Chronic Obstructive Pulmonary Disease and Lung Cancer in New Zealand

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Summary

Two tobacco-related respiratory diseases, Chronic Obstructive Lung Disease (COPD) and Lung Cancer, have substantial impacts on the health of New Zealanders.

More than 85% of the combined burden of these two conditions in New Zealand arises from tobacco, either from a history of smoking or indirectly from inhalation of second-hand smoke. Both conditions are largely avoidable.

In 1997, COPD was ranked third overall in its impact on the health of New Zealanders, after ischaemic heart disease and stroke: 2nd in men and 5th in women. The same report showed lung cancer ranked 8th overall: 5th in men and 12th in women. Both are significantly under-recognised and yet are predicted to have growing impact in countries such as New Zealand.

Evidence on the burden of COPD is sparse, but estimates based on best available data are that, each year in New Zealand, COPD:

- is responsible for 9,250 hospital discharges and 88,800 bed-days (1.5% of all bed-days)
- accounts for about 200,000 GP visits and more than 453,300 medications
- as a cause of death, ranks 4th after cancer, heart disease & stroke
- causes years of disability and of greatly reduced quality of life
- is estimated to cost between $102-$192 million in direct health care costs.

In New Zealand, lung cancer, each year:

- is responsible for 3,000 hospital admissions, and 25,000 bed-days
- accounts for an estimated 17,000 GP visits per year
- leads to about 1500 deaths, more than any other cancer deaths
- greatly shortens life: half die within 18 months of diagnosis and fewer than 10% live to 5 years
- is estimated to cost between $25-$40 million in direct health care costs.

Interventions are urgently required to reduce the prevalence of smoking in New Zealand: such efforts have stalled in recent years. For the large cohort of current and former smokers, greater attention is needed to detect and treat disease earlier. Improvements in management of COPD could reduce its impact.

This report describes the burden of the respiratory diseases COPD and lung cancer in New Zealand. Prevalence of tobacco smoking is outlined in Chapter 1. Background information about COPD and lung cancer forms Chapter 2. Separate chapters describing the health impacts (Chapter 3) and cost burden (Chapter 4) of COPD in New Zealand follow. Chapters 5 and 6 serve a similar purpose for lung cancer. Implications for the future and interventions are addressed in Chapter 7. Two appendices provide background material for estimation of prevalence of COPD.
Contents

Summary 4

Contents 5

Table of Figures 7

Table of Tables 9

1 Smoking in New Zealand 10
  1.1 Tobacco smoking 10
  1.2 Cannabis smoking 13

2 Common Lung Diseases 15
  2.1 Asthma 15
  2.2 Chronic Obstructive Pulmonary Disease 15
  2.3 Lung Cancer 19

3 Health impacts of COPD 22
  3.1 Prevalence and incidence 22
  3.2 Quality of life 26
  3.3 Morbidity 27
  3.4 Survival 31
  3.5 Mortality 31
  3.6 Long-term trends in New Zealand 33
  3.7 Trends in causes and risk factors for COPD 36
  3.8 Projections and prevention 36

4 Cost Burden of COPD 38
  4.1 Basis of costings 38
  4.2 Summary of costings 38
  4.3 Direct costs to patients and families 40
  4.4 Indirect costs 40
  4.5 Changing costs 41

5 Health impacts of Lung Cancer 43
  5.1 Prevalence and incidence 43
  5.2 Morbidity 45
  5.3 Mortality 47
  5.4 Long-term trends and projections 49
  5.5 Prevention 49

6 Cost Burden of Lung Cancer 51
  6.1 Basis of costings 51
  6.2 Summary of costings 51
6.3 Indirect costs 52
6.4 Changing costs 53

7 Implications for New Zealand 54
7.1 Health promotion and prevention 54
7.2 Recognition and diagnosis 54
7.3 Reporting and monitoring 56
7.4 Economic burden 57
7.5 Research opportunities and possibilities 57
7.6 Messages 57

Appendix 1 Potential data sources for measuring the prevalence of COPD 59
Appendix 2 Prevalence of impaired lung function and COPD 60

References 63
Acknowledgements 62
Table of Figures

| Figure 1-1 | Prevalence of cigarette smoking among men and women aged 15 and over, by age group (3-year moving average), per 100,000 | 11 |
| Figure 1-2 | Age-standardised prevalence of cigarette smoking among people aged 15 and over (%), by ethnicity (3-year moving average) | 11 |
| Figure 1-3 | Tobacco released per adult 15 years and over, 1970-2000 | 12 |
| Figure 3-1 | Estimates of COPD rates in four population-based studies in USA, including both diagnosed and undiagnosed, by smoking status, sex and age group | 25 |
| Figure 3-2 | Numbers of COPD Hospital discharges in New Zealand, by sex, age group and ethnicity, 1998/1999 | 28 |
| Figure 3-3 | Rates (per 100,000) of COPD hospital discharges in New Zealand, by sex, age group and ethnicity, over period 1997-1999 | 29 |
| Figure 3-4 | Numbers of COPD deaths in New Zealand, by sex, age group and ethnicity, 1998 | 32 |
| Figure 3-5 | COPD mortality rates (averaged over 3 years) in New Zealand, by sex, age group and ethnicity (per 100,000) | 33 |
| Figure 3-6 | Age-standardised mortality rates for COPD, selected countries, 1995/1996 (per 100,000) | 34 |
| Figure 3-7 | Trends in COPD hospital discharges in New Zealand, age-standardised* rates per 100,000 person years, by sex and ethnicity | 35 |
| Figure 3-8 | Age-standardised death rates of COPD and other major causes of death, New Zealand 1980-1999 (per 100,000) | 35 |
| Figure 3-9 | Age-adjusted mortality for COPD in USA, 1979-1998, by sex | 36 |
| Figure 3-10 | Projected prevalence of COPD in New Zealand in 2025, from models developed by Stang and applied to projected NZ population and using 1996 smoking prevalence | 37 |
| Figure 5-1 | Lung cancer registrations in New Zealand 1999, by age and sex | 44 |
| Figure 5-2 | Estimated age-standardised rates of lung cancer incidence worldwide, by WHO region, 1990 (per 100,000) | 45 |
| Figure 5-3 | Numbers of lung cancer public hospitalisations in New Zealand, by sex, age group and ethnicity, 1999 | 46 |
| Figure 5-4 | Lung cancer hospitalisation rates in New Zealand, by sex, age group and ethnicity (per 100,000) | 46 |
Figure 5-5  Numbers of lung cancer deaths in New Zealand, by sex, age group and ethnicity, 1999 .............................................................. 48

Figure 5-6  Lung cancer mortality rates in New Zealand, by sex, age group and ethnicity, 1999 (per 100,000).................................................. 48
Table of Tables

Table 2-1  Classification of COPD severity under criteria employed in the Australian and New Zealand Management Guidelines ............... 18
Table 2-2  Classification of COPD severity under GOLD criteria .................. 18
Table 3-1  Estimates of prevalence of COPD in New Zealand, based on WHO regional estimates .......................................................... 24
Table 3-2  Codes used in sourcing hospitalisation & death data for COPD .... 27
Table 3-3  Numbers of COPD discharges, and associated mean lengths of stay in 1999/2000 according to ICD-10 sub-group, by sex (mean days) ........................................................................................................ 30
Table 3-4  Numbers of discharges and associated mean lengths of hospital stay (days) in patients over 45 years, where COPD is, or is not, a co-morbid condition .................................................................................. 30
Table 4-1  Estimated direct costs to the health services of COPD in New Zealand, 2002 ('000s) .......................................................................................................................... 38
Table 5-1  Number of discharges and associated mean lengths of public hospital stay (days) in patients over 45 years, for lung cancer in New Zealand ........................................................................................................ 47
Table 6-1  Estimated health-related costs of lung cancer in New Zealand, 2002 ('000s) ....................................................................................................................................... 51
1 Smoking in New Zealand

1.1 Tobacco smoking

1.1.1 Health effect of tobacco smoking

Tobacco smoking and environmental tobacco smoke contribute to illness and death from a number of respiratory diseases including lung cancer, asthma, chronic obstructive pulmonary disease (COPD), as well as many cardiovascular diseases, and are believed to be a causal factor for conditions as diverse as fire injuries, maternal and perinatal ill-health.

Across developed countries, greater burden on health is attributable to tobacco smoke than to any other single condition.\textsuperscript{2} Burden to societies is estimated by the “disability-adjusted life year” (DALY), a conceptual measure of the impact a disease or health condition has on a population through its inclusion of the combined burden from early death and from disability while living with the condition. One DALY is defined as the loss of one year of healthy life to disease.

In developed countries, tobacco smoke has been estimated to be responsible for 12.2% of DALYs in the population. In comparison, 10.9% of DALYs are lost through high blood pressure, 9.2% from alcohol, 7.6% from high cholesterol and 7.4% from high body mass index (BMI).\textsuperscript{2} In New Zealand, approximately 15% of DALYs among men is attributed to tobacco, and 9% among women.\textsuperscript{3}

1.1.2 Tobacco smoking trends in New Zealand

Historically in New Zealand, men were the main users of cigarettes and tobacco, with large increases in the proportion smoking during and after the world wars when servicemen were provided with tobacco as part of their rations. Following World War 2, women began to take up smoking more, so that now they have levels very close to those in men.

Currently, there is a clear age-related gradient in prevalence of smoking: about 13% of those aged over 55 years smoke tobacco, 22% of those aged 35-54, 32% of those aged 25-34, and 33% of those aged 15-24 years. Gender differences in prevalence of smoking are now small, but years of smoking and tobacco quantities consumed are still less among women.

Smoking trends are monitored through regular population-based surveys. Figure 1-1 shows the falling prevalence of smoking in the New Zealand population (data sourced from Ministry of Health, 2002\textsuperscript{4}). The decline is particularly marked in people aged over 35 years. Prevalence among men and women in the 25-34 year age group has changed little since the mid-1980s.

However, high rates in the youngest age group, 15-25 years, are also seen in Figure 1-1. For young women, the data show a slight fall from the very high rates seen around 1980 but which remain high, but, since 1997, increasing prevalence in young men remains unchecked.

Not only overall rates of smoking have been reducing, but also the numbers of cigarettes smoked per day by smokers. In 1976, 51% of men and 34% of women smokers consumed at least 20 cigarettes, or the equivalent tobacco, per day. In 1992-93 this had reduced to 15% and 12% respectively.
Figure 1-1  Prevalence of cigarette smoking among men and women aged 15 and over, by age group (3-year moving average), per 100,000

Figure 1-2 shows marked differences in smoking prevalence between Maori, Pacific peoples, and non-Maori, again as measured by self-report in population-based surveys. Only part of the differences arise from the younger ages of the Maori and Pacific populations. The high prevalence among Maori shows little decline over time, even in the presence of steadily declining prevalence among European and other people.

Figure 1-2  Age-standardised prevalence of cigarette smoking among people aged 15 and over (%), by ethnicity (3-year moving average)

Cigarette consumption nationally is monitored regularly by ACNielsen Ltd. Their data show an overall continuing decline in cigarette consumption per adult since 1976, seen in Figure 1-3. Steepest declines occurred between 1984 and 1993, after which tobacco consumption per person levelled off. These data do not include tobacco brought in the baggage of international travellers or illegally.
There has been a steady decrease in the numbers of manufactured cigarettes, as measured by government duty paid. This reduction in tobacco consumption over the 1985-1995 period appears unmatched in any other country.\(^5\)

There has also been a small rise in the volume of loose tobacco, suggesting that consumers may be switching from tailor-made cigarettes to relatively cheaper loose leaf tobacco. Between 10 and 20% is now consumed as loose tobacco.\(^6\)

There is a clear inverse association of smoking with household income and socio-economic status.\(^4\) Interpretation of any association of income or socio-economic status with COPD should consider smoking as the most likely cause.

### 1.1.3 Effect of tobacco smoking on lung function

Amongst non-smokers, normal ageing accounts for a loss of lung function: forced expiratory volume in one second (FEV\(_1\)) falls approximately 30ml per year of age.\(^7\) Smokers lose an extra 33ml/year on average, though this is very variable.

There is wide variation in susceptibility to the airway-damaging effects of cigarette smoke. In a population of smokers, about 15% can be expected to develop clinically significant and diagnosed COPD, 50% will develop chronic bronchitis and/or undiagnosed COPD, 16% of men and 9% of women lifelong smokers will develop lung cancer, and about 50% will not develop any symptomatic physiologic deficit.\(^8\)\(^9\) There are also increased risks such as other cancers and cardiovascular disease which may also involve respiratory problems but are not covered in this report.

There are many unanswered questions. It is not yet clear why some smokers seem protected from respiratory disease; which of the carcinogens and toxins in tobacco smoke are responsible for respiratory problems; or whether using filters or low tar cigarettes is protective for COPD or lung cancer; and whether loose tobacco has a different mix of toxins and carcinogens than manufactured cigarettes. These all have implications for introduction or promotion of various form(s) of tobacco control.
1.1.4 Smoking cessation

Once damage to lung function is established it is largely permanent, although further damage is to some extent avoidable if smoking stops. Those who quit smoking soon revert to a similar annual loss in lung function to non-smokers.\(^7\)\(^{10}\)

1.2 Cannabis smoking

Most information about the impact of recreational smoking on health is obtained from studies of tobacco smoking; relatively little information is available about cannabis smoking primarily because of tobacco’s greater use historically. Unbiased data are difficult to obtain for the use, production or distribution of an illegal substance, and there is little consumption data.

1.2.1 Health effect of cannabis smoking

Cannabis smoke is similar in constitution to tobacco smoke, but contains a substantially higher proportion of particulate matter and of some carcinogens than does tobacco smoke.\(^{11}\)\(^{12}\) While there are few good quality studies which inform us of the risks associated with cannabis smoking, the contents of the smoke are good reason to believe that chronic heavy cannabis smoking has adverse effects upon the respiratory system.

Studies that have compared tobacco smoking with cannabis smoking indicate that marijuana affects mainly the large airways, and tobacco mainly the peripheral airways and regions around the air sacs. Among cannabis smokers, the common practice of also smoking tobacco is likely to lead to both types of damage.\(^{13}\) One longitudinal study showed that “non-tobacco” smoking (which is assumed to be mainly cannabis smoking) was associated with the development of cough, phlegm and wheeze. Changes in FEV\(_1\) and forced vital capacity (FVC) were least among non-smokers, then, in decreasing order, among current tobacco smokers, then cannabis smokers and worst among those who smoked both tobacco and cannabis.\(^{14}\)

Although cannabis smokers typically smoke fewer joints a day than tobacco smokers smoke cigarettes, they inhale a larger volume of smoke, inhale more deeply, take more particulate matter per puff, and hold their breath four or five times longer.\(^{15}\) Therefore the respiratory burden of carbon monoxide and smoke particulates such as tar is greater than when smoking a similar quantity of tobacco. It is claimed that 3 - 4 joints a day are associated with the same evidence of acute and chronic bronchitis and the same degree of damage to the bronchial mucosa as 20 or more tobacco cigarettes.\(^{13}\) Current evidence is that chronic cannabis smoking does increase the prevalence of bronchitic symptoms and reduces respiratory function, it probably increases the risk of developing respiratory tract cancer and it possibly influences the development of COPD more greatly than tobacco.\(^{16}\)\(^{17}\) Upper respiratory tract diseases also occur as a result of cannabis smoking but are not covered in this report.

