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Occupational respiratory health surveillance at Minara Resources, Murrin Murrin mine site

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OCCUPATIONAL RESPIRATORY HEALTH SURVEILLANCE AT MINARA RESOURCES, MURRIN MURRIN MINE SITE

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MPH, Hons Toxicology

This thesis is presented in fulfilment of the requirements for the award of the Degree of Doctor of Philosophy

Faculty of Computing, Health and Science
Edith Cowan University

May 2011

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ACKNOWLEDGEMENTS

I am grateful to Edith Cowan University and Minara Resources for providing me with the opportunity to complete this PhD study. I would like to acknowledge Associate Professor Jacques Oosthuizen, for establishing the collaborative agreement under which this study was conducted; and Minara Resources for agreeing to the collaborative agreement and supporting the research outlined in this thesis.

I wish to acknowledge the Murrin Murrin employees, the subjects of this study, who made this study possible.

Finally, I would like to acknowledge and thank my supervisors, Associate Professor Jacques Oosthuizen and Dr Janis Jansz, for their guidance, constructive criticism and support throughout my PhD journey.

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ABSTRACT

This thesis outlines the results of occupational respiratory health surveillance at Minara Resources, Murrin Murrin mine site. The research was conducted as part of a collaborative agreement between Edith Cowan University and Minara Resources, the overarching title of which was 'Establishing best practice protocols in the management of occupational and environmental health in a high-risk mining and ore-processing environment'.

To form the basis of this research it was hypothesised that although the chemical hazards had been adequately identified, and the occupational exposures in each work area at Murrin Murrin were generally well below their respective occupational exposure levels, it was still possible that additive, or even synergistic biological effects could cause adverse respiratory health effects due to the exposure to a combination of these atmospheric contaminants. This was the perception and a concern voiced by the Murrin Murrin workforce.

Therefore, in working through the hypothesis, a literature review concentrating on the gaps in current knowledge and research for the early detection of occupational respiratory diseases was conducted, and the research tool and experiment design determined. The case for using pulmonary function tests in conjunction with a respiratory questionnaire in assessing early respiratory changes due to occupational exposures was established.

Over a period between 17 February 2004 and 21 June 2006, a longitudinal study was conducted to ascertain the prevalence of respiratory symptoms and lung function of employees at the Murrin Murrin Operation, and compared with a local control group consisting of catering staff who resided at the accommodation camp approximately eight kilometres from the mine site. Lung function data were also compared to established predicted normal values from a reference population with normal lung function. Lung function data were analysed to determine whether there was an effect due to the area worked, and the employee's length of service. The lung function parameters of the study group, corrected for age and height were compared using linear regression analysis with both the control group and the predicted normal values. Repeat lung function tests were conducted on a sample of the original study group approximately two years after the initial study and statistically analysed to determine

whether there was an effect on lung function over this time period. In addition, lung function tests were conducted for a cohort of refinery workers at the start and end of their two-week work period to determine whether there was a before-and-after effect due to their working conditions.

The prevalence of respiratory symptoms was less in the study group compared to the controls; and these respiratory symptoms were determined to be non-work-related. On statistical analysis, for the 'presumed healthy' workers (minus the smokers and those with known non-work-related respiratory symptoms) there was no overall decrement in lung function. Similarly, there was no overall statistically significant decrement in lung function for the 'presumed healthy' workers in the repeat study conducted approximately two years after the initial study. There was no decrement in lung function associated with area work; nor was there a decrement in lung function for the cohort of refinery workers from the start to completion of their two-week work period. However, there were decrements in lung function for the smokers in the study and control groups. There was a significant difference in FEV_1 between non-smokers and smokers with length of service ($p < 0.05$); and a significant difference ($p < 0.05$) in FEV_1 from first spirometry test compared with the repeat spirometry test for the smokers/asthmatics sub-group in the repeat study. This in effect acted as 'internal validity' indicating that spirometry was sensitive enough to detect a decrease in lung function due to smoking in the initial and repeat studies; and that if there were adverse respiratory health effects due to the exposure of atmospheric contaminants at the Murrin Murrin Operation, that this would have been detected.

Hence the concern shown by the employees at the Murrin Murrin Operation, that workplace emissions may be harming their respiratory health, appears to be dispelled by this study.

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1. INTRODUCTION

The extent to which occupational respiratory disease contributes to work-related illness in Australia remains unknown although estimates have been made. Two main reasons for this are that the aetiology of occupational respiratory disease is difficult to establish largely due to the long latency period, and the effect confounding such as environmental exposures and lifestyle factors such as smoking (Morrell, Kerr, Driscoll, Taylor, Salkeld, & Corbett, 1998). The magnitude of occupational injury is far easier to establish than for occupational diseases as there is a clear cause and effect for an injury. Worldwide it has been estimated that approximately two million people die every year as a result of work-related injury and/or illness (CCH, 2009). The National Occupational Health and Safety Commission (NOHSC, 2003) estimated there are more than 2,000 workplace-related fatalities every year in Australia. This figure is thought to be only an estimate, because the long latency period of some diseases and the difficulty in relating some conditions to periods of work make it difficult to quantify the precise number of deaths. Nevertheless, it has been estimated that 85% of workplace deaths worldwide are due to occupational diseases and, apart from cancer, the most common job-related health problems are respiratory diseases, musculoskeletal disorders, hearing loss, circulatory diseases and communicable diseases (CCH, 2009).

To add to the complexity of this issue, available statistics (such as workers' compensation data) suggest that musculoskeletal injury and dysfunction are the main cause of occupational abnormalities in Australia. However, these data sources have been reported to be misleading, because the more recognisable problems (such as musculoskeletal disorders) are more likely to be reported and recorded. By contrast, health problems related to substances (for example, toxic dust exposure) are often hard to identify because the early effects can be subtle and the time between exposure and the development of symptoms can be substantial (Morrell, Kerr, Driscoll, Taylor, Salkeld, & Corbett, 1998).

Although it is well known that exposure to irritant gases, aerosols and particulates in ambient air can cause adverse effects in the respiratory system (Balmes, 2002) the Parliament of Australia (2004) reported a paucity of data in Australia on morbidity and mortality associated with workplace toxic dust exposure. It also highlighted the need for robust surveillance systems and early accurate diagnosis of loss of lung function.

On inhalation of toxic agents there is a continuum of effects which may range from acute reversible effects through to chronic respiratory disease (Meldrum, 2001; McCance & Huether, 1999) and even fatality at concentrations immediately dangerous to life (National Institute for Occupational Safety and Health [NIOSH], 2005). This embodies the '*threshold concept'* where a small amount of a toxic agent produces little or no effect, but as the dose increases the incidence of a health effect in an exposed population exceeds that of a control population not exposed to the toxic agent (Cohen, 2002, p. 126). At the cellular level, injured cells affected by inhaled toxic agents may recover (i.e., a reversible effect) or die (i.e., an irreversible effect) (McCance & Huether, 1999). Such effects have been reported to be dependent on the chemical and physical properties of the toxic agent (or mixture of agents); the concentration in the air; the duration and frequency of exposure; the respiratory rate; and the susceptibility of each individual (Witschi, Pinkerton, Van Winkle, & Last, 2008; Cohen, 2002).

Inhalation of irritant gases or particulates can result in inflammation of the airways and lung parenchyma and can therefore affect lung function and initiate respiratory disease (Mitchell, 1997; Schwartz, 2002). The Australian Safety and Compensation Council (2006, p. 2) defined occupational respiratory disease (in contrast to non-work-related respiratory disease) as "caused or exacerbated by work factors". Moreover, the Australian and New Zealand Society of Respiratory Science (ANZSRS) considered it feasible to provide early detection of occupational respiratory disease (Parliament of Australia, 2004).

It is not, however, always clear whether respiratory disease is in fact occupational as there are normally confounding non-occupational factors that contribute to the disease burden in the individual. Cigarette smoking has been regarded as the classic example of this phenomenon in that regular smoking is widely regarded as the major risk factor for respiratory disease (Wewers et al., 2010). Likewise, asthma may be initiated and/or exacerbated by occupational exposures. In combination, mixed exposures to such non-occupational and occupational factors have been suggested to be additive or even synergistic (Tranter, 2004; Burge, 2002)

This research examined the respiratory health of workers at the Murrin Murrin lateritic nickel and cobalt extraction and processing operation in Western Australia. This chapter provides the rationale and background to the study including information on the

occupational setting, an overview of the respiratory health hazards associated with this occupational setting, and a summary of the possible pathophysiological effects of exposure to airborne contaminants. This is followed by the justification, significance, and purpose of the study.

1.1 Problem Statement

The respiratory health effects of concurrent and repeat exposures to complex mixtures of low-level airborne hazardous substances at the Murrin Murrin Operation remain unknown. Traditionally, health risk assessments are based on measurement of the airborne concentrations of hazardous substances independently. Such monitoring was conducted as a precursor to this study to determine the occupational exposures in each work area at Murrin Murrin (Oosthuizen & Cross, 2004). Although they were generally well below their respective occupational exposure levels, it is possible however, that additive, antagonistic, potentiated or even synergistic biological effects could occur due to exposure to a combination of these atmospheric contaminants (Zeliger, 2008). Moreover, it was the perception of the Murrin Murrin workforce that such exposures may result in adverse health effects not predicted by atmospheric monitoring (Oosthuizen & Cross, 2004).

1.2 Background

The Minara Resources' Murrin Murrin mine site is located approximately 60 km east of Leonora and 60 km west of Laverton in the north eastern Goldfields region of Western Australia. The primary business at Murrin Murrin is to produce nickel and cobalt from laterite ore. The operation exists to extract lateritic ore through open cast mining and then to process the ore using the Sherritt International Pressure Acid Leach (PAL) technology to recover nickel and cobalt (Mining-Technology.Com, 2010) whilst at the same time preventing or minimising unwanted releases of hazardous materials that could expose employees. This biological monitoring study was commissioned by Minara Resources to monitor the process safety and control measures at the Murrin Murrin Operation.

Atmospheric exposure measurements (also known as occupational hygiene monitoring) have been conducted since the Murrin Murrin mine operation commenced in 1999, in order to evaluate the adequacy of the primary preventive measures. As a

precursor to this study, in 2004 a systematic approach to occupational health and hygiene was adopted in order to develop best practice protocols for the management of occupational hygiene hazards at the Murrin Murrin mine site (Oosthuizen & Cross 2004; Wing, 2005; Wing & Oosthuizen, 2007). During the systematic identification and evaluation of all occupational health and hygiene hazards associated with the complex chemical extraction process of nickel and cobalt from ore at the Murrin Murrin mine site, a number of potentially irritant gases, aerosols and dusts were identified, the most notable being hydrogen sulphide, ammonia, sulphur dioxide/trioxide, oxides of nitrogen, sulphur dust, nickel dust, cobalt dust, calcrete dust and *red dirt* dust (dust from the ground in this area). Occupational hygiene monitoring has demonstrated that the mean occupational exposures were generally well below their respective occupational exposure levels (Wing, 2005; Wing & Oosthuizen, 2007). Despite this, there was concern expressed by the staff that, in combination, the various contaminants may present an additive or even a synergistic deleterious health effect (Interdepartmental Group on Health Risks from Chemicals, 2008).

1.3 Justification and Significance

1.3.1 Justification

Toxicological reviews of the potentially irritant gases, aerosols and dusts identified via the occupational hygiene surveys at the Murrin Murrin mine site, determined that the predominant route of exposure would be via inhalation, with the lung being the main target organ, possibly resulting in respiratory disease (Witschi, Pinkerton, Van Winkle, & Last, 2008; Nemery, 2002; Cowie, 2002). If this were the case, this would potentially cause a significant, short-term and long-term, health and economic impact. The aim of this biological monitoring was therefore to detect possible adverse effects at an early stage in order to prevent potential long-term occupational respiratory disease (Hendrick, Burge, Beckett, & Churg, 2002; Parliament of Australia, 2004).

As stated by Morgan and Seaton “All occupational lung disease represents a failure of preventive measures” (1995, p. 9). Atmospheric exposure measurements and biological monitoring are therefore used to determine whether existing preventive measures are adequate, and if not, that additional controls are implemented (Plog, 2002; Hendrick et al., 2002). Prevention of disease is a fundamental principle of occupational epidemiology (Checkoway, Pearce, & Kriebel, 2004). Hence an occupational

epidemiological study of the workforce at the Murrin Murrin Operation was conducted. The prevalence of respiratory symptoms and lung function for each worker and each independent workgroup – that is, mining, ore leach, refinery, utilities, calcrete, pastoral and administration – were investigated.

1.3.2 Significance

An epidemiological study of complex mixtures of low-level respiratory irritants in an Australian lateritic ore mining and processing plant did not appear to have been conducted previously. Each work area/department at the Murrin Murrin Operation has its own unique profile of potential respiratory irritants, with limited periods of low-level exposure not experienced in any other industry. Thus this epidemiological study enabled the measurement of low-level exposures of these complex mixtures in each work area as well as for the whole site. The findings therefore have the potential to identify any additive or synergistic effects which may occur through interaction of a combination of the potential respiratory irritants.

Although the outcome of this study is specific to the Murrin Murrin Operation, it may also have some relevance to other lateritic mining operations using the high pressure acid leach (HPAL) method of extraction such as those in Indonesia, Brazil, Cuba, Colombia and New Caledonia (Intec, n.d.; Barnes, 1998; Mining-Technology.Com, 2010).

1.4 Purpose of the Study

This study was a longitudinal study of the Murrin Murrin workforce, looking for possible early respiratory health effects due to potential exposure to various hazardous substances associated with the mining and processing of ore for production of nickel and cobalt. The purpose of the study was to conduct respiratory health surveillance of the Murrin Murrin workforce to:

- detect possible adverse respiratory health effects at an early stage, in order to
- prevent potential long-term occupational respiratory disease, and then, if necessary,

- to recommend interventions to prevent untoward health effects, and to enable management to have a proactive approach for the protection of the workforce (Hendrick et al., 2002).

1.5 Organisation of Thesis

Chapter 2 provides a critical review of existing literature regarding how air quality can affect lung function and cause respiratory disease. The impact of respiratory disease is discussed and the literature review then focuses specifically on occupational respiratory disease, its aetiology, the health surveillance methodology to determine occupational respiratory disease, and the potential confounding factors. It identifies the gaps in current knowledge and research for the early detection of occupational respiratory diseases, and states the case for pulmonary function tests in conjunction with a respiratory questionnaire in assessing early respiratory changes due to occupational exposures, which was the main aim of this study.

Chapter 3 provides an overview of the Murrin Murrin Operation process, from mining of the ore, through its processing to produce the final nickel and cobalt products. In doing so, the chemical hazards that workers may face on a daily basis are identified.

Chapter 4 focuses on the respiratory health issues presented by each hazardous substance associated with the mining, the process plant, and specific work areas, and relates this to the personal exposure assessments that were determined through the occupational hygiene component of this study. It also discusses the effects of mixed exposures on the respiratory system.

Described in Chapter 5 are the methods and procedures used to collect the data for this biological effects monitoring study. This includes the study design, administration of the respiratory questionnaire, the measurement technique, the equipment and quality control, and statistical analysis.

Presented in Chapter 6 are the results of this study. The prevalence of respiratory symptoms of the study group of workers at the Murrin Murrin Operation is compared with those of a control group of caterers located close to the operation. The lung function of the study group is compared both with their predicted values, and the control group, and analysed with the sequential removal of the confounding factors known to affect lung function. Also presented in Chapter 6 are the results of the initial and repeat

lung function study, as well as the lung function for a cohort of refinery workers before and after a work period. The results chapter also presents an analysis of the effect of length of service, and the effect of smoking on lung function.

Chapter 7 provides a discussion of the results and the limitations of this research and, finally, Chapter 8 provides the conclusions and recommendations that emerged from this research.

2. LITERATURE REVIEW

2.1 Introduction

It is known that occupational respiratory disease is a major contributor to the burden of lung disease in Australia but the exact causes and statistics remain unknown (Morrell, Kerr, Driscoll, Taylor, Salkeld, & Corbett, 1998). The extent to which the occupational air quality at the Murrin Murrin Operation affects lung function remains unknown. This study investigates this. This chapter provides the background for this research and contains a critical examination of published epidemiological, experimental and theoretical evidence relevant to this research.

The main literature review was conducted prior to the start of the study to shape the experimental design and, therefore, the methodology is based on references current at the start of the study in 2004.

2.2 Air Quality and Respiratory Impairment

It has been widely reported in the literature that air quality affects lung function and thus morbidity and ultimately mortality (Ostrowski & Barud, 2006; Samet et al., 2000). According to the European Lung Foundation (n.d.a) respiratory disease is the second biggest killer globally after cardiovascular diseases (British Lung Foundation, n.d.) and excessive exposure to hazardous substances at work via inhalation is known to cause occupational lung disease, morbidity and mortality. Perhaps the most classic case in history was documented by Ramazzini who uncovered the association of stonemasonry and the exposure of silica dust, to silicosis (Wright, 1964; Checkoway et al., 2004). Similarly, pneumoconiosis in miners was reported by Agricola in 1556 (Cantrell & Volkwein, n.d.). A more recent case where poor air quality caused a significant number of deaths was the London Smog Episode of 1952 where mortality in London was affected by air pollution (Amdur, 1980). This was largely attributed to the burning of coal by house owners wanting to keep warm during a cold winter, and industries such as coal-fired power stations, resulting in an atmosphere dense with smoke particles, soot and sulphur dioxide. This event influenced public perception of poor air quality ultimately leading to the introduction of air quality guidelines (Bell, Davis, & Fletcher, 2004).

Subsequently, there have been many studies of air pollutants measured both in cities and other geographical areas throughout the world where air pollutants have been found to be associated with respiratory impairment and mortality (Souza, Saldiva, Pope, & Capelozzi, 1998; Wieringa et al., 1998; Samoli et al., 2008; Wong, Vichit-Vadakan, Kan, Qian, & the PAPA Project Teams, 2008). Urban air pollution remains a significant public health problem in many countries and is known to cause and aggravate existing respiratory disorders and cardiovascular disease (Anderson, Atkinson, Peacock, Marston, & Konstantinou, 2004). The main sources of air pollutants for the general Australian population are considered to be:

- particulates;
- ozone (O_3);
- nitrogen dioxide (NO_2);
- carbon monoxide (CO);
- sulphur dioxide (SO_2).

(Australian Bureau of Statistics, 2009)

2.3 Impact of Occupational Lung Disease

2.3.1 Worldwide

According to Driscoll et al. (2005) in the year 2000 chronic obstructive pulmonary disease (COPD) asthma and pneumoconioses caused by airborne particulates were estimated to have caused up to 386,000 deaths worldwide; and the sum of years of potential life lost due to premature mortality and the years of productive life lost due to disability (Disability Adjusted Life Years – DALY's) was estimated to be 3.7 million and 1.6 million respectively. Nelson et al. (2005) estimated for the year 2000 13% of COPD, 11% of asthma, and 9% of lung cancer, was occupationally related and accounted for a sizable proportion of the global burden of disease, which could be substantially reduced through application of proven risk prevention strategies. The annual cost of occupational COPD in the U.S. population alone was estimated to be $5 billion, based on an assumed attributable fraction for COPD deaths due to occupational exposure of 15% (Leigh et al., 2002). According to Blanc, et al., (2002) exposure to workplace contaminants such as dust, gases, vapours, or fumes was associated with a 2.0-fold (95% CI [1.6–2.5]) increase in the risk of COPD.

The European Lung Foundation (n.d.a) predicts that in 2020, there will be 11.9 million (community and occupational) deaths worldwide caused by lung diseases.

2.3.2 Europe

Sigsgaard et al., (2010) reported that in Europe in 2000, it was estimated that COPD, asthma and pneumoconioses caused 52,700 deaths (39,300, 6,200, and 7,200 respectively). Therefore occupational respiratory disease was ranked third overall, with mining occupations having the highest prevalence of occupational lung disease. Furthermore, male manual workers have been reported to have twice the risk of mortality from occupational respiratory disease than non-manual workers (Sigsgaard et al., 2010).

2.3.3 Finland

The Finnish Institute of Occupational Health (2010) reported that in 2005 the number of notified asbestos-induced cases was 807 and the number of cases of allergic respiratory disease was 746. Karjalainen, Kurppa, Martikainen, Karjalainen, and Klaukka (2002) determined the attributable fraction of work-related asthma to be 29% for men and 17% for women. The overall incidence of pneumonia in Finland was estimated to be 10.8 per 1,000 adults per year (European Lung Foundation, n.d.a).

2.3.4 Norway

Eagan, Gulsvik, Eide, and Bakke (2002) conducted an 11-year cohort study of a population of 2,819 Norwegian subjects to evaluate the influence of occupational exposure on the incidence of respiratory symptoms and asthma. They determined that occupational exposure accounted for 14% of asthma cases and 6-19% of respiratory symptoms.

2.3.5 USA

The most recent report from the NIOSH Work-Related Lung Disease (WoRLD) Surveillance System (2009b) presents detailed data on occupationally-related respiratory disease in the USA. Selected extracts quoted from the highlights are provided below.

2.3.5.1 Hypersensitivity pneumonitis

The annual number of hypersensitivity pneumonitis deaths in the USA has been generally increasing, from less than 20 per year in 1979 to over 60 in 2004.

2.3.5.2 Work-related asthma

There were 4,132 cases of work-related asthma in the USA during 1993–2002. About 68% of which represented asthma caused by occupational exposure, while 20% represented pre-existing asthma aggravated by occupational exposure.

Nearly 20% of asthma cases were associated with miscellaneous chemicals, 13% with mineral and inorganic dust, twelve percent with cleaning materials, 11% with indoor air pollutants, and 4% with exposures to polymers, among others.

2.3.5.3 COPD

Mining industries were in the top five USA industries for COPD mortality, as were trucking service and automotive repair and related services.

2.3.5.4 Respiratory conditions due to toxic agents

The estimated number of cases of respiratory conditions due to toxic agents in 2000 in the USA was 14,700.

The estimated rate of respiratory conditions due to toxic agents in the primary metals industry (CIC 33) was 3.8 per 10,000 full-time workers in 2000.

2.3.5.5 Asbestos

Data from the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA) indicate a trend towards lower asbestos exposure levels from 1979 to 1999, concomitant with mandated reductions in the OSHA permissible exposure limit (PEL). However, data indicate a steady increase in asbestos exposure levels in the mining industry for the years 2000 through 2003 and a slight rise in all other industries in the 2 years previous to 2003. Olsen et al (2011, p. 273) state that “in [Western Australia] WA, 1631 people (1408 men, 223 women) were diagnosed with malignant mesothelioma between 1960 and 2008”. Asbestos may be intersected during exploration drilling and unearthed during mining operations as it is widely

distributed in WA particularly in the “greenstone belts” (Department of Mines and Petroleum, 2010c)

2.3.5.6 Silica

Over the past several decades in the USA, silicosis mortality has declined, from well over 1,000 deaths annually in the late 1960s to fewer than 200 per year in the mid 2000s. The construction and mining industries accounted for at least one third of deaths attributable to silicosis from 1990 through 1999 (Centers for Disease Control & Prevention, 2008).

2.3.6 Germany

According to Loddenkemper (2006) 37% of all compensated occupational diseases are attributable to occupational lung disease in Germany. Baur and Latza (2005, p. 597) reported that the confirmed cases of non-malignant occupational respiratory diseases in Germany were:

- benign asbestos-associated diseases (1,975 cases);
- silicosis/coal-worker's pneumoconiosis (1,158 cases);
- obstructive airway diseases due to allergens (935 cases);
- chronic obstructive bronchitis and/or emphysema in hard-coal miners (414 cases);
- obstructive airway diseases due to irritants and toxic agents (181 cases);
- diseases caused by ionising radiation (171 cases);
- diseases due to isocyanates (55 cases); and
- 22 cases of other rare occupational lung and airway diseases.

Baur and Latza (2005, p. 597) also reported that miners, bakers, chemical workers, hairdressers and health care workers were affected the most.

2.3.7 United Kingdom

In the 1999 SWORD surveillance of work-related and occupational respiratory disease report an increase was seen in most respiratory diseases in the United Kingdom. There were 4,393 incident cases. Benign pleural disease was the single most frequently reported condition (28% of all diagnoses reported) there were 1,168 (26%) occupational

asthma cases and 1,032 (23%) cases of mesothelioma (Meyer, Holt, Chen, Cherry, & McDonald, 2001).

The British Thoracic Society (2006) determined that mortality and morbidity due to occupational lung disease was increasing, with a 70% rise in mortality due to mesothelioma since 1992. It was estimated that 4,000 COPD deaths every year may be associated with work exposures. Also, in 2006 the Health and Safety Executive, UK, estimated that the true total lifetime cost of occupational asthma for male workers was between £53.6 and £78.0 million (British Lung Foundation (n.d.a). Ayres, Boyd, Cowie, and Hurley (2010) estimated the cost of occupational asthma to be as large as £70—£100 million however, and, due to underreporting, may be as large as £95—£135 million.

The Health and Safety Executive (n.d.) determined that in the UK in 2009-2010 there were 38,000 workers who had breathing or lung problems caused or exacerbated by work.

2.3.8 South Africa

In South Africa, despite incomplete reporting, diseases with long latency periods made up 76.2% of the cases of work-related respiratory disease. This is reflected in the study by Girdler-Brown, White, Ehrlich, and Churchyard (2008) who determined that in a cohort of former gold miners, 18 months after cessation of work, 50% had either silicosis, tuberculosis or COPD. Moreover, Esterhuizen et al. (2001) reported that pneumoconiosis was the most frequently reported disease, followed by inhalation accidents; and that occupational asthma was the fourth most reported disease.

Overall it can be seen that the profile of occupational lung disease varies with each country, geographical region, and type of industry, and is in constant flux due to changes in industrialisation. According to Hendrick et al. (2002) occupational asthma and chronic bronchitis are currently more prevalent than alveolar and interstitial diseases. To this end, the World Health Organization (2010) has developed a strategy for the prevention and control of chronic respiratory disease; and many workplace regulatory authorities throughout the world have developed preventative strategies (WorkSafe WA, 2004; Department of Mines and Petroleum [DMP], 2010a; Safe Work Australia, 2010d; Health and Safety Executive, UK, n.d.; NIOSH, 2009a).

2.3.9 The background general respiratory health of Australians

According to the Australian Bureau of Statistics (2009) lung disease is a significant and growing health issue in Australia with 2.6 million cases of lung disease reported in 2007–2008. The Australian Bureau of Statistics also stated that more than 2 million Australians reported having asthma during 2007–2008; and that each year lung disease causes 19,200 deaths in Australia. According to the Australian Lung Foundation (2009) one in five Australians over 40 is affected by COPD.

However, the degree to which occupational exposure to respiratory hazards affects these statistics appears at present to be unknown. Although Safe Work Australia (2010c, p. 3) states that "occupational respiratory diseases did not display a clear overall trend of increase or decrease", and it is difficult to separate work-related from non-work-related respiratory disease (NIOSH, 2007).

Australia ranked fourth highest out of 16 high-income countries in the prevalence of moderate to severe COPD (de Marco et al., 2004) and according to the Australian Institute of Health and Welfare (2008) about 9–15% of asthma in adults may be caused by occupational exposures, which can result in a significant time off work for those suffering from the disease.

2.4 Occupational Respiratory Disease

The Australian Safety and Compensation Council (2006, p. 2) defined occupational respiratory disease as "respiratory disease that is caused or exacerbated by work factors", and asserted that the causative agents are airborne contaminants "such as dust, mist, fibres, fume, vapour or gas" which if inhaled and are "small enough in size to gain access to the deeper, pulmonary areas of the lung" are likely to cause occupational respiratory disease.

Except for inhalational accidents where excessive exposures results in obvious acute lung injury (Sallie & McDonald, 1996; Shakeri, Dick, & Ayres, 2008) occupational respiratory disease is often hard to distinguish from respiratory disease observed in the general population (Hendrick et al., 2002) as respiratory disease may be caused by environmental or occupational exposures and the contribution that each plays is often not determined. These combined effects may be additive or even synergistic, as in the case of smoking and asbestos exposure (Selikoff, Hammond, & Churg, 1968). Even in

one discrete industry it is not easy to determine the causative agents of respiratory disease. For example; Ross and Murray (2004) pointed out there are different airborne exposures in the mining industry even for the extraction of one type of mineral due to the different tasks and equipment used. In 1997, Mitchell called for an Australian register of all occupational lung diseases, and again in 2006 Sim, Abramson and Radi commented on the lack of data regarding occupational respiratory disease in Australia. More recently, to address this Safe Work Australia (2010d) conducted a *National Hazard Exposure Worker Surveillance: Exposures to dust, gases, vapours, smoke and fumes and the provision of controls for these airborne hazards in Australian workplaces*. Hence it is work in progress. The extent to which occupational factors contribute to respiratory disease does not appear to have been determined and is difficult to ascertain.

2.5 Epidemiology – Cause and Effect

All diseases have causes and consequences. The aetiology of workplace exposures to airborne contaminants may be obvious if an apparent disease cluster or rare disease is observed. For example, vinyl chloride was found to cause angiosarcoma, a rare cancer of the liver (Creech & Johnson, 1974). Determining the aetiology of lung disease may be obvious, for example, in the case where excessive exposure to silica dust causes silicosis. Similarly the smog and its constituent smoke particles, soot and sulphur dioxide that caused the London Smog Episode, caused and aggravated existing respiratory disorders and cardiovascular diseases (Amdur, 1980). Likewise the cause and effect of a single massive chemical exposure resulting in reactive airways dysfunction syndrome (RADS) (Shakeri et al., 2008) is obvious. These are case series reports where an apparent increase in a specific disease is reported and clear a cause and consequence determined in each case. However, for more complex cases more complete epidemiological studies are required with comparison groups and exposure assessments (Checkoway et al., 2004).

2.6 Aetiology of Lung Disease

Epidemiological studies aim to determine the aetiology of disease and can be utilised to determine the aetiology of occupational respiratory disease. Respiratory irritants via inhalation may cause airways inflammation and subsequent damage of the upper and lower respiratory tract, or chemical pneumonitis which is acute inflammation of the

pulmonary parenchyma. Asthmagens can cause diseases of allergic and immunological origin and provoke respiratory symptoms in sensitised individuals at very low concentrations (Ryon & Rom, 1998).

The aetiology of lung disease is due to the inhalation of hazardous substances such as dust, fibre, gas, vapour, mist, aerosol, fumes and smoke (Campbell, 2009) above a *threshold level* (United States Environmental Protection Agency, 2009). The toxic effect is reversible if the exposure (or dose) to the hazardous substance remains below the threshold (the no-observed-adverse-effect level [NOAEL]) level and irreversible if it exceeds this level. The American Thoracic Society (2003, p 787) states, "There are convincing data to show that the level of exposure is a critical risk factor for sensitizer-induced occupational asthma". Similarly Oudijk, Lammers and Koenderman (2003, p. 9) state that COPD:

> appears to start as a reversible self limiting inflammatory reaction … mediated by both monocytes and neutrophils …. After prolonged exposure … a switch to chronicity takes place and an irreversible inflammatory reaction is initiated which is clearly associated with neutrophils in the lung tissue.

According to Barnes, Shapiro, and Pauwels (2003) the switch to chronicity, or chronic inflammation, in the case of COPD results in fixed narrowing of small airways and emphysema. There are histopathological differences between COPD and asthma in that COPD mostly affects the bronchioles as well as the parenchyma, whilst there is an inflammatory response in all airways with asthma, however, this is usually without involvement of the lung parenchyma (Fabbri et al., 2003).

The pathophysiological effects of airborne contaminants on the respiratory system are dependent on the physicochemical properties, the toxicity, and the dose of the airborne contaminant or mixture of airborne contaminants (Tranter, 2004; Plog, 2002). Normally, as the dose of the airborne contaminant increases so does the response on the respiratory system that is, the dose-response relationship (Tranter, 2004). For gases, the water solubility of an inhaled gas will determine the site of deposition, whilst the duration of exposure and rate of breathing of an individual, and their susceptibility, will influence the severity of the toxic effect (Sullivan & Krieger, 2001). For particulates the aerodynamic diameter is of importance. Aerodynamic diameters in the 0.3-0.5 μm

range may be deposited in the lower airways and alveoli, often resulting in diffuse bronchiolar inflammation and obstruction as well as pulmonary oedema (Rosenstock, Cullen, Brodkin, & Redlich, 2004). Acute or subacute exposure to toxic chemicals, as described by Nemery (2002), results in inflammation of lung tissue. A vast number of substances can therefore cause inhalation injury.

The immunological aspects of COPD are described by Cosio, Seatta and Agusti (2009) as a cascade of inflammatory processes. The histopathological picture of a typical respiratory insult is loss of ciliated epithelial cells of the airway and of type I alveolar epithelial cells. Following this there is damage to the tight junction interface between epithelial cells, sequentially leading to subepithelial and submucosal damage, with effects on the smooth muscle and afferent parasympathetic sensory nerve endings causing bronchoconstriction. This in turn initiates an inflammatory response where neutrophils and eosinophils release mediators that cause further injury. According to Ryon and Rom (1998) the repair mechanism is initiated by type II pneumocytes and cuboidal cells.

2.7 Continuum of Respiratory Health Effects

As with most pathophysiological effects there may be a continuum of effects on the respiratory system. These may be an acute and reversible effect due to a single or a small number of sub-threshold exposures, or irreversible effects due to a single high-level acute exposure, or repeated chronic exposure (Plog, 2002). Delayed onset from acute exposure to toxic agents may occur days to weeks after initial insult (Schwartz, 2002). The consequences of such respiratory exposures may range from irritation, allergy, cell damage, fibrosis, oedema, emphysema, cancer or systemic effects (Winder & Stacey, 2004).

A continuum of effects on the pulmonary system begins with acute lung injury due to damage to the epithelial-endothelial barrier of the lung, which may be due to a variety of agents. This begins with an early inflammatory response with the release of humoral mediators, which, if left unchecked, may progress with the development of chronic lung injury (Jacono et al., 2006).

Meldrum (2001) and Banks (2001) both state that responses to irritants may be viewed as occurring along a continuum. At one end of the continuum no effects would

be observed, followed by reversible irritation, and ultimately irreversible effects would be observed. The aim of respiratory health surveillance is to detect possible adverse effects at an early stage in order to prevent potential long-term occupational respiratory disease. Ammonia, hydrogen sulphide, nitrogen oxides and sulphur dioxide have been shown to be associated with inflammatory changes in small airways. These respiratory irritants at low-level repeated exposure and acute high-dose exposure are capable of causing decrements in lung functions measured by spirometry (Boswell & McCunney, 1995).

Menzel and McClelland (1980, p. 264) categorise pulmonary responses to an array of toxicants into five categories as follows:

1. Irritation of the air passages which results in constriction of the airways. Oedema often occurs and secondary infection frequently compounds the damage.

2. Damage to the cells lining the airways, which results in necrosis, increased permeability, and oedema. This oedema is, in general, intraluminal (within the airways) rather than interstitial (within the cells of the airway).

3. Production of fibrosis, which may become massive and cause obliteration of the respiratory capacity of the lung. Local fibrosis of the pleura also occurs, restricting the movement of the lung and producing pain through the irritation of the pleural surfaces.

