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An investigation into dust, gases and vapours expulsed during the oxidation of pyritic black shale, and their potential for impacting on employee health

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AN INVESTIGATION INTO THE DUST, GASES AND VAPOURS EXPULSED DURING THE OXIDATION OF PYRITIC BLACK SHALE, AND THEIR POTENTIAL FOR IMPACTING ON EMPLOYEE HEALTH

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USE OF THESIS

ABSTRACT

Pyritic black shale (PBS) is occasionally extracted as a waste product of iron ore mining in the Pilbara region of Western Australia. Mineralogical studies such as Brocks, Summons, Buick, and Logan (2003) of the PBS located at Mount Tom Price Operations have established that PBS can contain elevated concentrations of pyrite, carbon, bitumens, polyaromatic hydrocarbons and volatile organic compounds (VOCs). The natural oxidative weathering of PBS can cause exothermic reactions leading to the shale reaching temperatures that cause the expulsion of toxic gases such as hydrogen sulphide, sulphur dioxide, methane and carbon monoxide.

Current management of the potential hazards of PBS is based on limiting employees' working time in PBS areas, using gas detection equipment and wearing respiratory protection for particulates and acid gases. This research investigates the potential risk of adverse health effects for employees working within PBS areas.

Three similar exposure groups (SEGs) have work activities located within pits that contain PBS. These groups are classified as: drill and blast; geologists/samplers; and heavy-mobile equipment operators. Historical exposure data for inhalable dust, respirable dust, respirable crystalline silica and toxic gases were obtained for each of the SEGs and analysed using the IHSTAT program. In order to identify what VOCs could potentially be released during the oxidation of PBS, thermal desorption tubes were used to collect positional air samples from burning PBS, and a laboratory trial was conducted where several PBS samples were taken from two waste dumps, crushed, milled and inserted into quartz tubes for thermal desorption. All thermal desorption tubes were heated to 340 °C and 500 °C and gas chromatography mass spectrometry (GCMS) was conducted on the resultant gas samples (WIENV 31).

Particulate results show that the only non-conformance with the Safe Work Australia occupational exposure limits (OELs) occurred among heavy mobile equipment operators and their exposure to respirable crystalline silica. The estimated arithmetic mean exposure for the SEG was 0.043 mg/m³ and the estimated 95th percentile was 0.164 mg/m³ compared to the OEL of 0.1 mg/m³. The toxic gas data demonstrate that of the 64 days sampled, hydrogen sulphide exceeded the OEL on 1 day and the short-term exposure limit (STEL) was not exceeded. Sulphur dioxide levels exceeded the OEL on 8 days and on 4 of those days the STEL was also exceeded. All carbon dioxide results were within acceptable ranges. Thermal desorption data showed that benzene, toluene, heptane, cyclohexene, phenol, acetic acid, carbon disulfide and hexane could potentially be produced.

The identification of organic vapours indicates the possibility for adverse health impacts not previously considered. Sustained exposure to large quantities of these substances has the potential to produce chronic health effects in the form of cancers and nervous system damage. As a result, recommendations for mining operations in PBS pits are: to provide respiratory protection cartridges that protect against VOCs as well as toxic gases; to carry out sampling for these organic vapours to assess worker exposure; and to maintain existing control measures that limit employees' exposure to PBS.

DECLARATION

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TABLE OF CONTENTS

1. INTRODUCTION

During 2007, a study entitled "An investigation into the health effects of occupational exposure to pyritic black shale (PBS) at Tom Price mine operations Western Australia" was commenced. The research was made possible following the signing of a collaborative agreement between Rio Tinto and Edith Cowan University. Under this agreement, the university provided support to Rio Tinto employees involved in the research.

1.1 Problem statement

Tom Price operations has a comprehensive occupational hygiene program which covers exposure to dust, gases and noise as well as many other occupational hazards. The geology, geochemistry and formation of PBS has been well documented in past research; however, health impacts associated with exposure to black shale dust and vapour have not been previously investigated.

1.2 Aim and scope

The aim of this project was to address the existing knowledge gap by implementing an occupational hygiene survey which assessed the potential for health impacts to open pit workers exposed to PBS at Tom Price Operations. This study included Rio Tinto employees and contractors working in and around black shale pits. Contaminants associated with mining iron ore with areas of black shale during normal production processes were assessed.