1.2.2 Cannabis smoking trends in New Zealand

The best data available for prevalence of cannabis use in New Zealand describes current and ‘ever’ users, but does not help ascertain the levels of cannabis smoking over the longer term, i.e. the lifetime risk patterns. About 24% of adults in New Zealand report using cannabis more than twice in the last 12 months, and just 3% report using 10 or more times within the past 30 days\(^{18}\). In Australia, regular cannabis smoking is reported as 11%.\(^{19}\)
Prolonged exposure to smoking (tobacco or cannabis) is recognised as contributing most to COPD and lung cancer. Consumption data for several decades are therefore needed to determine the relationship with COPD onset, but these data are not available. Trends in cannabis use in New Zealand are currently being measured by SHORE, the Centre for Social and Health Outcomes Research and Evaluation of Massey University. In New Zealand, more than 60% of the regular cannabis users were aged under 30 years, so the impact of smoking will not yet contribute to COPD or lung cancer statistics.18
2 Common Lung Diseases

Lung diseases are important from a public health perspective, since they are a common cause of ill-health and account for a large proportion of deaths. The three main lung diseases are asthma, chronic obstructive lung disease, and lung cancer. The latter two are attributable in large part to tobacco smoking.

2.1 Asthma

The burden from asthma in people aged under 45 years in New Zealand was described in a report published in 2002. Many current smokers and ex-smokers suffer from both asthma and COPD. In the elderly particularly, asthma is frequently confused with COPD, with errors occurring in both directions. This report does not cover asthma.

2.2 Chronic Obstructive Pulmonary Disease

2.2.1 Characteristics

COPD is characterised by airflow obstruction with breathing-related symptoms such as chronic cough, breathlessness, sputum and wheeze. Since the Global Burden of Disease project ranked COPD as 12th on the list of diseases impacting on health worldwide in 1990 and predicted further growth to 5th by 2020, the disease has received greater recognition. In 1997, COPD was ranked third overall in its impact on the health of New Zealanders, after ischaemic heart disease and stroke: 2nd in men and 5th in women.

COPD is a non-specific term describing airway obstruction due to chronic bronchitis (with cough and excessive sputum) or emphysema (with characteristic histological pathology). It is largely preventable by not smoking. If identified early it is treatable (i.e. relief from symptoms may be possible) and the course of progression may be slowed if smoking stops, but very little can be done to reverse or cure the condition.

Typically, patients present complaining of increasing and long-term shortness of breath, or have a history of frequent respiratory infections. Mild levels of airway obstruction are often asymptomatic. In consequence, by the time patients notice shortness of breath on exertion, they may already have significant impairment of airway function. Early signs of COPD include frequent coughing with sputum and/or shortness of breath.

Many people with COPD suffer acute episodes of exacerbation requiring repeated primary care visits or hospitalisation. Exacerbations may be precipitated by respiratory infection, environmental exposures and co-morbid conditions, but may be reduced through influenza immunisations and some drugs.

COPD often leads to reduced activity levels at work, at leisure and in daily living, and to decreased quality of life. Over a period of years, people with COPD may become increasingly disabled and prone to infection, and may become malnourished. Depressed mood and anxiety are common, and severe confusion cause substantial problems. One study of people living with severe COPD showed levels of oxygen which were insufficient for normal activities more than
25% of the time. Even if COPD is not identified as the primary cause of death, its effects make it a common contributing cause.

In an initiative to increase awareness of COPD and to decrease morbidity and mortality from this disease, an international collaborative project was set up under the auspices of the World Health Organisation (WHO) and the US National Heart, Lung and Blood Institute. It is known as the Global Initiative for Chronic Obstructive Lung Disease (GOLD). In its first major statement, COPD is described as “a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.” This definition supersedes a variety of previous and sometimes conflicting definitions, and is the first to arise from world-wide consensus. Because it is fairly recent, previous definitions have been used in some parts of this report.

### 2.2.2 Causes of disease

In most cases, COPD is a direct result of years of inhaling tobacco smoke. Only about 10% of those dying of COPD have no history of cigarette smoking, but latest estimates put the contribution of tobacco at 69% of the COPD burden after adjustment for other risk factors. Environmental tobacco smoke, indoor cooking smoke in developing countries and genetic pre-disposition (for example alpha1-antitrypsin deficiency) are also factors.

Being overweight was shown to increase the risk of diagnosis of COPD by 2.4 times for middle-aged women in Canada, but not for men, and low income was associated with 3.7 times higher rates among middle-aged men, but it is not clear whether either of these associations is causal.

Tobacco smoke is implicated in 85 - 90% of COPD cases.

Occupation-related COPD does occur, in perhaps 30% of never-smokers with COPD, as a result of long-term inhalation of air-borne particulates (organic or inorganic dust), or chemical agents such as are found in manufacturing, public utilities, the armed forces, construction, transport and health industries. There is clear evidence that occupational exposure to dusts accelerates loss of FEV1. Exposure to gases, fumes, and vapours are associated with respiratory conditions such as shortness of breath and chronic bronchitis, but not necessarily to airway obstruction. In a general adult population, lifestyle risk factors cluster: cigarette smoking was found more often than expected in combination with excessive alcohol intake and with low consumption of vegetables and fruit. Reported associations may be effected by confounding.

One study has described associations between respiratory admissions and weather and air-borne pollutants (including air temperature, relative humidity, barometric pressure, sulphur dioxide and products of the oxidation of nitrous oxide), but the admissions included respiratory diseases other than COPD.

Cross-sectional studies suggest that consumption of flavonoids (particularly catechins) may be protective against COPD, but this is yet to be proved in randomised trials. Use of topical beta-blockers for glaucoma are associated with increased risk both of exacerbations and of new COPD.

### 2.2.3 Diagnosis

Diagnosis of COPD rests upon spirometry and the demonstration of airflow limitation that is not fully reversible. Spirometric tests of breathing function are
therefore advocated before confirming a diagnosis. Frequently history and symptoms alone are used by GPs who have no ready access to spirometry. Because several other prevalent conditions (e.g. heart disease, asthma) also cause shortness-of-breath, these must be excluded before a diagnosis is made.

COPD is usually recognised late, is often difficult to diagnose, and is frequently confused with other respiratory conditions. The impact of many medical conditions is compounded by coexisting COPD.

However, clinicians are inconsistent when diagnosing COPD, emphysema and chronic bronchitis; a paper by McAlister showed considerable differences in how clinicians have been taught to diagnose COPD.37 As the new guidelines are adopted, these variations should reduce. Where diagnosis is not made consistently between clinicians, or coding is inconsistent, records of hospital discharge and death will vary by location artefactually, impacting on all reports of the burden of the disease.

2.2.4 Therapies and outcomes

COPD is a slowly progressing disease; about 50% survive 10 years from diagnosis and living with the disease twice that period is not uncommon.38 Only 45-63% of deaths among those with COPD are due to the disease itself.39 Those who have COPD are at higher risk of cardiovascular disease and lung cancer, possibly related to increases in inflammatory markers plasma fibrinogen and serum C-reactive protein.40 41

In smokers with COPD, smoking cessation is proven to slow the decline in ventilatory function.42 43 Some relief from symptoms may be obtained by bronchodilator (or other) therapy amongst those with both COPD and asthma, but for many affected, no relief is obtained.44-46 Some randomised controlled trials have shown benefit from use of inhaled corticosteroids,47 however others have shown no effect.48 Further work is needed before clear treatment guidelines are possible.49

Pulmonary rehabilitation relieves breathlessness and fatigue, and enhances patients’ sense of control over their condition, although only modest improvements in exercise capacity are obtained.50-52 Education programmes promoting self-management have proven effective for asthma, and may have potential for COPD.53-55

Peak expiratory volume, although less appropriate than spirometry for initial diagnosis, is as good or better a predictor for prognosis and monitoring as is \( FEV_1 \), and is much easier and cheaper to measure.56

In New Zealand, evidence-based guidelines for best practice in the management of stable COPD were developed and made available on the internet and in print in 1999.57 In the USA, guidelines for treatment of acute exacerbations from the USA were published in 2001.49 58 Rising mortality from COPD and results from recent randomised controlled trials prompted the development of an international strategy for the prevention, management, and research of COPD in 2001.59 60 More recently, a joint project in Australia and New Zealand developed management guidelines based on the GOLD recommendations, produced a COPD handbook, but did not adopt the GOLD definitions.61
2.2.5 Terminology and classification

The terms Chronic Obstructive Pulmonary Disease (COPD), Chronic Obstructive Respiratory Disease (CORD), Chronic Obstructive Lung Disease (COLD), Chronic Obstructive Airways Disease (COAD), Obstructive Lung Disease (OLD) and Chronic Airways Limitation (CAL) are used somewhat synonymously: this report follows the GOLD convention by using the term COPD.60

For survey and perhaps for monitoring purposes, the simple 5-point MRC dyspnoea scale may be used to classify breathlessness from self-report.62 This is not regarded as adequate for classification of severity or for clinical management although it is sometimes used for that purpose when spirometry is unavailable. Other monitoring measures include reported symptoms: exercise capacity, particularly walking on the flat, frequency of exacerbations, and quality of life.

For clinical and population survey purposes, various classifications of severity have been used, such as these categories used in the Australian and New Zealand Management Guidelines (Table 2-1).61 Other staging systems include those of the British, the European, and the American Thoracic societies.63 64

Table 2-1 Classification of COPD severity under criteria employed in the Australian and New Zealand Management Guidelines

<table>
<thead>
<tr>
<th>Severity</th>
<th>FEV₁ % predicted</th>
<th>Functional assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>60-80%</td>
<td>Few symptoms, no effect on daily activities, breathless on moderate exertion</td>
</tr>
<tr>
<td>Moderate</td>
<td>40-59%</td>
<td>Increasing shortness-of-breath, breathless when walking on the flat, increasing limitation of daily activities</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt;40%</td>
<td>Breathless on minimal exertion, daily activities severely curtailed</td>
</tr>
</tbody>
</table>

Source: Adapted from Burdon 2002

For clinical purposes, the GOLD initiative classifies severity using a 4-point scale, and this is expected to replace those published by the British and the American Thoracic Societies60 and by the Thoracic Society of Australia and New Zealand (TSANZ).61 Four classifications of severity are made, based on a combination of criteria: spirometric measures (such as FEV₁/FVC ratio or FEV₁ as a percentage of that predicted given a person’s sex, age and height), symptoms (cough, sputum production or dyspnoea) and signs of respiratory failure.23 They are shown in Table 2-2.

Table 2-2 Classification of COPD severity under GOLD criteria

<table>
<thead>
<tr>
<th>Stage</th>
<th>Symptoms</th>
<th>Spirometry results (where FEV₁ refers to post-bronchodilator FEV₁)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0: At risk</td>
<td>Chronic symptoms (cough, sputum production)</td>
<td>with Normal spirometry</td>
</tr>
<tr>
<td>I: Mild COPD</td>
<td>With or without chronic symptoms (cough, sputum production)</td>
<td>with FEV₁/FVC &lt; 70% and FEV₁ ≥ 80% predicted</td>
</tr>
<tr>
<td>II: Moderate COPD</td>
<td>With or without chronic symptoms (cough, sputum production, dyspnea)</td>
<td>with FEV₁/FVC &lt; 70% and 30% ≤ FEV₁ &lt; 80% predicted (IIA: 50% ≤ FEV₁ &lt; 80% predicted) (IIB: 30% ≤ FEV₁ &lt; 50% predicted)</td>
</tr>
<tr>
<td>III: Severe COPD</td>
<td>Presence of respiratory failure (definition provided) or clinical signs of right heart failure</td>
<td>or FEV₁/FVC &lt; 70% and FEV₁ &lt; 30% predicted</td>
</tr>
</tbody>
</table>

Source: Adapted from Pauwels 2001

There has been debate about the worth of the classification, Stage 0 “At Risk”, i.e. no diagnosis of COPD, but at risk of the disease. Some suggest that it is not predictive of outcome, and that most of these patients will not have obstruction if
tested using spirometry, and the stage is therefore unhelpful.\textsuperscript{55, 66} There is potential for a staging system that considers factors such as age, arterial blood gases, body mass index and distance walked, and that could yield more helpful prognostic information.\textsuperscript{67}

2.3 Lung Cancer

2.3.1 Occurrence

Lung cancer is one of the most common malignancies diagnosed in New Zealand, the United States, and other developed countries. Parkin estimated the worldwide incidence of 25 major cancers in 1990, and showed that overall, lung cancer was the second most common for men (after prostate cancer) and for women (after breast cancer).\textsuperscript{68} With lung cancer emerging as the most commonly occurring cancer worldwide, they concluded that tobacco smoking is at present the most avoidable cause of cancer.

Worldwide, it is estimated to account for 12.8% of the total cases of cancer and 17.8% of cancer deaths.\textsuperscript{68} Lung cancer was the leading cause of cancer death among men in the USA in the 1950s until the late 1990s, and became so for women sometime later, in 1986.\textsuperscript{69}

It is a particularly lethal cancer, with more than 90% of affected patients dying of the disease within 5 years of diagnosis.\textsuperscript{70} Although one of the most preventable, it is often diagnosed at an incurable stage, once metastasised (i.e. spread around to other parts of the body).\textsuperscript{71} As with all cancers, the greatest opportunity for cure is when detected early in its course; most often it is diagnosed too late.\textsuperscript{72}

Lung cancer is usually detected too late to change its course.

2.3.2 Characteristics

Signs and symptoms are related to the size and location of the primary tumour and to the presence of metastatic disease. First symptoms may include cough (with or without blood in the sputum), wheezing, shortness of breath and tiredness, although the disease may be detected in non-symptomatic patients though chest x-ray. As the lesions enlarge, additional signs may appear: stridor, painful breathing, pneumonia, and swelling of the face and neck.

Early signs of lung cancer include one or more of cough, wheeze, shortness of breath and tiredness.