4. Constriction of the airways through allergic responses. Allergic alveolitis is a widespread response to the inhalation of some simple compounds, as well as of complex organic materials capable of producing specific antigenic responses.

5. Oncogenesis leading to primary lung tumours.

Gee and Mossman (1995, p. 197) focused on the cellular and molecular mechanisms of occupational lung disease and describe the pathophysiology of *acute lung injury* as:

1. Direct toxic effects on type I cells and endothelial cells (ECs);

2. Compliment activation;

3. Coagulation factors activation; and

4. EC biochemical response and polymorphonuclear neutrophil recruitment and activation that release protease and oxidants.

Gee and Mossman (1995, p. 197) also emphasise that "patients with the first group of disorders, even when severe, frequently make a complete recovery, and lung fibrosis is not the rule".

There is a threshold level, which, if exceeded ultimately leads to irreversible lung disease, and at sub-threshold levels the effects are reversible, as in the continuum postulated by Meldrum (2001) and Banks (2001). The aim of occupational respiratory health surveillance is to detect respiratory effects (should they exist) prior to the development of chronic irreversible pulmonary disease.

The susceptibility of the target organ or tissue and its repair mechanisms also has an impact on the reversibility/irreversibility of a pathophysiological effect (Eaton & Gilbert, 2008). Higenbottam, Siddon and Demoncheaux (2001) recognise that environmental pollution in its various forms, such as dust, fibre, gas, vapour, mist, aerosol, fumes and smoke, may produce a variety of effects on the lungs, both localised and general. Whilst Checkoway et al. (2004) point out that normally the risk is proportional to the dose (exposure) for most pathophysiological mechanisms. However, there are occasions where this is not so and toxicity follows a "non-linear or dynamic" pattern, such as with occupational asthma where there is "development of specific sensitivity", and "in-migration of inflammatory cells following irritant exposure to the airways" where there is a "positive feedback" (Checkoway et al., 2004, p. 309). Notwithstanding, Crapo, Harmsen, Sherman and Musson (2000) emphasise that the lung's defence mechanism maintains lung homeostasis and keeps the inflammatory response to foreign substances and antigens in check. Lafferty, Qureshi and Schnare (2010, p. 1) capture this in their statement:

> Selective induction of inflammatory responses to harmful environmental exposures and tolerance to innocuous antigens are required to maintain tissue homeostasis and integrity. Conversely, dysregulated innate immune

responses manifest as sustained and self-perpetuating tissue damage rather than controlled tissue repair.

Once this defence mechanism is breached, "disordered inflammation and immune responses" occur, which ultimately leads to "progressive and chronic lung diseases" (Crapo et al., 2000, p. 1983) most notably COPD or chronic asthma. COPD is usually a combination of emphysema and chronic bronchitis, where the alveolar walls are gradually destroyed (emphysema) and excessive mucus is produced as a consequence of chronic bronchitis (Australian Lung Foundation, 2010). Asthma, known to be caused by a variety of chemical and biological substances, is an inflammatory response (hypersensitivity) that causes narrowing of the upper respiratory tract which results in difficulty in breathing and wheezing (Chang-Yeung & Malo, 1994).

2.8 Mixed Exposures

As already discussed, exposure to low levels of airborne contaminants in the workplace may cause acute inhalation injury and occupational asthma, although an acute excessive exposure may result in substantial lung injury (Banks, 2001; Hudson & Steinberg, 1999). Balmes (2002, p. 727) reports, however, that such acute excessive exposures do not frequently occur and that "chronic recurrent exposures to lower levels of irritants are much more common". Furthermore, Balmes et al. (2003, p.787) state that "the lungs of workers at risk are subjected to the total exposure burden of all airborne contaminants in any workplace". It therefore seems that many occupational diseases have multiple causes (Muir, 1995).

Lung injury often results from interactions between two or more toxic agents (Witschi & Hakkinen, 1984). Petsonk (2002) asserts that work-related asthma may be caused by mixed exposures. On reviewing the disease *suberosis,* Alegre, Morell, and Cobo (1990) believe that three different distinct diseases with three different aetiological factors occur simultaneously due to mixed exposure to cork dust, toluene diisocyanate and conidia.

It is now well established that certain occupational exposures are strongly associated with an increased risk of COPD, and that, in combination, smoking and occupational exposure may significantly magnify the risk of COPD. Blanc et al. (2009, p. 12) state,

"On a population level, prevention of both smoking and occupational exposure, and especially both together, is needed to prevent the global burden of disease".

2.8.1 Low-level mixtures of respiratory irritants

Occupational exposure to low levels of mixtures of respiratory irritants (i.e., each below its respective occupational exposure standard) is capable of causing respiratory symptoms and a decrement in lung function (Barnhart, 1994; Kortenkamp, Faust, Scholze, & Backhaus, 2007). Kremer, Pal, Boleij, Schouten, and Rijcken (1994) identified a decrement in lung function for employees working greater than 10 years exposed to a mixture of low-level lung irritants (i.e., polyester vapour and oil mist and vapour workgroups). Similarly, Mustajbegovic et al. (2000. p. 439) detected decrements in lung function associated with mixed exposures, in two chemical factories in Croatia, even when the atmospheric levels of chemicals were "for the most part within acceptable limits". The effect on the lung by interaction between respiratory irritants may be additive, synergistic or antagonistic (Witschi & Hakkinem, 1984). It is possible to predict the respiratory response of a respiratory irritant if the physical and chemical properties are known. It becomes more difficult, however, when there is a mixed exposure. Historic examples of 'cocktails' of respiratory irritants include London smog, characterised by sulphur dioxide and smoke from incomplete combustion of coal combined with temperature inversion. Equally, the Los Angeles oxidising/photochemical air pollution, was characterised by hydrocarbons, nitrogen oxides, and photochemical oxidants catalysed by intense sunlight and a meteorological inversion (Amdur, 1980). More recently, environmental air pollution studies have been conducted using spirometry and questionnaires. For example, the study by Yu et al. (2001, p. 310) addressed the *Adverse Effects of Low-Level Air Pollution on the Respiratory Health of Schoolchildren in Hong Kong*, determined that "children living in a more polluted district" had increased respiratory symptoms and "significantly poorer lung function". Balmes (2002, p. 727) states that "although the contribution of low-level irritant exposures to the overall burden of work-related asthma cannot be precisely estimated the available data indicate that it is not likely to be inconsequential". Further research into the toxicology of low-level mixed exposures affecting the respiratory system is warranted, particularly in the mining industry, in order to identify such hazards and prevent occupational lung disease.

2.9 Prevention of Lung Diseases

Both the World Health Organization and the International Labour Organization (ILO) have targeted occupational lung disease for their preventive strategies due to its high prevalence, the fact that it is severely disabling, and workplace exposures can be avoided. Key to such preventive strategies is health surveillance (Takahashi et al., 1998).

Occupational exposure to respiratory hazards still remains a significant worldwide problem (Blanc et al., 2009). Different patterns of respiratory disease are seen throughout the world, being more prevalent in newly industrialised regions (Hendrick et al., 2002). There are a number of occupational lung disease surveillance databases which highlight the impact of occupational lung disease, although direct comparison is made difficult due to differences in reporting, classification, and medical diagnosis (Hendrick et al., 2002).

Various countries throughout the world, including Finland, the UK, the USA, France and South Africa have developed occupational respiratory disease surveillance systems. For example, NIOSH (2009b) manage an occupational respiratory disease surveillance system in the USA. However, as Elder et al., (2004, p 395) state "there are few such comprehensive and systematic data collection systems in place to monitor the extent of occupational respiratory disease in Australia". Similarly, the Australian Safety and Compensation Council (2006, p. iv) states "there is limited information on the extent of work-related respiratory disease in Australia". Therefore, the true incidence of occupational respiratory diseases in Australia remains largely unknown. One such surveillance scheme, SABRE, has been introduced in Victoria and Tasmania and is currently being extended to New South Wales. This system will provide data on occupational respiratory disease in those states (Monash University, 2004). The feasibility of a national environmental health surveillance system for Australia was considered in January 2008 by the Western Australian Environmental Health Directorate, under guidance from the Australian Government Office of Health Protection (Mullan, Ferguson, & Paech, 2008). Such surveillance schemes are an important component in the development of comprehensive occupational respiratory disease prevention strategies.

Health surveillance programs in the workplace will help pinpoint potential causative agents and adverse health effects at an early stage, in order to prevent potential long-term occupational respiratory disease. Health surveillance is a secondary preventative strategy. However, when used in combination with primary preventive strategies that focus on the control of workplace exposures using the traditional hierarchy of controls including education and training programs (Tranter, 2004) then prevention of occupational respiratory disease is feasible. According to Hnizdo, Glindmeyer, and Petsonk (2010, p. 797) "there are documented examples of spirometry monitoring coupled with intervention successes". They cite Musk, Peters, Bernstein, Rubin and Monroe (1982) and Pahwa, Senthilselvan, McDuffie and Dosman (2003) where a decline in forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) was reduced after introduction of a hierarchy of controls for fire-fighters and granary workers respectively.

2.10 Distinguishing Occupational Respiratory Disease from Background Environmental Respiratory Disease and Lifestyle Factors

Several authors have determined that workplace exposure contributes to overall mortality and morbidity of COPD (Hnizdo et al., 2002; American Thoracic Society, 2003; Trupin et al., 2003).

In 1965, Sir Austin Bradford-Hill (1897—1991) established nine criteria to determine the strength of potential links between a causative agent and a disease (causality). These include:

- temporal relationship;
- strength;
- dose-response relationship;
- consistency;
- plausibility;
- consideration of alternate explanations;
- experiment;
- specificity;
- coherence.

Checkoway et al. (2004, p, 12) state that "determining the potentially harmful effects of occupational exposures typically involves estimating relative risks or changes in physiological function in relation to exposure types and levels". The assessment of respiratory health risk requires recognition and evaluation of hazards by reviewing the process, raw materials used, work practices and controls in place, followed by an assessment of exposures through the determination of airborne contaminants using air sampling techniques (Plog 2002; Safe Work Australia, 2010a). Following such a formal process provides reliable information that can be used to determine the causes of occupational respiratory disease and pave the way for prevention strategies and controls. Using a formal epidemiological approach, the impact of work-related respiratory disease can be established, by comparing incidences of respiratory disease in an exposed (study) and a non-exposed (control) population. Excess incidence in an exposed population (after correcting for confounding factors, such as smoking) allows for the calculation of the population attributable risk (PAR %) – assuming the proportion of exposed persons in the population is known (Health and Safety Executive, UK, 2004).

2.10.1 Confounding

To distinguish occupational respiratory disease from background environmental respiratory disease and lifestyle factors, such as the prevalence of smoking, it is necessary to identify and eliminate these confounding factors where possible. According to Checkoway et al. (2004, p. 89) "Confounding can be thought of as a mixing of the effects of the exposure being studied with effects of other factors on risk of the health outcome of interest".

Conversely there may be a *healthy worker effect* which can occur when comparing health outcomes between the study and control group. A potential exists for the health impacts in this study to have been underestimated among the study group (Checkoway et al., 2004). Age, gender and body mass index are all confounding factors that need to be adequately controlled in the study design or analysis (Checkoway et al., 2004).

2.11 Occupational Health Surveillance

There is an international directive, ILO C176 Article11 (International Labour Organization, 1998) for the provision of health surveillance in mining. The primary aim

of occupational health surveillance is to maintain and protect worker health (Cooper & Zavon, 1994). Health surveillance is the systematic collection, analysis, and interpretation of health data to enable detection of early disease in individuals, followed by interventions aimed at preventing further exacerbation (Centers for Disease Control and Prevention, n.d.).

Such a health surveillance program should:

- identify workers or work groups who are exposed to health hazards at work;
- assess their risk of suffering an adverse health effect from exposure to these hazards;
- evaluate their health to detect early signs and symptoms of adverse health effects;
- verify the effectiveness of workplace controls (National Offshore Petroleum Safety Authority , n.d.); or
- implement further workplace controls if the risk remains intolerable. (Tranter, 2004).

It is necessary to recognise when respiratory disease is work related, to manage the consequences and prevent its reoccurrence (Hendrick et al., 2002). The UK Health and Safety Executive assert that the key to the formulation of effective treatment and preventative strategies relies on the accurate diagnosis of occupational respiratory disease (Health and Safety Executive, UK, n.d.). Fortunately, in the workplace, levels of hazardous substances can be measured through air monitoring, and their respiratory effects can be monitored by lung function tests (Hendrick et al., 2002) and diagnosis can be made through a respiratory questionnaire and spirometry that complies with the ATS/ERS criteria (American Thoracic Society, 1995; Miller et al., 2005; DMP, 2010a).

2.11.1 Workplace health surveillance in Australia

A senate inquiry (Parliament of Australia Senate Committee, 2006) into workplace harm related to toxic dust and emerging technologies highlighted the need for robust surveillance systems and early accurate diagnosis of loss of lung function. The surveillance system in Australia is based primarily on workers' compensation claims (Safe Work Australia, 2011c). Safe Work Australia (2011c) has targeted occupational respiratory diseases as one of eight identified occupational diseases for priority action.

The current the Australian *National Guidelines for Health Surveillance [NOSHC: 7039]*, in NOSHC, 1995c, p. 1) state "these guidelines are intended for use by the appointed medical practitioner when planning and implementing a program of health surveillance". Schedule 3 of these guidelines requires that a workplace risk assessment be conducted when one or more of 17 specified hazardous substances exist at a workplace. The related *National Model Regulations for the Control of Workplace Hazardous Substances [NOHSC: 1005] 1994, Health Surveillance* (Safe Work Australia 2011d) require that a risk assessment be conducted in each workplace where Schedule 3 hazardous substances exist, where other hazardous substances may cause health effects, or "there is a valid biological monitoring procedure available and a reasonable likelihood that accepted values might be exceeded". The employer then is required to provide health surveillance (Safe Work Australia 2011d, Section 14 c).

2.11.2 Department of Mines and Petroleum MineHealth Surveillance System

Health surveillance is driven by the statutory requirement in Western Australia under the Mines Safety and Inspection Act 1994. The Department of Mineral and Petroleum Resources (DMPR) under the Health Surveillance Program for Mine Employees states "It is mandatory that all assessments required under the Act are completed in accordance with these approved procedures". This program has been established according to DMPR "to promote the implementation of an effective health surveillance system for mining industry employees" (Department of Consumer and Employee Protection, 2010, p. 3).

> The objectives of the health surveillance system for mining employees are to:
>
> - Assess the health status of all mining industry employees on a regular basis;
> - Analyse collected data to detect adverse health effects at the earliest opportunity;
> - Enable appropriate and timely corrective action to be taken in order to safeguard the health and well-being of mining industry employees;
> - Provide data for future epidemiological studies.
>
> (DMP, 2010a, p 2)

Mining industry employers in Western Australia are required under legislation to establish and maintain a health surveillance system for employees. This health

surveillance system requires a health assessment for mining employees which consists of:

- a work history;
- a respiratory questionnaire;
- a lung function test;
- an audiometric (hearing) test; and
- in some cases, a chest x-ray.

(DMP, 2010a, p. 2)

Further health assessments are required at least every five years for all mine employees. One of the main objectives of this health surveillance is to determine the respiratory health of the mine and process workers to "enable appropriate and timely corrective action to be taken in order to safeguard the health and well-being of mining industry employees" (DMP, 2010a, p. 2).

2.11.3 Spirometry and respiratory questionnaire: subjective instruments to measure respiratory health

Beach (2002, p.1009) states that "lung function measurements provide an important tool in the diagnosis and management of occupational lung diseases". Used in conjunction with a respiratory questionnaire, spirometry testing provides a powerful diagnostic tool with adequate sensitivity and specificity (Post et al., 1998). Bellia et al. (2003, p. 21) asserts that a validated respiratory questionnaire provides a "subjective instrument of measurement in respiratory epidemiology". That is why respiratory health surveillance utilises a combination of a work history, a respiratory questionnaire and a lung function test.

2.11.4 Health surveillance studies utilising the combined questionnaire and spirometry

Various occupational respiratory health surveillance studies have been undertaken utilising the combined questionnaire and spirometry tool. Some selected examples are provided:

- In a longitudinal follow-up study of manganese mine workers, Boojar and Goodarzi (2002) using a respiratory questionnaire and spirometry combination, observed a significant decrement in lung function in 145 manganese mine workers compared to a control population at

approximately 4 years and 7 years after baseline measurements made at the time of employment.

- Vogelzang, van der Gulden, Folgering, and van Schayck (1998) investigated lung function in swine-confinement workers. This longitudinal study utilised a combined questionnaire and spirometry approach and identified "a longitudinal decline in forced expiratory volume in one second (FEV_1) which was significantly associated with the use of quaternary ammonia compounds as disinfectants and the automated dry feeding system" (Vogelzang, van der Gulden, Folgering, & van Schayck, 1998, p. 1048). The study also demonstrated the value of the combined use of spirometry and a respiratory questionnaire. In this study the questionnaire alone would not have distinguished respiratory symptoms because a significant number of pig farmers ceased to report their chronic symptoms. However, there was a definite decrement in FEV_1 in the cohort of 171 pig farmers over a 3-year period.
- From referrals to family physicians, Mpofu, Lockinger, Bidwell, and McDuffie (2002) observed decrements in lung function in a study of farmers and their families. This research was predominantly about the evaluation of a respiratory health program for Saskatchewan farmers and their families in Canada. There are a myriad of exposures likely to affect the respiratory health of farmers and their families. This was addressed to a limited degree in this study through a qualitative analysis of exposure to dust or fumes/chemicals. The protocol for this program included spirometry and a respiratory questionnaire as well as a health promotional component. Thus the study analysed the association of pulmonary function and self reported respiratory symptoms. The main outcome of the study was that "Individuals with these symptoms or who smoked were more likely than individuals without these symptoms and non-smokers to have lower than predicted overall pulmonary function" (Mpofu, Lockinger, Bidwell, & McDuffie, 2002, p. 1069).

- In a case study pulmonary effects were evident in workers exposed to incinerator fly ash, when using the combined questionnaire and spirometry tool (Boswell & McCunney, 1995).
- In a longitudinal study conducted by Motley, Smart, and Valero. (1956. p. 265) there was poor correlation with lung function testing compared with radiography for diatomaceous earth workers. In the initial study a decrement in lung function was detected in compensation claimants employed in the diatomaceous earth industry. In the repeat study combining radiography, spirometry and a questionnaire, the decrement in lung function was not convincing, although "the best lung function was present in those with no radiographic abnormality".
- In longitudinal studies of cannabis smokers, Taylor et al., (2002) found a relationship between cannabis smoking and a decline in lung function. Taylor et al., (2002, p. 1060) concluded "Longitudinal observations over 8 years in young adults revealed a dose-dependent relationship between cumulative cannabis consumption and decline in FEV_1/VC". "Dispelling the myth that cannabis smoking is relatively safe" Taylor followed this up in a *Position Statement of the Thoracic Society of Australia and New Zealand* stating that adverse respiratory effects of smoking cannabis are similar to those of smoking tobacco… (Taylor, & Hall, 2003, p. 310).
- Case studies on the lung function of 30 fire-eaters prior to and after their daily activity showed a mild decrease of airflows, which partially correlated with number of years spent in this activity. After a 1-day fire-eating activity, further airflow limitation increased. Up to 63% of subjects tested improved some spirometric airflow value after salbutamol inhalation (Cabrera et al., 2003).
- A cross-sectional and cohort of respiratory morbidity study of long-term and former asbestos workers with substantial exposure in Brazil (Algranti et al., 2000) and a case control study of asbestos workers in China (Wang, Yano, Wang & Christiani, 2001) demonstrated decreased pulmonary function, pleura thickening due to asbestos exposure.

- In a cross-sectional study there were subtle decreases in pulmonary function detected for a sub-population of tea packers from the start to the end of a shift (Abramson et al., 2001).
- In a cross sectional study Musk et al. (2000) concluded that no major adverse respiratory health effects were associated with work in the Australian alumina refineries studied.
- In a cross-sectional cross-shift study Raulf-Heimsoth et al. (2007) found significant cross-shift declines in lung function due to the irritative effects of fumes and aerosols of bitumen.
- Peters, Demers, Sehmer, Karlen, and Kennedy (2010) studied the lung function of a cohort of 281 trades' apprentices and concluded that "early signs of respiratory trouble among young adults….are related to the development of asthma and other respiratory illness later in life" (Peters, Demers, Sehmer, Karlen, & Kennedy, 2010, p. 242).
- The Queensland Mines Inspectorate determined that some Queensland quarries had problems with the control of silica dust exposure, did not have adequate exposure assessments, or health surveillance in place for their workers. Hedges, Reed, Mulley, Djukic and Tiernan (2010) therefore conducted a cross-sectional study of Respirable Crystalline Silica (RCS) exposure in quarries in Queensland and determined loss of lung function using an EasyOne® spirometer. They recommended that frequent lung function measurements be made to detect developing health problems due to cumulative exposures of RCS.

2.11.5 Reliability and validity of spirometry

In the past some authors have questioned the practical value of pulmonary function tests for early changes for disorders such as chronic obstructive lung disease (West, 1987). The Health and Welfare Canada Task Force on Health Surveillance (Canadian Public Health Association, 1986, p. 106) went even further to infer that "Spirometry is indicated [only useful] to monitor established disease or for diagnostic purposes in symptomatic individuals". Rossignol, Seguin, and DeGuire (1996, p. 1259) found in a Regional Public Health Program, "there were too many sources of variation for spirometry to fulfil the objective of early detection of pulmonary function decline related to exposure to welding fumes. Sobaszek et al. (1998, p. 223) reported for

stainless steel welders that "there was no influence of the specific welding process on the spirographic parameters, but a decrease in spirographic values after 25 years of welding activity was evident". Ulvestad et al. (2001) did not detect a decrement in lung function, whereas they detected airways changes using acoustic rhinometry and detected increased levels of exhaled nitrous oxide, associated with underground construction workers exposed to dusts and gases. Nield and Burmas (n.d., Conclusion) representing DMP, Western Australia, Resources Safety, concluded that the "use of screening spirometry in healthy populations is questionable based on costs versus benefits to stakeholders, thus MineHealth methodology is currently under review". In their presentation Nield and Burmas discussed the phenomenon of an improved lung function response for individuals at a repeat/second test, which is considered to be due to an individual's ability to do better on a repeat spirometry test as they have mastered and improved their technique, hence adding a form of confounding although such intersession changes are likely to be considered measurement noise (Nield and Burmas, n.d.).

For lung function tests to be valid they must be performed rigorously to the American Thoracic Society and European Respiratory Society (ATS/ERS) criteria (American Thoracic Society, 1995; Miller et al., 2005) and the Thoracic Society of Australia and New Zealand (in Pierce and Johns, 1996). If these protocols are followed rigorously, issues such as reproducibility, reliability, sensitivity, specificity and quality control are addressed (Doherty, 2008). In so doing, the American College of Occupational and Environmental Medicine (2010, p. 1) state that, "Spirometry, the most frequently performed pulmonary function test (PFT) is the cornerstone of occupational respiratory evaluation programs". Spirometry has been recommended as an instrument for health surveillance to help determine and prevent occupational respiratory disease (Hankinson & Wagner, 1993; Harber & Lockey, 1993; American College of Occupational and Environmental Medicine, 2000; Townsend, 2005).

Finkelstein et al. (1993, p. 532) determine that measurement in the home using portable spirometers was valid when compared with the 'gold standard' of the pulmonary function laboratory; and Johns and Pierce (2003, p.1) promote spirometry as "the single most broadly useful non-invasive test for ventilatory lung function". With interpretation, spirometry is capable of detecting a range of decrements in pulmonary function such as:

- inflammation of the respiratory system;
- airway narrowing;
- obstruction;
- bronchospasm;
- COPD;
- asthma;
- emphysema.

Johns and Pierce (2003) conclude that spirometry is particularly useful for differentiating between obstructive and restrictive lung function problems.

2.11.6 Obstructive ventilatory defect

Obstructive ventilatory defects disturb air flow in and out of the lungs as pathophysiological effects reduce the diameter of the airways causing airflow resistance (usually from an inflammatory response) bronchospasm, oedema and increased mucus secretions (NIOSH, 2003). Obstructive disorders include asthma, bronchitis, and emphysema although the pathophysiological mechanisms are considered to be different (Johns Hopkins School of Medicine's Interactive Respiratory Physiology, 1995).

Obstructive ventilatory defects are associated with a reduction in FEV_1 in relation to FVC and other measures. This results in a low FEV_1/FVC%. The lower limit of normal is considered to be approximately 70—75% (Pierce & Johns, 1996). Beach (2002) asserts that FEV_1/FVC% values < 50% imply severe obstruction.

According to Pellegrino et al. (2005, p. 953):

> An obstructive ventilatory defect is described as a disproportionate reduction of maximal airflow from the lung in relation to the maximal volume (i.e., VC) that can be displaced from the lung. It implies airway narrowing during exhalation and is defined by a reduced FEV_1/VC ratio below the 5th percentile of the predicted value.

2.11.7 Restrictive ventilatory defect

Johns Hopkins School of Medicine's Interactive Respiratory Physiology (1995, p. 1) describes restrictive disorders as a “pulmonary deficit, such as pulmonary fibrosis

(abnormally stiff, non-compliant lungs)". However, it may also be caused by "respiratory muscle weakness, paralysis, and deformity or rigidity of the chest wall".

Such a restrictive pattern, according to Beach (2002, p. 1012) gives a "proportionate reduction in both FVC and FEV_1 and by reduction of TLC, VC, and other static lung volumes. Whilst Pellegrino et al. (2005, p. 955) defines a restrictive ventilatory defect as:

> characterised by a reduction in total lung capacity (TLC) below the 5th percentile of the predicted value, and a normal FEV_1/VC. The presence of a restrictive ventilatory defect may be suspected when VC is reduced, the FEV_1/VC is increased (85–90%) and the flow-volume curve shows a convex pattern.

Spirometry therefore is considered very useful as a screening test of general respiratory health, to establish as early as possible whether or not there is respiratory impairment.

2.11.8 Assessing early respiratory changes due to occupational exposures

Spirometry has been proven to be particularly useful in assessing early respiratory changes due to occupational exposures. Several researchers have demonstrated decrements in lung function, using spirometry, due to exposure to low-level irritants (Balmes, 2002; Dube, Puruckherr, Byrd, & Roy, 2002; El-Zein, Malo, Infante-Rivard, & Gautrin, 2003) and mixtures of low-level respiratory irritants (Barnhart, 1994; Kremer et al., 1994; Mustajbegovic et al., 2000; Ryon & Rom, 1998; Hendrick et al., 1996; White, 1996; Harber et al., 2007).

Pasker et al. (1997) demonstrated very subtle pulmonary function changes due to exposures to zinc oxide containing fumes. They demonstrated that it was possible to detect a subclinical response. This study, of 57 workers exposed to zinc oxide containing dust and 55 controls, not only used the questionnaire/spirometry approach but also compared the forced oscillation technique (FOT) with spirometry. The study investigated pre-shift and post-shift pulmonary function. The outcome was the observation of a subtle decrease in pulmonary function, observed during night shift with both spirometry and confirmed by FOT. Abramson et al. (2001) also detected an across-shift decline in FEV_1 among a cohort of tea packers. In a study of occupational asthma,

Anees, Moore and Burge (2010) conclude that sufferers of the condition demonstrated a rapid decline in FEV_1 due to exposure; however, after removal from exposure, the effect reverses and there is an improvement (a *step-up*) after which it then continues to decline at a slower rate similar to individuals with normal lung function.

Johns and Pierce (2003, p. 92) state that:

> Spirometry has the capacity to detect airway dysfunction long before symptoms develop, bringing the opportunity to remove harmful exposures before disability develops or becomes severe. These factors render spirometry a critically useful investigation in the detection, management and prevention of respiratory disease.

It has therefore been demonstrated conclusively in multiple studies, among a range of populations and occupational exposure groups that, when used in conjunction with a respiratory questionnaire, spirometry testing provides a powerful diagnostic tool with adequate sensitivity and specificity (Post et al., 1998; Bellia et al., 2003, Hankinson 1986; Utell, Frampton, & Morrow, 1993; Hankinson, Kinsley, & Wagner, 1996; White, 1996; Luo, Hsu, Hsieh, Wong, & Chang, 1998; Sobaszek et al., 1998; Baur & Latza, 2005 Algranti et al., 2000; Musk et al., 2000; Abramson et al., 2001; Kern et al., 2001; Meijer, Grobbee, & Heederik, 2001; Yu et al., 2001; Boojar & Goodarzi , 2002; Mpofu et al., 2002; Murphy , Harrison, & Beach, 2002; Cabrera et al., 2003; Chénard et al., 2007; Doherty, 2008; Forbes et al., 2009).

2.11.9 Reliability, validity, accuracy and precision of the EasyOne spirometer

After determining that spirometry in conjunction with a respiratory questionnaire provides a powerful diagnostic tool with adequate sensitivity and specificity, a review of spirometer's, in particular the EasyOne spirometer, was conducted to ensure its reliability, validity, accuracy and precision. According to Blanc et al. (2009, p. 8) The EasyOne™ spirometer (ndd Medical Technologies, Zurich) "has been recognised for its reliability, accuracy, and durability and has been widely used in epidemiologic research". Evaluations of the stability and accuracy of this specific spirometer have been conducted by Walters, Wood-Baker, Walls and Johns (2006), Coates (n.d.), Australian and New Zealand Horizon Scanning Network (2007), Skloot, Edwards, and

Enright (2010), Gallo, Crapo and Jensen (2009), and Barr et al. (2008), justifying its use in clinical, occupational and research settings.

2.11.10 Alternatives to spirometry

Alternative techniques to determine early effects of workplace exposures on the respiratory system were reviewed prior to the commencement of this study. In general it was found that such newer methods were yet unproven and access to most was restricted to specialised respiratory laboratories due to the need for using specialised equipment or invasive techniques such as lung biopsy, bronchoalveolar lavage, and induced sputum (Balbi et al., 2007).

A new development in the area of respiratory screening is fractional exhaled nitric oxide (FeNO) which functions as a "noninvasive, simple, well-tolerated, and reproducible marker of airway inflammation" (del Giudice et al., 2004, p. 759). Measurement of exhaled nitric oxide (eNO) in a breath test can be conducted to determine airway inflammation particularly asthma (Taylor, Pijnenburg, Smith, & Jongste, 2006). Portable instruments are becoming available making it more practical under field conditions. However, the main limitation of this technique is the need for predicted normal FeNO reference values for normal populations which are yet to be established (Balbi et al., 2007; Taylor, Pijnenburg, Smith and Jongste, 2006).

Biomarkers are potential candidates for further research, though few have been validated. Those showing most promise are sputum neutrophils, IL-8, serum tumour necrosis factor, and C-reactive protein (Tzortzaki, Lambiri, Valchaki & Siafakas, 2007; Kony et al., 2004).

2.12 Summary

This chapter has outlined that air quality can affect lung function and cause respiratory disease (Ostrowski & Barud, 2006). It presented a critical review of existing literature regarding the aetiology and impact of occupational lung disease, the possible effects of low levels of mixed exposures, the confounding factors, and the health surveillance techniques to enable detection of such effects.

The impact of respiratory disease is a major problem worldwide, and the total lifetime cost considerable, but it can be avoided (Takahashi et al., 1998). Several

countries have respiratory disease surveillance systems and some specifically for occupational respiratory disease. The surveillance system in Australia is based primarily on workers compensation claims (Safe Work Australia, 2011c). Hence there is a long latency period and these data reflect historical workplace exposures and not emerging new issues. Safe Work Australia (2011c) has targeted occupational respiratory diseases as one of eight identified occupational diseases for priority action. Prior to this the senate inquiry (Parliament of Australia Senate Committee, 2006) into workplace harm related to toxic dust and emerging technologies highlighted the need for robust surveillance systems and early accurate diagnosis of loss of lung function. During the inquiry process the Australian and New Zealand Society of Respiratory Science recommended regular lung function testing to detect early signs of respiratory symptoms. Therefore, a top-down, bottom-up approach, employing an Australian occupational respiratory health surveillance system to define the problem of work-related respiratory disease so that specific workplace hazards can be targeted, plus a risk-based approach at the local workplace level to identify, evaluate and control respiratory hazards, is advocated.

This review determined that pulmonary function tests, providing they are performed rigorously to the ATS/ERS criteria (Miller et al., 2005) detect airway dysfunction long before symptoms develop (Johns & Pierce, 2003). Several researchers have been able to detect such early respiratory changes. These studies have shown that pulmonary function tests used in conjunction with respiratory questionnaires, conducted by competent practitioners using reliable and accurate equipment, provide valid data. Thus such an approach was adopted with the aim of determining whether exposure to a specific spectrum of gases, aerosols and particulates present in each work area at the Murrin Murrin Operation presented a respiratory health risk.

The main gaps in this research area appear to be that the extent to which occupational respiratory disease contributes to work-related illness in Australia remains effectively unknown. The extent to which occupational factors such as dust, gases, vapours, smoke and fumes that contribute to respiratory disease also remains unknown. Very little is known about mixed exposures, particularly in the mining industry, and research is also needed into early detection of respiratory diseases in order to prevent them.

This literature review, along with an overview of the Murrin Murrin mine and processing plant processes (Chapter 3) plus the review of the respiratory health effects presented by the hazardous substances associated with these processes (Chapter 4) helped formulate the research methodology. This culminated in a longitudinal study of the Occupational Respiratory Health Surveillance at Minara Resources, Murrin Murrin Mine Site

3. OVERVIEW OF THE MURRIN MURRIN OPERATION PROCESS

3.1 Introduction

The Minara Resources' Murrin Murrin Operation (Figure 3.1) is situated approximately 60 kilometres equidistant from Laverton and Leonora, on the edge of the Gibson Desert in Western Australia, in the north east Goldfields region (Figure 3.2). The Murrin Murrin Operation mines lateritic ore in the locality, which is refined in to nickel and cobalt at the processing plant.

Figure 3.1 Murrin Murrin Operation

(Source: Minara Resources, 2004a)

The project, commissioned in May 1999 by Anaconda Nickel Ltd, is a joint venture between Murrin Murrin Holdings Pty Ltd, a wholly owned subsidiary of Minara Resources Ltd (60%) and Glenmurrin Pty Ltd, a wholly owned subsidiary of Glencore International AG (40%) (SPG Media Limited, 2007; Mining-Technology.Com., n.d.).

The source of the laterite mineralisation, with its concentration of nickel and cobalt in silicate minerals, was formed as a consequence of extensive weathering of olivine-rich ultramafic rocks (Gaudin, Decarreau, Noack, & Grauby, 2005; Dalvi, Bacon, & Osborne, 2004). The laterite ore, with its soft clay-like nature, lies close to the ground surface and is therefore relatively easily mined using open cut mining techniques.

The ore is processed at the Murrin Murrin processing plant based on the Sherritt International high pressure acid leach technology (Figure 3.3) (Taylor, 2000; Wellesley-Wood, 2002). In brief, the Murrin Murrin process prepares the ore prior to subjecting it to sulphuric acid in high-temperature, high-pressure autoclave vessels to extract nickel and cobalt from lateritic ores (Ozberka, Jankolab, Vecchiarellic, & Krysad, 1995; Mining-Technology.Com., n.d.). Following this, the solid wastes are thickened and neutralised, and the liquid containing nickel and cobalt is separated by counter current decantation (CCD). The nickel and cobalt are precipitated out as mixed sulphides, and eventually refined using solvent extraction and a range of chemical reagents.