1.3 Overview of the study

Chapter 2 provides the background information required to gain an understanding of the study that was conducted. Commencing with a discussion on the climatic and geological background of the Pilbara region,information on exposure standards and the relevant health information related to contaminants associated with iron ore mining areare also addressed. Chapter 2 concludes with an overview of the current exposure sampling that is relevant to workers operating within the pit environment.

Chapter 3 is a review of relevant literature related to the research hypothesis and chosen methods. This chapter includes a discussion of sampling methods and justification of the methodology used.

Chapter 4 contains the results of the study. This chapter outlines the entire sampling programme and the decisions made in the development of the sampling regime. A statistical summary of the sampling results is also presented.

Chapter 5 completes the thesis by discussing the meaning of the results as well as the conclusions drawn and recommendations made for future black shale management.

2. BACKGROUND INFORMATION

Rio Tinto's Tom Price iron ore mine is located 5 km from the town of Tom Price approximately 1200 km north east of Perth, in the Shire of Ashburton in the Pilbara region of Western Australia (Figure 1). Development of the mine began in 1966 (Rio Tinto, 2009). The mine currently produces approximately 20 million tonnes of iron ore and moves 30 million tonnes of waste material annually. The company operates 14 mines in the Pilbara region of Western Australia, integrated with dedicated railway and port facilities in Dampier and Cape Lambert.

2.1 Climate

The Pilbara is located in a semi-arid tropical region with Tom Price having an average annual rainfall of 371.4 mm (derived from 1971–1998 Bureau of Meteorology data, 1999 –2006 site weather station at Tom Price). Evaporation from free water surfaces in the area is approximately 3,600 mm per year. Tom Price recorded a total of 711 mm of rainfall during 2006 due to several tropical cyclones that occurred in the first quarter of the year. Annual regional rainfall shows a typical tropical wet-dry pattern with high rainfall in summer and little or no rainfall in winter.

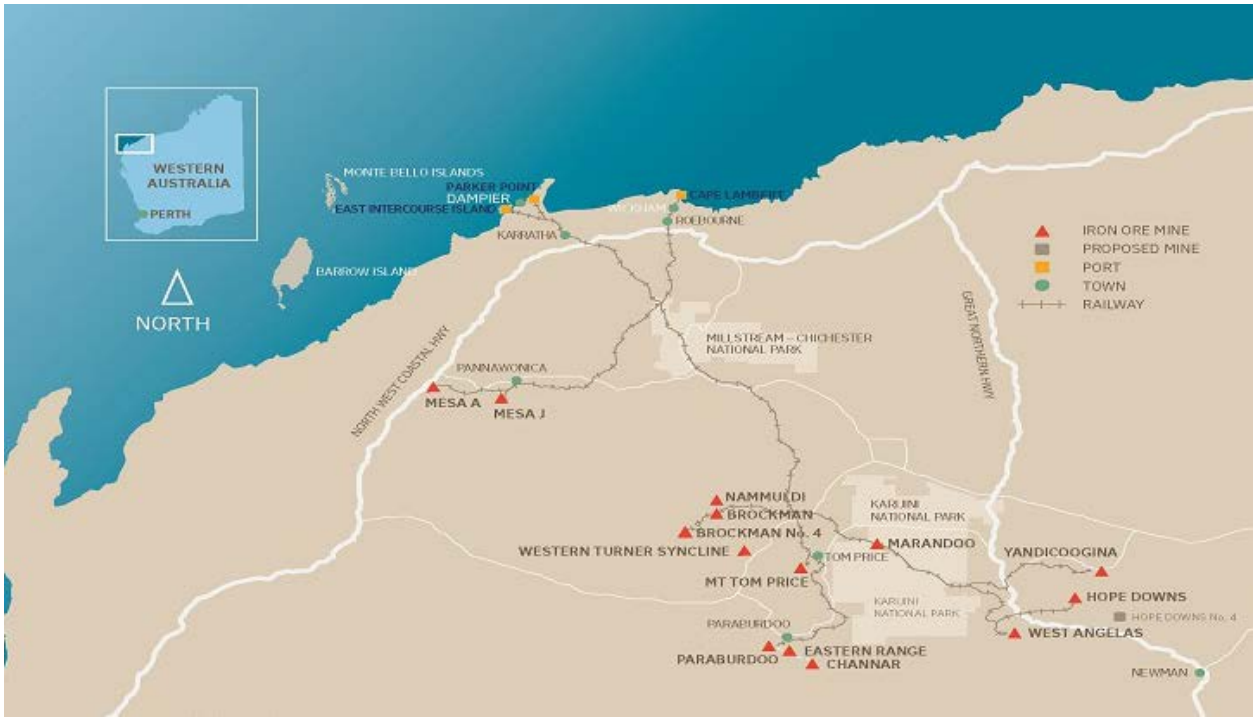

Figure 1. Location of the Pilbara region showing Tom Price operations (Rio Tinto intranet http://rtio.riotinto.org/Operations/)