2.3.3 Diagnosis

Diagnosis is normally through chest imaging (X-ray or CT) then sputum cytology or histology of tissue obtained at biopsy or bronchoscopy.\textsuperscript{72} It is hoped that recent advances in molecular methodologies will provide a useful tool in the early detection of lung cancer, through evaluating transformations in the bronchial epithelium (where earliest changes in onset of lung cancer occur) or through analysis of the gases in exhaled air.\textsuperscript{73}

2.3.4 Causes of disease

Tobacco smoking, either from actively smoking or from passive exposure to environmental tobacco smoke, causes almost all lung cancer.\textsuperscript{27, 74, 75} Cigarette
smoking is the primary cause of 75-80% of all lung-cancer deaths and at least 90% of lung cancer incidence.\textsuperscript{76-78} After adjusting for other known risk factors, at least 85% of the total lung cancer burden is ascribed to tobacco.\textsuperscript{26} Risk of lung cancer is most dependent on smoking status and duration of smoking: low tar cigarettes confer little advantage over normal cigarettes.\textsuperscript{76} Variations in disease rates between geographic regions and between genders are largely a function of historical patterns in cigarette smoking.\textsuperscript{79-81}

Among non-smokers, there is a consistent association of environmental tobacco smoke and lung cancer: there is a 20-30\% excess in risk of lung cancer disease and death among non-smokers who live with smokers.\textsuperscript{75}

\textbf{Tobacco smoke is implicated in over 90\% of lung cancer cases.}

Environmental toxins and irritants (for example asbestos and fine particles from combustive air pollution) also contribute, asbestos particularly in smokers.\textsuperscript{31 82 83} An observed association of lung cancer mortality with social-economic status appears to be related to patterns of cigarette smoking, having reversed from a positive to negative association in the period 1950 to 1998.\textsuperscript{84}

Risk factors regarded as contributing but probably not causal include genetic predisposition; low consumption of fruit, vegetables and cheese; previous lung disease (e.g. COPD, pneumonia, asthma and tuberculosis); and prior tobacco-related cancer.\textsuperscript{77 85 86} There is little evidence to support the argument that physical exercise or alcohol consumption is associated with lung cancer.\textsuperscript{87 88} Predisposition to lung cancer may be related to genetic aberrations of leucocytes and relate to familial aggregation of lung cancer.\textsuperscript{89}

Two major randomised controlled trials designed to test beta-carotene as a chemoprotective agent among current and former smokers were negative; in fact, those receiving beta-carotene were at slightly higher risk of lung cancer, an unexpected finding since non-intervention studies had led to the belief that carotenoids might be protective.\textsuperscript{90} A recent review of chemo-protective trials also showed no protective effects from five different substances.\textsuperscript{91}

\subsection*{2.3.5 Therapies and outcomes}

Prognosis in lung cancer is poor, but is better when diagnosed early. Fewer than 10\% survive 5 years from diagnosis, and 50\% will die by 18 months.\textsuperscript{92} Survival varies by smoking status: of former smokers, 50\% are likely to die by 18 months, and of those who continue to smoke, 50\% will die by 14 months.\textsuperscript{93} In contrast to that for COPD, the decline is rapid – as late as 5 months prior to death, patients generally have few functional limitations and little pain.\textsuperscript{94}

\textbf{More than 90\% of people diagnosed with lung cancer die of it within five years. If they do survive to five years with no recurrence, they are regarded as cured.}

Once diagnosed, lung cancer is treated by radiation therapy and chemotherapy, and rarely by surgery to remove the affected part of lung. If diagnosed early, these treatments may extend life, but too often diagnosis is made too late.\textsuperscript{72}
2.3.6 Terminology and classification

Lung cancer sub-types (adenocarcinoma, small-cell, squamous cell, small cell, non-small cell and others less common) are based on their histology. The most common are squamous cell tumours which occurs largely in central lung zones, and adenocarcinoma which occurs largely in the peripheral lung zones. Although there are some differences between sub-types in risk factors, time trends, survival time and case-fatality, tobacco smoking is so overwhelming a risk factor that further description of sub-types is unjustified for this report.\(^9^5\)

Tumours are graded according into stages according to spread: I being localised, II intrathoracic spread, III nodal spread, and IV being metastatic and regarded as having poor prognosis. Staging is not considered further in this report.
3 Health impacts of COPD

3.1 Prevalence and incidence

3.1.1 Measurement

The proportion of the population with conditions such as COPD, that have a slow onset and with symptoms that can be difficult to distinguish from other conditions, is not easily measured. Part if the difficulty relates to lateness of people seeking help, and tolerance of symptoms. There is a pattern of under-recognition of the seriousness of their condition (“just smoker’s cough”) evident in delays to obtaining medical help and late diagnosis.\(^{96}\) Prevalence of COPD is usually estimated through routinely collected hospital discharge data or through population surveys. Each method has its advantages and disadvantages, and neither is very accurate. Appendix 1 illustrates the strengths and weaknesses of various methods.

COPD prevalence reported in a population depends very much on the criteria used.\(^{94,97}\) Several phenomena work counter to each other. Those studies which utilise self-reported respiratory symptoms (rather than reported diagnosis) typically produce high rates of respiratory symptoms due to the lack of specificity of the symptoms. Prevalence estimates based on self-reported symptoms yield estimates which are in most cases higher than those derived either from doctor-diagnosis or from measuring lung function, and probably have limited usefulness to assess the burden of disease. Studies report wide geographic variations.\(^{98}\)

Surveys that measure airflow in a representative sample of the population tend to find 2 – 4 times as many cases as are found from self-reported diagnosis alone. One large population-based study in the USA found that 63.3% of participants with low lung function documented at the time of survey had no prior or current diagnosis of obstructive disease.\(^{99}\) Another conducted in Spain found that 78.2% were previously undiagnosed.\(^{100}\)

Surveys that measure actual airflow find more than twice as many cases of COPD than surveys using self-reported diagnosis.

Recent development of criteria to standardise assessment of airways obstruction should in time lead to improved measurement and estimation of burden.\(^{59}\) Even greater accuracy will be possible as public healthcare organisation chronic disease registers become operational.

3.1.2 Prevalence of COPD in New Zealand

No good population-based surveys conducted in New Zealand have adequately described the burden of COPD. The only large, well-designed population-based survey known to have reported COPD prevalence in a New Zealand population was as part of the European Community Respiratory Health Survey (ECRHS), an international collaboration.\(^{101}\) It aimed to compare occupational groups, and originally enrolled a random population sample of about 12,000 people. An attempt was then made to contact and resurvey those who had reported some respiratory symptoms at the initial survey. The detailed questionnaire was completed by over 1,600 adults aged 20 to 44 years and breathing function was measured by spirometry in 1,132 (70.8%). Among those who had spirometry, 19.8% had an FEV% ≤ 0.75 and of these, 18.8% had FEV\(_1\) ≤ 80% predicted.
given the subject’s age and sex. On the basis of this study, which measured actual airflow on a sample of all participants, 1.8% had COPD in this young age group (20-44 years). However, the condition affects mainly those aged over 65 years, so these results are misleading for the population as a whole.

The population-based National Health Survey conducted by Statistics New Zealand and the Ministry of Health asked about one symptom (being woken by shortness of breath) and about asthma, but did not test lung function nor record diagnosis of COPD. In the 1996-97 survey, after weighting to reflect the sex and age distributions of the New Zealand population who were living in private dwellings, 10.1% of participants reported being woken by an attack of shortness of breath during the last 12 months, due to any one of a number of possible causes.

This sparseness of New Zealand-specific data will soon be rectified with two studies currently gathering information on the prevalence of COPD.

In 2001 the Health Research Council approved funding for a population-based survey which is now underway in Auckland. It includes people aged 35 to 74 years, with both Maori and Pacific peoples represented. It will assess the prevalence of self-reported diagnoses and breathing problems and describe lung function as measured by spirometry (P. Black, personal communication). Another study of COPD is a Wellington survey of a random sample of 3,000 people aged 25 to 75 years. Following administration of a questionnaire, three sub-samples (one random, one of cigarette smokers, one with respiratory symptoms) will be invited to undergo further laboratory testing and be followed for a year or more to monitor progression of lung function. The relationship between respiratory symptoms, severity, and progression of the disease will be described (R. Beasley, personal communication).

Until results of these two studies become available, the prevalence of COPD in New Zealand is best estimated by generalising from studies carried out in other comparable countries. In the 1990 Global Burden of Disease project, the authors estimated prevalence of diagnosed COPD to be 834 per 100,000 (of any age) worldwide and 535 per 100,000 (of any age) in established market economies similar to New Zealand.

However, when both diagnosed and undiagnosed airways obstruction are combined, prevalence is higher. The WHO-supported Global Programme on Evidence for Health Policy estimated lung function obstruction for New Zealand from evidence of spirometrically-determined prevalence in Australia and northern American countries (age-standardised rate of 2-5%), and for northern European countries (3-7%). For physician-diagnosed COPD, they made no direct estimate for Australia or New Zealand. These WHO-derived rates are, in general, lower than those shown in rigorously-conducted population-based studies.

To produce more reliable estimates, Shibuya et al. used statistical models with data derived from COPD mortality, and applying what is known about incidence, duration and mortality. Prevalence estimates of diagnosed COPD for men and women were derived separately for the Western Pacific Region A countries including New Zealand and Australia, and are shown in Table 3-1. By applying these models to the population of New Zealand aged 45 years and over, COPD prevalence was estimated at 40,000 people in 2002. However, when the equivalent rates for northern American countries and for northern European
countries were applied to New Zealand population, estimates reached 50,000 and 75,000 respectively. Because the New Zealand and Australian estimates were based on so few small Australian studies, including one conducted 35 years ago, and because it is unlikely our rates would be so much lower than comparable countries such as the UK, Canada or the USA, the three estimates have all been used for costing purposes in this report.

Table 3-1  Estimates of prevalence of COPD in New Zealand, based on WHO regional estimates

<table>
<thead>
<tr>
<th></th>
<th>For WPRO A: Western Pacific Region, incl. New Zealand &amp; Australia</th>
<th>For EURO A: European Region incl. UK, Sweden, Italy, Finland, Denmark, Spain, the Netherlands etc</th>
<th>For AMRO A: American Region incl. Canada &amp; USA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>Prevalence per 1000</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>12.40</td>
<td>14.90</td>
<td>18.20</td>
</tr>
<tr>
<td><strong>All 45+ years</strong></td>
<td>31.70</td>
<td>38.10</td>
<td>49.70</td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-4 years</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>5-14 years</td>
<td>0.07</td>
<td>0.03</td>
<td>0.00</td>
</tr>
<tr>
<td>15-29 years</td>
<td>0.71</td>
<td>1.28</td>
<td>0.04</td>
</tr>
<tr>
<td>30-44 years</td>
<td>5.64</td>
<td>4.42</td>
<td>7.95</td>
</tr>
<tr>
<td>45-59 years</td>
<td>14.28</td>
<td>22.69</td>
<td>38.45</td>
</tr>
<tr>
<td>60-69 years</td>
<td>41.05</td>
<td>44.95</td>
<td>67.62</td>
</tr>
<tr>
<td>70-79 years</td>
<td>63.70</td>
<td>80.14</td>
<td>96.00</td>
</tr>
<tr>
<td>80+ years</td>
<td>113.31</td>
<td>100.89</td>
<td>84.73</td>
</tr>
<tr>
<td><strong>All men</strong></td>
<td>15.73</td>
<td>17.72</td>
<td>20.67</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-4 years</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>5-14 years</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>15-29 years</td>
<td>0.25</td>
<td>0.27</td>
<td>0.13</td>
</tr>
<tr>
<td>30-44 years</td>
<td>7.62</td>
<td>11.30</td>
<td>3.55</td>
</tr>
<tr>
<td>45-59 years</td>
<td>20.04</td>
<td>23.00</td>
<td>20.52</td>
</tr>
<tr>
<td>60-69 years</td>
<td>27.44</td>
<td>38.50</td>
<td>60.66</td>
</tr>
<tr>
<td>70-79 years</td>
<td>41.47</td>
<td>43.98</td>
<td>73.99</td>
</tr>
<tr>
<td>80+ years</td>
<td>58.84</td>
<td>68.72</td>
<td>64.91</td>
</tr>
<tr>
<td><strong>All women</strong></td>
<td>15.13</td>
<td>18.24</td>
<td>16.86</td>
</tr>
<tr>
<td><strong>Estimated counts for New Zealand in 2002</strong></td>
<td>40,000</td>
<td>50,000</td>
<td>75,000</td>
</tr>
</tbody>
</table>

Source: Adapted from Shibuya 2001

In an alternative approach, estimates of spirometrically-measured airway obstruction are possible. The most reliable estimates are likely to come from the work of Stang and others, who used four population-based surveys combining self-reported symptoms and spirometry, with the prevalence of tobacco smoking, to derive population estimates of those affected by COPD, even if currently undiagnosed. These rates are plotted in Figure 3-1.

However, Stang-based estimates for New Zealand produce counts that are considerably higher than other estimates: almost 223,000 prevalent cases of whom almost half (48%) would be aged under 55 years and perhaps 50,000 (22%) with the condition diagnosed. Although these are very high, the ratio of diagnosed to undiagnosed COPD is within the range reported in studies elsewhere.

For the purpose of the financial estimates later in this report, three estimates of prevalence of doctor-diagnosed COPD are presented: for 40,000, 50,000 and 75,000 people, about 3.3%, 3.8% and 5.0% respectively of those over 45 years.
In New Zealand, it is probable that:

- about 223,000 have obstructive disease, only 22% diagnosed,
- about 50,000 people 40+yrs have diagnosed COPD, and
- about 180,000 people 40+yrs have undiagnosed COPD.

3.1.3 Variations by age, sex and ethnicity in New Zealand

Increased age has long been known to be associated with risk of COPD, due to accumulated pack-years of smoking. In the absence of good New Zealand data, Figure 3-1 demonstrates well this relationship using the models of Stang.\(^{104}\) That the condition need not necessarily be only experienced only by older people was apparent in a recent survey from Australia using spirometry, in which 51.6% of people with mild COPD were aged under 40 years.\(^{105}\) Younger age of starting smoking may be leading to onset of COPD at younger ages.

It used to be thought that women were protected in some way from tobacco-related disorders. It is now apparent that if anything, women are more susceptible to COPD than are men, given the same levels of tobacco exposure.\(^{106}\)

*Figure 3-1 Estimates of COPD rates in four population-based studies in USA, including both diagnosed and undiagnosed, by smoking status, sex and age group*

Between ethnic groups, differences in prevalence may be related solely to differences in exposure to tobacco smoke and environmental toxins. This is not necessarily so, since there are familial aggregations of genetic markers known which are linked with higher rates of COPD, but as yet there is no evidence of race-specific differences which could not have been the result of exposure to tobacco or other causal agents.

3.1.4 Variations by occupation, deprivation and/or socio-economic status in New Zealand

The ECRHS study found that particular occupations (of 21 different categories) were at elevated risk of various respiratory symptoms compared to professional and administrative workers in New Zealand. Some of the symptoms suggested obstructive airways: bakers (26% higher risk of obstruction-related conditions), other food processors (3% higher risk of chronic bronchitis), spray painters
(about 15% higher risk of obstruction-related conditions) and chemical processors (18.8% higher risk of chronic bronchitis). There were many where occupation was not described adequately enough to allow clear categorisation, and this, with the small numbers in some categories, may have prevented other groups at higher risk from being identified.\textsuperscript{101}

3.1.5 Comparable other countries

Appendix 2 demonstrates how widely variable reported COPD prevalence is, even in relatively similar developed countries. Differences arise primarily from historical exposure to tobacco smoke, but much also is artefactual, resulting from differing methods of ascertainment (reported symptoms, self-reported diagnosis, or spirometry).\textsuperscript{97} In addition, there are methodologic variations in definition of COPD and selection of different age groups in the population, in particular the exclusion of older people from many studies.