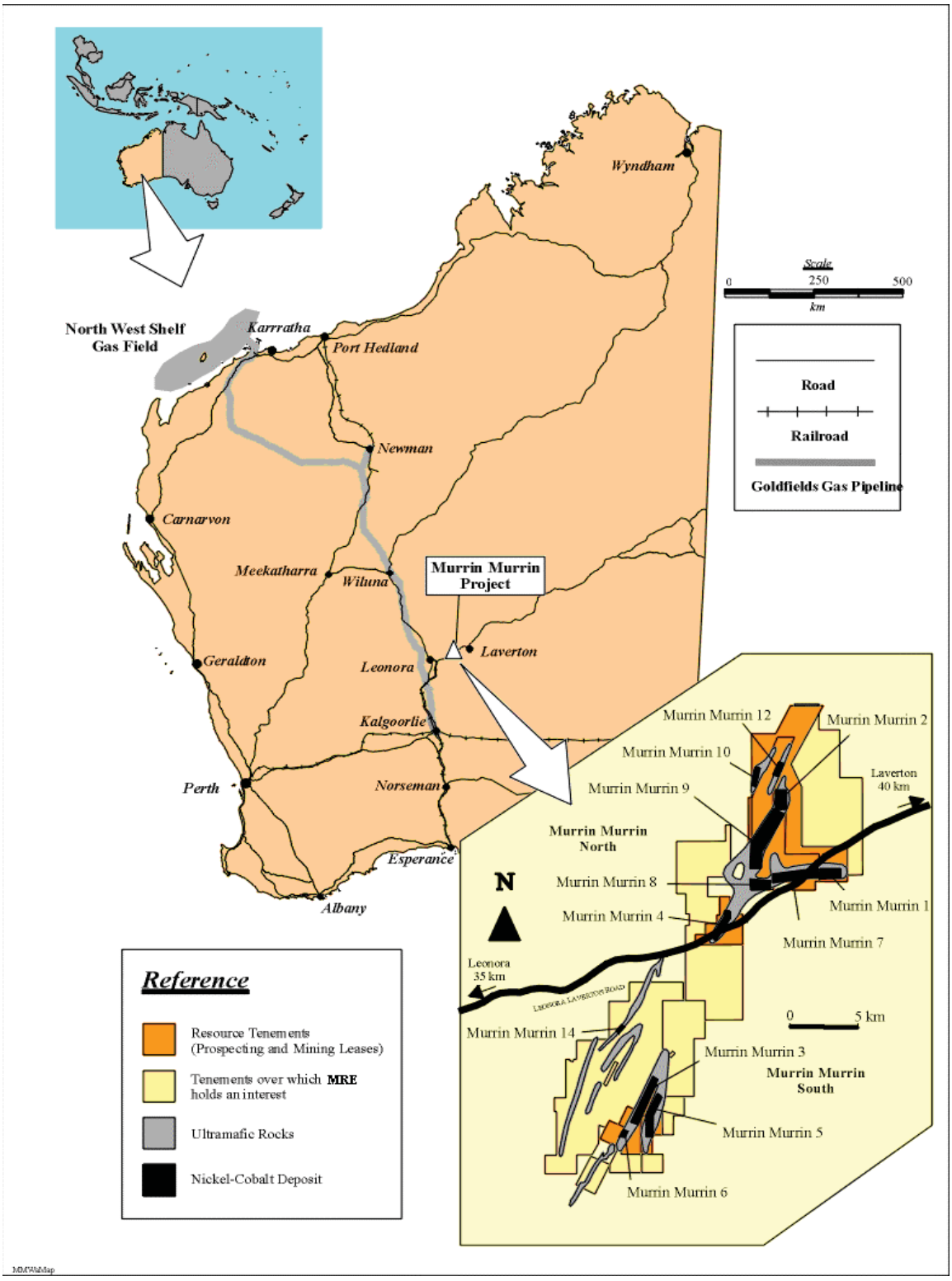

Figure 3.2 Location of the Murrin Murrin Operation

(Source: Minara Resources, 2004a)

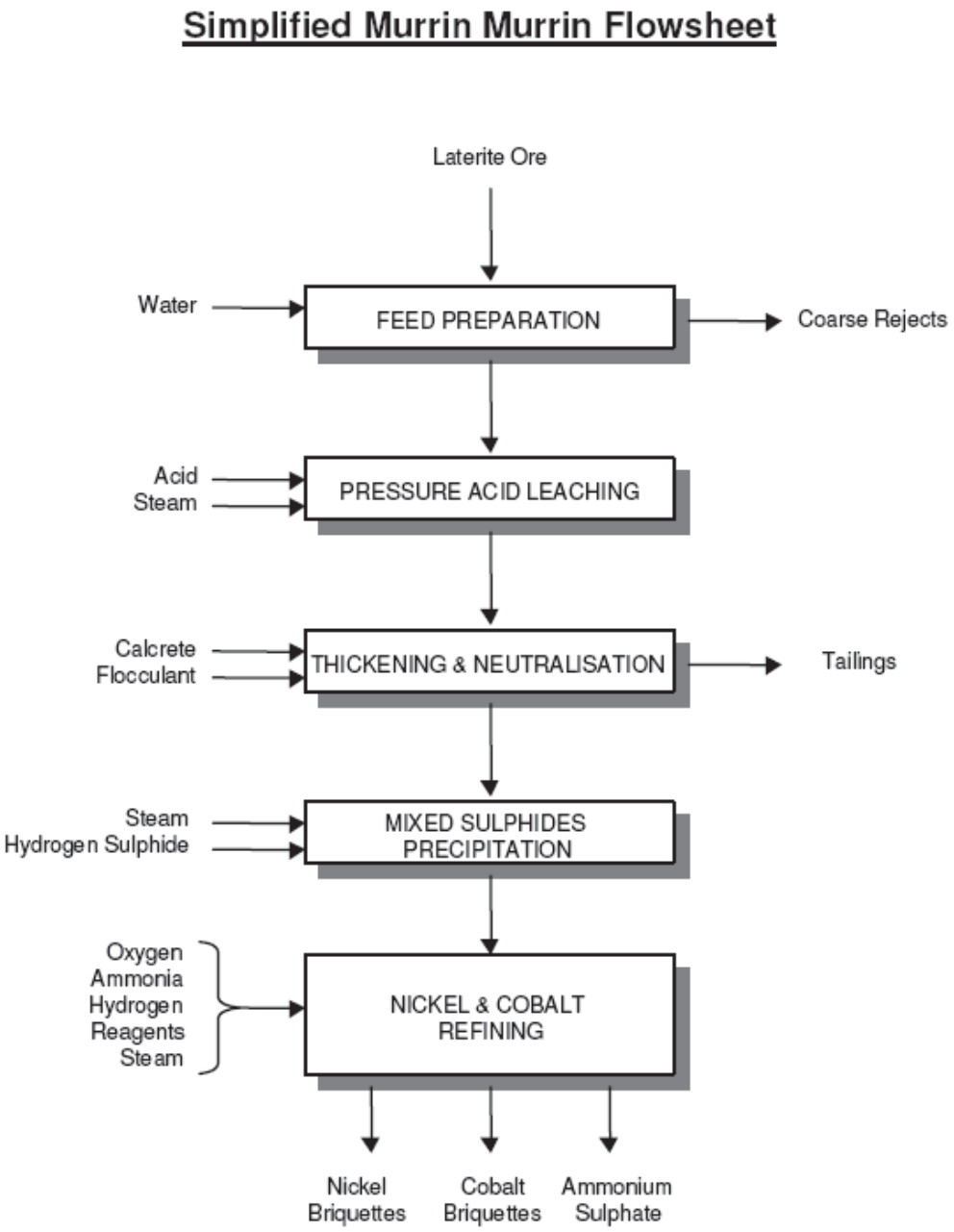

Figure 3.3 Murrin Murrin Process Flowsheet

(Source: Taylor, 2000)

3.2 Murrin Murrin Technology

3.2.1 Mining

The soft lateritic ore is mined by the open pit method as they are near-surface deposits. The pits are shallow because there is usually only an overburden of approximately 10 metres overlying the ore body of about 20 metres in depth. The areas of the ore body which have nickel-cobalt mineralisation occur in horizontal zones, which allows for bulk mining methods to be employed. Excavators are used to extract the ore which is transported by haul trucks to the Run of Mine (ROM) ore stockpile, ready to be processed. The simplified geology showing the horizontal zones of the ore body are pictured below.

- **Ferruginous Zone (FZ):** The top laterite unit contains mainly iron oxides as waste. A small amount of Ni/Co mineralisation appears at the bottom of this zone.
- **Smectite Zone (SM):** The middle laterite unit contains mainly smectite clays and medium to high grade Ni/Co mineralisation throughout the zone.
- **Saprolite Zone (SAP):** The deepest laterite unit contains mainly primary smectite clays and basic minerals referred to as serpentine minerals. The grade of Ni/Co mineralisation varies significantly in the saprolite zone.

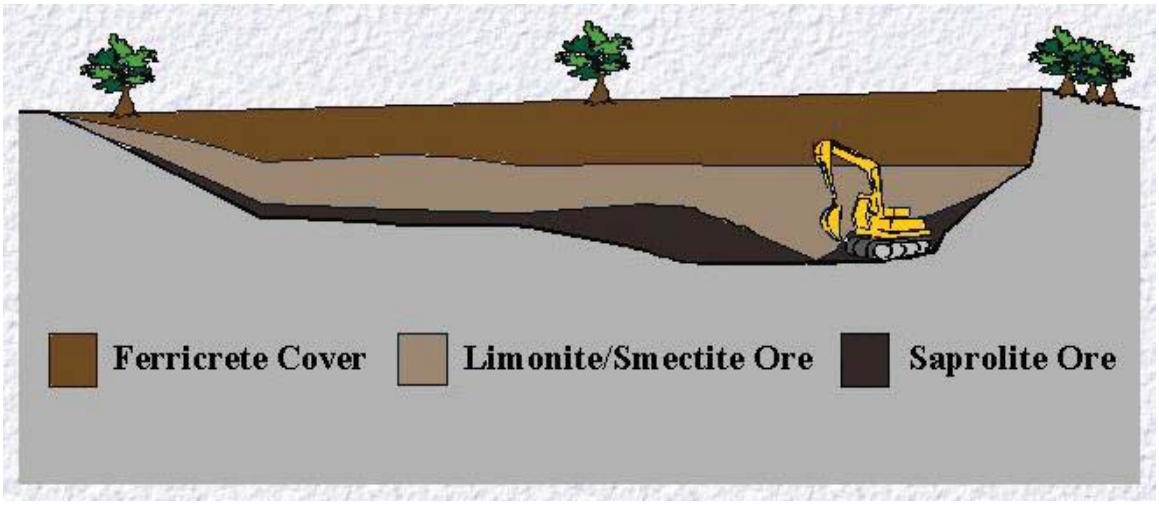

Figure 3.4 Simplified Geology

(Source: Minara Resources, 2004a)

The three ore types, shown in Figure 3.4, are mined separately and blended to achieve the optimum nickel concentration as an ore feed from the ROM stockpiles.

The activities required to mine the ore include:

- overburden removal by excavators and trucks;
- some blasting of occasional areas of cap rock;
- ore extraction by excavators and trucks.

The mining operations include the removal of ore from the ground and its transportation to the ROM pad. The ROM pad is the first point of contact with the processing plant.

3.3 Processing Plant

3.3.1 Ore leach

The purpose of the ore preparation facility is to crush the ore to a specified size and add water to produce a slurry feed at the required density prior to feeding it into the acid leach circuit. To achieve this, ore is blended from finger piles on the ROM pad by front-end loader. The ore is then loaded into a low-capacity ore bin which feeds a sizer (similar to a crusher). The MMD Group of Companies' sizer is the first point of size reduction and breaks up any large lumps of ore, crushing the ore to less than 150mm.

3.3.1.1 Slurrying mill

The sizer product is conveyed to the slurrying or semi-autogenous grinding (SAG) mill (Figure 3.5). The ore is prepared in the slurrying mill where it is ground to produce a slurry feed of the correct particle size and density to feed the pressure acid leach circuit, with a final slurry density of between 39% and 42% solids by weight, adjusted by adding water.

As the slurry flows from the mill onto a vibrating screen the larger particles are caught and then removed by conveyor to a low-grade stockpile. The slurry filtered through the screen (approximately 1.7% nickel and 0.07% cobalt) is pumped to a slurry storage tank which is eventually pumped into the acid leach section.

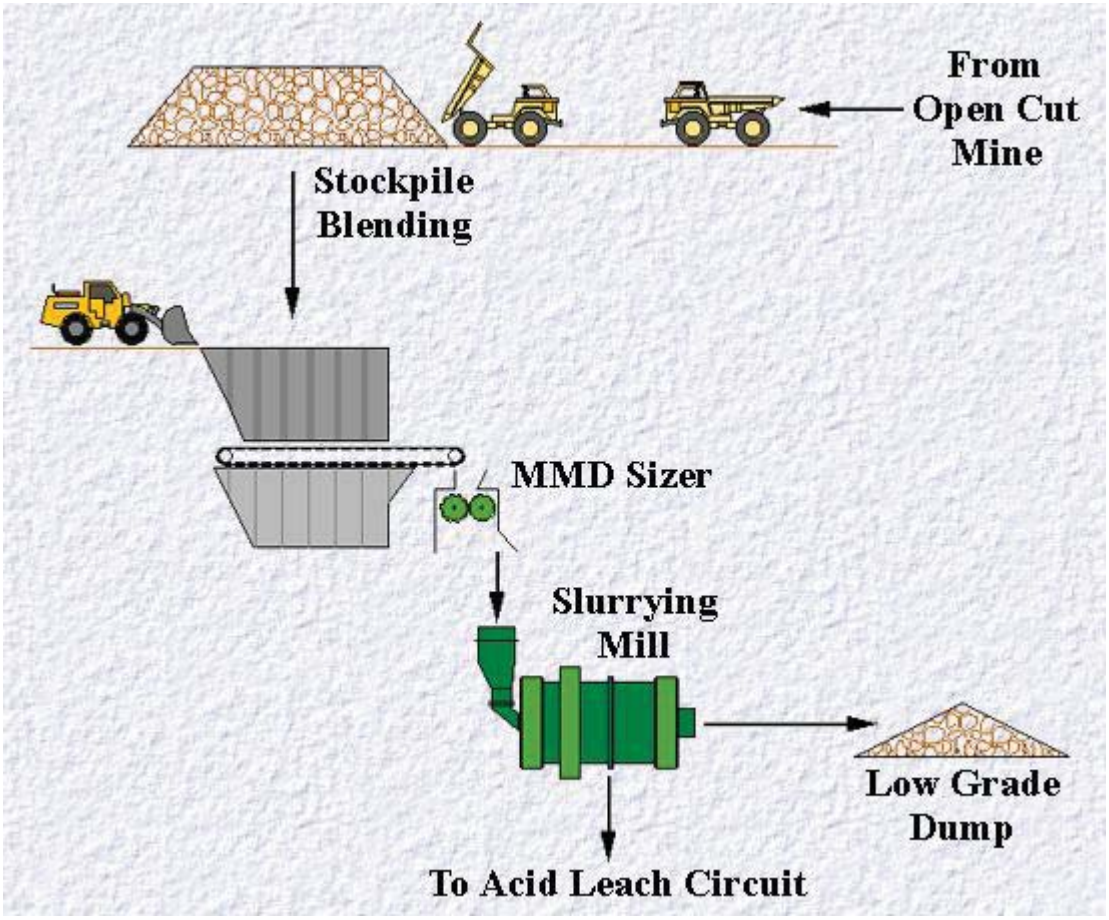

Figure 3.5 Ore from Stockpile to the Slurry Mill

(Source: Minara Resources, 2004a)

3.3.1.2 High pressure acid leaching

The high pressure acid leach circuit is shown in Figure 3.6. The high pressure leaching is carried out in titanium-lined autoclaves in which the ore slurry is mixed with concentrated sulphuric acid. There are four autoclaves which operate at 255°C and 42 bar pressure (4200KPa) to dissolve out the nickel (Ni) and cobalt (Co) as soluble sulphate salts.

The sulphuric acid for the leaching process is generated by the acid plant located close by on the site. The leaching reaction extracts nearly all of the nickel and cobalt into the solution (liquid) phase with the solids as residue. The nickel and cobalt is released from the ore leaving residual waste material, which is returned to the slurry ore preparation before ultimately being pumped out, after being neutralised, into the tailings dam.

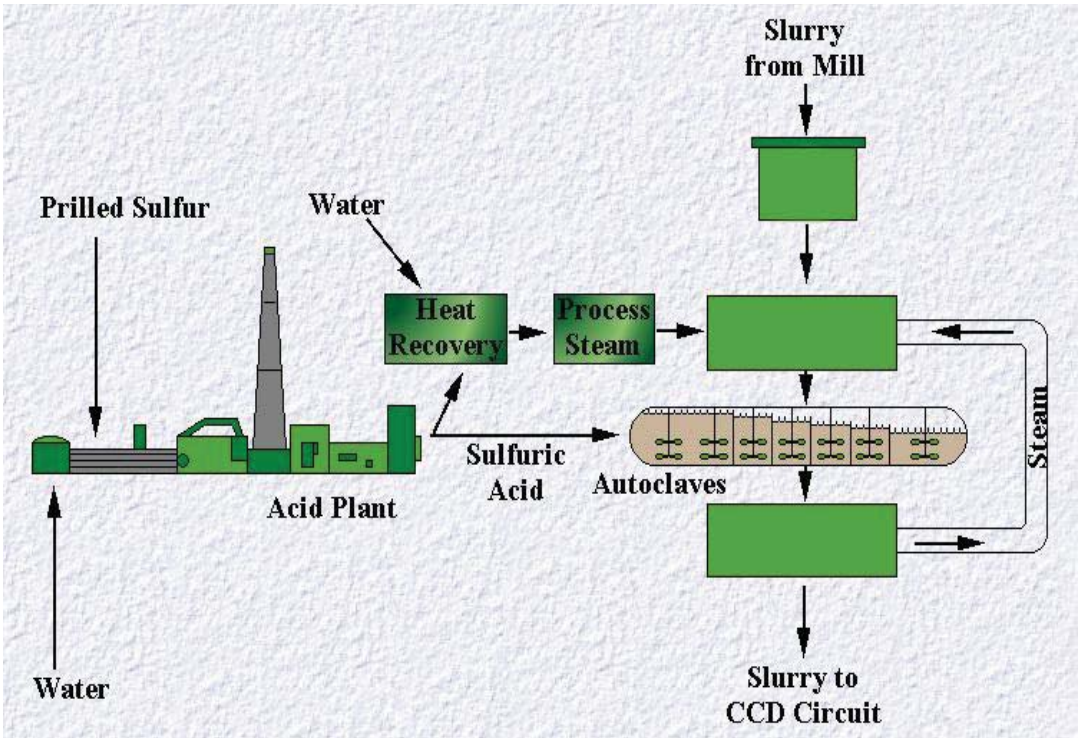

Figure 3.6 High Pressure Acid Leach Circuit

(Source: Minara Resources, 2004a)

3.3.1.3 Counter current decantation (CCD) circuit

The slurry from the pressure acid leach circuit is fed into the CCD circuit. The CCD circuit has seven 50-metre thickeners where the slurry is separated into two streams, the residue solids, and soluble nickel and cobalt solution.

The CCD and solution neutralisation circuits are outlined in the simplified flow diagram, (Figure 3.7).

Each CCD thickener mixes, washes and separates the solid residue waste from the sought-after nickel and cobalt solution. The residue solids from the last thickener are pumped to the tailings neutralisation circuit prior to disposal into a tailings dam, whilst the nickel and cobalt sulphate solution from the first wash thickener gravitates to the solution neutralisation circuit.

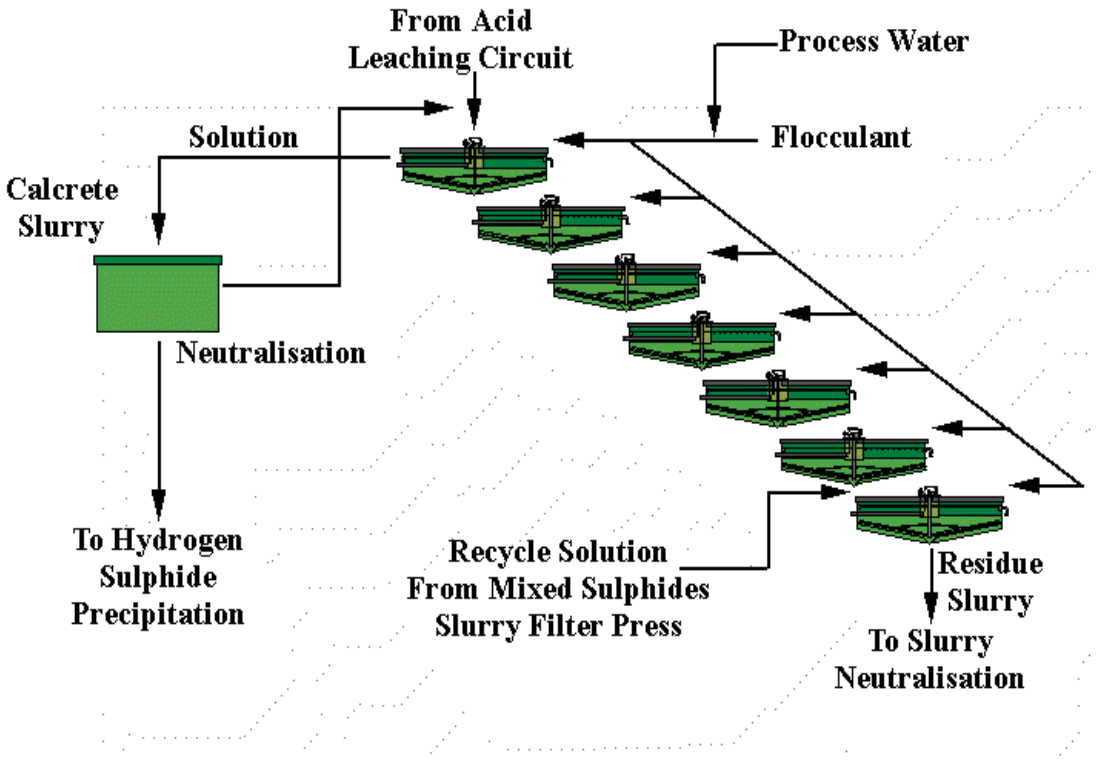

Figure 3.7 Counter Current Decantation (CCD) Circuit

(Source: Minara Resources, 2004a)

3.3.1.4 Solution neutralisation circuit

The pressure leach solution (which contains dissolved nickel and cobalt sulphates) passes into the neutralisation circuit to neutralise the pH which is less than pH 1.0. The solution has to be neutralised prior to the recovery of nickel and cobalt in the refinery because of its highly corrosive nature. Calcrete (a limestone-like mineral mined locally) is added to neutralise the acid. The solution then enters the mixed sulphides precipitation circuit, where hydrogen sulphide gas is introduced which converts the solution into a mixed nickel/cobalt sulphide.

Initially, hydrogen sulphide is mixed with the nickel/cobalt solution from the first wash thickener, to change the iron chemistry from ferric to ferrous iron which interferes with the process, in order to maximise the nickel and cobalt extraction in the following neutralisation stage.

As it flows through a series of four agitated tanks, the free sulphuric acid is neutralised by the addition of calcrete slurry, which forms a gypsum precipitate. The leachate is separated from the gypsum precipitate, filtered, and directed to the sulphide

precipitation area for further treatment. The thickened solids underflow from the thickener is returned to the CCD circuit to extract the residual nickel and cobalt liquor. Eventually, the gypsum is removed, along with the solid residue, for disposal into the tailings dam.

3.3.1.5 Mixed sulphide precipitation and slurry neutralisation circuits

Figure 3.8 below represents diagrammatically the mixed sulphide precipitation and tailings slurry neutralisation circuits.

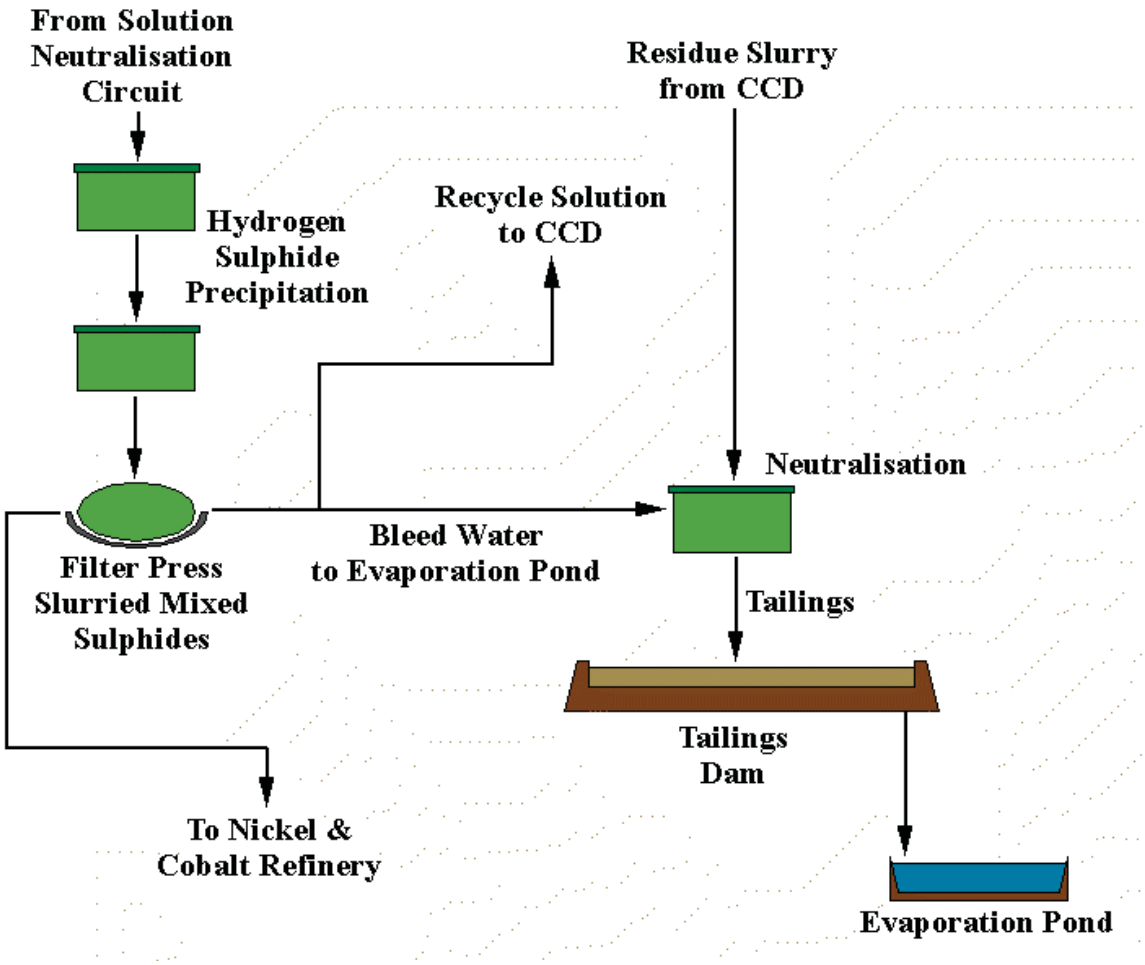

Figure 3.8 Mixed Sulphide Precipitation and Slurry Neutralisation Circuits

(Source: Minara Resources, 2004a)

3.3.1.6 Mixed sulphide precipitation

The aim of the mixed sulphide precipitation circuit is to transform the nickel and cobalt sulphate solution into solid mixed sulphides. In order to precipitate out the mixed sulphides, hydrogen sulphide is introduced into the neutralised solution in four agitated tank stages. The hydrogen sulphide reacts with the nickel and cobalt sulphate in solution and forms a mixed (nickel and cobalt) sulphide solid precipitate. The precipitation reaction also removes unwanted manganese and iron.

The solid precipitate is washed and then it thickens as it settles out of the solution. The washed precipitate, still a slurry, is next filtered and then stored before being refined on site or sold directly on the open market.

The wash (barren solution) is pumped to either:

- the last wash thickener in the CCD circuit (approximately 40% of flow); or
- an evaporation pond (approximately 60% of flow).

3.3.1.7 Slurry neutralisation circuit

The pH of the waste solids in the underflow from the CCD circuit is neutralised using calcrete slurry introduced into agitated tanks in the slurry neutralisation circuit. The solids are disposed of in the tailings dam, and the liquid flows to the evaporation pond.

3.3.2 Refinery

In the refinery, the mixed sulphides are refined to produce high-grade nickel and cobalt powder which is then bonded into briquettes ready for purchase. This process is carried out in numerous steps which are briefly outlined below and outlined in the simplified flow diagram, Figure 3.9.

3.3.2.1 Mixed sulphide leaching

The mixed sulphide precipitate is taken from storage or directly from ore leach to the refinery. Here the mixed sulphides are firstly washed and slurried with water to undergo pressure oxygen leach in a stainless steel autoclave, to manufacture a 99.5% nickel and cobalt solution.

3.3.2.2 Iron/copper removal

Unwanted iron is removed after leaching by adding ammonia to the nickel/cobalt. Zinc sulphide is also added to remove unwanted copper. Inevitably, during this process a small amount (+9%) of nickel is removed in the precipitate along with the iron. The solution is therefore thickened and filtered to remove the iron *cake*, and the residual nickel in solution is returned back into the process through the ore leach feed.

3.3.2.3 Zinc/copper removal

The nickel solution, now free of iron, is reacted with anhydrous ammonia and hydrogen sulphide which removes residual copper and zinc. Again the copper and zinc form a precipitate, which is thickened and filtered out of solution as copper/zinc cake waste.

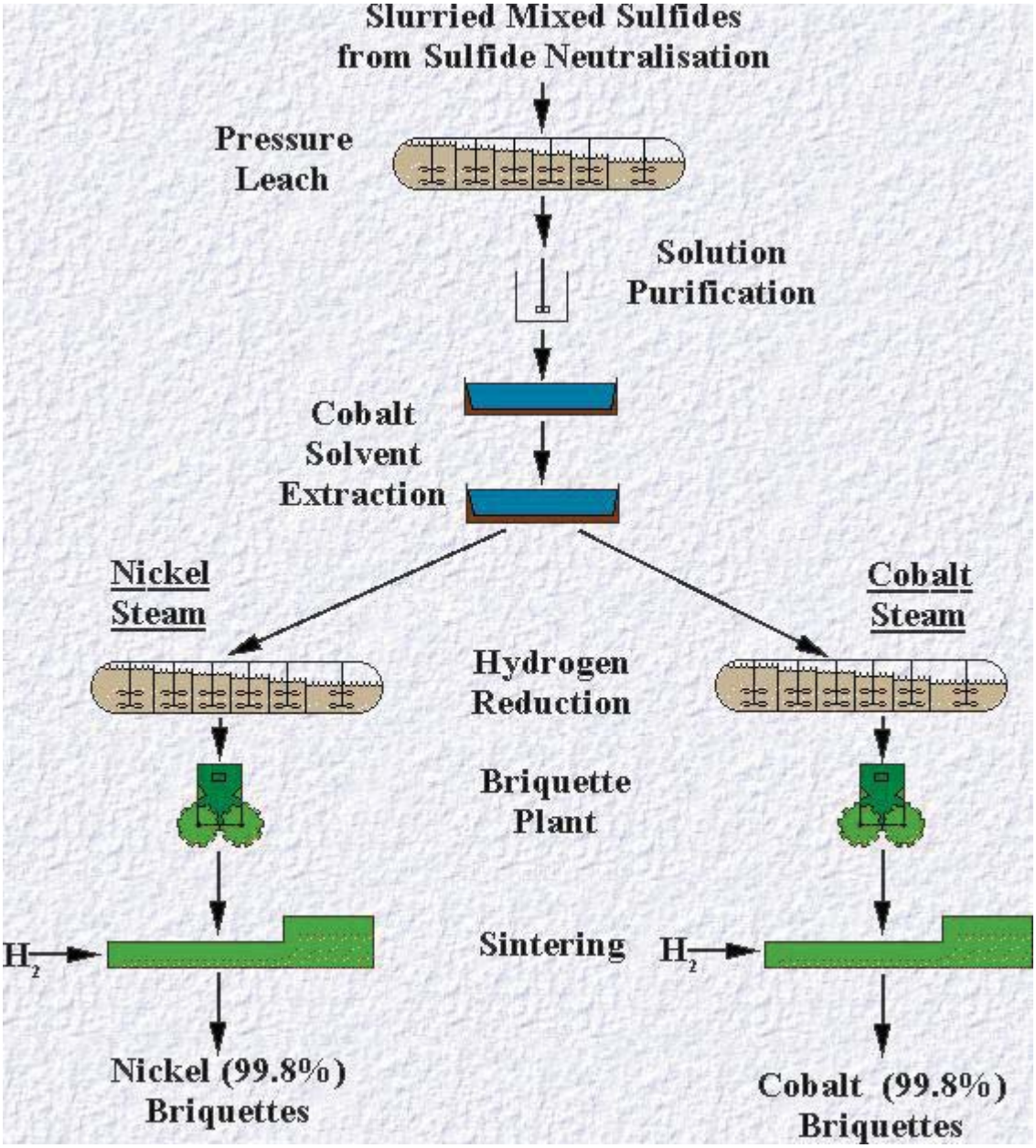

Figure 3.9 Refinery: Simplified Flow Diagram

(Source: Minara Resources, 2004a)

3.3.2.4 Solvent extraction

Cobalt is removed from the Ni/Co solution by using Cyanex 272, a solvent extractant. For this process, a large volume of solution is mixed with a smaller volume of organic extractant which separates out the cobalt in the organic phase and the solvent becomes loaded with cobalt. Cobalt is then removed from the solvent with sulphuric acid and the barren solvent is recycled through the cobalt hydrogen reduction circuit.

Once the cobalt has been removed from the Ni/Co solution and separated from the nickel, it is then concentrated into high-grade cobalt solution (*raffinate*) and pumped to the cobalt hydrogen reduction circuit.

3.3.2.5 Hydrogen reduction of nickel and cobalt solutions

The nickel raffinate and cobalt raffinate are now separated and processed separately by autoclave in two streams. Anhydrous ammonia and ammonium sulphate is added to both the high-strength pure cobalt and nickel solutions prior to hydrogen reduction. The nickel ammonium sulphate liquor is preheated prior to entering the nickel hydrogen reduction autoclaves. Hydrogen gas is introduced under pressure and the nickel is precipitated as a powder. The powder is recovered from the bulk liquor via a flash tank, pan filter and dryer. The cobalt raffinate is similarly processed and precipitated as a powder.

3.3.2.6 Briquetting and sintering

Next, the powdered nickel and cobalt from each reduction autoclave circuit is compressed into pillow-shaped lumps (briquettes) which are then sintered in a furnace at approximately 1120 degrees C prior to it being transported and sold to smelters around the world. There are separate briquetting and sintering furnaces for nickel and cobalt products.