2.2 Mining and processing

Ore is mined using conventional open pit mining methods of drilling and blasting with progressive extraction from 15m high benches. The material is blasted before it is loaded into haul trucks and transported to the primary crusher for processing. High-grade ore is crushed to less than 200 mm in the primary crusher and is then directed to the primary stockpile. Ore is reclaimed from the primary stockpile for secondary crushing to produce particles of less than 80 mm in diameter. The particles are then passed over vibrating screens and separated into lump ore and fine ore sizes. Ore material greater than 31.5 mm is directed to a tertiary crusher and the product is then re-screened. Low-grade ore is crushed, screened and directed to the beneficiation plant for upgrading. Upgrading involves:

- wet screening
- heavy media drum separation of the lump fraction
- heavy media separation of the 6 mm fines
- magnetic separation of the 0.5 mm fines
- removal of the slimes (less than 0.03 mm).

Products from the beneficiation plant are blended with the stockpiled high-grade plant products. Lump and fine products are stockpiled prior to being loaded onto trains and transported to the port of Dampier. The layout of the Tom Price mine and pit is illustrated in Figure 2.

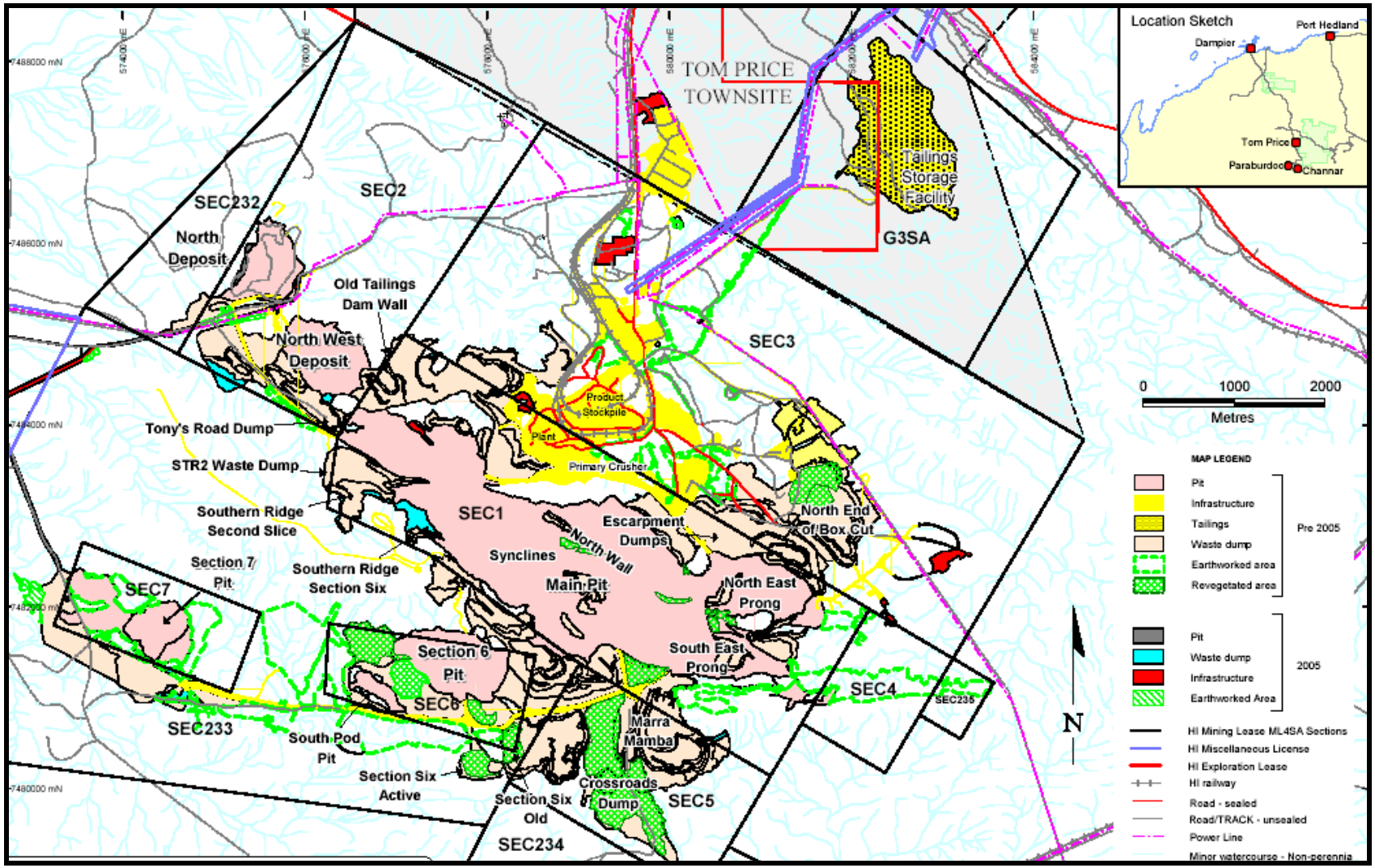

Figure 2. Tom Price mine and pit layout (Rio Tinto, 2009)

2.3 Hamersley Iron Province

Tom Price lies within the Hamersley Iron Province, a group of Lower Proterozoic sediments referred to as the Hamersley Group (see Figure 3). The Group comprises a sequence of banded iron formation, shale, dolomite and acid volcanic rocks. Banded iron formations (BIFs) consist of alternating layers of iron and silica-rich material deposited as chert. Iron minerals occur as oxides, carbonates and/or silicates. The Brockman Iron Formation is the thickest and most exposed formation of the Hamersley Group, and outcrops as rugged mountains and distinctive escarpments. Topography is dominated by the strong folding of the southern plateau region resulting in an irregular outcropping and predominantly linear arrangement of the hills in a north-west to south-east alignment. The stratigraphic sequence exposed in the Tom Price area ranges from the Fortescue Group through to the upper Hamersley Group in the core of the Turner Syncline. In the immediate mine area, outcrop is dominated by the lower units of the Hamersley Group from the Marra Mamba Iron Formation through to the Brockman Iron Formation. All low-phosphorous Brockman ore is restricted to the Colonial Chert Member of the Mount McRae Formation and the Brockman Iron Formation.