As an example of an oft-cited study, in the USA, the National Health and Nutrition Survey III (NHANES III) conducted from 1988 to 1994 included over 20,000 adults.\textsuperscript{107} A sub-sample of 5,743 aged 45+ years underwent spirometric measurement of breathing function, and FEV\textsubscript{1}\% calculated. Questionnaires were used to record any history of respiratory disease or symptoms and history of cigarette smoking, and rates calculated for an age-standardised USA population. Undiagnosed airflow obstruction (12.0\%) was substantially more common than either diagnosed COPD (3.1\%) or diagnosed asthma (2.7\%), indicating the extent of measurement differences. Airway obstruction was more prevalent in men (14.2\%) than in women (9.9\%), and was clearly associated with age.\textsuperscript{99}

3.2 Quality of life

The only published data on quality of life for people with COPD in New Zealand is for a small cohort of patients hospitalised for acute exacerbations of COPD. It showed patients had lower than expected scores on Physical Function subscale of the SF-36 (a measure of health-related quality of life).\textsuperscript{108} Many required help when they were breathless, and depended on family and friends for help with showering (43\%), shopping (43\%) and meal preparation (23\%).

In the NHANES III study described above, self-reported adverse effects on health and functional status increased with severity of FEV\textsubscript{1} impairment. The magnitude of association between extent of breathing impairment and health and functional impact was similar to or greater than those of being elderly, obese, or with conditions such as arthritis, cancer, cardiovascular disease or diabetes. Amongst those with undiagnosed obstruction, self-reported impairment was associated with the degree of airflow obstruction even after adjustment for clustering of co-morbidities in smokers.\textsuperscript{107}

In a study of seriously ill Yorkshire patients, 90\% of those with end-stage COPD were found to suffer clinically relevant anxiety or depression: in contrast, 52\% of patients with non-small cell lung cancer had anxiety or depression. The impaired quality of life and emotional wellbeing in COPD patients gave rise to needs which were not as well met as those of patients with lung cancer.\textsuperscript{109}
3.3 Morbidity

3.3.1 Hospital discharges in New Zealand

People with COPD tend to live in the community, at least until very late on the disease process when they may require residential or private hospital care. Acute exacerbations may however require admission to hospital. Discharges are coded routinely and submitted to the New Zealand Health Information Service (NZHIS) for summarisation and monitoring. Two main coding schemes are used, the International Classification of Diseases (ICD) for diagnoses, and Diagnostic-Related Groupings (DRG) for grouping people with similar care requirements for funding purposes. However, neither of these systems provides data which clearly distinguishes people with COPD.

In order to tabulate hospitalisations for this report, we obtained summaries of discharge data from all public hospitals in New Zealand for the conditions coded under the ICD systems shown in Table 3-2.

Table 3-2 Codes used in sourcing hospitalisation & death data for COPD

<table>
<thead>
<tr>
<th>ICD Revision</th>
<th>ICD-9CM</th>
<th>ICD-10CMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchitis, not specified as acute or chronic</td>
<td>490</td>
<td>J40</td>
</tr>
<tr>
<td>Chronic bronchitis</td>
<td>491</td>
<td>J41x &amp; J42</td>
</tr>
<tr>
<td>Emphysema</td>
<td>492</td>
<td>J43x</td>
</tr>
<tr>
<td>Chronic airway obstruction, not elsewhere classified</td>
<td>496</td>
<td>J44x</td>
</tr>
</tbody>
</table>

(x represents any one of several more finely classified ICD-10 codes not used in this report.)

To illustrate the difficulties in relying on discharge codings, a recent paper from Norway shows that in an audit of hospitalisations coded as COPD, 58% of hospital stays required recoding: 31% led to changes in DRG codes which were sufficient to mean the hospital was under-funded by on average 0.30 DRG points. There was a large variation in recording between coders and several problems related to interpretation of ICD-10 coding.110 Another report from the USA shows that much of the recent observed doubling of reported morbidity occurred as a result of changing the standard population used.111 The same report describes increases in reported chronic bronchitis and reduction in acute bronchitis, about 1992, when a new coding category was introduced for acute exacerbation of chronic bronchitis. Similar effects are likely on mortality codings.

Figure 3-2 shows hospital discharges for the year 1998/1999 which have one of these ICD codes as the principal diagnosis. During 1998/1999, approximately 7400 (1.05% of all) discharges from public hospitals were for COPD. Mean length of stay for these admissions in 1998/1999 was 9.3 days, but in 1999/2000 mean stay was 9.6 days (see 3.3.4).

Numbers of discharges have increased noticeably since 1997: in 1995 there were 6400 discharges, and in 1996, 6500. By 1997 numbers had jumped to 7000, then 7400 in 1998 and over 9000 in 1999. At least part of this increase is a result of changes to the ICD-10 coding systems for acute exacerbations of COPD.

Fewer than one in 20 COPD patients (4.8%) seen in public hospitals were treated as day-patients. Data for private hospital admissions show that their facilities are seldom used for acute exacerbations: in 1995, the last year for which data are available, only 152 discharges were for COPD and related conditions.112
COPD is a common comorbidity for hospitalisations for people admitted for other reasons, adding to the lengths of stay and therefore to costs. This is further described in 3.3.4.

**Figure 3-2 Numbers of COPD Hospital discharges in New Zealand, by sex, age group and ethnicity, 1998/1999**

3.3.2 Variations by age, sex and ethnicity in New Zealand

As seen in most tobacco-related conditions, COPD is age- and sex-related: the majority (about 40%) of COPD admissions are of those aged 65-79 years, with higher rates for men. The mean age for those admitted is 70.7 years.

Hospitalisations in Maori occur at earlier ages and rates increase more steeply with age than non-Maori, with Maori women bearing the greatest burden (Figure 3-3). At the present time, there is a drop-off in hospitalisation rates among very old women, but this is no longer the case for men. Mean age at hospitalisation for Maori is more than eight years younger than of non-Maori (63.4 and 71.7 years respectively). Much of the heavier burden of COPD Maori have over non-Maori is likely to result from their history of smoking, and perhaps also from greater use of loose tobacco by Maori.113

Hospitalisation rates among Maori women are more than double those of non-Maori at all ages; in men this difference is not so marked. A review of hospital admission data in the Auckland region by Garrett and others reported improved survival after admission among Maori compared to non-Maori. They reasoned that Maori people tended to present at hospital in more acute condition, and argued that clinicians would more likely admit them in order to be safe (Garrett, unpublished manuscript, 1999). However, alternative explanations are possible, for example misclassification of asthma as COPD among Maori. Evidence necessary for testing this explanation, such as data which describe presentations to hospital emergency departments, are not publicly available.

Rates of hospitalisation for Maori are more than double those for non-Maori. It is not known to what extent this reflects greater prevalence of COPD among Maori, or more frequent admissions for Maori patients who have the disease.
3.3.3 Variations by deprivation in New Zealand

A socio-economic gradient was described for COPD in hospitalisations in 1997-98 in a Ministry of Health publication. Compared with deciles 1-4 (the least deprived 40% of the population), the higher deciles showed an additional 1620 discharges, 41% of all discharges for COPD. This report was not able to control for smoking however, and overseas evidence does suggest that most of such differences are explainable by the association of smoking (or exposure to environmental tobacco smoke) and socio-economic status.

In the Auckland study mentioned above, the New Zealand Index of Deprivation, (NZDep), was obtained for patients hospitalised for COPD. Time to readmission or death was calculated and compared across deprivation categories; but no significant differences were found (J. Garrett, unpublished manuscript).

3.3.4 Hospital bed-days in New Zealand

Since 1995, average lengths of stay in public hospitals for discharges with COPD as the primary diagnosis have varied from a low of 7.4 days in 1997 to 9.6 days in 1999. The increase in 1999 may arise from greater clarity in instructions for coding admissions acute exacerbations; in the past these had sometimes been coded as acute bronchitis.

In private hospitals, much longer stays (e.g. 183.1 days in 1997) suggests that when they are used, it is for long-term, possibly end-of-life, care. A total of 86,370 bed-days were used in 1999/2000 for COPD in public hospitals and an estimated 28,000 in private hospitals, totalling over 114,400 days.

Length of stay for men with COPD as primary diagnosis is 10.6 days, 2 days longer than for women (8.6 days). As has been repeatedly shown previously and
for many conditions, lengths of stay for Maori are shorter on average, 5.8 days compared to 10.1 for non-Maori. Reasons may involve younger age of Maori patients, and for some, a preference to return home at an earlier stage.

Table 3-3  Numbers of COPD discharges, and associated mean lengths of stay in 1999/2000 according to ICD-10 sub-group, by sex (mean days)

<table>
<thead>
<tr>
<th>ICD-10 sub-group</th>
<th>Men</th>
<th>Women</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N mean stay</td>
<td>N mean stay</td>
<td>N mean stay</td>
</tr>
<tr>
<td>J40 Bronchitis, not specified as acute or chronic</td>
<td>215 3.2</td>
<td>300 3.7</td>
<td>515 3.5</td>
</tr>
<tr>
<td>J41 Simple &amp; mucopurulent chronic bronchitis</td>
<td>6 8.0</td>
<td>3 3.5</td>
<td>9 6.5</td>
</tr>
<tr>
<td>J42 Unspecified chronic bronchitis</td>
<td>24 7.4</td>
<td>24 4.2</td>
<td>48 5.7</td>
</tr>
<tr>
<td>J43 Emphysema</td>
<td>260 11.0</td>
<td>158 8.7</td>
<td>418 10.1</td>
</tr>
<tr>
<td>J44 Other chronic obstructive pulmonary disease</td>
<td>4158 8.6</td>
<td>3878 11.0</td>
<td>8036 9.8</td>
</tr>
</tbody>
</table>

Of the hospitalisations for sub-groupings of COPD in 1999/2000, emphysema had the greatest mean length of stay (10.1 days), and non-specific bronchitis had the shortest mean stay (3.5 days). The largest group is ‘Other chronic obstructive pulmonary disease’, which accounts for 89.0% of the discharges with mean stay of 9.8 days, and a total of 78,800 days (Table 3-3). Across all groups shown, the total days stay was 85,100, 1.5% of all public hospital bed-days in 1999/2000.

In those whose primary diagnosis is a condition other than COPD, coexisting COPD is associated with longer stays. Table 3-4 shows that among people aged over 45 years admitted for seven important other diagnostic groups, concurrent COPD is associated with increased length of stays. However this does not apply to all conditions.

Table 3-4  Numbers of discharges and associated mean lengths of hospital stay (days) in patients over 45 years, where COPD is, or is not, a co-morbid condition

<table>
<thead>
<tr>
<th>Selected condition and ICD 9 code</th>
<th>Without COPD N mean stay</th>
<th>With COPD N mean stay</th>
<th>Additional bed-days per capita nationally</th>
</tr>
</thead>
<tbody>
<tr>
<td>Other circulatory disease (440-459)</td>
<td>7268 7.0</td>
<td>695 11.3</td>
<td>4.2 2946</td>
</tr>
<tr>
<td>Diseases of the digestive system (520-579)</td>
<td>24437 5.9</td>
<td>1443 7.9</td>
<td>2.0 2880</td>
</tr>
<tr>
<td>Fractures and injuries (800-829)</td>
<td>8406 8.2</td>
<td>615 10.5</td>
<td>2.3 1428</td>
</tr>
<tr>
<td>Other musculoskeletal (720-779)</td>
<td>8287 13.6</td>
<td>430 15.8</td>
<td>2.2 939</td>
</tr>
<tr>
<td>Arthropathies and related disorders (710-719)</td>
<td>7679 14.2</td>
<td>320 16.4</td>
<td>2.2 693</td>
</tr>
<tr>
<td>Other injuries (830-995)</td>
<td>7675 4.6</td>
<td>353 6.5</td>
<td>1.9 660</td>
</tr>
<tr>
<td>Infectious &amp; parasitic diseases (001-139)</td>
<td>2943 7.7</td>
<td>275 9.5</td>
<td>1.8 498</td>
</tr>
</tbody>
</table>

This has been reported elsewhere - a study in Finland showed that among people hospitalised with pneumonia or influenza, those with COPD had stays averaging 14.7 days compared to 7.7 days if uncomplicated.

3.3.5 COPD hospitalisations in comparable countries

Length of stay for COPD in Australian private and public hospitals in recent years varied between 7.2 days in 1997/98 and 4.6 days in 2000/01, but there was no clear trend toward shorter stays. Kinnunen reported lengths of hospital stay in Finland between 1987 and 1998 as on average 8.9 days, and were significantly increased if there were co-morbidities. In the UK in 1994, COPD stays were 9.9 days. While acknowledging that there has been an international trend to shorter stays over time, valid comparisons between countries are difficult.
because of differences in heaviness of smoking over time, and institutional differences e.g. in admission criteria, diagnostic and coding practices.\textsuperscript{119}

3.4 Survival

Survival for those who have been hospitalised with COPD in New Zealand is currently being measured (P. Poole, unpublished data); interim findings suggest it is not dissimilar to those found overseas. In general, studies conducted overseas have shown that use of different inclusion criteria lead to very different results that are not readily applicable to other patient groups, particularly if a standardised instrument is not used to assess severity.\textsuperscript{39}

For example, in one study of elderly patients with stable COPD, less than 20% of patients had died one year after entry to the study.\textsuperscript{120} In another, of those admitted for an exacerbation with respiratory failure, 40% died within a year.\textsuperscript{121} However among those having had rehabilitation for serious COPD, only a third of patients lived more than six months after acute COPD exacerbation, and only half lived to two years.\textsuperscript{122}

3.5 Mortality

3.5.1 Measurement

Published mortality data are likely to significantly misrepresent the true burden of the disease for the following reasons:

- A quality-assurance study conducted in Denmark showed that at least 109 (50 to 60%) of 218 deaths ascribed to asthma during 1994/95 were in fact due to COPD.\textsuperscript{123} In contrast, in Ireland a similar study found that 25% of deaths certified and registered as COPD could have been called asthma using current standards of clinical diagnosis.\textsuperscript{124} If similar misreporting of COPD deaths is occurring in New Zealand as in Denmark, the true burden of fatal COPD could be anywhere between 25% lower or 2.5 times higher than published.

- The disease is more likely to be cited as a contributory than an underlying cause of death, or may not be cited at all.\textsuperscript{125} In one study, inclusion of COPD as a contributing cause of death more than doubled the number for COPD alone (Mueller et al. manuscript in preparation).

In combination, potential errors in reports could mean that true COPD mortality is up to 2½ times the published rates.

It is acknowledged that, because of substantial comorbidity, patients with COPD often die with their disease rather than from their disease. Perhaps only 45% to 63% of deaths in patients with advanced lung disease are directly due to the disease itself.\textsuperscript{39}

Faced with multiple chronic conditions, it is often not straightforward deciding which diagnoses to write as “direct”, “antecedent”, “underlying” or “other significant condition” cause of death on a death certificate. While the NZHIS does provide guidelines for the determination of underlying condition, it is dependent on the certifying medical practitioner to apply the rules and to provide adequate information.\textsuperscript{126}
3.5.2 COPD deaths in New Zealand

In New Zealand, COPD is the 4th leading cause of death after ischaemic heart disease, stroke and lung cancer. In 1999, there were 1443 deaths ascribed to COPD, 5.1% of all deaths. Of those aged 35-64 years, COPD accounted for 2.2% of deaths, and of those aged over 65 years, 7.1%. In the same year 1041 (72.1%) of COPD deaths were of people aged over 75 years, in the “oldest old”, a greater burden than is recognised (Figure 3-4).