3.3.3 Utilities

The Utilities Department or Utility Areas provide services such as electricity, gas, water, and chemicals used in ore leach and the refinery. These services are required to process and treat the crude ore and refine it into pure nickel and cobalt metal products. These consumable products, which assist the processing areas to extract the nickel and cobalt, are provided by a number of dedicated utility plants.

The plant layout of all facilities at the Murrin Murrin Operation is illustrated in the diagram (Figure 3.10). It shows the relationship between the processing and utility areas. A brief description of each of the utility areas is provided below.

3.3.3.1 Sulphuric acid plant

The Murrin Murrin sulphuric acid plant is one of the biggest in the world with 4400 M tons per day acid production. The acid is used in the HPAL processing circuit to leach the nickel and cobalt out of the ore in the ore leach area. The acid plant also provides most of the heat required to drive the power and steam generation plant.

3.3.3.2 Power and steam generation plant

Power is essential for every area of the plant, and steam is used in the ore leach and refining processes.

To meet the site's energy requirements there are 3 x 55 tonnes per hour steam boilers. The steam is used to drive 2 x 28-megawatt steam turbines. Additional power may also be generated by the 20-megawatt gas turbine, and there are six standby diesel generators and associated mechanical and electrical equipment. Energy is also recycled from the ore leach autoclave flash system to preheat feed slurry into the HPAL circuit.

3.3.3.3 Water supply and water treatment plant

The water supply, critical to the Murrin Murrin processing plant, is sourced from the borefields some 50 km away. Approximately one third of the raw water is treated by reverse osmosis in the water treatment plant to produce high-quality demineralised and potable water for use in the sensitive processing stages and for human consumption respectively. The remaining two thirds are used directly as process water.

3.3.3.4 Natural gas supply

Natural gas supplied by the Goldfields Gas Transmission Pipeline is used in many areas of the plant for various burners and as a heating source in the hydrogen sulphide plant. Natural gas is also the source or primary feed for the hydrogen plant.

3.3.3.5 Hydrogen plant

Hydrogen is produced in the hydrogen plant on site by reacting natural gas with steam. Hydrogen is used in the nickel and cobalt autoclave circuits (furnaces) to reduce the powdered nickel and cobalt into briquettes and to sinter them, and for the production of hydrogen sulphide gas produced in the hydrogen sulphide plant.

3.3.3.6 Hydrogen sulphide plant

Hydrogen sulphide (H_2S) is used in the process prior to and in the refinery, to produce a mixed nickel and cobalt solid sulphide product. H_2S is produced in the hydrogen sulphide plant by reacting hydrogen and sulphur together. The sulphur is pumped from the sulphur pit located in the sulphuric acid plant.

3.3.3.7 Sulphur stockpile

Sulphur is trucked in from external suppliers and stored as a large stockpile close to the acid plant and hydrogen sulphide plant. Approximately 500,000 tonnes/year of sulphur is used to produce sulphuric acid in the acid plant and hydrogen sulphide gas in the hydrogen sulphide plant.

3.3.3.8 Oxygen/nitrogen (air separation) plant

The air separation plant produces oxygen and nitrogen from the air of the local atmosphere. The plant uses a double column distillation/ pressure swing absorption system to produce oxygen and nitrogen which are used in various stages of the nickel/cobalt processing.

Nitrogen is used predominantly for the pressure acid leach autoclaves and for purging in the hydrogen and hydrogen sulphide plants, whilst oxygen is used mainly in the refinery area for the mixed sulphide pressure leach.

3.3.3.9 Air supply

Instrument air supply for the entire plant is provided from the air separation plant. Air compressors are installed in most areas of the operation to provide high and low pressure plant and dried instrument air.

3.3.3.10 Ammonia supply and storage

Ammonia is primarily utilised in the refinery in the hydrogen reduction areas. Ammonia is transported to the site and stored in pressurised bullet storage tanks to be distributed to the processing plant.

3.3.3.11 Ammonium sulphate

Ammonium sulphate is used as a catalyst for the refining of nickel and cobalt in the hydrogen reduction process. The excess of ammonium sulphate from the process is reclaimed in a three-stage evaporation process and sold predominantly for fertiliser.

3.3.4 Calcrete plant

Calcrete (similar to limestone) is a strong alkaline substance used in the solution neutralisation circuit to neutralise the acidic HPAL product prior to introducing it into the refinery.

Calcrete is mined locally at the calcrete pit approximately 50 km from the processing plant. This is transported to the calcrete plant close to the processing plant where it is made into fine slurry for use in the neutralisation circuit.

3.3.5 Final products

The following products are produced by the Murrin Murrin processing plant:

- nickel;
- cobalt;
- ammonium sulphate.

3.3.6 Packaging and transport of products

The final nickel product is packed in two tonne *bulka bags* or metal drums according to customer requirements, whilst cobalt is packaged in metal drums. These products are transported by road to the local port to be shipped to customers worldwide. Ammonium sulphate fertiliser is trucked to the close-by Malcolm Siding rail head for transportation to be sold locally.

In 2003/04, the Murrin Murrin Operation mined and processed 2.8 million tonnes per annum of laterite ore to produce about 27,950 tonnes per annum of nickel and 1,982 tonnes per annum of cobalt briquettes (Minara, 2004b). In 2005, 28,240 tonnes of nickel and 1,750 tonnes of cobalt were produced (Minara, 2005) and in 2006 approximately 32,000 tonnes of nickel were produced (Johnson, 2007).

3.3.7 The Processing plant layout at the Murrin Murrin Operation

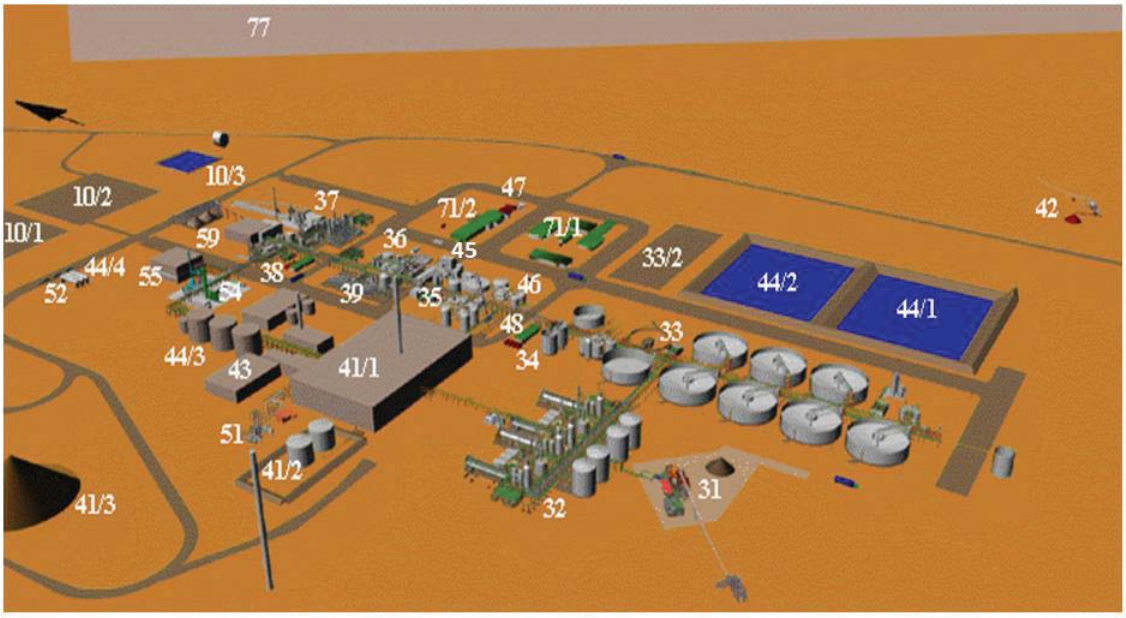

Figure 3.10 Processing Plant Layout

(Source: Minara Resources, 2004a)

The area names for each of the above area numbers are outlined in Table 3.1.

Table 3.1 Processing Plant Areas

10/1	Earthworks Contractors Area (during construction phase)	**43**	Power Plant
10/2	Temporary Batch Plant (during construction phase)	**44/1**	Raw Water Dam
10/3	Construction Water Dam (during construction phase)	**44/2**	Process Water Dam

31	Slurrying (SAG) Mill - Ore Preparation	**44/3**	Water Treatment Plant
32	High Pressure Acid Leaching	**44/4**	Caustic Storage
33	CCD Containment	**45**	Natural Gas Supply
33/2	CCD Containment Pond	**46**	Air Supply System
34	Neutralisation and Thickening	**47**	Fuel Farm
35	Ni/Co Precipitation and Barren Liquor Neutralisation	**48**	Main Pipe Racks
36	Sulphide Grinding, Dissolution and Impurity Removal	**51**	Hydrogen Sulphide Plant
37	Hydrogen Reduction	**52**	Ammonia Storage
38	Cobalt Reduction	**54**	Hydrogen Plant
39	Solvent Extraction	**55**	Oxygen Plant
41/1	Acid Plant	**59**	Ammonium Sulphate Plant
41/2	Acid Storage	**71/1**	Administration Office Area
41/3	Sulphur Storage	**71/2**	Workshop/Warehouse Area
42	Calcrete Plant	**77**	Tailings Dam

(Source: Minara Resources, 2004a)

Colloquially the areas are referred to by the area numbers above with a suffix of two further zeros. For example:

- ore leach – 3100, 3200, 3300, 3400 (thirty-one hundred, thirty-two hundred, etc.);
- refinery – 3500, 3600, 3700, 3800, 3900;

- utilities – 4100, 4300, 4400;
- BOC plant (air supply system) – 4600;
- calcrete plant – 4200.

3.4 Conclusion

The overview of the Murrin Murrin Operation process provides an indication of the chemical hazards that workers potentially face on a daily basis. The area names and numbers provide an indication of the processes occurring in that area and the chemical hazards associated with the tasks conducted in those work areas. The overview of the Murrin Murrin Operation process formed part of the exposure assessment strategy from which the health effects presented by the hazardous substances associated with the mining and process plant were determined. This is the subject of the next chapter.

4. THE RESPIRATORY HEALTH EFFECTS PRESENTED BY THE HAZARDOUS SUBSTANCES ASSOCIATED WITH THE MINING AND PROCESS PLANT

4.1 Introduction

In the precursor occupational hygiene studies prior to this study at the Murrin Murrin Operation a number of respiratory hazards associated with the complex chemical extraction process of nickel and cobalt were identified and the following exposure groups were identified as areas of concern:

- ore leach, area 3100, operators – inhalable dust, cleaning under the sizer;
- refinery, area 3700, furnace operators – nickel dust;
- refinery, area 3700, packaging operators – cobalt dust;
- refinery, area 3800, operators – cobalt dust.

(Oosthuizen & Cross, 2004; Wing, 2005; Wing & Oosthuizen, 2007).

Workers in these areas were required to wear P2 respiratory protection that complied with AS 1716 and was used in accordance with AS1715 as the work areas had been previously demarcated as potentially hazardous.

Wing (2005, p. 120) discussed the potential for exposure to sulphuric acid mist (from the acid plant in area 4100) "if operators were working near a leak in the process". Similarly, there is the potential for exposure to sulphuric acid mist at start-up of the acid plant especially during a cold start-up. These exposure scenarios are avoidable as they are "visible to the naked eye, so the identification of leaks prior to an exposure is possible" (Wing, 2005).

Wing (2005) also identified other possible exposures, mainly associated with problems with the process, including process failures or abnormal operating conditions. The areas highlighted included:

- hydrogen sulphide in utilities, Areas 4100/5100;
- ore leach, Areas 3400 and 3510;
- inhalable dust, hosing out the sizer tunnel – in ore leach, area 3100;

- nickel and cobalt dust – “working with dry production materials” – in the refinery, area 3500;
- “Exposure to high short term concentrations of Ammonia” – refinery, area 5900.

Wing (2005) did not include the mining operations and calcrete pit in the scope of his work.

Additional historical data from the Occupational Hygiene Database (Minara Resources, 2005b) was accessed to identify worker exposures that may have occurred prior to the study conducted by Wing. Most of these exposures were one-off incidents, usually under abnormal working conditions, and, as far as can be established, appropriate personal protective equipment was worn in each situation. These included:

- calcrete dust exposure in the calcrete pit and plant;
- intersection of chrysotile and anthophyllite asbestos in mining ore bodies and during exploration drilling;
- respirable dust levels for the mining blast crew and drillers;
- respirable dust levels during preparation of ore samples in the laboratory;
- grinding and welding in confined space;
- ammonia in the refinery sampling and analysis huts;
- reclaiming nickel powder/waste when loading nickel bags into hopper of the screening unit in the refinery;
- loading nickel powder into bins/hoppers in the refinery;
- welding fume during maintenance activities.

At the time when these data were collected it was noted that the majority of the dirty work was being done by contractors, particularly during scheduled shutdowns. These transient workers were not included in routine occupational hygiene monitoring and their exposure profiles could not be established, particularly since they worked at multiple mine sites. Regular contractors based on site were included in this study, these included calcrete workers, and maintenance workers employed in the central works and BIMS areas.

4.2 The Specific Exposures Present in Each Work Area at the Murrin Murrin Operation

The specific exposures present in each work area at the Murrin Murrin Operation were determined to be as follows:

4.2.1 Mining

- mineral dust;
- general (red dirt) dust;
- fibrous material (chrysotile and anthophyllite);
- silica (from blast and drilling);
- diesel emissions.

4.2.2 Ore leach

- mineral dust;
- sulphur dioxide/trioxide and sulphuric acid mist;
- caustic soda;
- hydrogen sulphide;
- general (red dirt) dust.

4.2.3 Refinery

- hydrogen sulphide;
- hydrogen peroxide mist;
- ammonia;
- nickel dust;
- cobalt dust;
- mixed sulphides (nickel/cobalt sulphides);
- organic solvent;
- ammonium sulphate;
- general (red dirt) dust.

4.2.4 Utilities

- sulphur dioxide/trioxide and sulphuric acid mist;
- sulphur dust;

- hydrogen sulphide;
- diatomaceous earth;
- lime dust;
- caustic soda dust.

4.2.5 Calcrete plant and pit

- calcrete dust;
- diesel emissions.

(Oosthuizen & Cross, 2004).

The predominant route of exposure across the mining and processing operation was considered to be inhalation, and the primary target organ the lung. This was determined from the toxicological profile of each health hazard. A summary of the respiratory health issues presented by each of these hazardous substances individually (a single chemical) and the issues surrounding the toxicity of a combination of chemical agents (chemical mixtures) is addressed below.

4.3 Respiratory Health Issues Presented by Each Hazardous Substance

4.3.1 The inflammatory response

Insult due to any toxic agent will initiate an inflammatory response (Rote, 1998). The extent of this response is dependent on the concentration, duration and frequency of exposure of the toxic agent (Eaton & Gilbert, 2008). There is a continuum of effects: the inflammatory response may resolve if the insult ceases, repair may occur with minimal pathophysiological results (Teder et al., 2002) or it may result in permanent damage, chronic injury, and possibly death.

4.3.2 Toxic effects of dusts and particulates

4.3.2.1 Dusts and particulates

Dust particle size is the major factor that dictates where particulates will be deposited within the respiratory tract, and the area of deposition is a factor that influences the eventual pathophysiological effect. The aerodynamic equivalent diameter (AED) is used to predict where the particle will be deposited in the respiratory system (Kelly, 2002).

Larger particles (greater than 10 μm) are generally deposited in the nares, particles in the region of 10-5 μm are deposited in the large conducting airways, whilst those between 0.5 and 5.0 μm are most likely to be deposited in the thoracic/bronchial region, and particles of between 0.5 and 2.0 μm are more likely to be deposited in the alveoli (World Health Organization, 1999; Witschi, Pinkerton, Van Winkle, & Last, 2008). The convention is that particulate matter of 10μm or below (named PM_{10}) will reach the bronchi and lower regions of the respiratory tract, and that particulate matter of 2.5μm or less ($PM_{2.5}$) can penetrate deep into the respirable part of the lung, the alveoli (World Health Organization, 1999). PM_{10} are the airborne particles with an aerodynamic diameter smaller than 10 microns, and $PM_{2.5}$ are airborne particles with an aerodynamic diameter smaller than 2.5 microns.

Fibres as long as 200 μm and with a diameter of 3 μm are able to penetrate deep into the lung (Winder & Stacey, 2004). The chemical nature of asbestos fibres renders them relatively insoluble and therefore difficult to remove from the lung via its natural defence mechanisms (Kelly, 2002).

The chemical properties of inhaled particulate matter has a significant effect on pathophysiology which is dependent on the toxic nature of the particulate, its concentration (level of exposure) and the duration of exposure (which, in combination, equals the dose) (Kelly, 2002).

Dust inhalation may initiate pulmonary fibrosis which is dependent on the physicochemical properties of the dust. Pneumoconiosis is the term used to describe interstitial disease of the lung due to prolonged exposure from inhalation of significant amounts of inorganic dust (Brichet, Desurmont, & Wallraet, 2002). A continuum of effects – from reversible on termination of exposure, slight effects with little decrement in lung function, to progressive massive fibrosis and the possibility of lung cancer – is dependent on the physicochemical/toxic properties of the dust and the dose (Gee & Mossman, 1995; Haspeter, Witschi, Pinkerton, Van Winkle, & Last, 2008).

There is strong evidence linking a significant decrease in lung function to long-term excessive dust exposure (Parliament of Australia Senate Committee, 2006). Despite the paucity of data in Australia on morbidity and mortality associated with toxic dust exposure, this association has been well established internationally (Downs et al., 2007).

Exposure to high levels of dust, irrespective of its physicochemical composition, will affect lung function. The degree of toxicity and the severity of the effects on lung function are dependent on the level of exposure and the physicochemical properties of the dust. For example, dust exposure in young coal miners (Carta, Aru, Barbieri, Avataneo, & Casula, 1996) and iron foundry workers (Gomes, Lloyd, Norman, & Pahwa, 2001) increases the incidence of respiratory symptoms and decline of lung function. Inhalable dust (PM_{10}) exposure is a significant predictor of decreased lung function (Downs et al., 2007).

4.3.2.2 Mineral dust and general (red dirt) dust

NIOSH (2001, p. 1) states that, "miners at noncoal surface mining operations are often exposed to high levels of respirable dust". The silica content of the respirable dust is of major concern hence DMP (2010b, p. 1) require dust suppression where dust is generated from operations that involve ground disturbance when using machinery, and "along each open pit road and vehicle operating area, during dumping operations, at all stockpile areas, stockpile stacking operations, stockpile tunnels and material reclaim operations, and at all crushing and screening plant".

Most of the dust from mining activities consists of coarse particles larger than 10 µm in diameter most of which will be trapped in the nasal region and expelled from the respiratory system (New South Wales Department of Health, 2007). However, operations such as drilling, blasting, loading, unloading, and transporting can generate sufficient respirable dust (Onder & Yigit, 2009) to warrant dust suppression controls, regular dust monitoring, and health surveillance.

Dust storms are a seasonal event at the edge of the Gibson Desert where the Murrin Murrin Operation is located. The main health concern associated with the red dirt dust from the gravel roads and the surrounding bushland, is its potential to trigger respiratory symptoms and exacerbate asthma. The New South Wales Department of Health (2003) and the Health Department of Victoria (2010) both state that particles from dust storms tend to be coarse and do not pose a serious health risk; and that they may worsen pre-existing conditions such as asthma and emphysema.

4.3.2.3 Fibrous material (chrysotile and anthophyllite)

In Western Australia, asbestiform minerals are found throughout the "greenstone belts", which host the state's major gold and nickel deposits (DMPR, n.d.). At the Murrin Murrin mine site, chrysotile and anthophyllite has been intersected during exploration drilling and identified in a few of the pits. Asbestos fibres are capable of causing asbestosis, lung cancer and mesothelioma (International Agency for Research on Cancer [IARC], 1998; Gibbons 2000; Winder & Stacey, 2004).

The risk of asbestos-related disease is dependent upon:

- the concentration of respirable fibres in the air;
- the length of time exposed;
- the type of fibre present (amphibole asbestos or serpentine); and
- the morphology of the fibres (fibre size and shape).

(DMPR, n.d., p. 5)

Asbestosis is a diffuse interstitial fibrosis of the lung parenchyma resulting from the exposure and retention of a high concentration of asbestos fibres. The lung function measured by spirometry in such cases presents as a restrictive ventilatory disorder (De Vuyst & Gevenosis, 2002).

It was considered that the potential for exposure to asbestiform material would be greatest during exploration drilling when asbestos is intersected. Asbestos has been observed in the pit areas and upon delivery of ore to the mill. Despite this, air monitoring has shown no significant risk to workers (Wing, 2005).

4.3.2.4 Silica

The mining industry has often been associated with silicosis (a pneumoconiosis) caused by the silica content of dust which may cause massive fibrosis and lung cancer (Haspeter et al., 2008; Winder & Stacey, 2004; IARC, 1986). Silicosis is considered to be associated with occupations that have very high exposures to respirable silica dust. On 1 January 2005 a revised national exposure standard of 0.1 mg/m^3 (time-weighted average [TWA], 8 hours) for quartz, cristobalite and tridymite came into effect (Parliament of Australia Senate Committee, 2006).

In Western Australia, particularly in open cut mining, the silica exposures are now mostly well below the occupational exposure standard which is reflected by the Health Surveillance Program for Mine Employees, Department of Industry and Resources, WA, which has shown no significant decrement in lung function observed for this group of WA mine workers over the past 5 years (Nield & Burmas, n.d.). The blast crew and drillers are perhaps at the highest risk of dust and respirable silica exposure in the open pit areas (Brichet, Desurmont & Wallaert, 2002) and the laboratory staff during preparation of ore samples for laboratory analysis (Minara Resources, 2005b). The potential for exposure to elevated levels of inhalable dust during operation of heavy mining vehicles and during charging and blasting at the Murrin Murrin mine site was identified (Minara Resources, 2005).

Exposure to silica dust has been associated with decreased lung function, and can be a cause of fixed obstructive airway disease at doses below that which lung fibrosis becomes clinically obvious (Parliament of Australia Senate Committee, 2006).

4.3.2.5 Calcrete dust

Calcrete, a limestone-like mineral mined in a local pit, is added to neutralise the acid in the neutralisation circuit. Calcrete consists of calcium carbonate, magnesium carbonate and silica. Although the silica content is often low, the exact proportion of these components in calcrete varies from location to location (Chen, Lintern, & Roach, 2002).

Dust monitoring at the calcrete plant and calcrete pit has shown that levels of silica were below the occupational exposure standard applicable at the time. However, the exposure standard for silica was reduced in 2004 (NOHSC, 2004) as the previous standards were not considered adequate for protecting workers from the risk of silicosis and lung cancer (Rice et al, 2001; Safe Work Australia, 2010b).

There have been occasional excursions above the occupational exposure standard for the respirable dust level, which were associated with the calcrete crushing circuit; however, the crusher operators were required to wear respiratory protection (Minara Resources, 2005b; Wing, 2005).

4.3.2.6 Diatomaceous earth

Diatomaceous earth is an amorphous silica, derived essentially from the remains of dead diatoms in marine sediments (Antonides, 1997; Harber, Dahlgren, Bunn, Lockey, & Chase, 1998). It was used as a filtering agent in the acid plant area (Wing, 2005; Minara Resources, 2001) but has since been substituted by perlite. Diatomaceous earth is known to cause silicosis and lung cancer (Rice et al., 2001).

Regular dust monitoring in the acid plant area indicated that the levels of diatomaceous earth dust have been well below the exposure standard (Minara Resources, 2005b).

4.3.2.7 Sulphur dust

Sulphur is a non-metallic dust known to cause irritation and inflammation to the nose and throat, and with long-term exposure it may cause chronic bronchitis (NIOSH, 2000). A large quantity of sulphur was stored as a large, open stockpile and used to produce sulphuric acid in the acid plant and hydrogen sulphide gas in the hydrogen sulphide plant. Operators unloading sulphur on delivery and loader operators removing sulphur from the stockpile for the process were at most risk of exposure to sulphur dust; however, respiratory protection was required in this area.

The sulphur stockpile was located at a safe distance from the main processing areas due mainly to the potential fire risk and to prevent exposure to sulphur dust on windy days. Eye and respiratory protection was mandatory in the vicinity of the sulphur stockpile.

4.3.2.8 Lime dust

Hydrated lime was identified through a formal risk assessment process as a potential health hazard in the utilities area 4100/5100, particularly for the operator and the delivery driver who unloaded the lime. Lime has the potential to irritate the nose, throat and respiratory system (International Program on Chemical Safety, 2001). However, occupational hygiene monitoring showed levels to be below the occupational exposure standard and were therefore deemed acceptable (Wing, 2005).

4.3.2.9 Caustic soda

Inhalation of low levels of caustic soda (sodium hydroxide) as a dust, mist or aerosol has the potential to cause irritation of the upper and lower respiratory system (Agency for Toxic Substances and Disease Registry, 2002). High-level, short-term exposures of sodium hydroxide via inhalation may be corrosive to the respiratory system and cause oedema of the lung (obstructive lung disease) often with a delayed onset (International Program on Chemical Safety, 2000; Hansen & Isager, 1991; Rubin, Bentur, & Bentur, 1992; California Office of Environmental Health Hazard Assessment, 1999). The risk associated with caustic soda when tipping bags of the material into the process was determined to be low through occupational monitoring (Wing, 2005).

4.3.2.10 Ammonium sulphate

Visible ammonium sulphate dust was generated during loading and unloading in the ammonium sulphate shed. However, it did not exceed the occupational exposure levels (Wing, 2005).

Ammonium sulphate dust is known to cause irritation of the respiratory tract and impair respiratory function, especially in asthmatics (Scorecard Organisation, n.d.) and the lung function in healthy adult volunteers was affected after a two- to four-hour exposure to 1 mg/m^3 (Organization for Economic Co-Operation and Development Screening Information Data Sets, 2004)

4.3.3 Toxic effects of metals

4.3.3.1 Nickel dust

Certain species of nickel have been shown to be carcinogenic (IARC, 1990; International Programme on Chemical Safety, 1991) and following the European Union Risk Assessment (2008) conducted by the Danish Government, more stringent occupational exposure standards for nickel and its compounds have been implemented (Safe Work Australia, 2010b). The carcinogenicity appears to be uniquely associated with occupational exposures during the smelting and refining of nickel (Nickel Institute, 2007). The Agency for Toxic Substances and Disease Registry (2005) state that:

> The most serious harmful health effects from exposure to nickel, such as chronic bronchitis, reduced lung function, and cancer of the lung and nasal

> sinus, have occurred in people who have breathed dust containing certain nickel compounds while working in nickel refineries or nickel-processing plants. (Agency for Toxic Substances and Disease Registry, 2005, p. 5)

To date, a number of epidemiological studies have established a link with nasal and lung cancer at high exposures to a mixture of nickel compounds found during the refining of sulphidic ores (Bates, 2008). Andersen, Berge, Engeland, and Norseth (1996) identified the synergistic effect of smoking and nickel exposure in the causation of lung and nasal cancer among nickel refinery workers. However, it has been argued that such cancers have not been observed in the extraction and processing of lateritic ore, which is the ore body mined and processed at the Murrin Murrin Operation (Doll, 1990; Goldberg et al., 1992).

Histologically, the early acute respiratory effect of inhalation of nickel dust is an inflammatory response, with effects on the epithelial and endothelial cells and an influx of alveolar macrophages (Klein & Costa, 2008). This may result in asthma, resolve if the insult ceases, or this may lead to fibrosis, (Bates, 2008; Agency for Toxic Substances and Disease Registry, 2005). In a study conducted by Cirla, Bernabeo, Ottoboni, and Ratti (1985) a significant decrease in lung function (FEV_1) was observed with six out of the seven asthmatics exposed to 0.3 mg/m^3 nickel sulphate for 30 minutes, whilst similar exposures to other metal salts did not affect lung function.

Safe Work Australia (2010b) Hazardous Substances Information System (HSIS) defines nickel and compounds as hazardous. The current relevant occupational exposure standards TWA (eight-hour, time-weighted average) exposure limit in the workplace for nickel metal is $1mg/m^3$. Soluble inorganic and insoluble inorganic nickel compounds have TWA's of 0.1 and $0.2mg/m^3$ respectively (American Conference of Industrial Hygienists, 2010)

On occasion, there have been exceedances of this standard at the Murrin Murrin Operation, most notably for the refinery 3700 area furnace operators, as indicated in precursor occupational hygiene studies conducted by Wing (2005) and Cross (2005). However, respiratory protection is mandatory for workers in this area.

4.3.3.2 Cobalt dust

The National Toxicology Programme concluded that there was clear evidence of carcinogenic activity via inhalation in mice and rats exposed to cobalt dust (National Toxicology Program, 1998; Bucher et al., 1999). IARC has determined that, with chronic exposure, cobalt and cobalt compounds are possibly carcinogenic to humans (IARC, 2006).

Acute inhalation effects of low doses of cobalt dust include respiratory irritation and asthma. Occupational exposure to various forms of cobalt can cause bronchial asthma (Swennen, Buchet, Stanescu, Lison, & Lauwerys, 1993; Linna et al., 2003) and effects on lung function (Nemery, Casier, Roosels, Lahaye, & Demedts, 1992).

At higher doses, cobalt may cause a progressive interstitial fibrosis (Liu, Goyer, & Waalkes, 2008). Respiratory symptoms may appear anywhere from a few months to several years from exposure; symptoms may be reversible if removed from the dust exposure, or there may be a delayed progression resulting in progressive interstitial fibrosis (Mapel & Coultas, 2002).

Morgan (1995) describes three respiratory effects produced by exposure to cobalt as: reversible airways obstruction, hypersensitive pneumonitis or alveolitis, and pulmonary fibrosis. The fibrosis is said to be reversible if exposure to cobalt dust is terminated early enough (Cugell, Morgan, Perkins, & Rubin, 1990). Swennen et al. (1993) established a dose-effect relationship with decrements in forced expiratory volume in one second/vital capacity (FEV_1/FVC) and the levels of cobalt exposure. After implementing effective exposure controls, a follow-up study was conducted after 13 years among the same workforce and cobalt exposure in this study was only influenced negatively by smoking (Verougstraete, Mallants, Buchet, Swennen, & Lison, 2004).

The current Australian occupational exposure standard for cobalt metal dust is 0.05 mg/m^3 for an eight-hour workday and 40-hour work week (Safe Work Australia, 2010b).

Monitoring results from regular occupational hygiene studies within the refinery at the Murrrin Murrin Operation showed that certain operator groups, particularly area 3800 (cobalt area) were being exposed to concentrations of cobalt exceeding this

exposure standard. However, it was common practice to wear respiratory protection whilst working in these areas (Oosthuizen & Cross, 2004; Wing, 2005).

4.3.3.3 Mixed sulphides (nickel/cobalt sulphides)

As the exact speciation of the nickel and cobalt compounds of the mixed sulphides in the process in area 3500 of the refinery remain essentially unknown, it is considered that the mixed sulphides material could pose a possible health risk. It has been argued that water-soluble nickel presents the greatest carcinogenic risk (Dunnick et al., 1995; Grimsrud, Berge, Haldorsen, & Andersen, 2002) although Heller, Thornhill and Conrad (2009) and Bates (2008) refute this. Cobalt sulphate is carcinogenic in rodents via inhalation (Bucher et al., 1999). Because such chemical species are likely to be in the mixed sulphides process material, the precautionary principle has been adopted. When there is a process stoppage and material has to be emptied into a bunded area, the product is maintained wet and the area hosed down to prevent any airborne dust until it can be reintroduced into the process. Full protective clothing is used in such instances. Dust levels in this area were invariably well below the occupational exposure standard due to the wetting procedure (Wing, 2005; Cross, 2005).

4.3.4 Toxic effect of gases, solvents and vapours

4.3.4.1 Gases/fumes from blasting

According to the Department of Minerals and Energy (1999) during the mining process workers could potentially be exposed to fumes from blasting. Such fumes consist of carbon monoxide, sulphur dioxide and oxides of nitrogen which, in high enough concentrations, may pose health effects, particularly for the respiratory system (Mainiero, Harris, & Rowland, 2007). The risk in open pit mining is considered negligible as the blasters move away from the area before detonation and fumes generally disperse with the wind prior to workers entering the blasting zone.

4.3.4.2 Diesel emissions

Diesel exhaust fumes may cause respiratory disorders such as bronchitis, emphysema, and lung cancer. According to NIOSH (2010) diesel exhaust is a potential human carcinogen, presumed to be due mainly to polycyclic aromatic hydrocarbons (Kelly, 2002).

Diesel fumes contain a cocktail of hazardous substances including carbon monoxide, nitrogen oxides, benzene, polycyclic aromatic hydrocarbons, sulphur dioxides, and particulate matter. Short-term exposure of healthy human subjects to diesel exhaust at high concentrations of diesel fumes induces airway inflammatory responses and affects lung function (Rudell et al., 1996). Moreover decreased lung function has been observed among garage workers, and bus drivers and conductors for a state transport corporation (Chattopadhyay, Alam, & Roychowdhury, 2003).

Various studies have shown associations between increased respiratory symptoms, including asthma, exacerbation of asthma, and exposure to diesel fume (Kagawa, 2002; Stenfors et al., 2004; Gluck, Schutz, & Gebbers, 2003; Riedl & Diaz-Sanchez, 2005). Respiratory effects – for example, goblet cell hyperplasia – were observed in a group of Swiss custom officers who worked clearing diesel trucks for 40 hours a week over 5 years, whilst a control group of office workers experienced no such effect.

Diesel exhaust fumes are considered to be a significant hazard in underground mining. The New South Wales Mines Safety Performance Branch (Driscoll, 2007) considers diesel exhaust fumes a high priority exposure needing to be urgently addressed.

The hazard of diesel fumes has been determined to be less significant for open pit mining as exhaust fumes are not restricted to a confined-space environment and dilution and dispersion of the emissions occurs readily. Nonetheless, drivers and mechanics of diesel plant and machinery may be exposed to diesel fumes during their daily routine.

At the time of the study there was no established occupational exposure standard or firmly established monitoring protocol for diesel fumes, therefore no atmospheric monitoring was conducted (Adeeb, 2010).

4.3.4.3 Ammonia

Some people are more sensitive than others to ammonia. The odour threshold for ammonia is reported to be between 5 and 50 ppm (parts per million) and upper respiratory irritation occurs at between 30 and 50 ppm (World Health Organization, 1986). Upon inhalation, highly water-soluble gases such as ammonia may cause immediate upper respiratory tract irritation (Schwartz, 2002). However, it has been reported that there is a human physiological response and adaption to ammonia

(Ferguson, Koch, Webster, & Gould, 1977). Ferguson et al. reported that workers could be acclimatised to 100 ppm with occasional excursions to 200 ppm and that after acclimatisation; exposures to ammonia at up to 100 ppm produced no discernable health effects. Despite this, others have reported an intense irritation to the eyes, nose, and throat that occurs at 100 ppm (Agency for Toxic Substances and Disease Registry, n.d.) and that ammonia may also cause chemical bronchitis, oedema, and cough often with blood-stained sputum (Tranter, 2004).