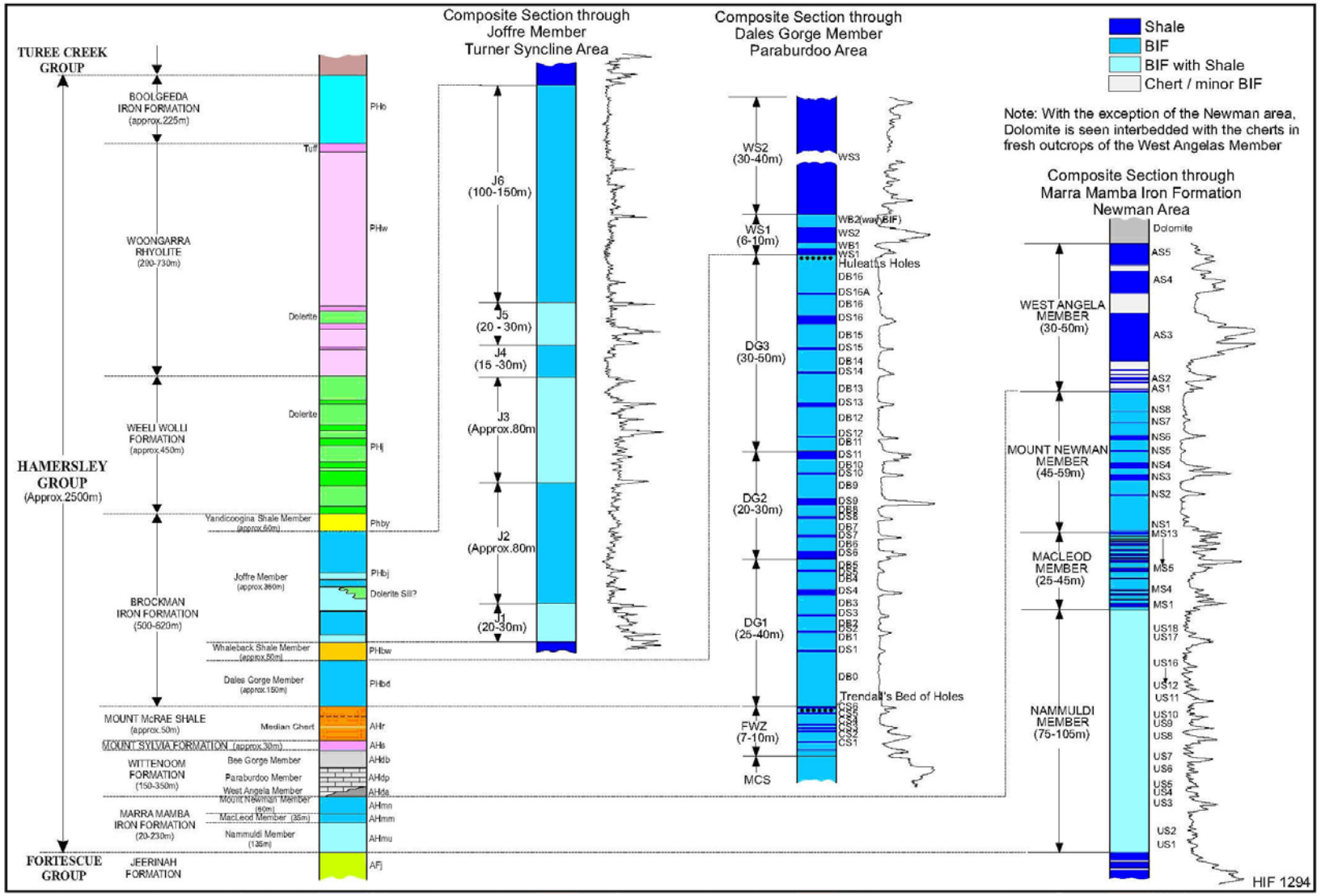

Figure 3. Stratigraphy of Hamersley Province iron ore deposits (Bitencourt, 2002).

2.4 Brockman Iron Formation

Regionally, the Brockman Iron Formation's thickness varies considerably, from 500 m at Paraburdoo and in the Newman area to 620 m at Tom Price. The sequence may lose up to 50 % total thickness when enriched from banded iron formation (BIF, ~30 % Fe) to ore (>60 % Fe) (Szulc, 2003). The Brockman Iron Formation consists of an alternating sequence of BIF, shale and chert. It is subdivided into the four members given below.

- The Dales Gorge Member (~150 m) is an alternating assemblage of 17 BIF and 16 shale macrobands. The member is informally divided into three units, DG1 through to DG3. The subdivision is based on the location of shale bands and is designed to maximise grade due to the abundance of shale (the major contaminant) in DG2.
- The Whaleback Shale Member (~50 m) has two zones: a lower zone of alternating thick bands of BIF and shale, which may be mineralised, and an upper zone of thin interbedded chert and shale.
- The Joffre Member (~360 m) is dominated by BIF with only minor shale interbands that are thinner and not as laterally persistent as those of the Dales Gorge Member.
- The Yandicoogina Shale Member (~60 m) is a sequence of interbedded chert and shale, intruded variably by dolerite.

2.5 Mount McRae Shale

Figure 4 is a stratigraphic column of the Mount McRae Shale (MCS) at Tom Price. The footwall zone is considered to be part of the MCS but it is composed of interbedded BIF and shale, and where enriched, it is mined as low-grade ore. It is generally oxidised and has a low sulphide concentration, but some thin green-to-black shale beds may contain more than 10 % total sulphur. In bulk, the footwall zone is low sulphur and is not classified or managed as black shale, but sulphur concentrations of up to 1 % have been observed in tailings generated from footwall zone ore. To date, these tailings have not required special handling because they represent such a small percentage of the total tailings volume.