Over 72% of COPD deaths in New Zealand in 1999 were among people older than 75 years. This is a group who are seldom included in population-based surveys or in randomised trials of therapeutic agents.

Figure 3-4 Numbers of COPD deaths in New Zealand, by sex, age group and ethnicity, 1998

3.5.3 Mortality rates in New Zealand

Mortality data were obtained using the same ICD codes as for hospitalisations. Plots of the rates for the three year period 1997/98 to 1999/2000 are shown (Figure 3-5). Again, it is seen that COPD impacts mostly on the old and on Maori (although small numbers render results for older Maori unreliable).

3.5.4 Variations by age, sex and ethnicity in New Zealand

COPD deaths occur largely in older people and, in general, mortality rates for men have been about twice those for women. Almost half the deaths reported from COPD (710 or 49.2% in 1999) were among people aged over 80 years, and less than 5% were aged under 60 years (in 1999, 64 deaths or 4.4%). Mean age at death was 77.4 years (77.9 for non-Maori, 70.5 for Maori).

The Ministry of Health has reported mortality rates separately for Maori and non-Maori. Among men, age-standardised mortality rates are 19.8 per 100,000 for non-Maori and 31.5 for Maori. Women show the greatest differences between the ethnic groups however: 9.1 per 100,000 in non-Maori and 35.9 for Maori.
COPD mortality rates for Maori women in New Zealand are higher than reported in any other known population of women worldwide.

Figure 3-5  COPD mortality rates (averaged over 3 years) in New Zealand, by sex, age group and ethnicity (per 100,000)

3.5.5 COPD death rates in comparable countries

Rates of COPD in New Zealand appear to sit among those countries with a roughly matching history of tobacco consumption (Figure 3-6). Highest rates are seen in the Eastern European and ex-USSR countries where tobacco smoking has been particularly prevalent. Data are not available for many Asian and African countries, such as China, India and the South African Republic. Marked disparities between rates for men and women are seen in most countries, though are less marked in New Zealand.

Interpretation of such data is difficult. Greece for example has historically had high prevalence of smoking and high per capita consumption of tobacco, yet reports surprisingly low COPD mortality rates. A number of reasons have been mooted: high fruit and vegetable consumption, genetic protection, tobacco smoking at later age, religious fasting or death certification and recording conventions. Comparisons between countries in cause of death data are subject to the same reservations for New Zealand data, with in addition, national variations in diagnosis and ascribing cause of death. Whilst Figure 3-6 can safely be interpreted as showing NZ mortality as generally in the same league as countries such as Australia, France or Sweden, should not be used for ranking purposes.

3.6 Long-term trends in New Zealand

3.6.1 Trends in prevalence

Just as there are no good data on trends in prevalence of COPD are available for New Zealand, there are no good data describing trends. In other countries, COPD prevalence for men has begun to fall, but those for women are still rising, although they have not yet reached the levels for men. Patterns in tobacco smoking over the last few decades in New Zealand suggest that prevalence
should soon decline in men, but will continue to increase for women for some time yet.

Figure 3-6  Age-standardised mortality rates for COPD, selected countries, 1995/1996 (per 100,000)

3.6.2 Trends in hospitalisations

Hospitalisation rates for COPD are still climbing among men and women, Maori and non-Maori. Population rates age-standardised to the 2000 total New Zealand populations show average annual increases over the past three years of 32-33 per 100,000 for non-Maori men and women, 46 per 100,000 for Maori men, and 61 per 100,000 for Maori women (Figure 3-7). Not only are rates for Maori already very high, they are also rising steeply, at twice the rate of non-Maori.

3.6.3 Trends in mortality

Age-standardised COPD mortality rates have declined slowly but steadily between 1980 and about 1992, and since then, have levelled off (Figure 3-8). Rates for men overall continue to decline, but rising rates for women make up the difference. The decline in men is driven by reductions in non-Maori men, as rates continue to rise in Maori. Again, it is possible that changes in coding practices (as well as patterns of tobacco smoking) may be partly responsible for changes over time.
Murray and Lopez, in their WHO-sponsored Burden of Disease study, reported that world-wide, COPD was the only major cause of death that still has a rising mortality, and predicted that by the year 2020, COPD will be fifth leading cause of DALYs lost worldwide. While that may yet be shown, evidence of a continuing rise for New Zealand is not convincing.

In Canada, death rates from COPD are starting to level off for men, but not for women. Figure 3-9 shows in USA mortality trends, the dramatic and continuing increase in mortality among women in comparison to the slightly falling rate in men since the mid-1980s. For men and women together, COPD mortality continues to rise, in contrast to the falling death rates for all other
major causes of death over recent decades.\textsuperscript{1} Vermeiere demonstrated the
dramatic rise in COPD mortality in the USA, with falls for all other main causes of
death.\textsuperscript{129} However, when these analyses were replicated using data from New
Zealand, trends in COPD were not as compelling.

Figure 3-9  Age-adjusted mortality for COPD in USA, 1979-1998, by sex.

3.7 Trends in causes and risk factors for COPD

In New Zealand, it is estimated that at least 65\% of female deaths and 79\% of
male deaths from chronic obstructive respiratory disease are due to tobacco
smoking.\textsuperscript{4} Given that 90\% of cases are believed to be smoking-related, many
smokers with COPD must die of other smoking-related conditions. Higher COPD
prevalence among Maori is likely to result from high smoking prevalence (around
50\%) of the adult Maori population compared to non-Maori adults (about 25%)
(see Figure 1-2). Whether or not increasing use of loose tobacco impacts upon
rates of morbidity or mortality is not yet known.

Long delays between the highest levels of tobacco consumption per capita
(around 1976) and the peak in hospitalisations among men show the smoking-
related impact occurs after about 20-25 years’ exposure to tobacco. Overall
rates of COPD are likely to rise again because following higher smoking levels in
women in recent decades.

Patterns in other modifiable independent risk factors may also impact. If not
checked, rising rates of overweight and obesity may increase the COPD burden.
An association of occupation-related exposure to dust has been demonstrated
amongst bakers, chemical processors and spray painters.\textsuperscript{101} These occupations
are not large employers of people in New Zealand, so it is unlikely they markedly
influence COPD rates. Nevertheless, there may be an opportunity to increase
efforts within these industries to reduce future disease.

3.8 Projections and prevention

3.8.1 Prevalence

Future trends are most influenced by patterns in tobacco smoking. A Dutch
group modelled prevalence of COPD based on known smoking levels, and
concluded that there will be an unavoidable increase in the burden of COPD over the 20 years to 2015, an increase which is almost three-fold higher for women (142%) than for men (43%). When those results are applied to New Zealand data with a projected population for 2025 (medium immigration, medium mortality) and current smoking levels, prevalence is projected to rise to 378,000, to 8.9% of women, and to 8.0% of men over 40 years.

Figure 3-10 Projected prevalence of COPD in New Zealand in 2025, from models developed by Stang and applied to projected NZ population and using 1996 smoking prevalence

3.8.2 Hospitalisations

Based on the predicted population of older people and present rates of COPD discharges, and if smoking levels continue as at present, hospitalisations can be expected to rise to over 12,000 per year by 2007, and almost 15,000 by 2012.
4 Cost Burden of COPD

4.1 Basis of costings

Economic evaluations which have been undertaken elsewhere have had reasonable population prevalence data on which to base their costings. In New Zealand this is not so, and the following estimates of the direct costs of health care have had to be based on poorly-measured data, not easily verifiable through alternative sources. They should therefore be regarded as indicative only.

It is clear from overseas studies and from the under-reporting of COPD, that costs of the disease are related to severity. In Sweden, costs per person for severe COPD were three times as high as costs for moderate disease, and more than ten times as high for moderate as for mild disease, but there are large individual variations. The following estimates consider severity by adjusting the proportion of patients who are likely to access each service. They do not include those with respiratory symptoms with no diagnosis of COPD.

4.2 Summary of costings

The main costs to the health system in New Zealand directly attributable to COPD are medications, hospital care (inpatient and emergency clinics), and visits to general practitioners. Laboratory services, provision of emergency department visits, smoking cessation programmes and laboratory testing also feature.

Direct costs to the New Zealand health care system for diagnosed COPD amount to a minimum of $102.6 million in the year 2002 (Table 4-1). More likely they will reach $128.3 million, and in the highest cost scenario, as much as $192.4 million. These exclude personal costs, those relating to domiciliary care, and less recognised health care costs (e.g. traditional healers, herbal therapies, dietary supplements etc). It also excludes the burden through indirect costs such as loss of earnings and quality of life. On average, direct costs per year for each patient are likely to be almost $2,600.

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<th>Item</th>
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<th>50,000 ('000s)</th>
<th>75,000 ('000s)</th>
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<td>Pharmaceuticals</td>
<td>16,320</td>
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<td>30,600</td>
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<td>GP visits</td>
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<td>9,000</td>
<td>13,500</td>
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<td>Emergency department visits</td>
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<td>3,400</td>
<td>5,100</td>
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<td>Smoking cessation programme</td>
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<td>2,880</td>
<td>4,320</td>
</tr>
<tr>
<td>Laboratory testing</td>
<td>2,200</td>
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<td>4,125</td>
</tr>
<tr>
<td>Pulmonary rehabilitation (course)</td>
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<td>3,550</td>
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<tr>
<td>Other direct health care</td>
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<td>6,833</td>
<td>10,250</td>
</tr>
<tr>
<td><strong>Total ('000)</strong></td>
<td><strong>102,630</strong></td>
<td><strong>128,288</strong></td>
<td><strong>192,432</strong></td>
</tr>
<tr>
<td><strong>Average per patient per year $</strong></td>
<td><strong>2,566</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 4-1 Estimated direct costs to the health services of COPD in New Zealand, 2002 ('000s)
4.2.1 Cost of hospital inpatient care

Hospitalisation costs are the highest cost item. They represent about 63% of the total costs, and are the most easily identified. Inpatient costs were costed at $925 per day, the current cost of a bed in a medical ward in a public hospital. If 50,000 people with COPD had 9,250 admissions for COPD, each of mean duration 9.6 days, the costs would be $80.6 million.

A study from Spain reported that COPD costs per patient were twice those for asthma. The same study showed that costs of drug treatment among those with COPD were almost as high as the costs of hospitalisations, so our pharmaceutical estimates may be understated. A UK report observed that hospitalisations for COPD cost 4.3 times as much as those for asthma. Further, they claimed that half the costs of COPD were in secondary care, of which 78% were admissions. If this were true for New Zealand, then total health care costs of COPD would reach over $193 million, close to our highest estimate of $192.4 million.

In the USA, hospitalisation costs represent 40.4% of total health care costs for patients with mild COPD, and 62.5% of total costs for patients with severe COPD. Of the total cost of care of COPD patients, 58% was for hospitalisation, very close to our estimate of 63%.

4.2.2 Cost of pharmaceuticals

Total estimated costs of medications for COPD are estimated at between $16.3 and $30.6 million, about 16% of total costs. Estimates are based on all currently diagnosed patients with diagnosed COPD having nine medications (antibiotics, bronchodilators and/or steroids) prescribed per year, at a cost of $45 per medication. However, these estimates are not easily verifiable. Estimates do not include costs for those people with respiratory symptoms but (as yet) undiagnosed COPD.

4.2.3 Cost of primary care

Based on estimates of four visits per patient per year to a general practitioner (GP), and 50,000 prevalent diagnosed COPD cases, there are an estimated 200,000 GP visits per year in New Zealand for COPD. At a cost of $45 per visit, GP costs are estimated at $9 million.

In relative terms, a UK study of GP visits showed that COPD was responsible for four times the number of visits as angina.

4.2.4 Cost of hospital emergency department visits

Inpatient stays are only one part of hospital costs for COPD patients. New Zealand data for emergency department visits are not available, and even if they were, it is likely that for many the diagnosis of COPD is missed. Conservative estimates assume that, on average, new patients visit an emergency department once early after diagnosis, and thereafter that 5% of patients with COPD visit an emergency department each year, with a costs of $600 per visit.

4.2.5 Cost of smoking cessation programmes

Estimates are based on the assumption that 40% of newly-diagnosed COPD patients, and 10% of those previously diagnosed, access a smoking cessation programme. These estimates are expected to vary widely geographically and by care provider, and they are based on advice of several experienced clinicians.
4.2.6 Cost of laboratory tests

It is expected that all new COPD patients will have initial blood tests to help rule out other similar conditions. Thereafter a blood test once a year is likely with a unit cost of $55.

4.2.7 Cost of pulmonary rehabilitation programmes

Although pulmonary rehabilitation has proven benefits, it is doubtful if more than 2% of new COPD patients, or 10% of existing patients in New Zealand access such a programme. Costings have been made on this basis at a cost of $500.

4.2.8 Cost of other direct health care

Other hospital-based services commonly used by COPD patients include X-rays, ambulance services and outpatient visits. None is estimated at more than 1% of the total average cost per patient per year.

Little information is available on use of oxygen therapy for COPD in New Zealand. A paper from Sweden suggests that oxygen therapy is used by about 20% of patients with \( \text{FEV}_1 < 40\% \), i.e. those most seriously affected by the disease.\(^{131}\) Although proven as cost-effective, New Zealand figures are likely to be lower.

Mobility aids such as wheelchairs seem to be used by few people with COPD. Home adaptations and education or occupational therapy are used even less and are not included in the assessment of costs.\(^{131}\)

Direct health care costs of COPD in New Zealand exceed $102 million annually, and are possibly almost twice that.

4.3 Direct costs to patients and families

4.3.1 Part-payments

Where patients make co-payments for subsidised care, for example GP visits, these are not included. However, where they pay full costs, for example for a private specialist, these costs are included in ‘Other direct care’.

4.3.2 Personal cares and other costs

Not included in these estimates are ‘respite’ or family relief care or long-term rest-home care, additional travel to access health services, loss of earnings of family members to give up work to care for parents, partners or other dependents. Such costs would fall mainly on the patient and the family, and not be included in the estimates above. These expenses are dependent on personal and family resources, and impact on the wider families’ quality of life.

4.4 Indirect costs

The following costs are not included above, but are likely to impact significantly on either taxpayers or on individuals and their families.

4.4.1 Pensions

Costs of pensions for people disabled by COPD in New Zealand have not been included in the estimates above. In Sweden, costs of pensions for people disabled by COPD are 1.25 times as high as from all direct health costs.\(^{131}\)
4.4.2 Work loss

Diagnosis of COPD is often made at older ages when many people are already retired. However, perhaps 20% of those diagnosed with COPD are still in paid work. In one study in USA, 45% of those aged under 65 years with COPD had to stay at home from work due to their COPD on some occasions during the year. As lung function decreases, the likelihood that a person will remain in paid work also decreases. Within 6 or 7 years of diagnosis, most people with COPD are no longer capable of productive work.

4.4.3 Quality of life

Often, it is decline in quality of life which first prompts individuals to seek medical advice, after living with respiratory symptoms and/or reduced lung function for some months or years.