When exposure to ammonia is high or prolonged the odour is no longer detected by the sense of smell (olfactory fatigue) which creates a high health risk. At extremely high doses, greater than the immediately dangerous to life and health (IDLH) standard of 300 ppm (NIOSH , 1996a) death from asphyxiation is possible (Ross, Seaton, & Morgan, 1995).

Chronic irritation of the respiratory tract, chronic cough, asthma, and lung fibrosis are the result of long-term repeated exposure to ammonia (Agency for Toxic Substances and Disease Registry, n.d.). Decreases in lung function have been reported for fertiliser workers exposed to ammonia (Ali, Ahmed, Ballal, & Albar, 2001; Rahman, Bråtveit, & Moen, 2007). In contrast, Sundblad et al., (2007) reported ammonia levels at 25 ppm for three hours did not significantly affect lung function. Ammonia was considered one of the main components of a mixed exposure that caused a decrease in FEV_1 among livestock farmers. The mixed exposure consisted of ammonia, hydrogen sulphide, and inorganic dust (Eduard, Pearce, & Douwes, 2009). Similarly, Preller, Heederik, Boleij, Vogelzang, and Tielen (1995) and Reynolds et al., (1996) have suggest a causal link between lung function decrements and exposure to a mixture of endotoxins and ammonia among swine production workers.

4.3.4.4 Hydrogen sulphide

The odour threshold of hydrogen sulphide is approximately 0.5 parts per billion (ppb) (Agency for Toxic Substances and Disease Registry, 2009). Hence, hydrogen sulphide can be detected at very low levels, much lower than levels acknowledged to cause health effects (Government of Western Australia, Department of Health, 2009) because the odour of hydrogen sulphide may be detected at such low levels, there was a perception on site that the emissions in the processing plant were likely to cause ill health. However, the lowest concentration for adverse health effects is at least 500 times

the odour detection limit (Government of Western Australia, Department of Health, 2009).

Hydrogen sulphide is a mucous membrane irritant, causing skin, eye and respiratory irritation. At low concentrations (≤50 ppm) it can quickly cause irritation of the nose, throat, and lower respiratory tract (Agency for Toxic Substances and Disease Registry, 2009). Richardson (1995) demonstrated a significant FEV_1/FVC decrease among sewer workers who were exposed to hydrogen sulphide as compared to a non-exposed cohort.

Airborne hydrogen sulphide levels above 100 ppm are considered immediately dangerous to life and health (IDLH) (NIOSH, 1996b) via inhalation, causing asphyxiation and possible unconsciousness. Delayed pulmonary effects such as oedema and pneumonia may occur up to 72 hours after exposure (Nemery, 2002). At high concentrations, inhalation of a small volume may lead to immediate loss of consciousness, respiratory paralysis, and death (Agency for Toxic Substances and Disease Registry, 2009).

4.3.4.5 Sulphur dioxide, sulphur trioxide and sulphuric acid mist

Sulphur dioxide, sulphur trioxide and acid mist are direct-acting respiratory irritants that cause mild bronchoconstriction (Costa, 2008) and affect the mucus membranes of the eyes nose and upper respiratory system. All have a pungent odour which can overwhelm the respiratory system at high concentrations. The odour threshold for sulphur dioxide is between 0.5 and 0.8 ppm (Tranter, 2004). There is a continuum of effects from slight irritation at low exposure levels, to death from acute over-exposure, and chronic respiratory disease due to long-term exposures (NIOSH, n.d.).

Bronchoconstriction has been demonstrated in exercising asymptomatic asthmatics at sulphur dioxide levels as low as 1 ppm (Horstman, Seal, Folinsbee, Ives, & Roger, 1988). Although not as sensitive as asthmatics, males aged 55 or more are more sensitive to the effects of sulphur dioxide than adolescents (Rondinelli, Koenig, & Marshall, 1987). Co-exposures of low levels of sulphur dioxide with other air pollutants have been shown to cause additive (Kagawa, 1983) and synergistic effects, as well as decrements in lung function (Kleinman et al., 1981). One of the aims of this study was to determine whether there may be an additive or synergistic effect of exposure to low

concentrations of air contaminants at the Murrin Murrin mine site. Conversely, it has been suggested that there is possible adaption to mixed pollutant exposures (Bell, 1977).

Winder (2004, p. 408) quotes the exposure-response to oxides of sulphur as:

- 1-5 ppm – mild bronchoconstriction;
- 5 ppm – alteration in lung function; and
- 5-10 ppm – some cases of bronchospasm.

IARC (1992, 5.5 Evaluation) has determined that "there is sufficient evidence that occupational exposure to strong inorganic acid mists containing sulphuric acid is carcinogenic to humans". Epidemiological studies have shown lung cancer and laryngeal cancer to be a risk in specific industries where there is exposure to sulphuric acid aerosols, although often in these studies there are co-exposures or other confounders such as cigarette smoking (National Industrial Chemicals Notification Assessment Scheme, 2003). The National Toxicology Program (2000) stated that sulphuric acid most often exists as a mist because of its low volatility and high affinity for water, and that proximity to the source of the acid mist is the main determinant of exposure to workers.

Proximity to the source is what Wing (2005, p. 119) was alluding to in relation to the Murrin Murrin processing plant. He stated that:

> All measured exposures to sulphuric acid mist were found to be below the limit of detection, and therefore well below the exposure standard …. It can be inferred that the exposures of [the area] 4100/5100 [sulphuric acid plant and hydrogen sulphide plant respectively] operators to sulphuric acid mist are acceptable under normal production conditions.

Process leaks may occur, and emissions are inevitable during start-up of the acid plant (Dames & Moore, 1997). However, under these circumstances the area is restricted to maintenance workers wearing the appropriate personal protective equipment.

4.3.4.6 Hydrogen peroxide mist

Hydrogen peroxide mist may be generated during unloading of vehicles or when hydrogen peroxide is introduced into the refinery process. Hydrogen peroxide is highly

corrosive and if inhaled may cause severe irritation and inflammation of the respiratory tract. Therefore full personal protective clothing was worn on delivery of consignments of hydrogen peroxide by the supplier (Plog, 2002; Degussa, 2011).

4.3.4.7 Organic solvent

Organic solvents (volatile organic compounds [VOCs]) via inhalation have been shown to cause respiratory symptoms (Koren, Graham, & Devlin, 1992). All organic solvents have the ability to irritate the respiratory tract to some degree (Queensland Health, 2002) and may affect the nose, throat and lungs and cause asthma-like symptoms (New Zealand Department of Labour, 2009). Jaakkola and Jaakkola (2002, p. 246) state that the commonly used measure of total VOC "is an ambiguous concept" and therefore may be misleading in determining adverse effects. Similarly (Rumchev, Spickett, Bulsara, Phillips, & Stick, 2004) state that measuring total VOC's has the potential to underestimate the risk. Hence it is better to monitor for the individual components of the solvent where possible as the respiratory effects are dependent on the type of solvent. Asthmatics appear to be more susceptible to effects of solvents on the airways. Often the solvent *per se* may not affect the lung; however, it may be a vehicle for other chemicals dissolved within it that causes effects on the respiratory system (Nemery, 2002). An organic solvent ("Shellsol 2046", White Spirits) was used in the process of extraction of nickel and cobalt in area 3900 of the refinery. Personal monitoring for volatile organic compounds (Total VOCs) and individual constituents was conducted in this area. Total VOCs were in the range of <0.1 to 20 (occupational exposure standard 175 ppm, Safe Work Australia, 2011b) and extremely small concentrations of toluene and naphthalene were also detected. Wing (2005, p. 110) concluded that "these concentrations were found to be so far below the exposure standards as to be insignificant".

4.3.4.8 Welding fume

Welding occurred regularly during maintenance activities across the Murrin Murrin Operation. Welding in confined spaces was highlighted as a hazard. Welding fume is a mixture of particles and gases, the constituents of which depend on the materials being welded, materials in the filler, electric parameters and shield gases, and surface contaminants. The acute effect of welding fume is considered to be irritation of nasal passages, throat and lungs (McMillan, 2002).

Inhalation of welding fumes has been shown to cause bronchitis and pneumonitis and the severity of symptoms appears to be dependent on the process or metals used. Occupational asthma is more prevalent among welders than the general working population and exposure to welding fume has resulted in short-term changes in pulmonary function. Obstructive changes were observed more frequently among older, smoking welders than controls; however, a restrictive pattern was observed more frequently among non-smoking welders. Significant decrements in certain pulmonary function parameters have been observed in some studies (Liss, 1996). Smoking appears to potentiate the effects from welding fume (Occupational Safety and Health Administration [OSHA], n.d.)

Studies of the long-term effects of welding fume indicate that lung cancer among welders is 30 to 40 % greater than among the general population; however, other co-exposures have also been implicated (NOHSC, 1990). However, IARC (1990) concluded that there is inadequate evidence for the carcinogenicity of welding fumes and gases in animals and limited evidence in humans, as there were confounding factors such as smoking and concomitant asbestos exposure.

4.4 Effects of Co-Exposures (Mixtures of Hazardous Substances) on the Respiratory System

Occupational hygiene monitoring at the Murrin Murrin Operation determined that exposure to single hazardous substances were invariably well below the occupational exposure standards (Oosthuizen & Cross, 2004; Wing, 2005). The concern of employees that a ‘chemical cocktail’ from the mining activities and the refinery process could result in adverse health effects was a major driver for this study.

Exposures to mixtures of hazardous substances have been shown to be more harmful than single substance exposures. In reality, occupational exposures are usually to low doses of a complex range of chemicals (Interdepartmental Group on Health Risks from Chemicals, (2009). Exposure may be concurrent or sequential, and their effects may be additive, antagonistic, potentiated or even synergistic (Zeliger, 2008).

It was recognised through a review of the toxicity of the majority of the occupational hygiene hazards at Murrin Murrin that most of the substances have the potential to affect the respiratory system and the main target organ is the lung (Hendrick, 2002). It is

thus possible that additive, antagonistic, potentiated or even synergistic biological effects could occur due to exposure to these combinations of atmospheric contaminants (Zeliger, 2008). Furthermore, chemical interactions of substances in the process or when released to the atmosphere could occur and secondary particles may be produced by intermediate reactions of gases and particulate matter in the atmosphere from gaseous emissions (World Health Organization, 2000). Some of the emissions at Murrin Murrin included:

- sulphur dioxide from the sulphuric acid plant and hydrogen sulphide circuit flares;
- oxides of nitrogen from the steam boilers and sintering plants;
- carbon dioxide from the neutralisation circuits, power production and hydrogen plant;
- carbon dioxide from the hydrogen plant;
- particulate emissions from the mining activities, stockpiles, conveyors, exposed areas and vehicular movement;
- process steam; and
- increased sulphur dioxide emissions during acid plant start-up.

(Dames & Moore, 1997)

It has also been recognised that lifestyle factors, particularly cigarette smoking (International Programme on Chemical Safety, 1999) may have an additive or synergistic effect with other agents particularly on the respiratory system. Such examples include:

- asbestos and smoking (Leigh, Berry, de Klerk, & Henderson, 1996);
- cobalt and smoking (Verougstraete, Mallants, Buchet, Swennen, & Lison, 2004);
- nickel and smoking (Heller, Thornhill & Conrad, 2009).

Like many other researchers, Mustajbegovic et al. (2000, p. 439) noted that "in a population of workers exposed to low levels of pollutants respiratory symptoms were primarily associated with smoking". Hence cigarette smoking has not only been shown to exacerbate respiratory symptoms, diseases, and disorders, but has camouflaged such symptoms, and conversely has led to occupational exposures being wrongly blamed for respiratory illness.

Human variability in susceptibility and sensitivity to toxic chemicals may also complicate the overall response to these exposures (Hattis, Erdreich, & Ballew, 1987).

4.5 Conclusion

Excessive exposures above the occupational exposure standards of NOHSC: 3008 (NOHSC, 1995b) and NOHSC: 1003 (NOHSC, 1995a) (Safe Work Australia 2010a) of all the aforementioned gases and dusts are known to cause a range of respiratory symptoms including:

- decrease in lung function;
- Asthma;
- upper respiratory tract symptoms;
- lower respiratory tract symptoms, and ultimately;
- chronic pulmonary diseases, and
- nasal or pulmonary cancer.

(Morgan & Seaton, 1995; Hendrick et al., 2002).

Exposures to any of these hazardous substances singly, or in combination, have the potential to cause significant health issues. However, at the generally low levels identified during the occupational hygiene monitoring, it is unlikely that any serious untoward effects would occur. It is considered that health effects at the cellular level of low-level exposure of each of these hazardous substances would be reversible, providing a threshold level is not exceeded, although this remains contentious for carcinogens (Stacey, 2004). The question of what these hazardous substances, in combination at low concentrations, do requires investigation. In the industrial situation, employees may be exposed to chemical mixtures at low concentrations (Carpenter, Arcaro, & Spink, 2002). There is evidence of occupational respiratory diseases from chronic low-level exposures to irritants below their occupational exposure standards (Balmes, 2002; Mustajbegovic et al., 2000).

This current study provides a direct respiratory health assessment of the low-level chemical mixtures that the employees in the various work areas were exposed to over their length of service at the Murrin Murrin Operation. The main aim was to determine whether there was any discernable effect on the respiratory system, and, if so, whether further intervention, on top of the control systems already in place, may be required.

The Senate Inquiry into Workplace Exposure to Toxic Dust highlighted the fact that in Australia, "we do not have research into early detection" (Parliament of Australia, 2004, p. 43) and there is a "need for health surveillance of employees exposed to toxic dust" and that "There is no point waiting until exposure has occurred and deleterious changes have occurred" (Parliament of Australia Senate Committee, 2006, p. 44). This study was based on this very sentiment. The methodology for early detection of respiratory health effects at Minara Resources, Murrin Murrin Mine Site is provided in the next chapter.

5. RESEARCH METHODOLOGY

This chapter describes the methods and procedures used to conduct this study.

5.1 Introduction

The main purpose of this study was to conduct respiratory health surveillance of the Murrin Murrin workforce to detect possible adverse respiratory health effects at an early stage, in order to prevent potential long-term occupational respiratory disease. This meant evaluating whether working at the Murrin Murrin Operation, or areas within the Operation, affected the respiratory health of mine and process workers.

To do this, an initial study was conducted using a questionnaire to determine the respiratory health of each member of the Murrin Murrin workforce, followed by a lung function test. These data were then compared with a local control group. The lung function of each individual, in the study and control groups, was also compared with their predicted normal lung function values (Zapletal et al, 1977). This was followed up by a repeat study of a sample (72) of the initial study group members approximately two years later.

The study took place at the Murrin Murrin Operation between 17 February 2004 and 21 June 2006. The protocols for the methodology used in this study were derived from the most current references available in 2004.

5.2 Application to Undertake Research Involving Human Subjects

The research proposal was submitted and accepted by the Edith Cowan University Human Research Ethics Committee. The confidentiality of the questionnaires and spirometry data was guaranteed by maintaining records for a minimum of five years in locked filing cabinets, and codifying the data for statistical analysis. The written consent of all employees participating in the study was obtained prior to commencement of the study.

5.3 Study Group

5.3.1 Inclusion criteria

The study group consisted of all Murrin Murrin Operation mine and process workers. For a population of 420 with a confidence interval of .99, the sample size required was 410, therefore the entire population was studied. Only two refused to participate therefore the final study group was 418 (99.5%).

5.3.2 Exclusion criteria

Excluded from the study were any contract workers on short-term contracts such as shut-down workers. Those temporarily occupying full-time positions were included.

5.4 Control Groups

5.4.1 Catering staff

A local group consisting of 40 catering staff who resided at the accommodation camp approximately 8 km from the mine site constituted the control group. Only a few members of the control group rarely, if ever, visited the mine site or processing plant. These few were limited to those transporting employees to and from site and were on site for a brief period away from the process areas. This control group was chosen for its close proximity to the Murrin Murrrin Operation with the aim of eliminating any location/environmental/climatic/geographical variables. They were chosen primarily for their absence of exposure to hazardous substances.

An occupational hygiene survey identified pool chlorine as the only hazardous substance at the accommodation camp. However, the controls were considered adequate. The cleaning agents used were selected as they were non-hazardous. Both the study group and control group worked a 12-hour shift; however, their work periods (swings) varied.

5.4.2 Predicted normal values

To remove some of this variability, lung function data were also compared to predicted normal values extracted from a European reference population of normal lung function, who were non-smokers and free from respiratory disease (Zapletal, Paul, & Samánek, 1977). These data were computed into the spirometer readings by the supplier

(NicheMedical, Leederville, WA). The manufacturers of the spirometer considered this reference population the best reference population for an Australian population. Predicted normal values were used to effectively remove confounding factors such has height, weight (BMI) and gender differences.

5.5 Study Design: Initial Study

5.5.1 Comparison of the respiratory symptoms and lung function of the Murrin Murrin study group with a control group.

Data derived through respiratory questionnaire and spirometry for the entire workforce (418 study group members) were compared with data from a control group of (40) catering staff residing close to the Murrin Murrin Operation.

5.5.2 Descriptive statistics of the study group compared with the control group

The parameters from the respiratory questionnaire for the study group were compared with the control group, and descriptive statistics for respiratory symptoms, smoking history and asthma status were determined.

5.5.3 Prevalence of lung disorders

The prevalence of lung disorders for the study group and control group were calculated by dividing the number of persons with respiratory disorders by the total number of individuals for that group and expressed as a percentage.

5.5.4 Linear regression analysis

The lung function parameters FEV_1 and FVC, corrected for age, gender and height, were compared using linear regression analysis with both the control group and the predicted normal values (Zapletal et al., 1977). The linear model of height, plotted against the lung function indices FEV_1 and FVC (Cotes, 1993) was employed to visualise the relationship between the study group, their predicted normal values (Zapletal et al., 1977) and the control group data. A series of scatter plots with regression lines (IBM® SPSS® PASW Statistics 18) were produced with sequential removal of the confounding factors in order to observe their effect on lung function.

Regression analysis enabled the sequential removal of confounding factors, to determine the goodness of fit (R^2) value, and the Pearson's correlation coefficient (r).

5.5.5 Comparison of lung function with predicted normal values

The lung function of both the study group and the control group was compared with their predicted normal values extracted from a reference population of 173 subjects with normal lung function, who were non-smokers and free from respiratory disease (Zapletal, Paul, & Samánek, 1977). This reference population was considered the most appropriate reference population for an Australian population and was computed into the spirometer readings by the supplier (NicheMedical, Leederville, WA).

Comparison with the predicted normal values removes the issue of confounding for factors such as age, gender, height and weight.

5.5.6 Work area/department

The lung function data for populations from each work area/department were analysed to determine if there were any statistically significant effects on lung function due to working in any specific work area. Both the production and the maintenance workers in each area/department were included in the initial and repeat study.

A dependent (paired samples) t-test was conducted to compare the lung function parameters (FEV_1 and FVC) for the never-smoker sub-group with their equivalent predicted normal values (Zapletal et al, 2007) for individuals in each work area.

5.6 Study Design: Repeat Study

5.6.1 Longitudinal study of lung function

Any changes in respiratory symptoms since the initial questionnaire were requested and noted.

Repeat spirometry measurements were conducted to establish rates of change in lung function over time, to provide an identifiable picture of lung function of the repeat study group. The advantage of utilising a longitudinal study is that it enables observations to be made on the same individuals to focus on changes occurring within subjects and to make population extrapolations that are not as prone to between subject variation (Yee & Niemeier, 1996).

Therefore repeat lung function tests were conducted on a sample of 72 of the original 418 members of the study group (i.e. 17%), approximately two years after the initial study. As the attrition rate was 51%, the 72 employees from the remaining 213 study population represented 34%. The time interval from initial spirometry test to the follow-up ranged from 173 to 845 days.

The change in lung function over time from the initial study to the repeat study was determined for the 72 employees. The mean change, range of change and overall mean change per year in lung function parameters (FEV_1 and FVC) was calculated. These calculations were made with the sequential removal of confounding factors (smoking, asthma and pre-existing respiratory disorders).

A dependent t-test was conducted to determine whether there was any significant difference from the initial lung function tests to the repeat tests.

5.6.2 Length of service at the Murrin Murrrin Operation

A disadvantage of longitudinal studies is the loss of study group members (or attrition, 51% occurred during this investigation). Therefore an addition statistical analysis of the effect of length of service (or period of employment) at the Murrin Murrin Operation on lung function was conducted for the original 418 members of the study group. The length of service for the 418 study group members ranged from one month to six years.

A multivariate analysis (MANOVA) was conducted to determine whether there was a significant difference in lung function with length of service.

5.7 Pre-Swing and Post-Swing Lung Function in a Cohort of Refinery Workers

Lung function tests for a cohort of refinery workers were conducted as they arrived for work on site prior to commencing work in the refinery, and on completion of their work period on site before returning home for their rest break. This was done in order to detect if there were any statistically significant decrements in lung function over a work period (colloquially known as a *swing*).

A dependent t-test was used to determine whether there was a significant difference for the pre-swing lung function when compared with the post-swing lung function of this group of refinery workers.

5.8 Study Instruments

5.8.1 Respiratory questionnaire

The prevalence of respiratory symptoms in the study and control groups was determined using the Government of Western Australia, Mining and Petroleum Resources (2004) respiratory questionnaire component of their health assessment form (modified British Medical Research Council questionnaire, 1986) (Appendix A). This was an internationally accepted standardised respiratory questionnaire.

The respiratory questionnaire was administered to each individual of the study and control groups prior to lung function testing by a competent *approved person*, approved by Resources Safety to carry out MineHealth Assessments on completion of compulsory training in spirometry (i.e., lung function testing) to standards required by Resources Safety and WorkCover WA. Data regarding each individual's work history, respiratory symptoms, smoking status and history, and asthma status was collected.

5.8.2 Measurement of lung function

5.8.2.1 Equipment

A portable handheld spirometer (EasyOne Model 2001 diagnostic spirometer, ndd Medizintechnik AG, Zurich) was used to determine various lung function parameters most notably forced expiratory volume in the first second (FEV_1) and forced vital capacity (FVC). These data were computed automatically by the spirometer and were also expressed as predicted values (Zapletal et al., 1977).

5.8.2.2 Technique

Prior to commencing spirometry, it was necessary to measure accurately each participant's height (measured without shoes) and weight. These data were entered into the instrument along with date of birth, ethnicity, gender, smoker status, and asthma status.

To gain the best possible lung function measurements it was necessary to explain and demonstrate the test procedure and coach each participant during the procedure. To gain optimum measurement of lung function the subjects were standing during the procedure. All subjects were warned of possible dizziness due to the procedure and all elected to stand, although precautions were taken for the possibility of a subject fainting.

As a noseclip is not mandatory for forced expiratory maneuvers, no nose clip was worn by any subject, although it is often recommended to prevent air escaping via the nose at the end of the manoeuvre. However, this did not pose a problem in any of the subjects (Spirxpert, n.d.; Lange, Mulholland, & Kreider, 2009). The participants were observed to ensure maximal effort was expended and a true result obtained, in addition to any liquid crystal display (LCD) message on the instrument (ndd Medizintechnik AG, 2001) which indicates such an event. The participants progressed through the series of lung function tests with a minimum of three manoeuvres until repeatability was obtained. Any suboptimal efforts were excluded from the study in accordance with the manufacturer's *EasyGuide* (ndd Medizintechnik AG, 2002, and the ATS/ERS guidelines (ATS, 1995; Miller, 2005; Johns & Pierce, 2003).

Records of all lung function data were maintained on the DMP respiratory health assessment form as a backup to the computerised spirometer data. Confidentiality of these documents was maintained by securing them in locked filing cabinets. The computer records were password protected and codified so that individuals were not identifiable.

5.8.2.3 Calibration and quality control

The spirometer accuracy was checked regularly during each batch of testing by a lung function specialist using a certified 3.00 litre syringe, despite the fact that the EasyOne spirometer maintains its calibration during routine use, has an in-built calibration system, and does not require daily calibration as specified in international spirometry guidelines (Walters, Wood-Baker, Walls, & Johns, 2006; Perez-Padilla et al., 2006).

No deterioration over time was detected.

The approved person's lung function was also monitored during each batch of testing to act as a biological control, and to check internal validity.

The manufacturer's *EasyGuide* (ndd Medizintechnik AG, 2002) instruction booklet was used to ensure the instrument was used correctly and to ensure quality of results. For example, the height above sea level of the Murrin Murrin Operation was pre-entered into the spirometer database to allow adjustment for barometric pressure (Spirxpert, n.d.). The EasyOne Model 2001 automatically adjusts for the standardization of gas volumes and environmental factors such as temperature, pressure and water vapour (Lange, Mulholland, & Kreider, 2009).

5.8.2.4 Protocol

Lung function was measured following the American Thoracic Society/European Respiratory Society protocol (American Thoracic Society, 1995; Miller et al., 2005).

5.8.2.5 Approved person

The study was conducted by a DMP competent approved person, approved by Resources Safety to carry out MineHealth Assessments on completion of compulsory training in spirometry (lung function testing) to standards required by Resources Safety and WorkCover WA (Department of Mines and Petroleum 2010a).

5.9 Exposure Assessment

Personal atmospheric monitoring of the workplace hazardous substances was conducted in each area of the site in a separate but associated study, and a detailed occupational exposure history was compiled for each work group. A qualitative review of the respiratory health effects presented by the hazardous substances associated with the mining and process plant were discussed in Chapter 4.

5.10 Statistical Analysis

The Statistical Package for the Social Sciences (SPSS) (IBM SPSS Statistics 18, 2010) was used for all data analysis.

The results using this methodology are outlined in the following chapter.

6. RESULTS

6.1 Initial Lung Function Study

6.1.1 Study group

The study population consisted of 384 male and 34 female (a total of 418) mining, processing and administrative employees working at the Minara, Murrin Murrin mine site.

6.1.2 Control group

The control group consisted of 27 male and 13 female (a total of 40) catering personnel at the Murrin Murrin accommodation village 8 km from the mine site.

6.1.3 Predicted normal values

Lung function data of the study and control groups were compared with predicted normal values (Zapletal et al., 1977) which were programmed into the spirometer. Predicted normal lung function values are derived from a population asymptomatic of lung function disorders and lifelong non-smokers. These values are computed into the spirometer and the results for each test subject compared with the predicted values relative to their gender, height and age. Both the study and control group test subjects were therefore compared to their respective predicted normal value for each lung function parameter (e.g., FEV_1 and FVC).

6.2 Profile of the Study Group Compared With the Control Group

The descriptive statistics for the study and control groups are compared in Table 6.1, below.

Table 6.1 Descriptive Statistics of the Study Group Compared with the Control Group

	Study Group (n=418)	**Control Group (n=40)**
Male	92% (n=385)	68% (n=27)
Female	8% (n=33)	32% (n=13)
Mean Height (cm) & Standard Deviation	177 ±7.99	173 ±8.86
Height Range (cm)	155-200	155-195
Mean Age (years) & Standard Deviation	39 ±9.1	39 ± 12.7
Age Range (years)	19-67	19-63
Current Smokers	34% (n = 144)	39% (n = 16)
Ex-smokers	25% (n = 104)	28% (n = 11)
Non-smokers	41% (n = 170)	33% (n = 13)

6.3 Prevalence of Respiratory Symptoms in the Study Group Compared With the Control Group

The prevalence of respiratory symptoms in the study and control group was determined using the Government of Western Australia, Mining and Petroleum Resources (MPR) questionnaire (2004) (modified British Medical Research Council questionnaire). Thirteen participants of the study group (3%) and one participant in the control group (3%) did not complete the questionnaire although all performed the lung function test. Thus there were 405 study group and 39 control group participants who completed the questionnaire. Any missing data was taken into account for the following calculations and statistical analysis. Table 6.2 below provides the prevalence of respiratory symptoms in the study and control groups.

Table 6.2 Prevalence of Respiratory Symptoms in the Study Group vs. Control Group

		Study Group n(%)	Control Group n(%)
	Cough		
1	Do you usually cough first thing in the morning?	19 (5%)	3 (8%)
2	Do you usually cough during the day or night?	31 (77%)	4 (10%)
3	Do you have a cough like this on most days for as much as three months each year?	12 (3%)	0 (0%)
	Phlegm		
4	Do you usually bring up phlegm from your chest first thing in the morning?	24 (6%)	3 (8%)
5	Do you usually bring up phlegm from your chest at any other time of the day?	26 (6%)	2 (5%)
6	Do you bring up phlegm like this on most days for as much as three months each year?	13 (3%)	1 (3%)
7	In the past three years have you had a period of increased cough and phlegm lasting for three weeks or more?	3 (1%)	2 (5%)
8	Have you had more than one such period?	4 (1%)	0 (0%)
	Breathlessness on Activity		
9	Do you get short of breath when hurrying on level ground or walking up a slight hill?	12 (3%)	2 (5%)
10	Do you get short of breath when walking with other people of your age on level ground?	3 (1%)	2 (5%)
11	Do you have to stop for breath when walking at your own pace on level ground?	0 (0%)	0 (0%)
	Breathlessness at Rest		
12	Do you ever get short of breath at rest?	5 (1%)	0 (0%)
13	Do you ever wake up in your sleep short of breath?	7 (2%)	1 (3%)
	Wheezing		

14	Does your chest ever sound wheezy or whistling?	46 (11%)	8 (20%)
15	Do you get this on most days or nights?	9 (2%)	1 (3%)
16	Have you ever had attacks of shortness of breath with wheezing?	11 (3%)	2 (5%)
17	Was your breathing absolutely normal between attacks?	7 (2%)	2 (5%)
	Therefore – converse of question 17 – not normal between attacks =	4 (1%)	0 (0%)
	Breathing Difficulty		
18	Does your chest ever feel tight or your breathing become difficult?	21 (5%)	3 (8%)
	Smoking History		
19	Do you, or did you, smoke more than 1 cigarette/day; a cigar/week; or 2 oz (50 g) pipe tobacco/month for at least one year? Smokers and ex-smokers	248 (61%)	26 (67%)
20	How much do you (or did you) smoke each day? (no. of cigarettes). Roll owns or pipes (number of grams/week)?	Used to calculate Pack Years	Used to calculate Pack Years
21	How old were you when you started smoking?		
22	If you are an **ex-smoker**, how old were you when you gave up smoking permanently?	104 (25%)	11(28%)
	Smokers = (Q19-Q22) =	144 (34%)	15 (39%)
	Therefore **Non-Smokers** =	154 (38%)	13 (33%)
	Past Chest Illness		
23	During the past three years have you had any chest illness which has kept you from usual activities for a week or more?	24 (6%)	1 (3%)
24	Did you bring up more phlegm than usual during this illness?	7 (2%)	0 (0%)
25	Have you had more than one illness like this in the past three years?	4 (1%)	0 (0%)
	Asthma		
26	Have you ever had asthma?	35 (9%)	4 (10%)
	Other respiratory illness		
27	Have you ever had any other respiratory illness?	37 (9%)	4 (10%)

Questions 20, 21 and 22 from the MPR (2004) questionnaire were used to determine pack years (Connolly & Alpert, 2008; National Cancer Institute, n.d.) to assess the effect of smoking on the FEV_1 for the Ever Smokers of the study and the control groups. The scatter plots showing these data are presented in the two figures below. The R^2 values and r values are given for each graph. The R^2 value i.e. goodness of fit of the regression line, and Pearson's correlation coefficient (r) which measures the strength of the relationship between two variables FEV_1 and Pack Years.

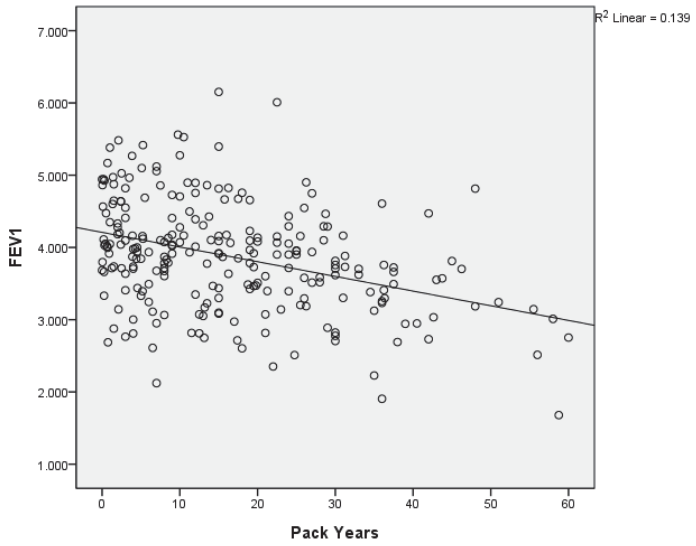

Figure 6.1 The Effect of Smoking (Pack Years) on FEV_1 (litres) for the Study Group Ever Smokers (n=242) (R^2 0.14) (r minus 0.46).

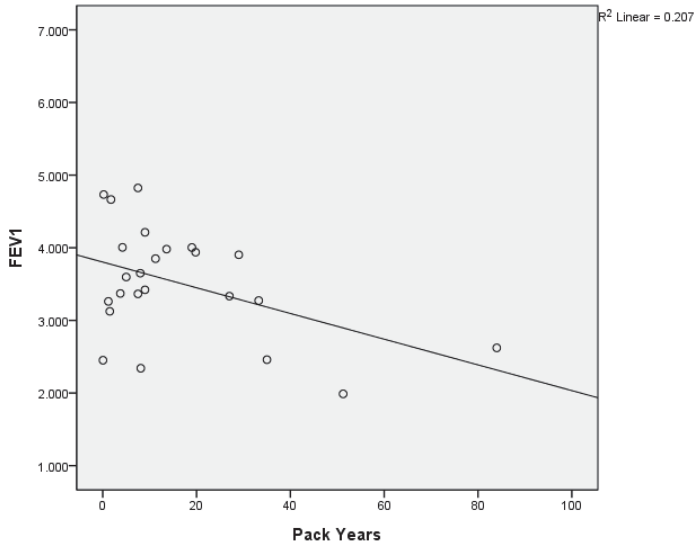

Figure 6.2 The Effect of Smoking (Pack Years) on FEV_1 (litres) for the Control Group Ever Smokers (n=24) (R^2 0.21) (r minus 0.37).

6.4 Prevalence of Respiratory Disorders

6.4.1 Control population

There were a total of five individuals in the control population (5/40 = 12.5%) with abnormal spirometry results (ndd Medizintechnik AG, 2002; Johns & Pierce, 2003); three with mild obstruction, one with mild obstruction and low vital capacity possibly due to restriction, and one with low vital capacity possibly due to restriction of lung volumes.