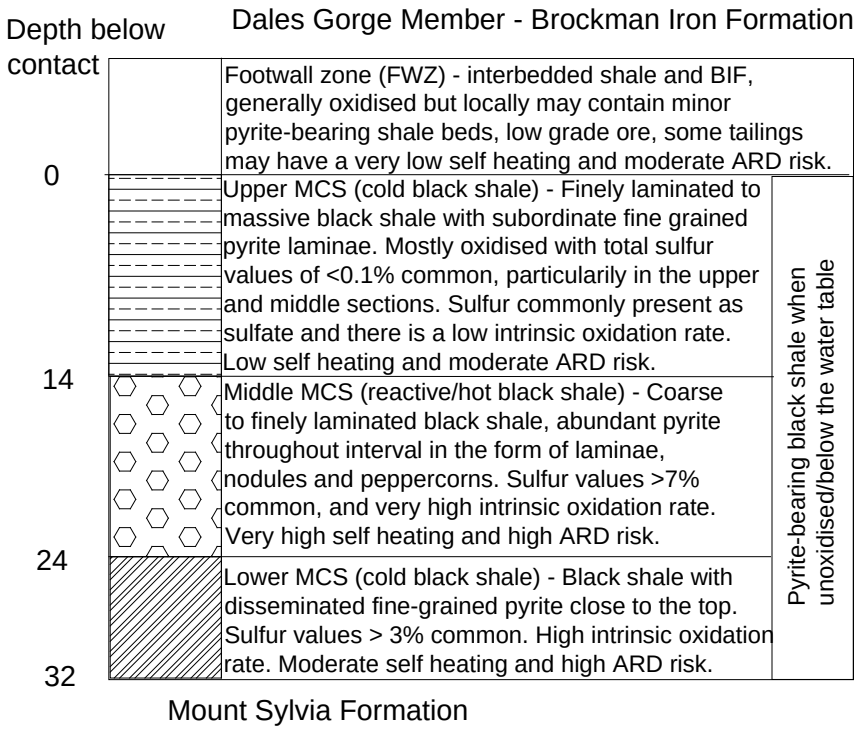

Figure 4. Stratigraphy of McRae Shale (MCS) at Tom Price (Bitencourt, 2002)

When it occurs as relatively un-oxidised black shale, the underlying MCS is divided into upper and middle zones at Tom Price. The middle zone is designated as reactive or hot black shale because it has the highest sulphide content and is the unit that is prone to self-heating and rapid acidification. The upper MCS is designated as cold black shale as it commonly contains less than 0.1 % total sulphur and poses a much lower self-heating risk than lower MCS.

Above the groundwater table, the MCS is generally oxidised. It contains few intact sulphides, generally less than 0.1 % sulphur, is characterised by red, yellow, orange and white colours and poses little threat of self-heating risk. Below the water table, and locally immediately above the water table, the MCS is a carbonaceous and sulphide-bearing shale that poses both an acid rock drainage (ARD) and self-heating risk. Although black shale is occasionally identified in the Whaleback Shale Member of the Brockman Iron Formation, to date it has not been found to pose a significant self-heating risk. Most Whaleback black shale samples

contain less than 0.1 % sulphur, although sulphur concentrations above 1 % (mostly as sulphate) have been identified (Rumball, 1984).

2.6 Pyrite

Rumball (1984) used Schwann's (1983, cited in Rumball, 1984) informal stratigraphic nomenclature to subdivide the MCS (Spodniewski, 2003). Schwann observed the MCS at Mt Whaleback and Tom Price mines and divided it into four units, of which the pyritic unit has been further divided into five zones on the basis of pyrite occurrence based on macroscopic scale (Spodniewski, 2003).

- *Massive Pyrite Zone:* appears to be solid pyrite, microscopic pyrite accounting for only 50 % of the shale, the remainder is mostly quartz.
- *Small Nodule Zone:* contains nodules of pyrite <5 mm in diameter scattered through a black shale matrix, the nodules accounting for less than 5 % of the shale.
- *Hematite-Pyrite Zone:* appears reddish coloured, friable, under microscope inspection no hematite was observed, possibly the weathered/oxidised zone.
- *Large Nodule Zone:* contains nodules of pyrite with a diameter of >1 cm, these nodules account for less than 5 % of the black shale.
- *Chert Triplet:* consists of three distinct chert bands each less than 1 m thick, in a black shale containing large to very large pyrite nodules.