Typically, quality of life in COPD becomes progressively worse until death. Patients tire more easily, and activities which have brought them pleasure become restricted or no longer possible – particularly activities which require physical exertion and use of their upper arms. Once diagnosed, most COPD patients report shortness of breath and coughing every day or most days. Respondents also report severe limitation of effort, even when simply walking upstairs. The WHO study showed that COPD affects quality of life in individuals as much on average as paralysis and AIDS.

The Ministry of Health estimated that in 1996 15,430 years were lost from premature death from COPD in New Zealand, ranking the condition sixth in the list of causes of early death.

Besides loss of productive life, loss of years lived through premature death causes pain and grief to families and friends, loss of guidance to younger family members, and deprives young families of many of their natural supports.

4.4.4 Disability-adjusted life years

Disability-adjusted life years (DALYs), a measure of loss of life through early death together with the loss of life lost due to reduced quality of life because of disability, has been calculated by the Ministry of Health for the major causes of disability. For the year 1996, ischemic heart disease was by far the largest, followed by stroke. Under their estimates, COPD accounted for approximately 27,800 (4.9%) of the DALYs lost in New Zealand. COPD was ranked third place overall, second for men and fifth for women. Preliminary estimates for the year 2003 show that the burden of COPD has grown markedly among women. It may now be the overall leading cause of death and disability in New Zealand.

4.5 Changing costs

4.5.1 Prevalence and survival

Increasing prevalence and improved survival will lead to more exacerbations, growing hospitalisation rates, and increased costs.

In Sweden, it is estimated that costs of COPD management alone increased almost 60% over 11 years to 1991, mainly due to costs of inpatient care relating to exacerbations. In the Netherlands, a dynamic life table model was used to project the future burden of COPD. Disease duration, aging of the population,
population growth, smoking behaviour and co-morbidity were taken into account. The models predict that marked increases are unavoidable in both the prevalence and burden of COPD by 2015: loss of life years are expected to rise by more than 60%, loss of disability-adjusted life years will increase by 75%, and health care costs are projected to rise by 90% over the 21 years from 1994 to 2015, with smoking accounting for 90% of the costs.

In New Zealand, as in most other countries, these models have not yet been attempted for COPD. Current initiatives to reduce smoking behaviour are unlikely to materially affect the projected increase in costs in the medium-term, until the toll from previous use of tobacco has peaked. The highest rates of smoking known in the western world are being recorded for young Maori women, and the prevalence of smoking in young men has been increasing very quickly. The consequences if left unchecked are enormous, for COPD and for many other conditions.

Nevertheless, initiatives in health care services have shown potential to reduce costs. A study conducted in the USA showed that if a 50% reduction in exacerbations could be achieved, costs ascribed to exacerbations of COPD could be reduced by 33%. Another showed that employing specialised respiratory care teams to provide in-home healthcare for patients with advanced COPD reduced overall costs through reductions in hospital care. Although differences in funding systems between USA and New Zealand make it unwise to generalise from these results to the New Zealand situation, clear cost benefits could be achieved in New Zealand.

4.5.2 Changing face of treatments

Greater use of drugs for COPD may lead to increased costs: more use of long-acting β₂-agonists and anti-cholinergics, inhaled steroids; α₁-antitrypsin replacement IV and any new drugs introduced.

Increased use of smoking cessation programmes would provide benefit even among patients hospitalised with COPD. A study in Denmark showed that smoking cessation was associated with a 40% reduction in the risk of COPD hospitalisations. Increased provision of smoking cessation programmes, while not expected to have major cost-benefit in terms of direct health care, could impact well on other aspects of the patients’ lives.

In addition, more use of pulmonary rehabilitation, long-term oxygen therapy and nocturnal ventilation for COPD are forecast to increase, with possibly more lung volume reduction surgery and lung transplants. In France, a public organisation known as ANTADIR, a network of regional associations for home care of chronic respiratory insufficiency, provides long-term oxygen therapy for about 60% at home, and positive pressure ventilation for about 40%. This model is suggested by some as a possible model for improving home-based oxygen supply.

Essentially however, the main driver of COPD costs will continue to be smoking rates, and it is by driving these down that diseases, and costs, will reduce.
5 Health impacts of Lung Cancer

5.1 Prevalence and incidence

5.1.1 Measurement

For lung cancer, incidence is a more useful measure of the burden of disease than prevalence, since the main burden is felt within months, or at most years, of diagnosis. Measuring the incidence of lung cancer has some of the difficulties of measuring COPD, in that late diagnosis means reported rates under-estimate true prevalence of the disease. However, unlike COPD, a definitive diagnosis is usually made, and the sub-type identified through histological testing.

In New Zealand, a cancer registry has been operating since 1975 to record and track newly diagnosed cases of cancer. Since 1994, new cases of all cancers in New Zealand have been required by law to be registered. Data for new registrations and for deaths are summarised and reported annually by the Ministry of Health.

5.1.2 Incidence of lung cancer in New Zealand

In 1999, 1526 new cases of lung cancer were registered, 9.1% of all new cancer registrations (10.5% for men, 7.4% of those for women). With a mean survival at most 18 months, the number of cases at any one time in New Zealand is likely to be about 2,250, the majority aged over 70 years.

In 1999, 1526 new cases of lung cancer were registered in NZ. Length of survival from diagnosis averages at most 18 months; few survive over 5 years.

A major report on cancer trends and projections published in 2002 showed the average annual age-standardised lung cancer incidence rate increased sharply from 39 per 100,000 in 1965, to reach a peak at just below 90 per 100,000 in the early 1980s. The rate has since fallen to 64 per 100,000 in 1996, reflecting the time lag since high tobacco consumption levels 20 to 40 years earlier.

5.1.3 Case-fatality and survival

Survival data are not published for New Zealand, but the fatality/case ratio is useful. In 1999, this was 0.93 for men and 0.97 for women, making it one of the most fatal cancers.

In the USA, 60% of deaths occur within a year of diagnosis and the 5-year survival rate after diagnosis with lung cancer is about 14%. In the UK fewer than 5% are reported to survive 5 years from diagnosis. Of those who do survive to 5 years from diagnosis, life expectancy is similar to that for those unaffected by lung cancer.

5.1.4 Variations by age, sex and ethnicity

As lung cancer primarily arises from long-term tobacco smoking, it is rarely diagnosed in those aged under 40 years, the majority being diagnosed aged between 65 and 79 years. In New Zealand, 70% of both registrations and deaths occur in older people (65 years and over), with rates rising exponentially after middle age.

145 147

70 149
Men have higher incidence rates of lung cancer than women, though with changing patterns of smoking, rates for men are falling (by about 1.1% a year since 1995). Conversely, those for women are climbing. After adjustment for age, incidence rates are almost three times higher among Maori men than non-Maori, and nearly four times higher for Maori women. These rates will mainly reflect higher tobacco exposure, poorer survival, and a lower rate of surgery.

Among men, incidence of lung cancer has been declining for over 10 years, while those for women are still rising. Maori men have almost 3 times, and Maori women almost 4 times, corresponding rates of non-Maori.

5.1.5 Variations by occupation and deprivation level

A recent Ministry of Health publication demonstrated a clear direct gradient of lung cancer incidence across deprivation deciles (using the New Zealand scale of deprivation, NZDep, where decile 1 is the least deprived, and 10 the most deprived). People living in the most deprived areas have over twice the incidence and mortality rates of lung cancer of those living in the least deprived. Similar conclusions were reached by Blakely in a large census-mortality study. Neither of these reports has adjusted for exposure to tobacco smoke, and since smoking history is associated with deprivation, it remains unclear the extent to which there differences relate to tobacco smoke.

Asbestos-related lung cancer is expected to increase steeply because of exposure to raw asbestos and asbestos products imported between 1962 and 1996.

5.1.6 Comparable other countries

Lung cancer is the most common cancer in the world, but there are large regional differences. Figure 5-2 shows the age-standardised rate of lung cancer incidence of the major world areas using the latest data available from the World Health Organisation website. Those data report Australia and New Zealand incidence rates as 47.6 per 100,000 men and 16.1 per 100,000 women, reportedly about
twice those of the average of all developed countries (24.1 and 7.8 respectively). More recent data from within New Zealand show age-standardised mortality rates show a substantial improvement for men of 32.9 per 100,000 and an increase to 19.3 per 100,000 for women.

Figure 5-2  Estimated age-standardised rates of lung cancer incidence worldwide, by WHO region, 1990 (per 100,000)

In comparison to New Zealand, the age range of peak incidence of lung cancer in the United Kingdom (UK) is slightly older, 75-80 years. This is probably a result of their older population.

Lung cancer incidence rates in Australia and New Zealand were about twice those of the average of all developed countries, but have reduced to comparable levels.

5.2 Morbidity

5.2.1 Hospital discharges in New Zealand

Hospitalisations of people with lung cancer typically occur for diagnostic purposes or because of acute infections, for remedial surgery, or for terminal cares. In 1999, there were 2707 public hospital discharges for malignant carcinoma of the trachea and lung (defined by ICD-9 code 162 or ICD-10 C33-C34x), up from 2400 in 1995. The 1999 hospitalisations had a mean length of stay of 8.6 days.
Care for people with lung cancer in private hospitals is unusual although hospice care would be utilised more often. In 1997, only 371 discharges from private hospitals were recorded, and while it is probable that these were mostly for diagnostic purposes, a proportion will have been long-term or perhaps terminal cares. The average length of stay in private hospitals was 20.1 days.

### 5.2.2 Variations by age, sex and ethnicity

*Figure 5-3 Numbers of lung cancer public hospitalisations in New Zealand, by sex, age group and ethnicity, 1999*

Of all discharges, 62.2% were of men. Age-specific public hospitalisations, by sex and ethnicity, are shown in Figure 5-3: the higher rates for men and the association with age are clear. Also visible is the increased rates of discharges among young women – rates for those aged 45 to 60 years now exceed those for men in the same age groups.

*Figure 5-4 Lung cancer hospitalisation rates in New Zealand, by sex, age group and ethnicity (per 100,000)*

In 1999, one in four (25.6%) lung cancer public hospitalisations were for people aged under 60 years, most are within the age range 55-80 years, and 316
(11.7%) over 80 years. Just 403 (14.9%) were aged under 55 years. Estimated mean age of hospitalisations was 67.3 years (68.5 for non-Maori, 60.7 for Maori).

Figure 5-4 shows public hospitalisation rates by age, sex and ethnicity over the period 1997-1999. Age-specific hospitalisation rates among Maori are comparable with those for non-Maori aged about 12 years older. Discharge rates for Maori women aged under 75 years are more similar to those for Maori men than for non-Maori women.

5.2.3 Hospital bed-days in New Zealand

While numbers of discharges related to lung cancer have increased in the interval 1997/1998 to 1999/2000, mean lengths of stay have reduced among men and non-Maori women, but increased in non-Maori women, as seen in Table 5-1 below. The overall impact of these changes has been a 34% increase in total hospital bed-days for lung cancer in just three years.

Table 5-1 Number of discharges and associated mean lengths of public hospital stay (days) in patients over 45 years, for lung cancer in New Zealand

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of admissions</td>
<td>Mean stay</td>
</tr>
<tr>
<td>Maori men</td>
<td>150</td>
<td>5.9</td>
</tr>
<tr>
<td>Non-Maori men</td>
<td>1245</td>
<td>7.8</td>
</tr>
<tr>
<td>All men</td>
<td>1395</td>
<td>7.6</td>
</tr>
<tr>
<td>Maori women</td>
<td>135</td>
<td>7.4</td>
</tr>
<tr>
<td>Non-Maori women</td>
<td>731</td>
<td>8.0</td>
</tr>
<tr>
<td>All women</td>
<td>866</td>
<td>7.9</td>
</tr>
<tr>
<td>Total</td>
<td>2261</td>
<td>7.7</td>
</tr>
</tbody>
</table>

5.3 Mortality

5.3.1 Lung cancer deaths in New Zealand

During each year in the period 1996 to 1999, approximately 1400 deaths have been ascribed to lung cancer (defined by ICD-9 code 162 or ICD-10 C33-C34x as before) in New Zealand. For men, most deaths occur in the 70-79 year age groups, but women who die of lung cancer tend to do so at younger ages, 65-74 years (Figure 5-5).

Although most result from long-term tobacco smoking, it is estimated that the number of lung cancer deaths increases by about 7 per year in New Zealand as a result of exposure to environmental tobacco smoke either at home or at work.152

5.3.2 Variations by age, gender and ethnicity

Mortality rates, like incidence rates for lung cancer are much higher for men than for women, though the gap is reducing. Rates increase with age except those for Maori women which are lower in the very old, though the population numbers are small for this group and so data are unreliable. In general, age-specific death rates in Maori women are similar to those seen in non-Maori women aged 20-25 years older. For men, this interval is about 15-20 years. Figure 5-6 shows age-specific death rates, for men and women separately and by ethnicity.145
Maori death rates are higher than any reported for any country or population group, including those of the Eastern European and former Soviet countries where smoking rates have long been high (see Figure 5-2). The Ministry of Health in 1999 reported age-standardised death rates per 100,000 of 75.3 for Maori men and 74.4 for Maori women, where comparable figures for non-Maori were 30.5 and 14.4 respectively.

Age-specific death rates in Maori women are similar to those seen in non-Maori women who are 25 years older.

5.3.3 Rates in last 10 years in New Zealand

Overall, mortality rates have been declining since the peak in lung cancer incidence for men in the early 1980s. This is a result of falling rates for men:
among women, mortality rates continue to rise.\textsuperscript{74} Because of the increasing population however, numbers of deaths have stabilised.

5.4 Long-term trends and projections

Ministry of Health forecasts show that among men, lung cancer incidence will continue to decrease, falling to 38 per 100,000 (858 registrations per year) in 2011/12.\textsuperscript{74} Mortality among men is forecast to drop to 35 per 100,000 (807 deaths) in the same year.\textsuperscript{4}

Among women however, incidence rates are expected to stabilise at their present level (33 per 100,000), but numbers will increase 42\% to about 825 registrations in 2011/12 because of the growth in the population of older people. Mortality in women is projected to increase slightly reaching 33\% per 100,000, about 860 deaths in 2011/12. If projected trends continue, incidence and mortality rates for women are likely to exceed those for men soon after that time.

The trends in lung cancer vary between countries, according to the patterns of tobacco smoking in each country’s recent past. In Italy for example, favourable trends in smoking since 1990 have contributed to a decrease in age-standardised rates of mortality among women, and stabilising rates among men.\textsuperscript{153} In a population-based study from Detroit, USA, lung cancer incidence rates have been declining among white men since 1983 and among black men since two years later. In women both white and black, rates seem to have levelled off by about 1993, then started to fall by about 1998. However, survival rates were worse for blacks than for white, at least in some cancer sub-types.\textsuperscript{154}

5.5 Prevention

Preventing diseases and ill-health is virtually always more cost-effective than treating developed disease. For lung cancer, this is clearly the case.

Of importance to policy-makers and individuals with a history of tobacco smoking, smoking cessation has been proven to reduce individuals’ risk of lung cancer and to reduce the burden for a population.