6.4.2 Study population

There were 26 individuals in the study population (26/418 = 6.2%) with abnormal spirometry results (ndd Medizintechnik AG, 2002; Johns & Pierce, 2003); 18 with mild obstruction, five with mild obstruction and low vital capacity possibly due to restriction, and three with moderate obstruction and low vital capacity possibly due to restriction. All were non-work-related, each with a history of respiratory illness and/or smoking. The conditions for these 26 individuals were:

- history of pneumothorax, former smoker;
- triple by-pass/sleep apnoea/10% lung removed due to thrombosis, BMI >35;
- smoker 30 cigarettes/day, BMI >40;
- smoker15 cigarettes/day, BMI >30;
- smoker 25 cigarettes/day and asthmatic, BMI >30;
- history of bronchitis, diabetic, asthmatic, BMI >30;
- known poor lung function (reason not provided) BMI >35;
- former smoker 20 cigarettes/day, BMI >30;
- former smoker, possible asthmatic, BMI >35;
- smoker 40 cigarettes/day;
- asthmatic, former smoker;
- asthmatic, occasional smoker;
- former smoker, 12 cigarettes/day;
- history of pneumonia, heart valve replacement, former smoker 30 cigarettes/day;
- former smoker 30 cigarettes/day;
- asthmatic, former smoker 20 cigarettes/day;

- known poor lung function (reason not given);
- smoker 15 cigarettes/day;
- smoker of a pack of hand-rolled cigarettes ("rollies") per week BMI >35;
- asthmatic, occasional smoker;
- two asthmatics, former smokers;
- former smoker 50 cigarettes/day;
- former smoker 30 cigarettes/day, asthma, history of pleurisy;
- bronchitis, smoker 25+ per day, asthmatic (died of lung cancer shortly after completion of the study);
- history of pneumonia, possible asthma.

BMI classification according to the World Health Organization (n.d.).

These were pre-existing respiratory disorders (non-work related) as determined through questionnaire.

6.5 Comparison of the Lung Function of the Study Group, with Their Predicted Values, and the Control Group - With Sequential Removal of Confounders

6.5.1 Lung function versus height

The linear model of height, plotted against the lung function indices FEV_1 and FVC (Cotes, 1993) was employed to visualise the relationship between the study group, their predicted normal values (Zapletal et al., 1977) and the control group data. A series of scatter plots with regression lines (IBM® SPSS® PASW Statistics 18) were produced with sequential removal of the confounding factors in order to observe their effect on lung function.

6.5.2 Sequential removal of confounders

A series of graphs are presented below to compare the FEV_1 and FVC of the study group, their predicted values, and the control group, initially looking at all subjects of the study group (n=418) and the control group (n=40) followed by sequential removal of confounding of smoking, smoking and asthma, then smoking, asthma and non-work-related respiratory disorders, ultimately ending up with comparison of the *presumed*

healthy non-smokers sub-population of the study group (i.e., with all confounders removed).

These are presented in a series of three figures, firstly comparing the FEV_1 for all subjects of the study group, their predicted values and control group; followed by FVC for all subjects of the study group, predicted values and control group; and then with the sequential removal of confounding as described above.

The R^2 values and r values are given for each graph. The R^2 value is the goodness of fit of the regression line, and Pearson's correlation coefficient (r) measures the strength of the relationship between two variables; which for this series of graphs are FEV_1 and height, and FVC and height.

6.5.2.1 All subjects study and control groups: FEV_1 plotted against height

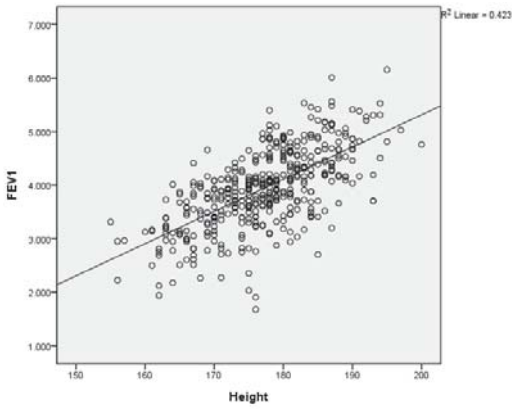

Figure 6.3 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for All Subjects (n= 418) of the Study Group. (R^2 0.42) (r 0.65).

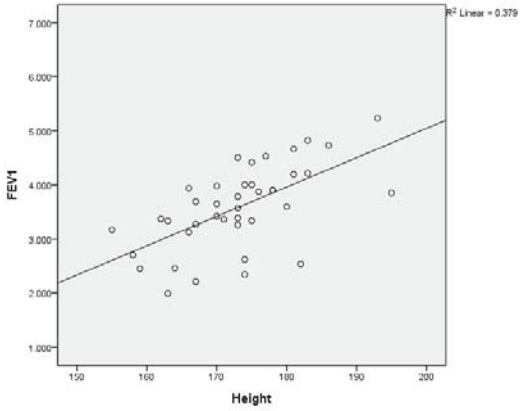

Figure 6.4 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for All Subjects (n=40) of the Control Group. (R^2 0.38) (r 0.62).

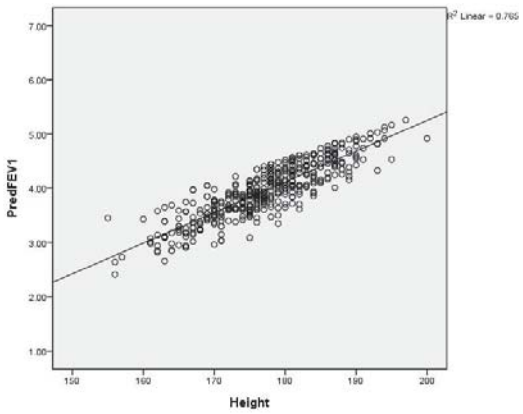

Figure 6.5 Scatter Plot Showing the Relationship of the Predicted Values of FEV_1 (litres) and Height (cm) for All Subjects of the Study Group (n=418) (R^2 0.77) (r 0.88).

6.5.2.2 All subjects study and control groups: FVC plotted against height

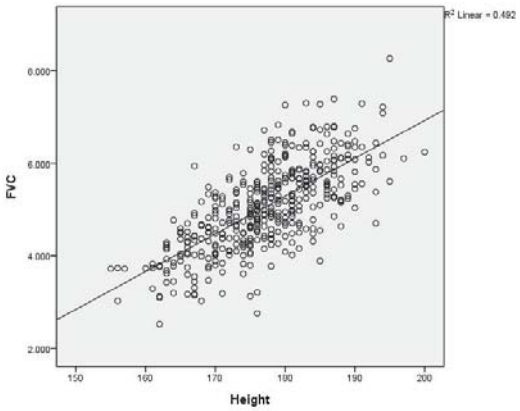

Figure 6.6 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for All Subjects (n = 418) of the Study Group. (R^2 0.49) (r 0.70).

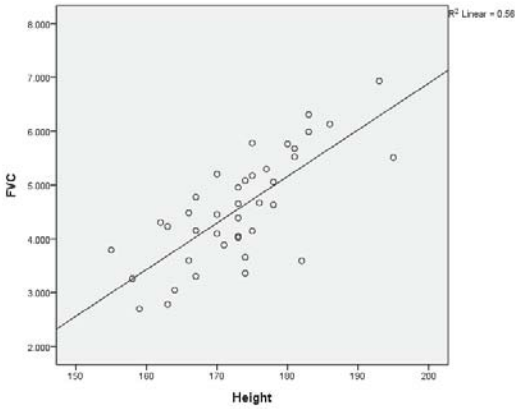

Figure 6.7 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for All Subjects of the Control Group (n = 40) (R^2 0.56) (r 0.75).

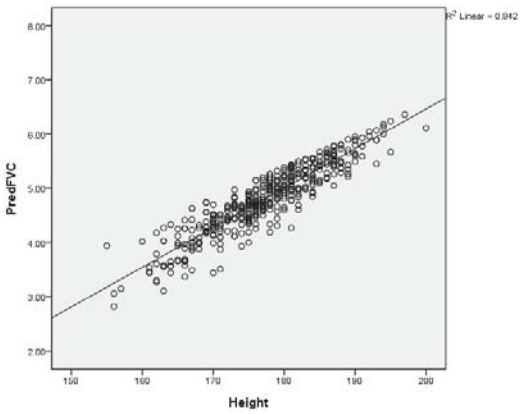

Figure 6.8 Scatter Plot Showing the Relationship of the Predicted Values of FVC (litres) and Height (cm) for All Subjects for the Study Group (n = 418) (R^2 0.84) (r 0.92).

6.5.2.3 Removal of confounding of smoking: FEV_1 plotted against height

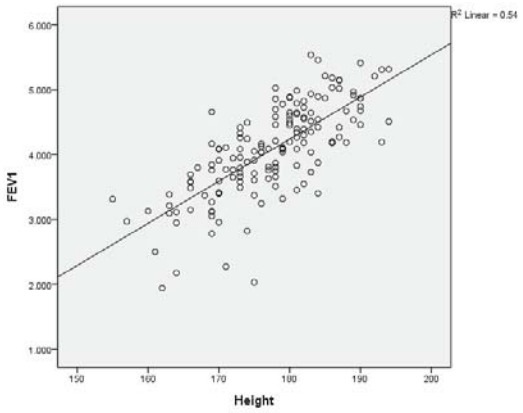

Figure 6.9 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Study Group Never Smokers (n = 153) (R^2 0.54) (r 0.74).

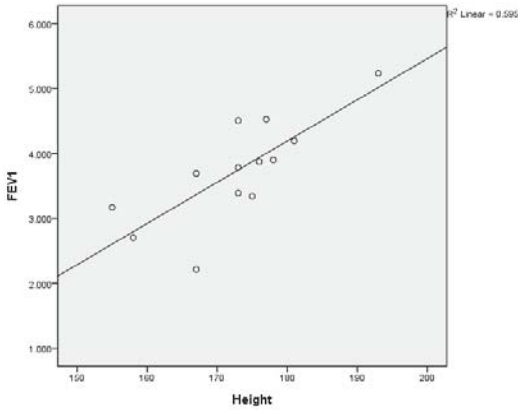

Figure 6.10 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Control Group Never Smokers (n = 13) (R^2 0.60) (r 0.78).

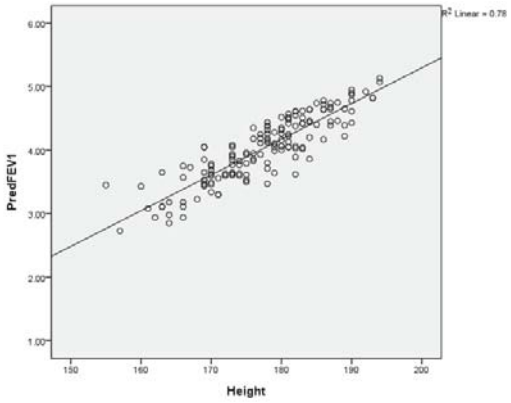

Figure 6.11 Scatter Plot Showing the Relationship of the Predicted Values of FEV_1 (litres) and Height (cm) for the Study Group Never Smokers (n = 153) (R^2 0.78) (r 0.88).

6.5.2.4 Removal of confounding of smoking: FVC plotted against height

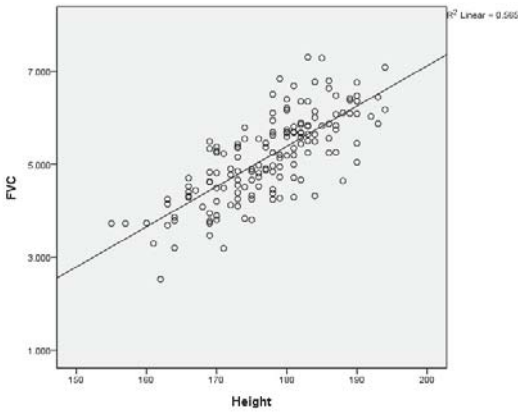

Figure 6.12 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Study Group Never Smokers (n = 153) (R^2 0.57) (r 0.93).

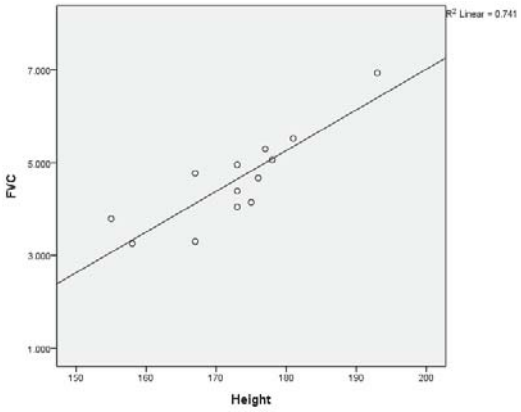

Figure 6.13 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Control Group Never Smokers (n = 13) (R^2 0.74) (r 0.94).

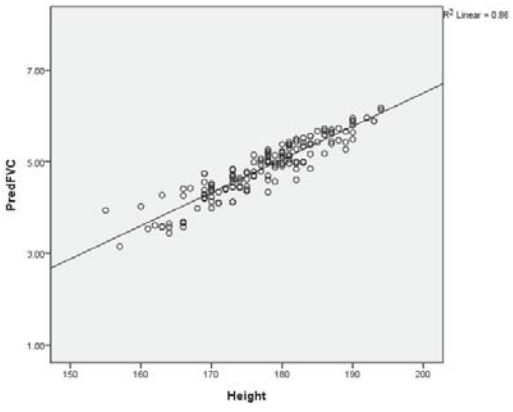

Figure 6.14 Scatter Plot Showing the Relationship of the Predicted Values of FVC (litres) and Height (cm) for the Study Group Never Smokers (n = 153) (R^2 0.86) (r 0.94).

6.5.2.5 Removal of confounding of smoking and asthma: FEV_1 plotted against height

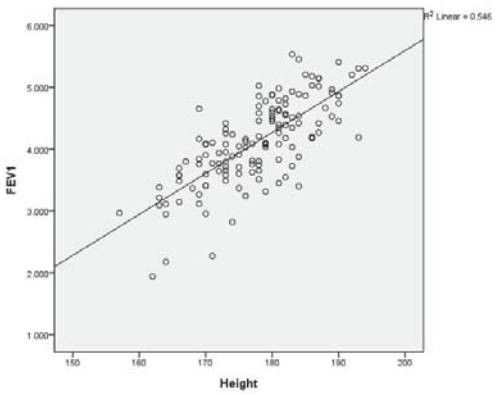

Figure 6.15 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Study Group Never Smokers/Non-Asthmatics (n = 136) (R^2 0.55) (r 0.75).

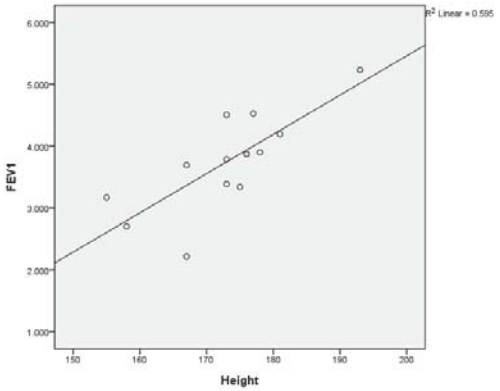

Figure 6.16 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Control Group Never Smokers/Non-Asthmatics (n = 13) (R^2 0.60) (r 0.78).

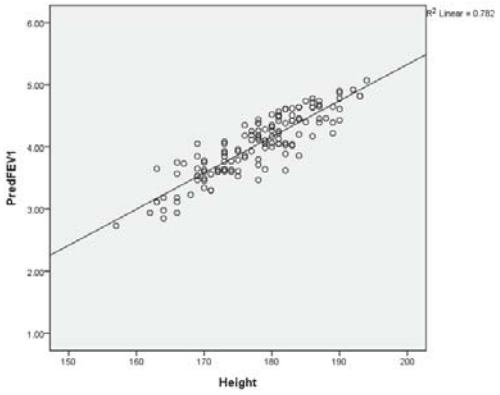

Figure 6.17 Scatter Plot Showing the Relationship of the Predicted Values of FEV_1 (litres) and Height (cm) for the Study Group Never Smokers/Non-Asthmatics (n = 136) (R^2 0.78) (r 0.89)

6.5.2.6 Removal of confounding of smoking and asthma: FVC plotted against height

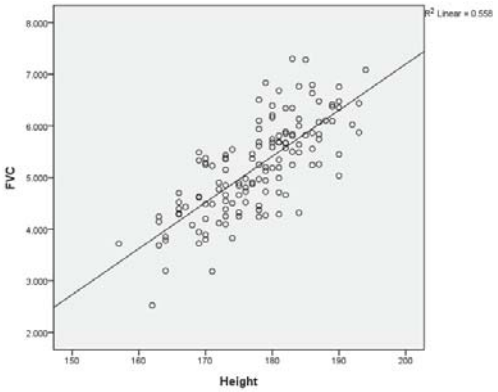

Figure 6.18 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Study Group Never Smokers/Non-Asthmatics (n = 136) (R^2 0.56) (r 0.75).

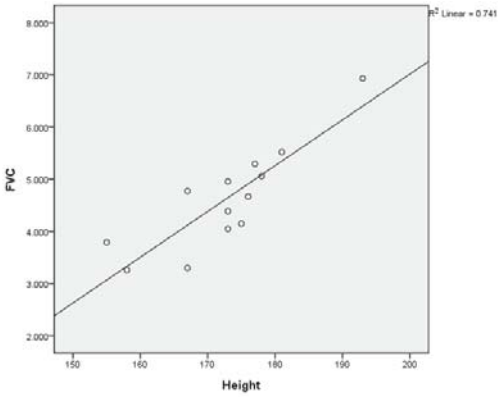

Figure 6.19 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Control Group Never Smokers/Non-Asthmatics (n = 13) (R^2 0.74) (r 0.88).

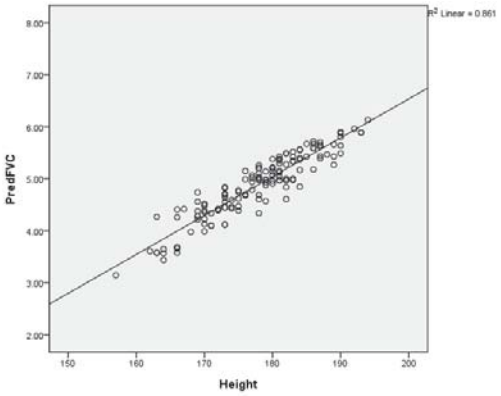

Figure 6.20 Scatter Plot Showing the Relationship of the Predicted Values of FVC (litres) and Height (cm) for the Study Group Never Smokers/Non-Asthmatics (n = 136) (R^2 0.86) (r 0.93).

6.5.2.7 Removal of non-work-related respiratory symptoms: FEV_1 plotted against height

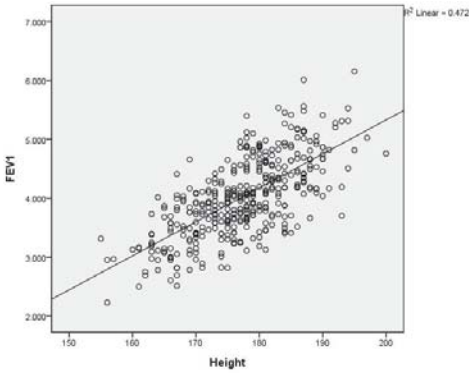

Figure 6.21 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Study Group on Removal of Individuals with Non-Work-Related Respiratory Symptoms (n = 392) (R^2 0.47) (r 0.69).

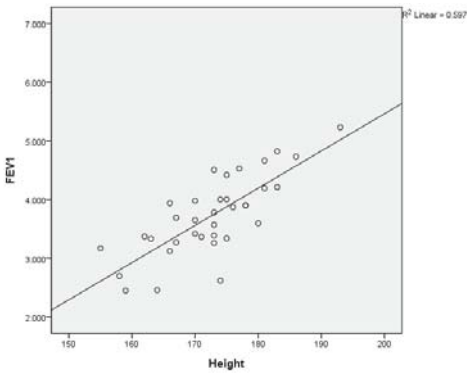

Figure 6.22 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Control Group on Removal of Individuals with Non-Work-Related Respiratory Symptoms (n = 35) (R^2 0.60) (r 0.77).

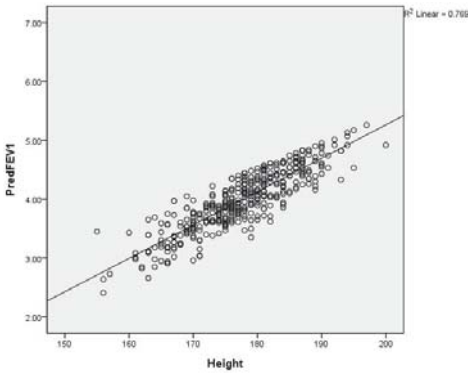

Figure 6.23 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Predicted Values of the Study Group on Removal of Individuals with Non-Work-Related Respiratory Symptoms (n = 392) (R^2 0.77) (r 0.69).

6.5.2.8 Removal of non-work-related respiratory symptoms: FVC plotted against height

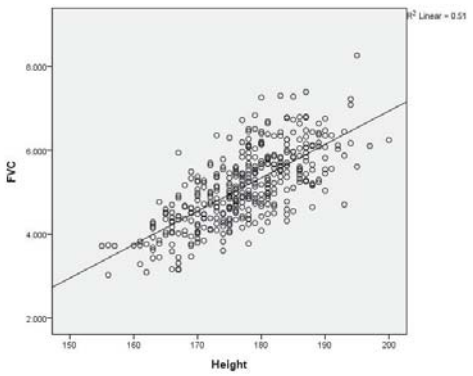

Figure 6.24 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Study Group on Removal of Individuals with Non-Work-Related Respiratory Symptoms (n = 392) (R^2 0.51) (r 0.71).

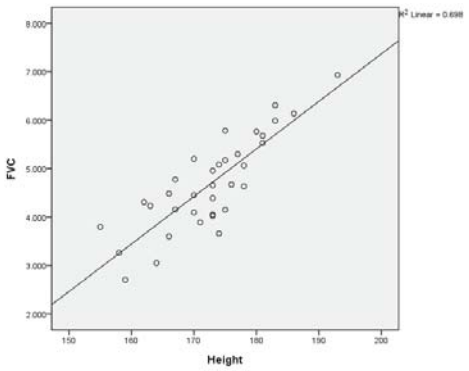

Figure 6.25 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Control Group on Removal of Individuals with Non-Work-Related Respiratory Symptoms (n = 35) (R^2 0.70) (r 0.84).

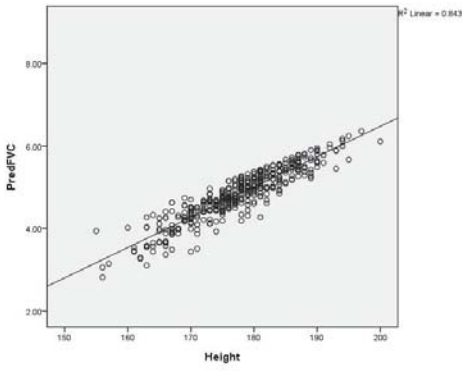

Figure 6.26 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Predicted Values of the Study Group on Removal of Individuals with Non-Work-Related Respiratory Symptoms (n = 392) (R^2 0.84) (r 0.92).

6.5.2.9 Removal of confounding of smoking, asthma, and non-work-related symptoms: FEV_1 plotted against height

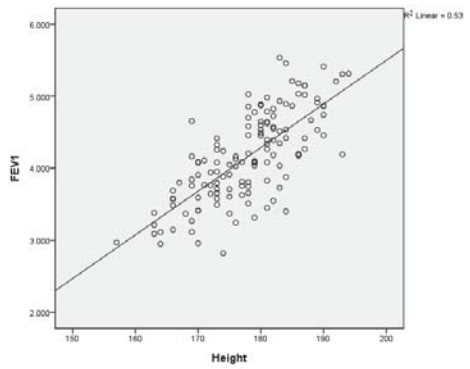

Figure 6.27 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Study Group on Removal of Smokers, Asthmatics, and Individuals with Non-Work-Related Respiratory Symptoms (n = 134) (R^2 0.53) (r 0.73).

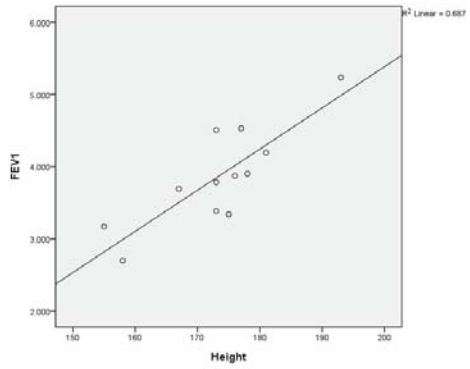

Figure 6.28 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Control Group on Removal of Smokers, Asthmatics, and Individuals with Non-Work-Related Respiratory Symptoms (n = 12) (R^2 0.69) (r 0.83).

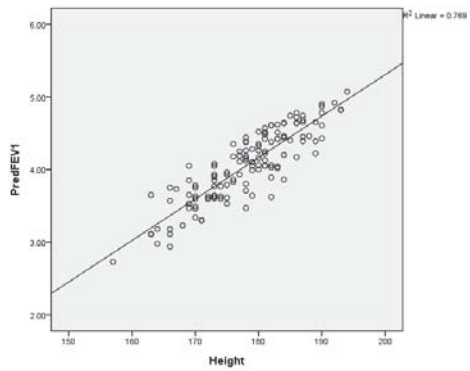

Figure 6.29 Scatter Plot Showing the Relationship of FEV_1 (litres) and Height (cm) for the Predicted Values of the Study Group on Removal of Smokers, Asthmatics and Individuals with Non-Work-Related Respiratory Symptoms (n = 134) (R^2 0.77) (r 0.88).

6.5.2.10 Removal of confounding of smoking, asthma and non-work-related symptoms: FVC plotted against height

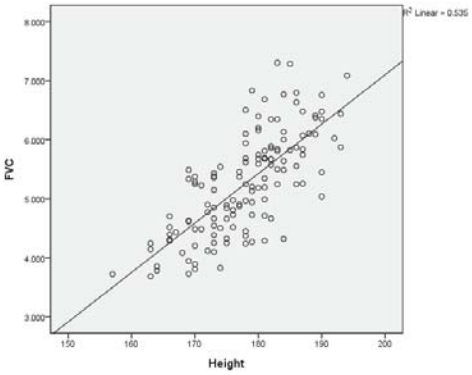

Figure 6.30 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Study Group on Removal of Smokers, Asthmatics and Individuals with Non-Work-Related Respiratory Symptoms (n = 134) (R^2 0.54) (r 0.73).

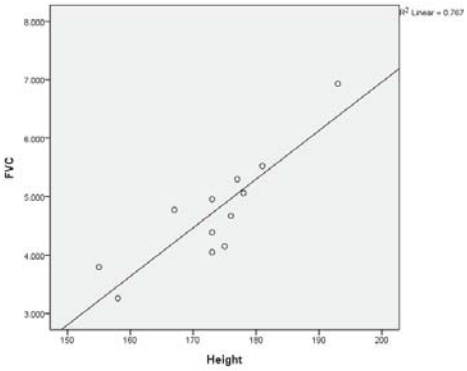

Figure 6.31 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Control Group on Removal of Smokers, Asthmatics and Individuals with Non-Work-Related Respiratory Symptoms (n = 12) (R^2 0.77) (r 0.88).

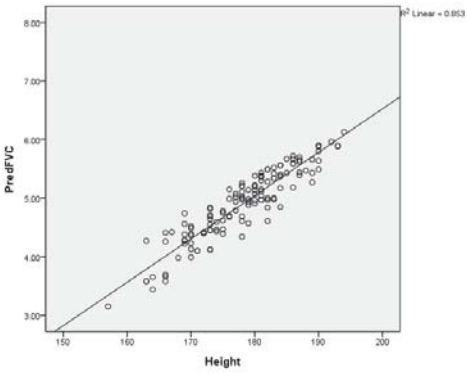

Figure 6.32 Scatter Plot Showing the Relationship of FVC (litres) and Height (cm) for the Predicted Values of the Study Group on Removal of Smokers, Asthmatics and Individuals with Non-Work-Related Respiratory Symptoms (n = 134) (R^2 0.85) (r 0.92).

6.5.3 Analysis of sequential removal of confounders

6.5.3.1 Goodness of fit (R^2) values of FEV_1 and FVC versus height as the confounders are removed

Table 6.3 Goodness of Fit (R2) for the Regression Plots for FEV_1 and FVC for the Study Group, as the Confounders are Removed, Compared with Their Predicted Values

Linear Regression	All Subjects	Minus Non-Work-Related Respiratory Disorders	Never Smokers	Never Smokers/ Non-Asthmatic	Never Smokers/ Non-Asthmatic No Non-work-related Respiratory Disorders	Predicted Value
n =	418	392	154	137	134	
FEV_1 v Height	$R^2 = 0.42$	$R^2 = 0.47$	$R^2 = 0.54$	$R^2 = 0.55$	$R^2 = 0.53$	$R^2 = 0.77$
FVC v Height	$R^2 = 0.49$	$R^2 = 0.51$	$R^2 = 0.57$	$R^2 = 0.56$	$R^2 = 0.54$	$R^2 = 0.85$

It can be seen that there was an improvement in goodness of fit (R^2) for the regression plots of the study group relative to the predicted values as confounders were removed. The confounders were smoking, asthma and non-work related (pre-existing) respiratory disorders, and therefore the converse descriptions – never smokers, non-asthmatic, no non-work related respiratory disorders – are headings in the table above.

6.5.3.2 r values of FEV_1 and FVC versus height as the confounders are removed

Table 6.4 Pearson's Correlation (r) for the Regression Plots for FEV_1 and FVC Plotted Against Height for the Study Group, as the Confounders are Removed, Compared with their Predicted Values

Linear Regression	All Subjects	Minus Non-Work-Related Respiratory Disorders	Never Smokers	Never Smokers/ Non-Asthmatic	Never Smokers/ Non-Asthmatic No Non-work-related Respiratory Disorders	Predicted Value
n =	418	392	154	137	134	
FEV_1 v Height	r = 0.65*	r = 0.69*	r = 0.74*	r = 0.75*	r = 0.73*	r = 0.88*
FVC v Height	r = 0.70*	r = 0.71*	r = 0.93*	r = 0.75*	r = 0.73*	r = 0.92*

Note. * Correlation is significant at the 0.01 level.

It can be seen that there is an improvement in the Pearson's correlation (r) for the regression plots of the study group relative to the predicted values as confounders were removed.

6.5.3.3 Independent t-test of FEV_1 for the study and control groups as the confounders are removed

Table 6.5 Independent t-test – Comparison of FEV_1 Between the Study and Control Groups

	All Subjects		Never Smokers		Never Smokers / Non-asthmatics	
	Study	**Control**	**Study**	**Control**	**Study**	**Control**
n=	418	40	154	13	137	13
mean	3.9L	3.6L	4.1L	3.7L	4.1L	3.7L
S.D.	0.74L	0.77L	0.71L	0.8L	0.69L	0.80L
p	<0.05		>0.05		>0.05	

Initially the FEV_1 for the study and control groups appear to be significantly different, but as the confounding was removed there appeared to be no significant difference for FEV_1 for these populations.

6.5.3.4 Independent t-test of FVC for the study and control groups as the confounders are removed

Table 6.6 Independent t-test – Comparison of FVC Between the Study and Control Groups

	All Subjects		Never Smokers		Never Smokers / Non-asthmatics	
	Study	**Control**	**Study**	**Control**	**Study**	**Control**
n=	418	40	154	13	137	13
mean	5.1L	4.6L	5.2L	4.6L	5.2L	4.6L
S.D.	0.93L	1.01L	0.93L	0.99L	0.91L	0.99L
p	<0.05		<0.05		<0.05	

Overall, there appeared to be a significant difference in FVC between the study and control group, even after the confounders were removed.

6.5.3.5 Dependent t-test of FEV_1 for the study group and their predicted values as the confounders are removed

Table 6.7 Dependent t-test – Comparison of FEV_1 for the Study Group and their Predicted Values as the Confounders are Removed.

	All Subjects n=418		**Minus Non-work-related Respiratory Disorders n=392**		**Never Smokers n=154**		**Never Smokers/Non-asthmatics n=137**		**Never Smokers, Non-Asthmatic No Non-work-related Respiratory Disorders n=134**	
	Study	Predicted	Study	Predicted	Study	Predicted	Study	Predicted	Study	Predicted
Mean	3.94L	3.96L	4.0L	4.0L	4.1L	4.0L	4.1L	4.0L	4.2L	4.1L
S.D.	0.74L	0.52L	0.67L	0.51L	0.71L	0.51L	0.69L	0.50L	0.62L	0.49L
p	>0.05		<0.05		>0.05		<0.05		<0.05	

There were mixed results on comparison of the FEV_1 for the study group and predicted values. There was no significant differences ($p > 0.05$) between the study and predicted values for FEV_1 without removal of any confounders and for removal of ever smokers; whilst there was a significant difference ($p < 0.05$) when ever smokers and asthmatics, and ever smokers and asthmatics plus those with respiratory disorders were removed.

6.5.3.6 Dependent t-test of FVC for the study group and their predicted values as the confounders are removed

Table 6.8 Dependent t-test – Comparison of FVC for the Study Group and their Predicted Values as the Confounders are Removed.

	All Subjects n=418		**Minus Non-work-related Respiratory Disorders n=392**		**Never Smokers n=154**		**Never Smokers/Non-asthmatics n=137**		**Never Smokers, Non-Asthmatic No Non-work-related Respiratory Disorders n=134**	
	Study	Predicted	Study	Predicted	Study	Predicted	Study	Predicted	Study	Predicted
Mean	5.1L	4.8L	5.1L	4.8L	5.2L	4.9L	5.2L	4.9L	5.3L	4.9L
S.D.	0.93L	0.64L	0.88L	0.63L	0.93L	0.63L	0.91L	0.62L	0.86L	0.60L
p	<0.05		<0.05		<0.05		<0.05		<0.05	

There were significant differences ($p < 0.05$) between the study and predicted values for FVC even on removal of confounding factors.