Rumball (1984) noted that under microscopic examination the pyrite occurs in three major zones showing no correlation with the five zones. These zones were identified as solid pyrite, poppyseed pyrite and subhedral pyrite. Furthermore, Kakegawa, Kawai and Ohmoto (1998) considered a similar scheme from the examination of drill core samples from the Whaleback mine, within a drill core that penetrated strata from the lower MCS to the upper Sylvia formation. They found that the lower section of the MCS contained fine grains (~10 μm) of subhedral and euhedral pyrite, as well as veins and pods of pyrite which were all concordant with bedding (Kakegawa et al., 1998). The zone of fine-grained pyrite is separated by a thin,

1 mm band of chert, which is the boundary of a younger pyrite zone and an older zone of pyrite which forms the lower and middle sections of the MCS. This older zone of pyrite (5-26 m) is characterised by the presence of coarse-grained pyrite (>100 μm) and pyrite nodules (~1 cm – 10 cm in diameter) which appear in great quantity in the lower section of the MCS (Kakegawa et al., 1998).

2.6.1 Formation of pyrite

There are two modes of formation for pyrites by bacterial sulphate reduction based on the supply and consumption of sulphate: (1) a closed-bottom system, with respect to sulphate where the rate of reduction is equal to or faster than the rate of sulphate supply so that all the sulphate that enters the system is converted to pyrite, and (2) an open-bottom system, with respect to sulphate, where the rate of sulphate supply is greater than the rate of sulphate reduction so that a small portion of sulphate that enters the system at a time is converted to pyrite. It should be noted that natural systems are almost always open with respect to hydrogen sulphide (Kakegawa et al., 1998).

The open-bottom system is found in two areas: (1) the upper part of a sediment column in modern normal marine sediments, and (2) in the anoxic water column of a euxinic basin such as the Black Sea or the Baltic Sea.

Three possibilities have been considered for the origin of early pyrite (disseminated and laminated pyrite) in the MCS: (1) direct precipitation from a hydrogen sulphide or HS^- rich water, syngenetic pyrite, in which the hydrogen sulphide or HS^- could have been produced by hydrothermal or biological processes; (2) bacterial reduction of seawater sulphate during early stages of sedimentation; and (3) hydrothermal (thermochemical) reduction of seawater sulphates. Kakegawa et al.. (1998) suggest that sulphate reduction in pore fluids were responsible for pyrite formation in the MCS.

2.6.2 Health effects of exposure to pyrite

Health impacts from exposure to pyrite dust have been extensively investigated in coal mining, however, as far as can be established, not in iron ore mining. A study by Cohn, Laffers, Simon, O'Riordan and Schoonen (2006), investigated the potential for reactivity from coal that contains pyrite. Experiments were performed to specifically evaluate the role of pyrite in coal dust reactivity. Coal samples containing various amounts of Pyrite were compared for differences in their generation of reactive oxygen species (ROS) and degradation of ribonucleic acid (RNA). The authors found that coals that contain iron also show the presence of pyrite, generate ROS and degrade RNA. Coal samples that do not contain pyrite do not produce ROS or degrade RNA. The concentration of generated ROS and degradation rate of RNA both increase with pyrite content in the coals. The prevalence of coal workers' pneumoconiosis can be correlated to the amount of pyrite in the coals. Considering the harmful effects of inhaled ROS particles, it has been suggested that the toxicity of coal may be explained, in part, by the presence of pyrite.

2.7 Reactivity of PBS

The large areas of PBS that are exposed on the pit walls in the STR and South East Prongs pits are reactive and sulphur dioxide may be detected in their vicinity (see Figure 2). In April 1983, a wall of PBS was reported as "turning white" after heavy rain. This was due to the formation of a thin coat of a white efflorescence, possibly potassium sulphate (Spodniewski, 2003).

Blasting of ore/waste rock enhances the reactivity of freshly fractured surfaces, and the high permeability allows oxidising agents like air and water to easily penetrate enabling the well-fractured rock to undergo rapid oxidation and possibly ignition. AN-FO (ammonium nitrate/fuel oil) explosives used in the charging of blast holes may replace the role of air and water and could possibly have a higher oxidative potential. Rumball (1991) proposes that there are stages of the reaction mechanism between weathered pyritic shale and ammonium nitrate. This includes the reduction of nitrate to nitrous acid by ferrous sulphate and pyrite

(initial stage), the evolution of nitrogen dioxide and a moderate amount of heat (intermediate stage) which will catalyse the explosive decomposition of ammonium nitrate (ignition stage).