In the United Kingdom, between 1950 and 1990 the numbers of lung cancers almost halved compared to expected rates had ex-smokers continued smoking.\textsuperscript{9} The introduction of tobacco control measures in some states of the USA has been credited with a 14\% reduction in incidence of lung cancer over 10 years, compared to a fall of only 2.7\% nationally.\textsuperscript{155} Another study sought to assess the impact of the American anti-smoking campaign of the 1970s by associating death rates with smoking rates over time. It determined that the campaign promptly reduced significant smoking among younger men and that this was reflected in a drop in lung cancer rates. For those who did quit, exposure to smoking cessation messages for 5-10 years seemed to yield the best changes in smoking rates.\textsuperscript{156}

Despite reductions in smoking prevalence, tobacco use is still common, and can not be feasibly eradicated. Higher risks of lung cancer persist in former smokers compared to never-smokers. Measures to slow smoking initiation, as well as smoking cessation, are still regarded as the best means of lowering the levels of lung cancer. It is inevitable that the health impacts of smoking over the past 20 or more years will lead to many more deaths and disabling diseases and events.
This comment about lung cancer and tobacco smoking was made by Peto:

“People who stop smoking, even well into middle age, avoid most of their subsequent risk of lung cancer, and stopping before middle age avoids more than 90% of the risk attributable to tobacco. Mortality in the near future and throughout the first half of the 21st century could be substantially reduced by current smokers giving up the habit. In contrast, the extent to which young people henceforth become persistent smokers will affect mortality rates chiefly in the middle or second half of the 21st century.”

“People who stop smoking, even well into middle age, avoid most of their subsequent risk of lung cancer, and stopping before middle age avoids more than 90% of the risk attributable to tobacco.” Peto
6 Cost Burden of Lung Cancer

6.1 Basis of costings

For lung cancer, there is reliable incidence data and less good utilisation data, but costings have greater reliability.

6.2 Summary of costings

Hospitalisations are the main costs to the health system in New Zealand for lung cancer, with GP visits the second largest. Medications, outpatient care and specialist visits are also major cost items.

The direct costs to the New Zealand health care system for lung cancer amounts to at least $18.0 million in the year 2002, but may be as much as $28.1 million. Costs per patient per year are estimated at $7500 on average.

Table 6-1  Estimated health-related costs of lung cancer in New Zealand, 2002 (‘000s)

<table>
<thead>
<tr>
<th>Lung cancer prevalence (3 estimates)</th>
<th>2,400</th>
<th>3,000</th>
<th>3,750</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estimated number of prevalent cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Item</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inpatient stays (beddays)</td>
<td>12,063</td>
<td>15,079</td>
<td>18,849</td>
</tr>
<tr>
<td>Palliative care</td>
<td>1,120</td>
<td>1,400</td>
<td>1,750</td>
</tr>
<tr>
<td>Prescriptions</td>
<td>835</td>
<td>1,044</td>
<td>1,305</td>
</tr>
<tr>
<td>Hospice or private hospital care</td>
<td>700</td>
<td>875</td>
<td>1,094</td>
</tr>
<tr>
<td>Hospital outpatient visits</td>
<td>625</td>
<td>781</td>
<td>976</td>
</tr>
<tr>
<td>GP visits</td>
<td>612</td>
<td>765</td>
<td>956</td>
</tr>
<tr>
<td>Private specialist visits</td>
<td>586</td>
<td>732</td>
<td>915</td>
</tr>
<tr>
<td>Blood tests</td>
<td>528</td>
<td>660</td>
<td>825</td>
</tr>
<tr>
<td>Other direct health care</td>
<td>559</td>
<td>699</td>
<td>873</td>
</tr>
<tr>
<td>Surgery</td>
<td>357</td>
<td>447</td>
<td>559</td>
</tr>
<tr>
<td>Total (‘000)</td>
<td>17,985</td>
<td>22,481</td>
<td>28,102</td>
</tr>
<tr>
<td>Average per patient per year $</td>
<td>7,494</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

6.2.1 Cost of public hospital inpatient care

Hospitalisation estimates are based on lengths of stay in 1999 where the primary diagnosis was lung cancer. For lung cancer, 2700 discharges were recorded with an average length of stay of 8.6 days at a cost of $857 per bed-day. If half the lung cancer patients stay in a public hospital, about $15 million is used by lung cancer hospitalisations each year.

6.2.2 Costs of palliative care

The Ministry of Health’s Palliative Care Strategy summarises the range of services and needs of people who are dying, including domiciliary nursing services, night nursing, counselling services and social work. Patients also may need to hire equipment such as adjustable beds, pain pumps etc. From data provided in the Strategy, an estimated 70% of people dying of lung cancer receive palliative care, using a range of funding sources. The costs of the package (excluding
hospice care) have been arbitrarily but conservatively set at $2000 per person for these costings, and total $1.4 million.

6.2.3 Cost of prescriptions
Analgesics, anti-emetics and steroids are required by almost all patients. Chemotherapy is used by only a minority. Costings have been estimated at $45 per script, each of which may prescribe several medications. In total, pharmaceuticals for lung cancer are estimated to cost $1.0 million annually.

6.2.4 Cost of hospice or private hospital care
Although hospice cares are funded through various sources, including voluntary and charitable trusts, they, with private hospitals, form a significant part of the costs of care for perhaps 25% of lung cancer patients, either for relief or terminal care. At an estimate cost of $500 per day, and average stay of 7 days, about $875,000 is used by lung cancer hospitalisations each year.

6.2.5 Cost of hospital outpatient visits
Based on the assumption that more than half the patients have 6 outpatient visits, costs would reach $781,000.

6.2.6 Cost of GP visits
Costing calculations have been estimated on the principle that all patients visit their GPs on average 8 times in the first few months, for investigations and developing care plans, and thereafter the number of visits reduces. Each visit is costed at $45, reaching a total of $765,000.

6.2.7 Cost of specialist services
Calculations are based on the premise that all patients with lung cancer see a thoracic specialist early after first seeing their GP for their respiratory problems. Estimates of out-of-hospital specialist care include all new lung cancer patients an average of four visits each initially, followed by quarterly visits thereafter. At a cost of $244 each, this represents a total of $732,000.

6.2.8 Other direct costs
Included as other direct costs are imaging (X-rays and CT scans), emergency department visits, ambulance services and other smaller cost items. Not included are travel to access health services, loss of earnings and inability to care for partners or other dependents. Again, such costs would fall mainly on the patient and the family.

6.3 Indirect costs
6.3.1 Work loss and premature death
By the time a diagnosis of lung cancer is made, many people are already retired. However, 30% to 35% with lung cancer are likely to be in paid employment, with consequent loss of production and support for families which often the state has to cover. Premature death causes pain and grief to families and friends, loss of guidance to younger family members, and deprives young families of many of the natural supports. These are not costed in this report.
6.3.2 Disability-adjusted life years
Lung cancer does not rank among the top contributors to the burden of disability since survival is typically short.

6.4 Changing costs

6.4.1 Accessibility to pharmaceuticals and services
Poorer survival among Maori may be partly related to lesser use of treatments – lung resection and medications. Arguably, this may result from seeking help later in the disease process or from a lower level of treatment, but may also arise from known preference for home care.

6.4.2 Changing face of treatments
In the USA, surgery for lung cancer has been forecast to increase since it has been shown to have proven benefits, but surgery will not necessarily increase in New Zealand. Development of newer and probably more expensive drug therapies will impact on costs, but no major changes are expected in the short term.
7 Implications for New Zealand

7.1 Health promotion and prevention

7.1.1 Recognition a priority

In New Zealand, there is widespread lack of appreciation of the scale of COPD. It places a heavy burden on our health services and on those who suffer from it. Although the evidence of the size of the burden is not reliable, there is clear evidence that the problem of COPD is growing and will become even more costly. Small falls in the rates of lung cancer will not be sufficient to reduce numbers with that condition, given the increasing age of the population.

7.1.2 Smoking a priority

To reduce that burden, use of tobacco must be further reduced. Of particular concern is that the COPD and lung cancer rates reported for Maori are likely to have been underestimated, owing to a low standard of ethnicity reporting on hospitalisation records and death certificates. In particular, there is an urgent need in New Zealand to introduce and support effective schemes to help Maori reduce tobacco consumption given their very high smoking prevalence.

Without doubt, smoking cessation is the most important approach to management in all obstructive lung diseases.

It is clear from several randomised controlled studies that there can be long-term benefits from smoking cessation initiatives, both to prevent disease and to reduce the rate of decline in those already with disease. For the young men and women vulnerable to pressure to begin tobacco smoking, there needs to be clear and continual messages about its harms, and to provide strategies to avoid smoking and so reduce the additional risks associated with commencement of smoking at an early age.

7.1.3 Management of other risk factors

Efforts to reduce work-place exposure to dust particles may feasibly assist in reducing the burden of COPD, as could measures to reduce obesity and overweight and reducing exposure to carcinogens.

7.2 Recognition and diagnosis

7.2.1 Awareness, early recognition and seeking help

Patients and their doctors tend to under-recognise COPD, and to detect lung cancer fairly late in the disease process. Efforts to contain the growth in human and financial burden should include smoking cessation and deterrence programmes, earlier recognition of the diseases, and searches for better management plans and therapies, on the basis that expenditure in these areas in the near future could lead to reduced costs in the longer term.

People with persistent cough or sputum production need medical evaluation: their friends and family members should encourage a visit to their GP.
7.2.2 Screening

By identifying people with preclinical disease early, diagnosis and treatment could potentially be accessed sooner through the use of population-based screening. Three fundamental principles are recognised by the World Health Organisation as necessary before adopting a screening programme.

- The target disease should be common, with high associated morbidity and/or mortality.
- Effective treatment, capable of reducing morbidity, should be available.
- Test procedures should be acceptable, safe and relatively inexpensive.\(^{159}\)

Further, screening programmes should meet more detailed criteria described elsewhere.\(^{160}\)\(^{161}\)

Screening that is targeted to a high-risk population, such as those who have ever smoked the equivalent of a pack of cigarettes a day for 10 years or more, has been successful to the extent that participants can be recruited, and airway obstruction measured.\(^{162}\) People identified as at high risk of COPD include all smokers aged 45 years of older, and anyone with a chronic cough, shortness of breath on exertion, production of high amounts of mucous secretion or phlegm, or wheeze at any age.\(^{163}\) In spite of claims that elderly people cannot perform spirometry properly, this has been proven incorrect in people without dementia.\(^{164}\)\(^{165}\)

For lung cancer, screening seems attractive for at least three reasons:

- once symptomatic, lung cancer is generally lethal, but if found when still localised it may be cured;
- new technology may detect early disease more efficiently; and
- a high-risk cohort is easily identified (tobacco-smokers).

It has been advised that high risk individuals should be tested routinely using spirometry.\(^{163}\)\(^{166}\) However, there is no validated screening tool, there is potential for enormous cost, and there are consequences for those who are found to be positive to the screen but not found to be positive in diagnostic tests.\(^{167}\) Recent reviews of the literature for chest x-ray, sputum cytology and low-dose CT scanning to screen for lung cancer concluded that their use is not supported by the published evidence.\(^{168}\)\(^{169}\) Although there continue to be calls for the introduction of screening of high risk groups, randomised trials of population screening for lung cancer have failed to identify its efficacy.\(^{166}\)\(^{169}-171\) A pilot study of spiral CT has been initiated to determine the feasibility of a larger, longer randomised trial.\(^{172}\)

With currently available diagnostic tests and treatment, untargeted population-based screening is not justified for either COPD or lung cancer.

7.2.3 Diagnosis

Recent evidence from a multicentre study in Italy suggests that misdiagnosis of asthma and COPD is related to older age and to a greater degree of disability.\(^{21}\)
They concluded that asthma in the elderly is frequently confused with COPD, with errors occurring in both directions. Even without that confusion, there remains the difficulty of clarity in clinical diagnosis (based on spirometry and/or signs and symptoms) and in epidemiologic studies. Nevertheless, some areas can be identified as justifiably deserving attention.

Targeted assessments by general practitioners could be more useful than population-based screening, and have been advocated for all smokers and others at high risk. Spirometry, like blood pressure and cholesterol measurements, could be widely applied for the assessment and measurement of those with undiagnosed obstructive lung disease. Calverley proposed that GPs should review case records to identify people who have required one or more courses of antibiotics during winter, and who may be at greater risk of COPD, but no evidence was presented that this approach is effective.

There is uncertainty as to whether primary care providers should be encouraged to obtain spirometers and become trained in its use and/or provide training for practice staff. On the one hand, calls are made for all GPs to use spirometry on high risk patients. On the other, evidence from New Zealand suggests that, at least at the present time, results of spirometric testing within a general practice setting are not reliable. An alternative may be to establish community-based clinics specialising in monitoring patients with respiratory diseases including asthma and COPD, similar to clinics for hearing and vision problems.

Diagnosis of COPD can be achieved only through community-based spirometry services with quality assurance programmes.

7.3 Reporting and monitoring

The lack of valid data on prevalence of COPD makes it impossible to reliably describe the true burden of this disease in New Zealand. This is not the case for lung cancer, which for which data is routinely gathered through the cancer register.

Two new initiatives may improve the reliability of data on the burden of the two diseases. The introduction of chronic disease registries within primary healthcare organisations will enable provision of better information of currently-diagnosed COPD and the cost of its care. There may also be opportunities to utilise information routinely gathered from an electronic national formulary proposed by PHARMAC by linking drug dispensing information with diagnostic codings. Utilisation data for hospital emergency departments is another untapped resource. Publication of internal audits of the national morbidity and mortality reporting systems would be useful to indicate the extent and direction of misreporting.

Introduction of ICD-10 coding is has already led to changes in reported rates, increasing those for COPD but showing no change for lung cancer. ICD-10 more clearly reports COPD distinct from chronic bronchitis and emphysema. Additional efforts to improve the quality of diagnosis coding and reporting COPD and related conditions as a cause of death may be worthwhile, possibly through Continuing Medical Education.
7.4 Economic burden

Even if all tobacco smoking were to miraculously stop in New Zealand tomorrow, numbers of new cases and numbers of deaths from COPD and lung cancer will increase next year and for some years to come, as a result of years of exposure to tobacco smoke. Best efforts to minimise the burden over future decades will be those which reduce the numbers of people taking up smoking and in encouraging existing smokers to stop as soon as possible. To reduce the financial burden on tax-payers in future years, it is necessary to spend on primary prevention now.

7.5 Research opportunities and possibilities

In the USA, COPD receives proportionally less funding for research when compared with most other illnesses, based on mortality, disability and years of life lost. This may reflect an attitude among funding bodies that regard tobacco smoking as singly the major cause of the disease and that there is little else to investigate. Recent research has focussed on the use of steroids for treatment of COPD and genetic markers for lung cancer. Work conducted at the University of Auckland has found genetic markers which appear related to protection from lung disease. Perhaps this will help explain the reasons some smokers get COPD and lung cancer, and some not.

Development of guidelines for best practice have identified areas of uncertainty related to acute care which require further research, for example the use of antibiotics during acute exacerbations.

Much remains to be learned about COPD and its impact on health in New Zealand. Increased prevalence, especially among Maori, requires that even greater efforts need to be put into education and recognition of lung disease, and in researching and implementing interventions which reduce the numbers of people smoking and the number of cigarettes smoked. The fact that knowledge of the main ‘culprit’ – tobacco – is well established should not deter from effort to reduce its impact.