6.6 The Effect of Length of Service and Lung Function

6.6.1 Effect of length of service on the lung function for the presumed healthy sub-group of the study group

The effect of length of service (DateDiff) on lung function for the presumed healthy non-smokers of the study group (32%) is represented in the two regression plots below:

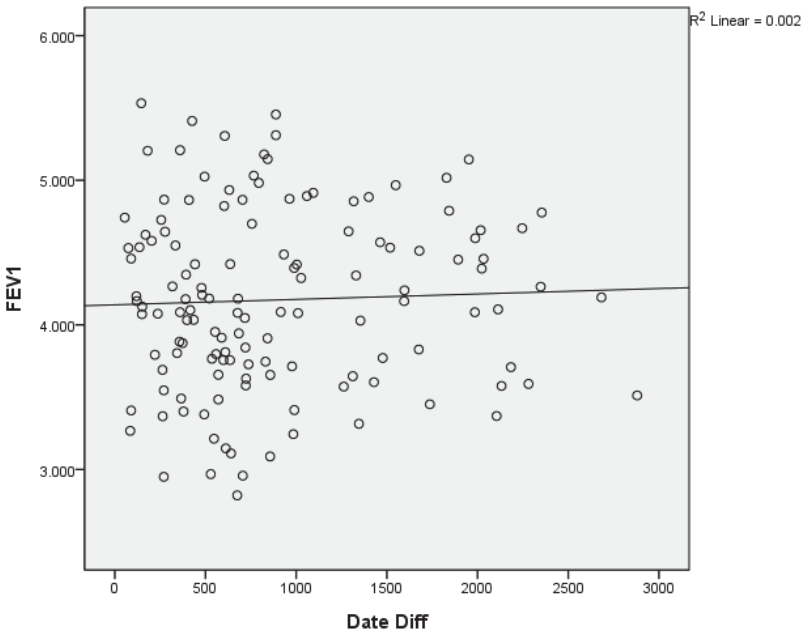

Figure 6.33 Scatter Plot Showing the Relationship of FEV_1 (litres) and Length of Service (DateDiff – days) for the Presumed Healthy Sub-Group of the Study Group (n = 134) (R2 0.002) (r 0.04)

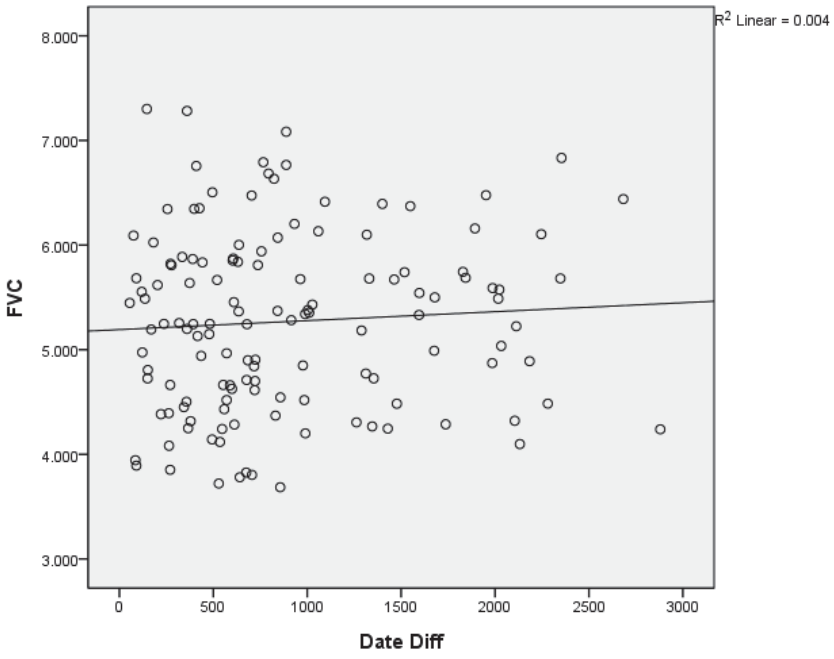

Figure 6.34 Scatter Plot Showing the Relationship of FVC (litres) and Length of Service (DateDiff – days) for the Presumed Healthy Non-Smokers of the Study Group (n =134) (R2 0.004) (r 0.07).

6.6.2 Comparison of the effect of length of service on the FEV_1 for the non-smoker and smoker sub-groups of the study group

The effect of length of service and FEV_1 for the study group non-smokers was compared with the study group smokers in the two plots below:

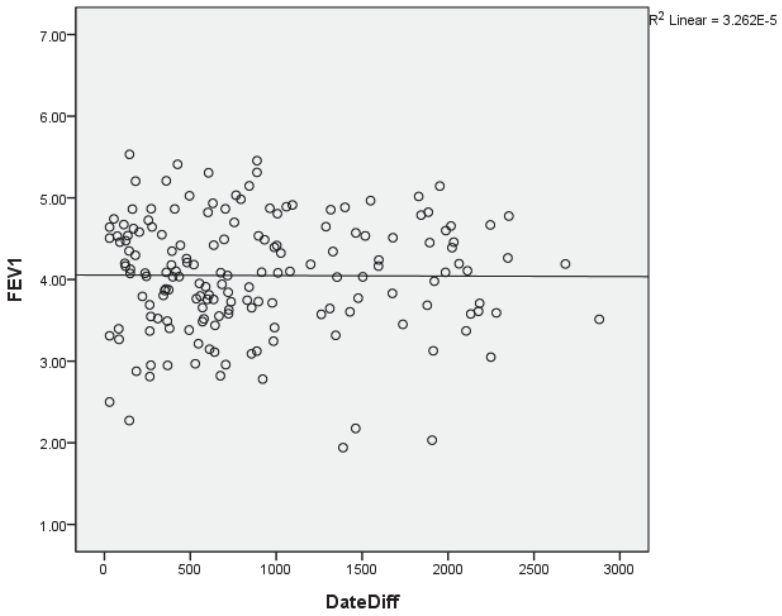

Figure 6.35 Scatter Plot Showing the Relationship of FEV_1 (litres) and Length of Service (DateDiff – days) for the Non-Smoker Sub-Population of the Study Group (n = 174) (R2 3.262E-5) (r 0.01).

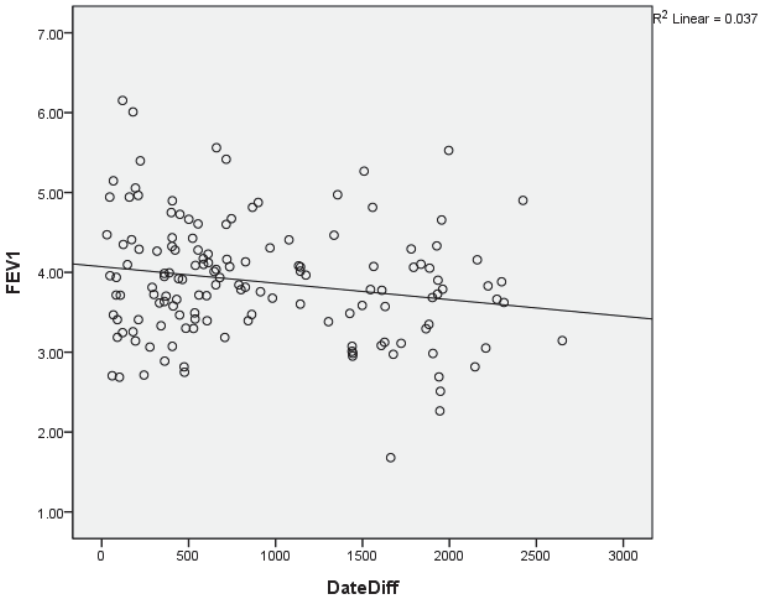

Figure 6.36 Scatter Plot Showing the Relationship of FEV_1 (litres) and Length of Service (DateDiff – days) for the Smoker Sub-Population of the Study Group (n = 143) ($R^2$0.037) (r minus 0.19).

6.6.2.1 MANOVA for the comparison of the effect of length of service on the FEV_1 for the non-smoker and smoker sub-groups of the study group

A multivariate analysis (MANOVA) showed there was no significant difference ($p > 0.05$) with length of service between the study group non-smokers and the study group smokers. However, this analysis showed a significant difference ($p < 0.05$) in FEV_1 between the non-smoker and smoker sub-groups of the study population. There was no similar effect for the FVC between the non-smoker and smoker sub-groups of the study population.

6.7 The Effect of the Area Worked and Lung Function

6.7.1 Comparison of the FEV_1 with the FEV_1 predicted values for individuals in each work area

A dependent (paired samples) t-test conducted to compare the FEV_1 and FEV_1 predicted values for the never-smoker sub-group in each work area is shown in the following table.

Table 6.9 Dependent t-test Comparing the FEV_1 with the FEV_1 Predicted Values for Individuals in Each Work Area.

Work Area	Mean	Standard Deviation	95% Confidence Limits		df	Sig. (2 –tailed)
			Lower	Upper		
Administration	-.060	.527	-.300	.180	20	.608
Mining Production	.102	.506	-.129	.332	20	.368
Mining Geologists	-.205	.393	-.830	.421	3	.374
Mining Maintenance	-.037	.386	-.650	.577	3	.862
Ore Leach Production	.140	.360	-.089	.369	11	.206
Ore Leach Maintenance	.012	.404	-.259	.283	10	.923

Refinery Production	-.063	.413	-.268	.142	17	.526
Refinery Maintenance	-.046	.421	-.279	.187	14	.679
Utilities Production	-.227	.623	-.748	.293	7	.336
Utilities Maintenance	.007	.572	-.522	.536	6	.974
Laboratory	.255	.508	-.135	.646	8	.170
Warehouse	.644	.479	-.118	1.405	3	.075
General Maintenance	.208	.369	-.040	.456	10	.092
Electrical Maintenance	.202	.208	.028	.377	7	.029*

Note. *p <0.05

The mean, standard deviation, degrees of freedom, and lower and upper confidence limits for all individuals in each specific work area are provided. A significant difference (p <0.05) was observed for the electrical maintenance work area. No other differences were observed for any other work groups (p >0.05).

6.7.2 Comparison of the FVC with the FVC predicted values for individuals in each work area

A dependent (paired samples) t-test was conducted to compare the FVC and FVC never-smoker sub-group in each work area is shown in table 6.10.

Table 6.10 Dependent t-test Comparing the FVC with the FVC Predicted Values for Individuals in Each Work Area

Work Area	Mean	Standard Deviation	95% Confidence Limits		df	Sig. (2 –tailed)
			Lower	Upper		
Administration	.198	.526	-.041	.438	20	.099
Mining Production	.376	.695	.060	.693	20	.022*
Mining Geologists	-.400	.708	-1.526	.727	3	.341
Mining Maintenance	.473	.791	-.786	1.731	3	.318
Ore Leach Production	.322	.518	-.007	.651	11	.054
Ore Leach Maintenance	.105	.409	-.170	.380	10	.415
Refinery Production	.327	.587	.035	.618	17	.030*
Refinery Maintenance	.235	.471	-.026	.495	14	.074
Utilities Production	-.128	.610	-.638	.381	7	.570
Utilities Maintenance	.395	.817	-.361	1.150	6	.248
Laboratory	.493	.705	-.049	1.034	8	.069
Warehouse	.829	.801	-.446	2.103	3	.130
General Maintenance	.512	.510	.170	.855	10	.008*
Electrical Maintenance	.512	.421	.160	.864	7	.011*

*p <0.05

A significant difference ($p < 0.05$) was observed for the following work areas:

- mining production;
- refinery production;
- general maintenance;
- electrical maintenance.

No significant difference was observed for the other work groups ($p > 0.05$).

6.8 Repeat Spirometry of 72 Mine Site Workers Involved in the Initial Study

Repeat lung function tests were conducted on a sample of 72 individuals from the initial 418 mine site workers. This population consisted of 25 non-smokers/non-asthmatics, 43 smokers and four asthmatics from the original study group. Five of the 72 individuals had known non-work-related respiratory disorders. Not all repeat testing was conducted at the same time interval. The time intervals from the initial spirometry test to the follow-up ranged from 173 days to 845 days.

The following table shows the

- mean change;
- range of change;
- overall mean change per year; of

FEV_1 and FVC as the confounders are sequentially removed.

Table 6.11 The Change in Lung Function Over Time for the Repeat Study Group with Sequential Removal of Confounders

	Number of Individuals (n)	Lung Function Parameter	Mean ± Standard Deviation (litres)	Range (litres)	Mean Time Period± Standard Deviation (days)	Overall Mean Change (ml/year)
All Subjects	72	FEV_1	-0.036±0.21	-0.54 to 0.39	623±198	-21
	72	FVC	-0.007±0.33	-0.74 to 0.79	623±198	-4
Smokers/ Asthmatics	47	FEV_1	-0.068±0.22	-0.54 to 0.39	616±192	-40
	47	FVC	-0.003±0.35	-0.74 to 0.79	616±192	-2
Non-Smokers/ Non-Asthmatics	25	FEV_1	0.024±0.17	-0.33 to 0.39	637±211	+14
	25	FVC	-0.013±0.29	-0.60 to 0.40	637±211	-7
Presumed Healthy, Non-Smokers	24	FEV_1	0.033±0.17	-0.33 to 0.39	633±215	+19
	24	FVC	<0.001±0.29	-0.60 to 0.40	633±215	0

6.8.1 Comparison of the change in FEV_1 over time for the presumed healthy, non-smoker and the smokers/asthmatics sub-groups from the repeat study

The comparison of the change in FEV_1 for the study group presumed healthy subjects, and the smoker/asthmatic sub-group, over the period from the initial to the repeat study is shown in Figures 6.37 and 6.38.

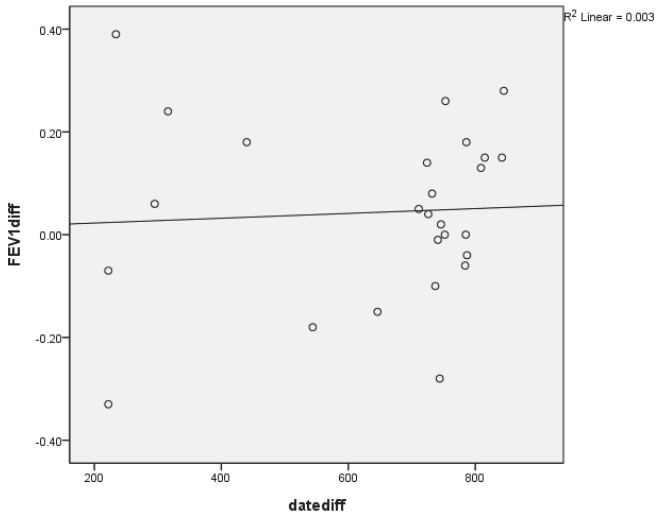

Figure 6.37 Scatter Plot Showing the Change in FEV_1 (FEV_1diff) in Litres, with the Period of Time Between Initial and Repeat Spirometry for the Presumed Healthy, Non-Smoker Sub-Group (n = 26) (R2 0.003) (r 0.058) (p >0.05).

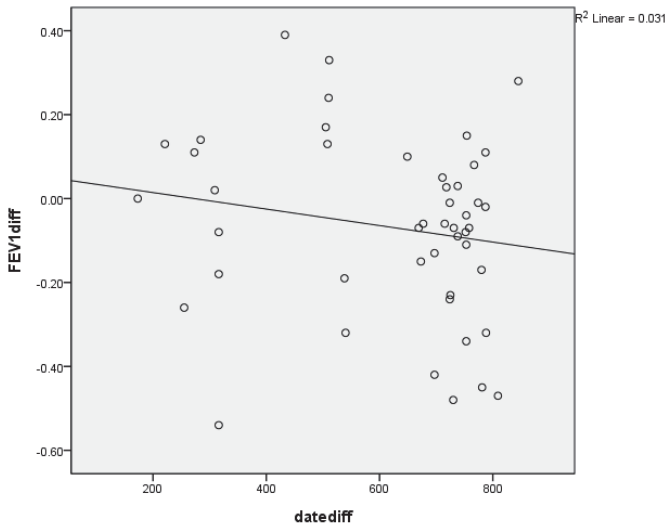

Figure 6.38 Scatter Plot Showing the Change in FEV_1 (FEV_1diff) in Litres, with the Period of Time Between Initial and Repeat Spirometry for the Smokers/Asthmatic Sub-Group (n = 47) (R^2 0.031) (r minus 0.18) (p <0.05).

In a dependent t-test there was no significant difference in FEV_1 from the first spirometry test compared with the repeat spirometry test for presumed healthy, non-smoker sub-group ($p > 0.05$). However, there was a significant difference ($p < 0.05$) in FEV_1 from first spirometry test compared with the repeat spirometry test for the smokers/asthmatics sub-group.

6.9 Cross-Swing Lung Function of a Cohort of Refinery Workers

Lung function tests for a cohort of refinery workers were conducted as they arrived for work on site prior to commencing work in the refinery, and on completion of their work period on site before returning home on their rest break. There was a mixture of dayshift and nightshift workers. There were 32 workers in total; however, five workers were repeated on a second swing. This equated to seven production workers and five maintenance workers from Crew A; 12 production workers from Crew B, three production workers from Crew C, and 10 maintenance workers from Crew D; giving a total of 37 pre-swing/post-swing observations.

6.9.1 Cross-swing change in lung function (FEV_1 and FVC) all 37 observations

Table 6.12 Cross-Swing Change in Lung Function (FEV_1 and FVC)

	Range (Litres)	Mean (Litres)	Standard Deviation	p
FEV_1	-0.58 to +0.52	-0.03	0.25	>0.05
FVC	-0.72 to + 0.95	+0.01	0.35	>0.05

There was no significant difference ($p > 0.05$) between the FEV_1 and FVC values for the 35 individuals (with 37 observations) from the start of a swing to the end of the swing.

6.9.2 Repeat cross-swing change in lung function (FEV_1 and FVC) for five individuals

There was no significant difference ($p > 0.05$) between the FEV_1 and FVC values for the five individuals from this cohort for their combined initial and repeat cross-swing lung function data.

6.10 Internal Reliability – Biological Control

As well as calibrating regularly during each batch of testing using a certified three litre syringe as per the ATS/ERS recommended standard; the lung function of the trained researcher was measured 41 times throughout the investigation to act as a biological control, for calibration purposes and to demonstrate internal reliability. The figures below (Figures 6.39 and 6.40) are scatter plots, and the table of descriptive statistics (Table 6.13) of the FEV_1 and FVC of the trained researcher over the study period.

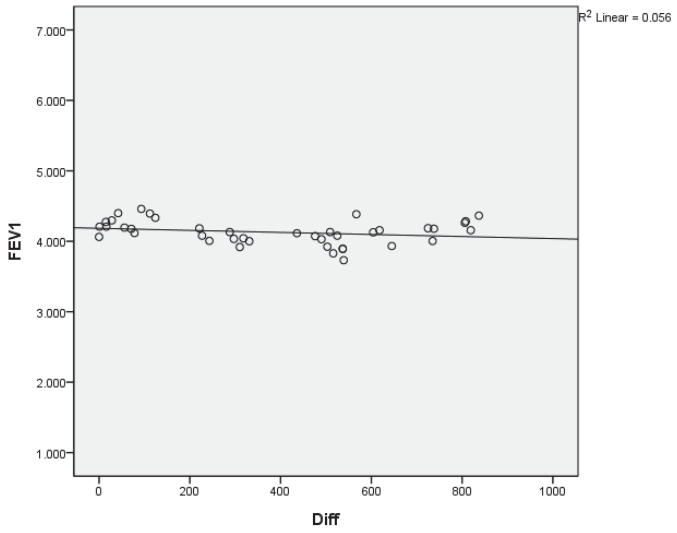

Figure 6.39 Scatter Plot Showing the FEV_1 (litres) Measured at Various Time Intervals [Diff] (days) Over the Study Period. (R^2 0.056) (r minus 0.237) ($p > 0.05$).

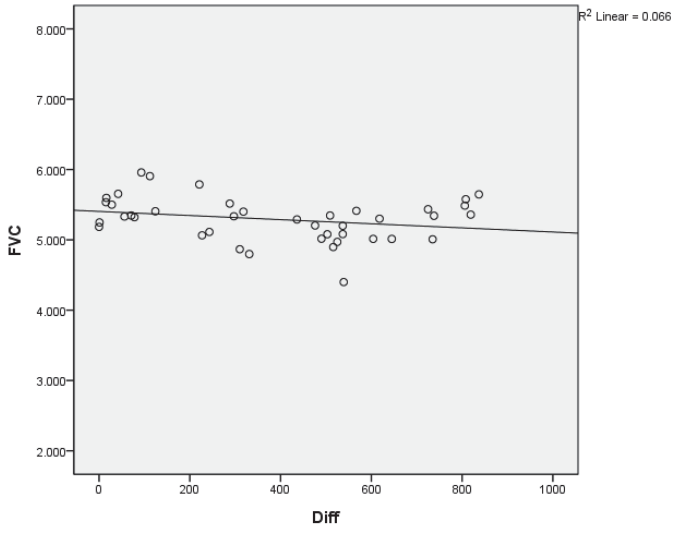

Figure 6.40 Scatter Plot Showing the FVC (litres) Measured at Various Time Intervals [Diff] (days) Over the Study Period. (R^2 0.066) (r minus 0.237) ($p > 0.05$).

Table 6.13 The Range, Mean and Standard Deviation of the Lung Function of the Biological Control over the Study Period

	Minimum	Maximum	Mean	Standard Deviation
FEV_1 (litres)	3.73	4.46	4.13	0.166
FVC (litres)	4.40	5.96	5.29	0.307

The outcome of these results for the:

- Initial study of the respiratory health surveillance of 418 employees at the Murrin Murrin Operation, compared with two control populations;
- Repeat study of 72 of these subjects; as well as the;
- Cross-swing study of a cohort of refinery workers;

is discussed in the following Discussion chapter.

7. DISCUSSION

7.1 Profile of the Study Group Compared With the Control Group

A total of 418 mining, processing and administrative employees based at the Murrin Murrin mine site constituted the study group. This group was compared with the control group of 40 catering personnel at the Murrin Murrin accommodation village some 8 kilometres from the mine site. The caterers were chosen for their geographic proximity to the mine site. The caterers rarely if ever visited the mine site or processing plant; the only exceptions were perhaps the bus drivers or delivery personnel where their time on site was limited. The control group were chosen for their proximity to the mine site to minimise any respiratory effect due to geographical location (Hendrick, 2002; National Institute for Occupational Safety and Health, 2009a).

7.1.1 Potential confounding due to the differences in the study and control populations

The potential confounding due to differences in the profiles of the study and control groups were:

- the male to female employee ratio in the study group was 92% to 8%; and for the control group it was 68% to 32%;
- the height range and mean height was different, with the study group range 155-200 cm, mean = 177 cm, and the control group range 155-195 cm, mean = 173 cm; and
- the smoking status was different in the study group compared with the control group (i.e., 38% non-smokers and 33% respectively).

7.1.2 Factors influencing lung function

There are positive and negative factors that affect lung function. It is well recognised that gender and height are the most important predictors of lung function with a linear correlation, whilst the relationship between age is more complex with a non-linear correlation (Pellegrino et al., 2005). It is known that excess BMI can impair lung function (Cotes, 1993). Genetic factors indeed influence lung function, as does a healthy lifestyle, body composition and respiratory muscle strength. Asthma is caused by both genetic and environmental factors and has a pronounced effect on lung function (Ryon

& Rom, 1998). Subjects with asthma or other respiratory disorders may well exclude themselves from such a mining operation (healthy worker effect). However, the most obvious negative factor is cigarette smoking, which has been determined to be the single most preventable risk factor for COPD (Blanc et al., 2009). Diabetes mellitus and obesity are amongst the disease states that have an indirect affect on lung function (Ostrowski & Barud, 2006).

To overcome the effects of confounding, the study design included predicted normal values (Zapletal et al., 1977) for gender, age, and height, computed into the software of the spirometer, against which to compare each individual's data. Potential confounding due to smoking status, being an asthmatic and pre-existing respiratory disorders were identified through the respiratory questionnaire and addressed during statistical analysis.

As to be expected, on statistical analysis (Simple Linear Regression, IBM® SPSS® PASW Statistics 18) there was a positive correlation between lung function and height and conversely a negative correlation with lung function and age (data not shown) for both the study and control groups.

The lung function of the study group was considered significantly different from both the control group and the predicted norm group (Zapletal, et al., 1977) as there was almost a consistent significant difference ($p < 0.05$) on comparison of their respective FEV_1s and FVCs. However, the R^2 values (goodness of fit) for the regression analysis appeared to become closer as the confounders related to poor lung function were removed (logistic regression). The difference of 5% more smokers in the control group was likely to account to some degree for the difference in prevalence of the non-work related respiratory disorders, which was 12.5% for the controls compared with 6.2% for the study group. There was a significant difference in both FEV_1 and FVC between the study group and their predicted values as all the obvious confounders (smokers, asthmatics and those with non-work related respiratory disorders) were removed. This may indicate the difference between a West Australian cohort (circa 2006) (i.e. the study group) and a European cohort (circa 1977) (i.e. the predicted normal values).

7.2 Initial Study

Data from the initial study consisted of the responses to the respiratory symptoms questionnaire and data from the lung function tests for both the study and control

groups. The questionnaires and measurements required for spirometry were initially conducted at the medical centre by appointment; however, as this proved impractical for most employees, the investigator conducted the study in the various work-area offices, and the accommodation office at the camp was used for the control group assessments. The procedures and instruments for measuring height, weight and lung function remained consistent and according to the ATS/ERS Guidelines (American Thoracic Society, 1995; Miller et al., 2005) and the Thoracic Society of Australia and New Zealand (Pierce and Johns, 1996).

7.2.1 Prevalence of respiratory symptoms in the study group compared with the control group

A comparison of the prevalence of respiratory symptoms in the study and control groups is given in Table 6.2 of the results chapter. These symptoms were determined by administration of a respiratory questionnaire.

7.2.1.1 Summary of the comparison of the study and control group respiratory symptoms

The pattern of respiratory symptoms determined from the questionnaire for the study group differed only slightly from that of the control group, differing predominantly at sub-question level where more detail was requested. The following contrasting responses to the respiratory questionnaire where the responses were greater for the study group are reported by exception.

- Although a slightly larger proportion of the control group reported they had a cough, proportionally more individuals in the study group reported that they usually coughed during the day and night (77% vs 10%). Also, 12 of the study group versus none in the control group reported it as a persistent cough;
- Five of the study group reported that they had experienced being short of breath at rest, in contrast to none in the control group;
- Proportionally more of the study group (6%) compared with the control group (3%) reported that during the past 3 years they had a chest illness which kept them from their usual activities for a week or more; and 2% of the study group reported that they brought up more phlegm than usual during this illness.

All these symptoms could be related back to individuals who were ever smokers or had known non-work related respiratory disorders. Cigarette smoking is known to cause coughing and particularly a persistent chronic cough that last longer than two to three weeks (Kerstjens, Rijcken, Schouten, & Postma, 1997) and to result in cough and phlegm (Heijdra, Pinto-Plata, Kenney, Rassulo, & Celli, 2002). Dyspnea, or shortness of breath, may be due to asthma or other respiratory or cardiovascular disorders (American Thoracic Society, 2003). The respiratory questionnaire was able to determine that none of these symptoms were work related.

7.2.2 Prevalence of respiratory disorders

The prevalence of respiratory disorders was determined by administration of a respiratory questionnaire and confirmed by spirometry (Section 6.4). In the control population there were five individuals (5/40 = 12.5%) with poor spirometry results (ndd Medizintechnik AG, 2002); three with mild obstruction, one with mild obstruction and low vital capacity, possibly due to restriction, and one with low vital capacity possibly due to restriction of lung volumes. In the study population there were 26 individuals (26/418 = 6.2%) with poor spirometry results: 18 with mild obstruction, five with mild obstruction and low vital capacity possibly due to restriction, and three with moderate obstruction and low vital capacity possibly due to restriction. All were pre-existing non-work-related (prior to working at Murrin Murrin) each with a history of respiratory illness and/or smoking.

Subjects with interpretations on the spirometry report with mild obstructive respiratory disorders included subjects with known asthma, bronchitis, and emphysema; these disorders were also determined prior to spirometry through interview using the respiratory questionnaire. Those diagnoses with low vital capacity possibly due to restriction were mainly smokers however there were three cases where other respiratory disorders that had been identified and medically treated. These interpretations provided on the report from the EasyOne spirometer not only correspond with the detail provided by the study group subjects but also to the pathophysiological profile of these respiratory disorders (Johns Hopkins School of Medicine's Interactive Respiratory Physiology, 1995).

A detailed analysis for the study group determined that there were 26 cases of non-work-related respiratory disorders each either with a history of respiratory illness, or

smoking, and elevated BMI, or a combination of these. These are listed in section 6.4.2 of the results section.

7.2.3 Comparison of the lung function of the study group, with their predicted values, and the control group – with sequential removal of confounders

The lung function of the study group (Figure 6.3) was compared, with the:

- control group (Figure 6.4); and
- predicted values for the study group individuals (Figure 6.5).

This was followed by a series of evaluations after sequential removal of the obvious confounding factors such as smoking, asthma and known non-work-related respiratory symptoms. The confounding of age and gender was addressed by comparison with their predicted values (Zapletal et al., 1977).

The series of analysis began with all subjects with no data removed from the study group (418 individuals on site) or control group (40 caterers off-site). Next, the ever smokers data were removed, followed by ever smokers and asthmatics, and finally the ever smokers, asthmatics and the 26 individuals with known non-work-related respiratory symptoms (Figures 6.3 – 6.32).

Two analyses were employed. Firstly, the FEV_1 and FVC were plotted against height to visualise the relationship of the study group versus the control group, and the predicted normal values, and a simple linear regression employed. Secondly, comparison of the means of the study group data versus the control group data (independent t-test) and the predicted normal values (dependent t-test) was also conducted (IBM® SPSS® PASW Statistics 18).

It can be seen from the first in the series of regression plots (Figures 6.3 – 6.5) that the lung function data (FEV_1 and FVC) for all subjects in the study group (418) and control group (40) were more disperse than the predicted normal values (i.e., many outliers). This was considered mainly due to confounding factors such as smoking and individuals with known non-work-related respiratory disorders; whereas the predicted values (Zapletal et al., 1977) were derived from a representative sample of a healthy, non-smoking population.

On more detailed examination of the respiratory questionnaire, the factors contributing to this disperse nature (scatter) for the study group data compared with the predicted values for these group individuals were outliers: those with poor lung function, including the 26 cases determined to have non-work-related respiratory disorders, a history of smoking, elevated BMI, or a combination of these; and those with excellent lung function who reported a high level of physical activity, such as scuba diving – known for 'large lungs' (Tetzlaff et al., 2006) – which were associated with the best lung functions. The increased diversity in spirometry results for both the study and control groups, as compared with the predicted values for the study group data, was reflected in the R^2 (linear regression goodness of fit) values for the FEV_1 or FVC versus height linear model as shown in Figures 6.3 – 6.5.

There was an improvement in the (goodness of fit) R^2 values and the Pearson's correlation (r) values for the series of regression plots depicted in Figures 6.3 through to 6.32, for the study group relative to the predicted values, as the confounders of

- individuals with known non-work-related respiratory disorders;
- ever smokers; and
- asthmatics,

were sequentially removed, and they more closely resembled the predicted values. These data are summarised in Tables 6.3 and 6.4. It can be seen that the study group lung function data never fully resembles the predicted value data, that could be due to the fact that the study group was from an Australian population whereas the Zapletal et al. (1977) lung function data was derived from a cross-section of healthy children, adolescents and adults from a European population. These predicted values were the manufacturer's recommendation as best reflecting the Australian population and were programmed into the spirometer, as there are no similar Australian predicted values. This population difference in lung function between the study group and their predicted values was also demonstrated through a series of t-tests summarised in Tables 6.7 and 6.8. The results demonstrate that overall there was a consistent significant difference in FVC even as the confounders were removed ($p < 0.05$) and a significant difference in FEV_1 when the confounding of smoking, asthma and other non-work-related respiratory symptoms were removed from the data ($p < 0.05$) thus demonstrating that overall there was a significant difference in FEV_1 and FVC between the study group and the predicted values. This is consistent with the linear regression where there was more

scatter of data around the line of best fit for the study group data compared with the predicted values for these data (comparison of Figures 6.27 with 6.29 for FEV_1, and Figures 6.30 and 6.32 for FVC).

7.2.4 Length of service

7.2.4.1 Study group

The effect of length of employment at the Murrin Murrin site (length of service) on lung function was investigated using linear regression plots, and by Multivariate Analysis of Variance for the main study group *presumed healthy workers* (i.e., non-smokers, non-asthmatics, and no non-work-related respiratory symptoms) (IBM® SPSS® PASW Statistics 18).

The regression plots for FEV_1 and FVC for the presumed healthy workers were relatively flat indicating no decrease or increase in FEV_1 or FVC with length of service (Figures 6.33 and 6.34). In addition, the Pearson's Correlations were not significant ($p > 0.01$) also indicating there was no effect of length of service on the lung function for the study group.

7.2.4.2 Contrast between the non-smoker and smoker sub-groups of the study group

On analysis, a contrast was shown between the non-smoker (Figure 6.35) and smoker (Figure 6.36) sub-groups. There was an evident decrease in FEV_1 with length of service for the smoker sub-group whilst there was no decrease for the non-smoker sub-group with length of service (IBM® SPSS® PASW Statistics 18). Again, the dispersion of the data was evident as the R^2 values were small (R^2 3.262E-5 and R^2 0.037 respectively). However, the Pearson's correlation for length of service for the smoker sub-population was significant at the 0.01 level indicating a decrement in FEV_1 over time for the smokers. In addition, the Multivariate Analysis of Variance (MANOVA) (Section 6.6.2.1) showed no significant difference in FEV_1 with length of service between the non-smokers and smokers ($p > 0.05$). However, there was a significant difference in FEV_1 between non-smokers and smokers ($p < 0.05$).

Therefore there was no decrease in FEV_1 for the study group presumed healthy workers with length of service at the Murrin Murrin mine site and processing plant. In

contrast there was a decrement in FEV_1 due to smoking associated with time (length of service) indicating that there was no work-related effect using this study protocol for this study period.

7.2.5 Effect of area worked and lung function

A dependent t-test (IBM® SPSS® PASW Statistics 18) was conducted to determine if there were any statistically significant changes in FEV_1 and FVC (Tables 6.9 and 6.10 respectively) compared with their predicted values for the never-smoker populations in the 16 work areas studied. There appeared to be a degree of variation in the mean values of FEV_1 (Table 6.9) and FVC (Table 6.10) across the workgroups; however, this was considered to be within the between-subject variation in lung function (Spirxpert, n.d.).

There were insufficient observations to conduct statistical analysis for two of these work areas, calcrete and pastoral, when the ever smokers were removed from the data. Statistical analysis revealed that there was no significant difference in lung function for most work areas. There were significant findings which indicated a slightly positive increase in lung function. There was a significant difference ($p < 0.05$) in FEV_1 for the electrical maintenance group; and a significant difference ($p < 0.05$) in FVC for the mining production, refinery production, general maintenance and electrical maintenance workers.

These increases were considered to be slight increases as the various regression plots relating to lung function over time for the non-smokers and presumed healthy worker groups were essentially flat (horizontal) indicating no decrease in lung function. Therefore there was no decrease in lung function for the never-smoker populations in 14 work areas studied at the Murrin Murrin mine site and processing plant.

7.3 Repeat Study

The initial aim was to conduct the repeat spirometry study at a 1-year interval. However, due to work commitments this was not possible; therefore this was extended to a 2-year interval. Ultimately the mean time period for the repeat spirometry tests was 1.7 years (range 173-845days) because the Murrin Murrin workforce had a high attrition rate at the time (i.e., workers leaving employment) of approximately 51% (Minara Resources, Human Resources, personal communication). Hence repeat lung function tests were conducted on a sample of 72 of the initial 418 mine site workers to include a

cross-section of workers known to have been employed for approximately six months or longer, purposely including those known to have poor lung function (five of the 72). This sample consisted of 29 non-smokers (four of these were asthmatic) and 43 smokers, from the original study group. Best attempts were made to gather a cross-section of workers from all work areas. A comparison of their initial and repeat lung function tests were statistically analysed to determine if there was a decrement in lung function over time from initial to repeat test. It has to be noted that the difference in time interval (173-845days) has the potential to create bias as there may be a dilution of a possible effect on lung function associated with the shorter exposure time on site.

7.3.1 Difference in lung function over time

Table 6.11 shows the change in lung function over time for the repeat study group, plus the change in lung function with time on sequential removal of the confounding factors of smoking, asthma, and other non-work-related respiratory symptoms. The data indicate that there was a decrease in both FEV_1 (21 ml/year) and FVC (4 ml/year) for all 72 cases of the repeat study group. The decreases were most marked for the smokers and asthmatic sub-group (with FEV_1 a decrease of 41 ml/year and FVC 2 ml/year). However, as the confounders were removed, ultimately resulting in the presumed healthy sub-group, there was no decrease in FEV_1 and FVC. In fact, there was a slight increase in FEV_1 of 19 ml/year. This slight increase was possibly due to a learning effect; that is, improved spirometry technique due to an individual's ability to do better on a repeat spirometry test as they have mastered and improved their technique (Nield & Burmas, n.d.).

