health effects may occur, it would be of value to compare outdoor and indoor air pollution levels in order to assess the need for more refined exposure estimates.

For **carbon monoxide** the air monitoring could be complemented by measurements of COHb in blood of selected groups of people, for instance retired people living in homes with no smokers and spending most of their daily hours within the neighbourhood where they live. This type of exposure monitoring would make it possible to check that the air quality monitoring truly identifies a health risk associated with CO. Monitoring badges can also be worn to estimate personal exposure.

Because of the decreasing air levels of **lead** it would not seem a high priority to monitor lead exposure in relation to air pollution, but the paint lead problem is still of importance in areas with houses more than 35 years old. Blood lead is a very effective way of measuring lead exposure at the personal level.

For other pollutants, such as **benzene**, specific metabolites can be measured in urine or the pollutant itself can be measured in exhaled breath. However, these are likely to be expensive and cumbersome approaches to establish exposure. Estimates based on ambient concentrations are more convenient, particularly as the health concerns arise out of very long-term exposures. Research to compare personal exposure and estimates from ambient concentrations may be able to establish the validity of the latter method. The correlation between air levels of benzene and the major pollutants should be studied, in order to assess whether a more easily measured pollutant can be used as an ‘indicator’ of the benzene level.

3.2 Indicators of health effects

The health effects that were mentioned in Table 3 would be the focus of health monitoring. Data on **mortality and hospital/emergency room admissions** are routinely collected but there is normally a major delay before the data become available, so for health monitoring purposes, special data collection systems need to be established.

The greatest problem with these effects is that they are generally rare events (within the population of Christchurch only about 7 people die each day and about 14 people are admitted to hospital for respiratory or cardiac diseases; Foster, 1996). It is not possible to establish that a small change (say 10 % due to an increase of the particulate level of 100 ug/m^3) in mortality or morbidity has taken place during a single day. Any health monitoring of these effects would have to extend over longer time periods, maybe even over several years (as in the epidemiological studies referred to above).

However, using *State* air quality indicators, such as the air concentrations of pollutants, to monitor air quality change over time normally also require multi-year databases to draw meaningful conclusions. Thus, the establishment of a multi-year mortality rate indicator (rolling average for different seasons) in a defined geographic area as an indicator may be a useful too for long-term air quality management.

The occurrence of the various **symptoms** referred to in Table 3 are likely to be much more common in the general population and could therefore be tested in “panel studies” for health monitoring. The occurrence of symptoms could also be expressed in terms of **restricted activity days**. In these studies selected people keep a health diary, in which specified symptoms are recorded. In order to assess the feasibility of this approach studies in areas with low pollution would need to establish the background prevalence of each symptom. Another approach would be to study absenteeism from school or work. The specific symptoms or illnesses could be recorded and associations with air pollution could be analysed.

The target groups for each health effects monitoring have to be considered. As mentioned above some of the effects have been reported mainly in asthmatics and some mainly in people with ischaemic heart disease. In addition, very young children and the elderly are particularly at risk for most of the air pollutants.

3.3 Consideration of the size of the population exposed

In order to assess the statistical feasibility of documenting changes in exposure or effects of air pollutants in New Zealand, the relatively small population has been considered an important obstacle. This is particularly true for the relatively rare severe effects of death and hospitalisation. However, if health monitoring takes a more long-term perspective by extending the period of data collection over a whole season or several years (as in the studies in the USA), it should be possible to establish an increase of mortality or morbidity in a city of the size of Christchurch. Simple statistical power calculations cannot be done for the multiple regression or time-series analysis required to study the effects. Only modeling with different sets of assumed data can clearly show the length of the period of data collection required.

However, as an illustrative example, we can make the power calculation on the basis of testing higher than average daily mortality on polluted days. If we assume a Poisson distribution of daily mortality and we are seeking the number of ‘polluted’ days we have to include in the study (N), the following formula applies:

$$(N \times P - N \times A)(N \times P - N \times A) / (N \times P + N \times A) = 7.84$$

where: A = average number of deaths per day
P = number of deaths each polluted day

The number 7.84 is based on a 80 % probability of establishing a difference between polluted and average days at $p < 0.05$.

Set F = P / A or the relative increase of mortality on polluted days, a ratio

Then: $N = (7.84 / A) \times ((F + 1) / (F - 1)(F - 1))$
In Christchurch: $A = 7$ deaths per day.

Thus:	For	F = 1.01	1.1	1.3	1.5	2.0
		N = 22512	235	29	11	3.4

So, a study of the 30 most polluted days of a year should be able to show with statistical significance a 30 % increase of mortality during those days. The average number of deaths during the 30 days would be 210, and the number needs to increase by 63 additional deaths to make it ‘measurable’. If the true increase during the 30 days is about 20 deaths (or 10 % of the average) then 235 days have to be studied, or all the most polluted days for 8 years (which would give 240 most polluted days). Such studies can be carried out in Christchurch in order to confirm whether the risk assessment (Foster, 1996) is supported by real observations. It is noteworthy that the recent mortality study in Christchurch (Hales et al., submitted) covered 6 years and produced results just within the boundary of "statistical significance".

For the biological monitoring variables, such as COHb, changes from a background average population level of 1 % to an increased level of 2.5 % in non-smokers is likely to be detectable in very small groups of people. However, studies to establish the background standard deviation of COHb values need to be carried out for accurate power calculations to be carried out.

4 Recommendations for further work

Recommendation 1

Human health indicators of air quality in New Zealand should be further developed, targeting the specific air pollution concerns in different regions. A common national approach needs to be flexible, in order to allow for development of regional indicators that are informative and cost-effective, depending on the priority issues in each region.

Recommendation 2

For some air quality health problems, such as mortality during the most polluted days in Christchurch, the statistical power of analysis of daily data on a cumulative basis (e.g. 30 worst days) may be sufficient for continuous health monitoring, particularly if rolling multi-year averages of mortality are used as indicators.

Recommendation 3

Further epidemiological studies should be carried out in New Zealand to identify the best health indicators for each major air quality concern; e.g. home heating, motorcars, industry. Collaborative studies involving cities in other countries with higher air pollution levels than those occurring in New Zealand would be of value in making comparisons with more extreme situations and adding points to the dose-response curve.

Recommendation 4

Studies of the 'burden of disease' due to air pollution in relation to other health concerns should be carried out, in order to set priorities for preventive actions. The health impacts of preventive policies (such as the one proposed for Christchurch) should also be studied and assessed.

Recommendation 5

Efforts should be made to ensure that all agencies that can contribute data to future health monitoring of air pollution effects, develop systems that make data sharing and analysis cost-effective. Census data, health statistics data, weather data and air pollution data, all need to be in compatible formats and based on common geographic units.

Recommendation 6

The application of GIS to air pollution health effects monitoring should be further explored. Ideally, the different agencies interested in or involved in this type of work should be encouraged by the MfE to collaborate in developing new methods and capabilities. Training in this field should be arranged at a national level.

Recommendation 7

Further analysis of routinely collected air pollution data should be carried out to identify if ‘indicator’ pollutants can be used in monitoring of health risks of e.g. benzene. In addition, the link between weather conditions and air pollution peaks should be studied further to assess whether air pollution warnings can be made a part of the weather reports.

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