7.6 Messages

The impact of these two conditions on lives in New Zealand is large. Coordinated efforts are required to minimise their impact.

- To everyone – in homes, workplaces and leisure venues - stop or reduce smoking. The earlier one quits smoking, the lower the risk compared to continuing smokers.

- To people with symptoms of obstructed airflow, seek medical help. Early diagnosis and intervention have proven benefits. Waiting until quality of life is affected before seeking help makes slowing the decline more difficult.

- To friends and families, help those with symptoms to seek help. Your support and encouragement can reduce delays in diagnosis.

To avoid large increases in the burden of COPD in future years, greater efforts need to be put into education and recognition of lung disease, and in finding interventions which further reduce smoking, particularly among Maori.
• To GPs, actively manage COPD - encourage and support smoking cessation efforts, screen smokers aged over 45 years with spirometry, and in those patients with COPD, avoid or delay acute exacerbations e.g. by vaccinating against influenza.

• To hospital respiratory teams, invest in approaches which will reduce exacerbations and length of stay in hospital. Encourage self-management programmes.

• To DHBs, help reduce smoking in your populations, and ensure evidence-based care is provided for patients with COPD and lung cancer.

• To research institutions and universities, seek effective preventive and therapeutic options.

• To national government, include COPD and lung cancer as priorities in policy and research agenda, monitor their burden to health and costs.

• To national, regional and local government bodies, set and chase targets for smoking cessation as high priority.
## Appendix 1 Potential data sources for measuring the prevalence of COPD

<table>
<thead>
<tr>
<th>Method</th>
<th>Strengths</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital discharges</td>
<td>Collected and summarised routinely for public hospitals.</td>
<td>Most people with COPD are not admitted to hospital in any one year, and some are admitted many times, so numbers cannot be used to report prevalence. In the past, coding variations between institutions and over time have meant discharge data were not reliable; recent initiatives mean that in the future they will be more so.</td>
</tr>
<tr>
<td>GP visits</td>
<td>Has potential to inform prevalence of diagnosed COPD if collected through chronic disease registers.</td>
<td>Data have been collected only in small studies or particular service providers, &amp; results not in public domain. Coding of conditions is often unreliable, especially if comorbidities are present, leading to measurement errors.</td>
</tr>
<tr>
<td>Prescriptions filled</td>
<td>Could inform cost burden.</td>
<td>Data is collected but not published routinely: issues of privacy limit use. Medications prescribed for COPD are not specific to COPD, so would overestimate the burden.</td>
</tr>
<tr>
<td>Population-based surveys using self-reported diagnosis of COPD</td>
<td>Relatively easy to conduct, can be undertaken by phone to keep costs down. Can seek information about impact on life and non-medical costs.</td>
<td>Underestimates true prevalence because only diagnosed cases are reported - late diagnosis is common, as is other terms for the condition. Overestimates result from misunderstanding of the questions – e.g. “chronic bronchitis” is confused with “acute bronchitis”.</td>
</tr>
<tr>
<td>Population-based surveys using self-reported symptoms of breathing impairment</td>
<td>Relatively easy to conduct, can be undertaken by phone. Has potential to capture information about numbers with symptoms but no diagnosis, and to record non-medical costs.</td>
<td>Overstates true prevalence of COPD because other respiratory and cardiovascular conditions are not distinctive, e.g. upper respiratory tract infections, ischaemic heart disease, pulmonary hypertension, shortness of breath through lack of fitness or anaemia, smokers cough, or other conditions.</td>
</tr>
<tr>
<td>Population-based studies using spirometry</td>
<td>Best estimate of prevalence, especially if in conjunction with reported symptoms, reproducible.</td>
<td>More expensive than other methods, because trained staff are required to administer spirometry, and an extra 20-30 minutes is needed per person in a face-to-face setting. Excludes those unable to provide reproducible lung function measures – often those most affected by disease.</td>
</tr>
</tbody>
</table>
## Appendix 2 Prevalence of impaired lung function and COPD

This table demonstrates the very wide variations in prevalence of COPD reported in countries comparable to New Zealand. Of note is the importance of method of measurement and smoking history – prevalence rates vary far more according to methods used and smoking status than they do by age group or country.

<table>
<thead>
<tr>
<th>Method employed</th>
<th>Criteria</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Reported doctor’s diagnosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>USA 2001, NHIS 18+ yrs. (American Lung Association 2003)</td>
<td>Reported doctor-diagnosed chronic bronchitis</td>
<td>65+yrs: 6.7% 45-64: 6.5% 18-44 yrs: 4.5%</td>
</tr>
<tr>
<td></td>
<td>Reported doctor-diagnosed emphysema</td>
<td>65+yrs: 5.1% 45-64: 1.8% 18-44 yrs: 1.8%</td>
</tr>
<tr>
<td>USA 1988-1994, NHANES III, aged 17+ yrs, either ‘white’ or ‘black’. (Mannino 2002)</td>
<td>Reported doctor-diagnosed current chronic bronchitis or ever emphysema</td>
<td>Smokers: 12.5%* Ex-smokers: 9.4%* Non-smokers: 5.8%* All 17+ yrs: 8.5%*</td>
</tr>
<tr>
<td>USA 1988-1994, NHANES III, 17+ yrs. (Coutts 2001)</td>
<td>FEV1/FVC ratio &lt; lower limit of normal (LLN)</td>
<td>Undiagnosed airflow obstruction: 12.0%* COPD: 3.1%* Asthma: 2.7%*</td>
</tr>
<tr>
<td>USA 1997, NHANES III 25+ yrs. (Mannino 2002)</td>
<td>Reported doctor-diagnosed chronic bronchitis</td>
<td>All 25+ yrs: 5.9%*</td>
</tr>
<tr>
<td>USA 1971-1984 NHANES I &amp; II combined, non-smokers aged 18-74 yrs only. (Whittemore 1995)</td>
<td>Reported doctor-diagnosed chronic bronchitis or emphysema</td>
<td>70-74 yr: 7.2%* 50-69 yr: 6.7%* 18-49 yr: 4.4%* All 18-74 yr: 4 to 6%*</td>
</tr>
<tr>
<td>USA, Canada, France, Italy, Germany, the Netherlands, Spain &amp; United Kingdom 2000, aged 45+ yrs, with ≥10 pack years of smoking and with COPD. (Rennard 2002)</td>
<td>Reported diagnosis of COPD, emphysema or chronic bronchitis</td>
<td>Smokers 45+ yrs: ~4.0%</td>
</tr>
<tr>
<td>Canada 1980–1995, aged 12+ yrs. (Lacasse 1999)</td>
<td>Chronic bronchitis or emphysema diagnosed by a health professional</td>
<td>75+ yrs: 8.3% 65-74 yrs: 5.4% 55-64 yrs: 4.7%</td>
</tr>
<tr>
<td>Canada 1994-1995, NPHS aged 35-64 yrs. (Chen 2000)</td>
<td>Chronic bronchitis or emphysema diagnosed by a health professional</td>
<td>Men - Smokers: 3.5% Ex-smokers: 2.9% Non-smokers: 0.8% Women - Smokers: 8.2% Ex-smokers: 2.7% Non-smokers: 2.1%</td>
</tr>
<tr>
<td>Sweden, Norbotten region c.1995, aged 20-69 yrs. (Lindstrom 2001)</td>
<td>Diagnosed by a doctor as having chronic bronchitis or emphysema</td>
<td>Men - Smokers: 5.2% Ex-smokers: 4.8% Non-smokers: 2.2% Women - Smokers: 6.3% Ex-smokers: 4.0% Non-smokers: 2.9%</td>
</tr>
<tr>
<td>Sweden 1992, Skane county, aged 20-59 yrs. (Montnemery 2001)</td>
<td>Diagnosed by a doctor as having chronic bronchitis or emphysema</td>
<td>Smokers: 6.2% Non-smokers: 3.5%</td>
</tr>
<tr>
<td>Location</td>
<td>Diagnosis</td>
<td>Men</td>
</tr>
<tr>
<td>---------------------------------------</td>
<td>------------------------------------</td>
<td>------------------------------</td>
</tr>
<tr>
<td>United Kingdom 1990-1997, patients in general practice research database (Soriano 2000)</td>
<td>Physician-diagnosed COPD</td>
<td>1.7%</td>
</tr>
<tr>
<td>Australia c. 1998, population sample from Melbourne electoral rolls, aged 45-69 years (Abramson 2002)</td>
<td>Diagnosis of chronic bronchitis confirmed by a doctor</td>
<td>All 45-69 yrs: 11.6%</td>
</tr>
<tr>
<td></td>
<td>Diagnosis of emphysema confirmed by a doctor</td>
<td>All 45-69 yrs: 1.1%</td>
</tr>
<tr>
<td>Finland, Sweden &amp; Estonia capital cities, aged 20-64 yrs. (Pallasaho 2002)</td>
<td>Diagnosed by a doctor as having chronic bronchitis or emphysema</td>
<td>Tallinn: 10.1%*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stockholm: 3.0%*</td>
</tr>
<tr>
<td>Finland, Lapland region c.1995, aged 20-69 yrs. (Lindstrom 2001)</td>
<td>Diagnosed by a doctor as having chronic bronchitis or emphysema</td>
<td>Men - Smokers: 3.0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-smokers: 2.4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Women - Smokers: 3.5%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-smokers: 2.3%</td>
</tr>
<tr>
<td>Israel 1998, general practice patients, aged 20+ yrs. (Rennert 2001)</td>
<td>GP's files show COPD</td>
<td>All 20+ yrs: 1.1%</td>
</tr>
</tbody>
</table>

**Reported symptoms**

<table>
<thead>
<tr>
<th>Location</th>
<th>Diagnosis</th>
<th>All 17+ yrs: 36.8%</th>
</tr>
</thead>
<tbody>
<tr>
<td>USA 1988-1994, aged 17+ yrs, either ‘white’ or ‘black’. (Mannino 2002)</td>
<td>Reported cough, phlegm, wheeze or shortness of breath</td>
<td></td>
</tr>
<tr>
<td>USA, Canada, France, Italy, Germany, the Netherlands, Spain &amp; United Kingdom 2000, aged 45+ yrs, with ≥10 pack years of smoking and with COPD. (Rennard 2002)</td>
<td>Had diagnosis of COPD, emphysema or chronic bronchitis or “persistent coughing with phlegm or sputum for last 2 years or more”.</td>
<td>All smokers 45+ yrs, either diagnosed or symptoms: ~6.0%</td>
</tr>
<tr>
<td>Australia c. 1998, population sample from Melbourne electoral rolls, aged 45-69 years (Abramson 2002)</td>
<td>Shortness of breath walking with others of own age</td>
<td>All 45-69 yrs: 10.3%</td>
</tr>
<tr>
<td>Finland, Lapland region c.1995, aged 20-69 yrs. (Lindstrom 2001)</td>
<td>Chronic productive cough</td>
<td>Men - Smokers: 14.8%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-smokers: 8.2%</td>
</tr>
<tr>
<td>Finland, Sweden &amp; Estonia capital cities, aged 20-64 yrs. (Pallasaho 2002)</td>
<td>Chronic productive cough</td>
<td>Helsinki: 12.1%*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stockholm: 5.6%*</td>
</tr>
<tr>
<td>Sweden 1992, Skane county, aged 20-59 yrs. (Montnemery 2001)</td>
<td>Long-standing cough during the last years</td>
<td>Smokers: 16.1%</td>
</tr>
<tr>
<td>Sweden, Norbotten region c.1995, aged 20-69 yrs. (Lindstrom 2001)</td>
<td>Chronic productive cough</td>
<td>Men - Smokers: 11.4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-smokers: 6.2%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Women - Smokers: 8.7%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-smokers: 6.5%</td>
</tr>
<tr>
<td>Study</td>
<td>Diagnostic Criteria</td>
<td>Men (%)</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>--------------------------------------------------------------------------------------</td>
<td>---------</td>
</tr>
<tr>
<td>Finland (year not stated), aged 30+ yrs.</td>
<td>Any degree of airway obstruction</td>
<td>64%*</td>
</tr>
<tr>
<td></td>
<td>Study-diagnosed chronic bronchitis and/or emphysema</td>
<td>22.1%*</td>
</tr>
<tr>
<td></td>
<td>FEV$_1$/FVC% ≤ 69%</td>
<td>11%*</td>
</tr>
<tr>
<td>North Italy 1988-1991, aged 25-73 yrs,</td>
<td>FEV$_1$/FVC &lt;75%</td>
<td>57.0%</td>
</tr>
<tr>
<td>rural dwelling. (Viegi 2000)$^{64}$</td>
<td>FEV$_1$/FVC &lt;70%</td>
<td>28.7%</td>
</tr>
<tr>
<td></td>
<td>FEV$_1$/VC &lt;88% predicted (men) or FEV$_1$/VC &lt;89% predicted (women)</td>
<td>12.2%</td>
</tr>
<tr>
<td>Spain 1997, 7 areas aged 40-69 yrs.</td>
<td>FEV$_1$/VC &lt;88% predicted (men) or FEV$_1$/VC &lt;89% predicted (women)</td>
<td>10.2%</td>
</tr>
<tr>
<td>(Pena 2000)$^{100}$</td>
<td>As above, plus negative bronchodilator test</td>
<td></td>
</tr>
<tr>
<td>USA 1988-1994, NHANES III aged 17+ yrs,</td>
<td>GOLD definition of COPD</td>
<td>23.6%*</td>
</tr>
<tr>
<td>either ‘white’ or ‘black’. (Mannino 2002)$^{184}$</td>
<td>ERS definition of COPD</td>
<td>14.3%*</td>
</tr>
<tr>
<td></td>
<td>Low lung function – FEV$_1$/FVC ratio &lt;0.70 and FEV$_1$ &lt; 80% predicted</td>
<td>6.8%*</td>
</tr>
<tr>
<td></td>
<td>ATS 1987 definition of COPD</td>
<td>2.9%*</td>
</tr>
<tr>
<td></td>
<td>FEV$_1$ &lt;50% predicted</td>
<td>1.4%*</td>
</tr>
<tr>
<td>Australia 2002?, aged 18+ yrs.</td>
<td>FEV$_1$ &lt;87.1% for men and &lt;89.1 for women (mild ≥70%, moderate 50-69, severe ≥49%)</td>
<td></td>
</tr>
<tr>
<td>(Ruffin 2003)$^{105}$</td>
<td>Mild COPD:</td>
<td>20.11%</td>
</tr>
<tr>
<td></td>
<td>Moderate COPD:</td>
<td>0.83%</td>
</tr>
<tr>
<td></td>
<td>Severe COPD:</td>
<td>0.36%</td>
</tr>
<tr>
<td>Asia-Pacific countries 2000, model projections for population aged over 30 years in each country. (Regional COPD Working Group 2003)$^{186}$</td>
<td>Model estimates of moderate-severe COPD, defined as FEV$_1$ &lt;80% predicted and FEV$_1$/FVC ratio &lt;70% predicted.</td>
<td>Australia: 4.7%</td>
</tr>
</tbody>
</table>
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Despite best efforts, any errors or omissions that will have occurred are those of the authors.