The range of change in FEV_1 and FVC was narrower for the non-smokers/non-asthmatics than for the smokers/asthmatics, which matched the range for all subjects (72) individuals, indicating that smokers, asthmatics and those with known non-work-related respiratory symptoms were mostly responsible for the largest decreases and increases (outliers) in lung function. This was confirmed by reviewing the respiratory questionnaires of these individual outliers. These were for individuals with either known non-work-related respiratory disorders or heavy smokers. This was reflected in the standard deviation for all subjects which were comparatively large (Table 6.11). The greatest decrement in FEV_1 was observed for an individual with known non-work-related respiratory symptoms (recent pneumothorax, and a smoker). The greatest

decrement in FVC was observed in an asthmatic diabetic individual. The outliers in FEV_1 and FVC for the presumed healthy sub-group were for one individual whose questionnaire indicated nothing abnormal, and the spirometry reports were normal on both occasions. The only distinguishing difference was that one spirometry session was quality *B* and the repeat spirometry *C*, both considered to be acceptable (ndd Medizintechnik AG, 2002). There were also improvements in FEV_1 and FVC for the presumed healthy sub-group for two different individuals. The reasons for these improvements were not determined, but may be due to increased physical activity, improved spirometry technique, or a recovery from a respiratory illness which was not ascertained during interview for the respiratory questionnaire.

As demonstrated in Table 6.11, the mean change for FEV_1 of minus 40 ml/year for the smokers/asthmatic sub-group was statistically significant ($p < 0.05$). This decrement was also considered biologically significant, significant beyond the natural decrease in FEV_1 with age (Kerstjens, Rijcken, Schouten, & Postma, 1997; Oasys, 2006). This significant decrement over time appeared to be associated with the smokers and asthmatics because on analysis when these confounders were removed (i.e., the non-smokers/non-asthmatics sub-group) there was no significant difference in the means for FEV_1 from the initial to the repeat study ($p > 0.05$) (IBM® SPSS® PASW Statistics 18).

To remove further confounding, the data from five individuals with known non-work-related respiratory symptoms were removed. One of these individuals had a decrement of 330 ml over a 732-day period (equivalent to 165 ml/year). On removal of all known confounding data, the resulting presumed healthy sub-group had a mean change of plus (an increment of) 19 ml/year and no change in FVC (neither a positive or negative change).

In contrast, for the smokers/asthmatics sub-group there was a decrease in FEV_1 (minus 40 ml/year) and a relatively small decrease in FVC (minus 2 ml/year) (Table 6.11) which appears to be the typical profile of the early effects of mild smoking (Kerstjens et al., 1997; Heijdra, Pinto-Plata, Kenney, Rassulo, & Celli, 2002).

Overall, there was no decrease in lung function for the presumed healthy sub-group, over the time from initial spirometry to the repeat spirometry, for the Murrin Murrin Operation personnel, whereas there was a decrease in lung function for the smoker/asthmatic sub-group in this repeat study.

7.4 Cross-Swing Lung Function in a Cohort of Refinery Workers

Lung function tests for a cohort of refinery workers were conducted as they arrived for work on site prior to commencing work in the refinery, and on completion of their work period on site before returning home on their rest break (known as a *swing*) (Section 6.9 of the results chapter). All but three of these were smokers. Despite this, there was no significant change in lung function from the start of swing to the end of swing for this cohort of refinery workers.

Twenty-nine of the 32 refinery workers were smokers. Three members of this cohort had mild obstruction as diagnosed by spirometry, one had a history of pneumothorax and was a current smoker, and one was an asthmatic and a smoker. Another with normal spirometry had a history of non-work-related pneumonia and smoked 20 cigarettes per day.

7.4.1 Cross-swing FEV_1

The cross-swing FEV_1 results in Table 6.12 in the results chapter show that the maximum decrease in FEV_1 was minus 0.58 L, with a maximum increase in FEV_1 of 0.52 L. The maximum decrease in FEV_1 was observed for a smoker with a history of (non-work-related) pneumonia. The maximum increase (improved) FEV_1 was observed in a heavy smoker with a history of (non-work-related) bronchitis.

There was a mean decrease for the cohort of minus 0.03 L, from the start to completion of the work period, for the cross-swing refinery worker cohort. This 30 ml decrease was not significant ($p > 0.05$).

7.4.2 Cross-swing FVC

The maximum decrease in FVC for this cohort was minus 0.72 L, with a maximum increase in FVC of 0.95 L. The 0.72 L decrease was observed for a mild smoker, and the improvement in FVC of 0.95 L was observed for a heavy smoker with a history of non-work-related bronchitis. Despite these large volume changes, both were recorded as normal spirometry (ndd Medizintechnik AG, 2002) on the spirometer records.

The maximum negative change (minus 0.72 L) was a 12% change over a period of 14 days. Although it was recorded as normal spirometry (ndd Medizintechnik AG,

2002) a follow-up of this individual was requested on the grounds that such a decrease is considered of clinical significance even in normal subjects (American Thoracic Society, 1995; Wang & Petsonk, 2004; Mason, Broaddus, Murray, & Nadel, 2005). The spirometry from this individual has since been consistently classified as normal spirometry (Mary Morrissey, Minara Resources, personal communication, March, 2011).

Despite the large standard deviation, with some individuals showing changes of up to 17% in their lung function, there was no statistical significant difference ($p > 0.05$) in lung function for this cohort from the beginning of the swing to the end of the swing for either FEV_1 or FVC for these 37 observations (Table 6.12). Nor was there a statistical significant difference ($n = 10$, $p > 0.05$) in the repeat cross-swing lung function for five of these refinery workers.

7.5 Limitations

No matter how well a study is conducted there will always be limitations, such as study design, attrition, missing data, unknown confounding factors, instrumentation, and data analysis (Checkoway et al., 2004).

7.5.1 Main study

It was considered that there was no selective failure to participate as all the control group members agreed to participate and only two of the 420 study-group members refused to participate. However, there was attrition during collection of the initial study-group data as workers were joining and leaving employment at the Murrin Murrin Operation. Work rosters were used to capture all employees in each work area until the final number of 418 was reached. This may have produced a 'healthy worker effect' (Checkoway et al., 2004) whereby individuals with work-related respiratory disorders left the Murrin Murrin workforce, which was considered unlikely.

7.5.2 Repeat study

Follow-up of workers leaving the workforce was purposefully not included in the study design and in the ethics protocol hence there was loss of follow-up in the repeat study. However, best attempts were made to gather a cross-section of workers from all work areas hence there may have been some selection bias, as well as a potential 'healthy worker effect' due to the possibility of individuals with work-related

respiratory disorders leaving the workforce. Attrition is reported to be a major limitation of longitudinal studies (Checkoway et al., 2004). Attrition was an issue with this study. The attrition rate of approximately 51% over this period would have introduced new workers with less time on site (causing a dilution of the effect) therefore attempts were made to focus on the longer-serving members of the workforce and limiting it to those with service of greater than six months; in the event, the range of length of service was from 0.47 years to 2.3 years, with a mean of 1.7 years. Smokers and those with known non-work-related respiratory symptoms were included with the intention of seeking any potential synergistic effect.

Due to the attrition rate impeding the repeat study, the length of service (time worked on site, [minimum 1 month, maximum 8 years with a mean of 2.5 years]) in the initial study was also analysed.

7.5.3 Missing data

Missing data can seriously affect the outcome of a study, and can distort or miss-represent the sample (IBM SPSS Statistics 18, 2010).

Every attempt was made to acquire all data. It was very rare for individuals not to divulge information and, since anonymity was assured, it is likely that the questionnaires completed by interview by an 'approved person' were answered truthfully. For example, Patrick et al. (1994) states that reporting of smoking is usually accurate.

Two sets of data were excluded as the quality rating for the spirometry tests did not meet the quality criteria; one person rushed the test and did not complete it, and the other was disqualified due to inadequate effort, which was also indicated as a substandard quality rating on the spirometer read-out (ndd Medizintechnik AG, 2002). The remaining spirometry data met the strict protocol requirements.

Therefore most data was missing at random and the IBM® SPSS® PASW Statistics 18 excluded these cases for that particular value during statistical analysis.

7.5.4 Variability

Variability may occur due to misclassification of the spirometry results due to the difference in the outcome between sessions, where in one session the data for an

individual are within the normal range, but at another session they are outside that range. This is likely to create within- and between-subject variability. The intra-individual standard deviation of repeated measurements of FEV_1 and FVC in a healthy adult is considered to be about 200 ml and about 340 ml respectively (Rozas & Goldman, 1982). To reduce within-session variability the ATS/ERS criteria (American Thoracic Society, 1995; Miller et al., 2005) and the manufacturers (ndd Medizintechnik, 2002) guidelines were rigorously applied. Moreover a single competent person collected all data using the same instrument to conduct the spirometry tests.

7.5.4.1 Intra-individual variation

Intra-individual variation is mainly due to physiological issues, environmental issues, the instrumentation and the conduct of the lung function tests. Physiologically lung function is known to vary mainly due to stature and age (Chinn, Cotes, & Martin, 2006). However, the largest variability in this study was due to confounding factors: non-work-related factors such as smoking, non-work-related respiratory diseases such as asthma, effects of overweight and cardiovascular diseases such as diabetes, which are known to have a pronounced effect on lung function (Poirier et al., 2006). The standard deviation of results on occasions was considered high (for example, in the cross-swing study of the refinery workers) but observed to be caused by the confounders introduced by individuals with known non-work-related respiratory disorders, and smoking. The study protocol was strictly adhered to in order to limit all but the physiological issues.

7.5.4.1 .1 Internal validity – biological control

A biological control with known normal lung function was incorporated into the study protocol to monitor intra-individual variation in lung function. The lung function of the trained researcher was repeatedly measured throughout the investigation to act as a biological control to demonstrate internal validity. There was no significant ($p > 0.05$) decrease in lung function for this individual over the study period, and although there was an apparent decrease normally attributed to age this was not significant (r - 0.24 for both FEV_1 and FVC) (Figures 6.39 and 6.40). This was consistent with the study findings. The standard deviation in FEV_1 was 166 ml, and for FVC was 307 ml, which is consistent with the intra-individual standard deviation, quoted by Rozas & Goldman (1982).

7.5.4.2 Between-subjects variation in FEV_1

Between-subjects variation in lung function is largely affected by subjects with respiratory disorders, although there have been shown to be diurnal variation (Spirxpert, n.d.).

As demonstrated in Tables 6.5 and 6.7:

- The mean ± SD for FEV_1 for all subjects in the study group was 3.94 ± 0.74;
- The mean ± SD for FEV_1 for cases with no known non-work-related respiratory symptoms in the study group was 4.0±0.67;
- The mean ± SD for FEV_1 for all subjects in the control group was 3.6±0.77.

7.5.4.3 Between-subjects variation in FVC

As demonstrated in Tables 6.6 and 6.8:

- The mean FVC for all subjects in the study group was 5.1±0.93;
- The mean ± SD for FVC for cases with no known non-work-related respiratory symptoms in the study group was 5.1±0.88;
- The mean ± SD for FVC for all subjects in the control group was 4.6±1.01.

7.5.4.4 Effect of learning

It has been suggested that the effect of learning the spirometry technique may improve the lung function results of a repeat test (Nield & Burmas, n.d.). Such variation is more likely to be due to the difference in equipment used and the variety of people conducting the spirometry test. However, if spirometry is conducted to the ATS/ERS criteria this variation should be eliminated.

7.5.4.5 Climate or other factors

It is possible that climate or other factors may have confounded the results. To eliminate the possible effect of climate a control population close to the Murrin Murrin operation was chosen.

To reduce possible bias of atmospheric pressure during lung function testing, the spirometer was pre-programmed with the altitude of the Murrin Murrin operation. The model of spirometer used for this study was pre-programmed to remove any effects of temperature and relative humidity at the time each lung function test was performed.

7.5.5 Internal validity

Internal validity is the assurance that can be given to a cause and response relationship in a study (Checkoway, et al., 2004). In this study the lung function data of smokers acted effectively as a positive control, whilst the lung function of a healthy non-smoking person (biological control) effectively acted as a negative control for internal validity.

7.5.5.1 Smokers decrease in lung function

A consistent negative effect on FEV_1 among smokers was demonstrated throughout this study. This was seen on analysis of the ever smokers for both the study and control groups with FEV_1 plotted against pack years. Here, a significant decrement in FEV_1 was observed (Figures 6.1 and 6.2). Both of these gave a negative correlation significant at the 0.01 level. Similarly, there was a decrement in FEV_1 for smokers with length of service (time worked on site) (Figure 6.36) significant at the 0.05 level. Again, a decrement in FEV_1 was observed in the repeat study. Here, there was a significant difference ($p < 0.05$) in FEV_1 from the first spirometry test compared with the repeat spirometry test for the smokers/asthmatics sub-group (Figure 6.38).

As the confounder of smoking was removed from most data an improvement appeared with lung function data, specifically FEV_1, as smoking appeared to affect FVC to a lesser extent.

7.5.5.2 Biological control (lung function of the approved person)

The lung function of the approved person conducting the lung function tests was taken on 41 occasions during each batch of testing for calibration purposes and for internal validity. The linear regression plots of both FEV_1 and FVC versus time difference between spirometry were effectively horizontal indicating that there was little to no change in FEV_1 and FVC with time (Figure 6.39 and 6.40). The dispersion of the

data was evident as the goodness of fit R^2 values were small ($R^2 = 0.056$ and $R^2 = 0.066$ respectively).

On further analysis, the Pearson's correlation for:

- FEV_1 with time was r = minus 0.237; p >0.05; and
- FVC with time was r = minus 0.258; p >0.05.

This indicates that there was no significant difference in both lung function measures and that there was a negative relationship, perhaps starting to indicate the effect of age (Sharma & Goodwin, 2006).

It is considered that this study protocol was sensitive enough to detect a decrement in lung function due to smoking over the study period, and that there was no significant difference in FEV_1 and FVC with time for the biological control.

7.6 Correlation with the Known Work-Area Exposure Levels

Because there was no overall decrement in lung function for the Murrin Murrin Operation employees for this study period, the correlation with the known occupational exposure levels in each work area became redundant. The absence of an effect on lung function is considered to be reflective of the actual work-area exposure levels which were invariably well below the regulatory occupational exposure standards.

7.7 Reasons for the Absence of an Effect on Lung Function for the Main Study and Repeat Study and the Cohort of Refinery Workers

The outcome of this study was that there was no overall decrement in lung function for the Murrin Murrin Operation employees for the period each individual worked on site, or when working in specific work areas at the mine site or the processing plant for this study period. This result was consistent in all phases of the study. Statistical analysis showed that with length of service on site, in the repeat study, and the biological control over the study period, there was no overall decrease in lung function. In contrast, the effect of smoking was detected across all phases, acting as internal validity (positive control) that an effect could be detected by these research tools.

The results of the study provide evidence that primary preventive measures aimed at protecting workers are effective. Point source emissions are contained within the

environmental operating licence limit and the workplace exposures are generally maintained to levels below their respective occupational exposure standards. In instances where exceedances were likely, adequate personal protective controls were implemented. The data therefore suggest that the mine and process plant safety and environmental programs were effective. These primary preventive measures include:

- plant and process design;
- plant integrity and maintenance;
- controlled process chemistry;
- plant and process controls;
- process operation and control, including instrumentation, training/competency, and operating procedures.

Secondary preventative measures (backup controls) were also in place to detect process upsets and equipment failure, for example, control-room instrumentation and control-room operators. Tertiary preventative measures included strategically placed emission monitoring alarms around the processing plant, emergency response planning and training, and finally emergency evacuation and respiratory protection as a last resort in their hierarchy of control. Improvements to their program have been implemented such as dust reduction projects in the nickel and cobalt buildings.

It is essential, however, that Minara Resources continuously monitor and review these controls to ensure their effectiveness and to make further improvements where possible.

7.8 Addressing the Smoking Issue

It is important not to ignore the effect of smoking – although this is a lifestyle choice, not strictly a workplace issue – by providing the opportunity for employees to enter a smoking cessation program, as Australian workplaces will ultimately benefit in the long term.

DMP (2010b) recognises that smoking is a contributory risk factor to worker health, and promotes the health of people engaged in mining operations. Implementing a Health Ownership Model (Cameron, 2010) by addressing both workplace health needs and individuals' health needs, in this case by encouraging and supporting employees to quit smoking, will be beneficial to both the company and the individual.

7.9 Summary

This research set out to determine if there was a possible adverse respiratory effect of concurrent and repeat exposures to complex mixtures of low-level airborne chemicals at the Murrin Murrin operation. The overall outcome of this study was that there was no overall decrement in lung function for the Murrin Murrin Operation employees for the period each individual worked on site, or when working in specific work areas at the mine site or the processing plant for this study period. It could be argued that this time period may be too short to pick up a long-term effect, however, since commissioning the processing plant had been open for more than 5 years, and mining activity prior, with the maximum length of service in this study of 8 years. The length of service of participants ranged from 1 month to 8 years, (mean of 2.5 years). Also, this result was consistent in all phases of the study. Statistical analysis showed that length of service on site, in the repeat study, and the biological control over the study period, was not related to a decrease in lung function. In contrast, the effect of smoking was detected across all phases, acting as internal validity (positive control) that an effect could be detected by these research tools.

The conclusions and recommendations arising from this study are provided in the next and final chapter.

8. CONCLUSION AND RECOMMENDATIONS

The concern shown by the employees at the Murrin Murrin Operation that workplace emissions may be harming their respiratory health appears to be dispelled by this study.

The purpose of this study was to detect possible adverse respiratory health effects at an early stage in order to prevent potential long-term occupational respiratory disease in the Murrin Murrin workforce, and then, if necessary, to recommend interventions to prevent untoward health effects, and enable management to have a proactive approach to the protection of the workforce. No work-related respiratory health effects were detected for this workforce relative to the workforce's length of service and over the study period. There are, of course, limitations to this study as it really only addresses some of the more acute respiratory health issues and insufficient time elapsed to examine any long-term effects.

8.1 Initial Study

8.1.1 Prevalence of respiratory symptoms in the study group compared with the control group

In combination, the respiratory symptoms established from the questionnaire and from spirometry determined that in the control group population there were 12.5% individuals with non-work-related respiratory disorders compared with 6.2% in the study group. All these symptoms were related back to individuals who were Ever Smokers or had known non-work-related respiratory disorders. A detailed analysis of the data from the 418 participants in the study group discovered that there were 26 cases of non-work-related respiratory disorders.

8.1.2 Length of service

On statistical analysis, with these 26 individuals and the smokers' data removed (i.e., the presumed healthy workers sub-group) there was no overall decrement in lung function for the Murrin Murrin Operation employees with length of service ($p < 0.01$).

8.1.3 Effect of area worked

In addition, there was no decrease in FEV_1 associated with the Never Smokers in the 14 work areas studied at the mine site or processing plant when compared with their predicted FEV_1 values.

8.2 Repeat Study

Similarly, in the repeat study, there was no significant difference in FEV_1 from the first spirometry test compared with the repeat spirometry test for the presumed healthy sub-group ($p > 0.05$).

8.3 Cross-Swing Study of a Cohort of Refinery Workers

The cross-swing study of a cohort of 35 refinery workers indicated there was no (before and after) decrement in lung function from the start of a swing to the end of a swing ($p > 0.05$). In addition, for a repeat cross-swing study for five of these individuals, again, there was no significant difference ($p > 0.05$) between the FEV_1 and FVC values.

8.4 Effect of Smoking

A constant theme throughout the findings of this study was the negative effect due to cigarette smoking. There were decrements in lung function measured for the smokers in the study and control groups. There was a significant difference in FEV_1 between non-smokers and smokers with length of service ($p < 0.05$) and a significant difference ($p < 0.05$) in FEV_1 from first spirometry test compared with the repeat spirometry test for the smokers/asthmatics sub-group in the repeat study. This in effect acted as internal validity indicating that spirometry was sensitive enough to detect a decrease in lung function due to smoking in the initial and repeat studies (Kerstjens, Rijcken, Schouten, & Postma, 1997).

8.5 Summary

It has been demonstrated in this research that, when used in conjunction with a respiratory questionnaire, spirometry testing provides an effective diagnostic tool with adequate sensitivity to detect effects on lung function. It must be noted that this study was not about validation of spirometry, but the detection of possible adverse respiratory health effects at an early stage using the respiratory questionnaire in conjunction with

lung function testing, which it appears to do well. Asthmatics (non-work related) were identified both through the questionnaire and by spirometry. In this study, an effect due to smoking was observed while there was no obvious effect on lung function for the presumed healthy sub-group, for the study period. This combination (spirometry plus questionnaire) was able to identify individuals with non-work-related respiratory symptoms. Furthermore it was determined that there was no overall decrement in lung function for the presumed healthy workers with length of service or any specific work area. That there was no significant difference in FEV_1 from the first spirometry test compared with the repeat spirometry, and there was no cross-swing decrement in lung function for this cohort of refinery workers.

Thus it can be concluded that spirometry in combination with a respiratory questionnaire is sensitive enough to detect an effect on lung function, however, no workplace effect was noted, using this study protocol.

8.6 Recommendations

The reasons for the absence of a work-related effect on lung function at the Murrin Murrin Operation using this study protocol, over this study period, would appear to be due to the primary, secondary and tertiary preventative measures implemented at the mining and process areas. These preventative measures included a hierarchy of controls including isolation, engineering, administrative and personal protective equipment, as well as occupational hygiene monitoring, and health surveillance. However, it cannot be assumed that these preventative measures will remain adequate over the longer term. The aim of occupational hygiene is to ensure exposure to hazardous substances does not affect employee health. Therefore it is recommended that, through continuous improvement, this level of protection is maintained, and even improved upon where possible by proactively reviewing workplace health risk assessments and conducting health monitoring.

As asthma and other pre-existing respiratory disorders may be exacerbated by occupational exposures, these subjects should be monitored more closely than other employees for possible respiratory health effects. It is equally important not to ignore the effect of cigarette smoking, although this is a lifestyle issue rather than a workplace issue. Individual health ownership to protect those with asthma and, to help smokers

quit smoking should be encouraged within the workplace this will be beneficial to both the company and the individual.

Suggestions for further research;

- extend this longitudinal study to identify long term trends at the Murrin Murrin operation;
- expand this study to review the respiratory health of people who have worked in the WA mining industry for periods in excess of 15 years;
- examine the respiratory health of shutdown workers such as the boilermakers, welders, and confined space workers who carry out the majority of the 'dirtier' maintenance work and are more likely to be exposed to higher levels of respiratory health risks.

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TABLE OF ABBREVIATIONS

AED	aerodynamic equivalent diameter
ANZSRS	Australian and New Zealand Society of Respiratory Science
ATS	American Thoracic Society
BIMS	Brambles Industrial Maintenance Services
BMI	body mass index
CCD	counter current decantation
CCH	Commerce Clearing House (a Wolters Kluwer business)
CI	confidence interval
CIC 33	census industry code (primary metal industries)
cm	centimetre = one hundredth of a metre
Co	Cobalt
CO	carbon monoxide
COPD	chronic obstructive pulmonary disease
DALYs	disability adjusted life years
DMP	Department of Mines and Petroleum
DMPR	Department of Mineral and Petroleum Resources
EC	endothelial cells
eNO	exhaled nitric oxide
ERS	European Respiratory Society
FeNO	fractional exhaled nitric oxide
FEV_1	forced expiratory volume in one second
FEV_1/FVC	the ratio of forced expiratory volume in one second to forced vital capacity
FEV_1/FVC%	the ratio of forced expiratory volume in one second to forced vital capacity – as a percentage
FOT	forced oscillation technique
FVC	forced vital capacity
FZ	ferruginous zone
HPAL	high pressure acid leach
HSIS	hazardous substances information system
H_2S	hydrogen sulphide
IARC	International Agency for Research on Cancer
IBM	International Business Machines – a company that manufactures and sells computers and computer software
IDLH	immediately dangerous to life and health
ILO	International Labour Organization
IL-8	interleukin 8
km	kilometres
L	litre(s)
LCD	liquid crystal display
MANOVA	multivariate analysis of variance
m^3	cubic metre
mg	milligram = one thousandth of a gram
mg/m^3	milligram per cubic metre
ml	millilitre = a thousandth of a litre
MPR	Mining and Petroleum Resources
MSHA	Mine Safety and Health Administration
n	sample size
Ni	nickel
NIOSH	National Institute for Occupational Safety and Health
NOHSC	National Occupational Health and Safety Commission

NO_2	nitrogen dioxide
^oC	degrees centigrade
OSHA	Occupational Safety and Health Administration
O_3	Ozone
p	(or p-value) a measure of how likely the sample results are, assuming the null hypothesis is true; the smaller the p-value, the less likely the sample results
PAL	pressure acid leach
PAPA	public health and air pollution in Asia
PAR%	population attributable risk – as a percentage
PEL	permissible exposure limit
PFT	pulmonary function test
pH	a measure of the acidity or alkalinity of a solution
$PM_{2.5}$	fine particles in the (ambient) air 2.5 micrometres or less in size
PM_{10}	fine particles in the (ambient) air 10 micrometres or less in size
ppb	parts per billion
ppm	parts per million
RADS	reactive airways dysfunction syndrome
RCS	respirable crystalline silica
ROM	run of mine
r	Pearson's correlation coefficient – a standardised measure of the strength of relationship between two variables
R^2	coefficient of determination. The goodness of fit of a statistical model describes how well it fits a set of observations
SABRE	surveillance of Australian workplace based respiratory events
SAG	semi-autogenous grinding
SAP	saprolite zone
SD	standard deviation
SO_2	sulphur dioxide
SM	smectite zone
SPSS	Statistical Package for the Social Sciences
SWORD	surveillance of work-related and occupational respiratory disease
TLC	total lung capacity
TWA	time-weighted average
UK	United Kingdom
µm	micrometre(s) = one millionth of a metre
US	United States
VOC	volatile organic compounds
VC	vital capacity
vs	versus
WoRLD	work-related lung disease
≤	equal to or less than
>	more than
<	less than

GLOSSARY OF TERMS

Allergy	A reaction of the immune system to something that does not bother most people.
All subjects	All individuals in the study (and control) group.
Approved Person	A person approved by the Department of Mines and Petroleum, Resources Safety, Western Australia, to carry out MineHealth Assessments on completion of compulsory training in spirometry (lung function testing).
Asthmagen	Any substance that is causally related to the development of asthma symptoms.
Biological Control	The lung function of a subject with stable respiratory function recorded regularly as part of an ongoing quality control program.
BOC Plant	The production and supply of compressed and bulk gases.
Bulka Bags	Large lightweight bags, usually made of woven polypropylene, with four cross corner loops, capable of holding $1m^3$ or 1 tonne, can be handled by crane, Hyab, forklift.
Crew	A group of workers operating and maintaining (in this instance) the refinery.
Cross-Swing	Lung function tests for a cohort of refinery workers were conducted as they arrived for work on site prior to commencing work in the refinery, and on completion of their work period on site before returning home on their rest break.
Dependent *t-test*	A test using the *t-statistic* that establishes whether two means collected from the same sample (or related observations) differ significantly.
Ever Smokers	A person who has ever been a cigarette smoker or cigar smoker.
FEV_1diff	Change in FEV_1 from the initial to the repeat lung function test.
Independent *t-test*	A test using the *t-statistic* that establishes whether two means collected from independent samples differ significantly.
Length of Service	The duration of service in the company (at Minara Resources, Murrin Murrin mine site).
Never Smokers	A person who has never been a cigarette smoker or cigar smoker.
Non-work-related respiratory symptoms	Respiratory symptoms determined to be other than work related.
Pack Years	A way to measure the amount a person has smoked over a long period of time.
Predicted (normal) Values	Spirometry reference values from groups of relatively healthy persons (non-smokers, with no known respiratory disorders).
Presumed Healthy	The sub-group of the study (and control) group on removal of those subjects who were ever-smokers, and those with known non-work related respiratory disorders. Therefore the never-smokers, with no known non-work related respiratory symptoms (I.E., The known confounders removed).
Rollies	Hand-rolled cigarettes.
Sig	Statistical significance.

Spirometry	A test that can help diagnose various lung conditions.
Swing	A work pattern – from arrival for work on site, to completion of work period on site, before returning home for a rest break.

APPENDIX A: HEALTH ASSESSMENT FORM, DMP, WA

HEALTH ASSESSMENT FORM **CONFIDENTIAL**

Government of **Western Australia**
Department of **Mines and Petroleum**
Resources Safety

Resources Safety
303 Sevenoaks Street, Cannington WA 6004
Phone: 08 9358 8461
www.dmp.wa.gov.au/ResourcesSafety
minehealthreporting@dmp.wa.gov.au

HEALTH ASSESSMENT FORM

MINES SAFETY AND INSPECTION ACT 1994 Section 75 (1)

TYPE OF HEALTH ASSESSMENT: ☐ INITIAL ☐ PERIODIC

Health Surveillance Number
(To be assigned by DMP)

PLEASE PRINT IN BLOCK LETTERS

EMPLOYEE'S PERSONAL DETAILS (AS PER CURRENT ID)

Surname: ..... ☐ MALE ☐ FEMALE
(include former name, if name has changed)
Given names: ..... Date of birth: ...../...../.....

Contact address: .....
(Health Surveillance Card will be sent to this address) ..... Post code: .....

Home/Mobile Number: .....
(Mandatory)

Name and address of private doctor: .....
..... Post code: .....

Signature: ..... Date: ...../...../.....

EMPLOYER DETAILS (CURRENT)

Company: .....
Site: .....
Contact Person: .....
Address: .....
Contact Number: ..... Post code: .....

APPROVED PERSON OR MEDICAL PRACTITIONER DETAILS

Approved Person .....
Medical Practitioner .....
Address: .....
Contact Number (mandatory): ..... Date: ...../...../.....
Approved Person No. or Provider No.: .....

Please send the completed health assessment forms including chest x-ray (if required) to: Mines Occupational Physician, Resources Safety, DMP, *100 PLAIN STREET, EAST PERTH WA 6004.*

SECTION I – WORK HISTORY

To be completed by the approved person or medical practitioner only

Note:
i. Enter all past work history both mining and non-mining from when you left school.
ii. Enter specific job descriptions, .e.g air leg operator, plant operator, driller, fitter, truck driver, electrician, laboratory operator, mine manager
iii. Record duration and "from – to" dates as accurately as possible.
iv. Minesite column – Enter name of mine; if outside WA specify location; if not a minesite leave blank.

Usual occupation or trade:_____

Description of current occupation / job	Period of Time (fill In either of the following) Duration (yy/mm)	From – To (mm/yy – mm/yy)	Name of employer	Name of minesite
	___/___	___/___ - ___/___		
Previous Jobs (most recent job first)	Period of Time Duration (yy/mm)	From – To (mm/yy – mm/yy)	Name of employer	Name of minesite (use "u/g" to indicate if underground)
1.	___/___	___/___ - ___/___		
2.	___/___	___/___ - ___/___		
3.	___/___	___/___ - ___/___		
4.	___/___	___/___ - ___/___		
5.	___/___	___/___ - ___/___		
6.	___/___	___/___ - ___/___		
7.	___/___	___/___ - ___/___		
8.	___/___	___/___ - ___/___		
9	___/___	___/___ - ___/___		
10.	___/___	___/___ - ___/___		
11.	___/___	___/___ - ___/___		
12.	___/___	___/___ - ___/___		

SECTION II – RESPIRATORY QUESTIONNAIRE

To be completed by the approved person or medical practitioner only

Please instruct the employee to give you quick (spontaneous) answers to the questions listed below.

	YES	NO
Cough		
1. Do you usually cough first thing in the morning?	☐	☐
2. Do you usually cough during the day or at night?	☐	☐
If **NO** to questions 1 and 2, go to question 4. If **YES** to questions 1 or 2:		
3. Do you have a cough like this on most days for as much as three months each year?	☐	☐
Phlegm		
4. Do you usually bring up phlegm from your chest first thing in the morning?	☐	☐
5. Do you usually bring up phlegm from your chest at any other time of day or night?	☐	☐
If **NO** to questions 4 and 5, go to question 9. If **YES** to questions 4 or 5:		
6. Do you bring up phlegm like this on most days for as much as three months each year?	☐	☐
7. In the past three years have you had a period of increased cough and phlegm lasting for three weeks or more?	☐	☐
If **NO** to question 7, go to question 9. If **YES** to question 7:		
8. Have you had more than one such period?	☐	☐
Breathlessness on activity		
9. Do you get short of breath when hurrying on level ground or walking up a slight hill?	☐	☐
If **NO** to question 9, go to question 12. If **YES** to question 9:		
10. Do you get short of breath when walking with other people of your age on level ground?	☐	☐
If **NO** to question 10, go to question 12. If **YES** to question 10:		
11. Do you have to stop for breath when walking at your own pace on level ground?	☐	☐
Breathlessness at rest		
12. Do you ever get short of breath at rest?	☐	☐
13. Do you ever wake up in your sleep short of breath?	☐	☐

	YES	NO
Wheezing		
14. Does your chest ever sound wheezy or whistling?	☐	☐
If **NO** to question 14, go to question 18. If **YES** to question 14:		
15. Do you get this on most days or nights?	☐	☐
16. Have you ever had attacks of shortness of breath with wheezing?	☐	☐
If **NO** to question 16, go to question 18. If **YES** to question 16:		
17. Was your breathing normal between attacks?	☐	☐
Breathing Difficulty		
18. Does your chest ever feel tight or your breathing become difficult?	☐	☐
Smoking History		
19. Do you, or did you, smoke more than 1 cigarette/day; a cigar/week; or 2 oz (50g) pipe tobacco/month for at least one year?	☐	☐
If **NO** to question 19, go to question 23. If **YES** to question 19:		
20a. How much do you (or did you) smoke each day? (no. of cigarettes/cigars)	_____	
20b. Roll-your-owns or pipes (number of grams/week)?	_____	
21. How old were you when you started smoking?	_____	
22. If you are an ex-smoker, how old were you when you gave up smoking permanently?	_____	
Past Chest Illness		
23. During the past three years have you had any chest illness that has kept you from your usual activities for a week or more?	☐	☐
If **NO** to question 23, go to question 26. If **YES** to question 23:		
24. Did you bring up more phlegm than usual during this illness?	☐	☐
25. Have you had more than one illness like this in the past three years?	☐	☐
26. Have you ever had asthma?	☐	☐
27. Have you ever had any other chest illness, injury or surgery?	☐	☐

If yes, provide details:

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SECTION III – LUNG FUNCTION TEST

Height_____(cm) Age_____ (years) Weight_____ (kg)

(must be measured)

Lung Function (Spirometry) **Room Temp. _____ °C**

Make:

Model:

Date of calibration (3-litre syringe) _____/_____/_____ Mandatory for all spirometers

Measurement Results:
(attach all spirometry printouts with flow volume graphs to this form)

Three acceptable and reproducible results (within ± 0.15L)

	Test 1	Test 2	Test 3
FEV_1			
FVC			

Bronchodilator use: Yes ☐ No ☐

If yes, how long before test? _____ minutes/hours.

Comments (especially if any difficulty with spirometry):

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