2.8 Self-heating

Self-heating of the PBS can occur when the pyrite (FeS_2) and carbon in the black shale is exposed to the atmosphere and begins to oxidise. Under some conditions, the heat generated by the oxidation reactions cannot be dissipated rapidly enough, so the temperature reaches a point where spontaneous combustion and significant sulphur dioxide gas generation can occur (Rio Tinto, 2007). A study at BHP's Whaleback mine showed that waste rock self-heating is initialised by the oxidation of carbon. Gas analysis performed as part of the oxidation test work indicates that detectable sulphur dioxide emissions do not commence until a temperature of 160 ºC has been reached (Davies, 2002). Rumball (1984) noted that black shale undergoes two modes of oxidation: a wet and a dry phase. Wet-phase reactions occur in temperatures ranging from ambient to 340 ºC. Rumball suggests that as the temperature increases, water boils off, leaving a sulphuric residue behind, and hydrogen sulphide is the likely product of pyrite oxidation. The boiling off of hydrogen sulphide will enable dry oxidation to occur. The dry reaction has been monitored at temperatures as high as 672 ºC and, at these high temperatures, other components of the black shale are oxidised resulting in the black shale burning.

2.9 Hydrogen sulphide

Hydrogen sulphide (H_2S) is a colourless gas easily recognised at 0.025 ppm by its distinctive rotten egg odour, and is well known to affect diffuse areas of the human nervous system including the cortex, sub cortex, cranial nerves and peripheral nervous system (Hirsch and Zavala, 1999). An acute effect of H_2S on the olfactory nerve is temporary paralysis or olfactory fatigue, which disables the sense of smell rendering it a poor warning system for detecting the continued presence of gas (Hirsch and Zavala, 1999). H_2S is rapidly absorbed from the lungs following inhalation, and absorption through the skin is minimal (Topping,

2001). Following adsorption, H_2S is widely distributed around the body, primarily as undissociated H_2S or as HS^- ions. H_2S binds reversibly to metalloenzymes, including those involved in aerobic cellular respiration such as cytochrome oxidase. The main detoxification pathway for H_2S is oxidation to sulphate, occurring primarily in the liver and to a lesser extent in the blood, followed by excretion in the urine in free or conjugated form. Another minor metabolic route for H_2S occurring primarily in the intestinal mucosa and liver, is methylation to methanethiol and dimethylsulfide. Metabolism to sulphate is relatively rapid and hence H_2S is unlikely to bioaccumulate (Costigan, 2003).

2.9.1 Effects of single exposure

Several case reports of fatal poisonings in workers exposed to H_2S have been documented with the data clearly indicating that short-term single exposures to concentrations of 500 ppm and above may be fatal (Turner & Fairhurst, 1990). Studies in animals confirm the human evidence regarding the acute toxicity of H_2S. An LC_{50} (concentration causing lethality in 50 % of animals) of 444 ppm was measured in rats following a four-hour exposure period (Costigan, 2003). Human evidence suggests that exposure to higher concentrations in the region of 1000 ppm and above are rapidly fatal (within minutes). Deaths at such high exposure concentrations are caused by inhibition of cytochrome oxidase causing a blockage of the mitochondrial electron transport system and inhibition of cellular respiration. This leads to inactivation of the respiratory centres in the brain leading to respiratory arrest, unconsciousness and death. In addition, more prolonged exposures to somewhat lower concentrations can cause pulmonary oedema and congestion which can be fatal. Hence there is a duality of the mechanisms leading to death, involving both centrally mediated respiratory depression and lung damage. The relative significance of these mechanisms depends on the exposure conditions. Neurological sequelae such as memory loss are common in survivors following periods of unconsciousness caused by H_2S exposure (Health and Safety Executive, 2003).

Exposures to high concentrations of H_2S also cause direct damage to the upper respiratory tract epithelium. Inhalation studies in rats showed that a four-hour exposure to 400 ppm caused cell death and hair loss of the nasal and respiratory epithelium, with indications that an inflammatory response was occurring (Lopez, Prior, Yong, Lillie, & Lefebvre, 1988). However, below 200 ppm these effects were minimal. Exposure of rats to 50 ppm for four hours caused inhibition of lung cytochrome oxidase. It seems possible that the inhibition of lung cytochrome oxidase may account for the pulmonary oedema observed at high exposure concentrations in animals and humans (Khan, Coppock, Schuler, & Prior, 1998).

2.9.2 Effects of repeated exposure

A cross-sectional study of Finnish pulp mill workers showed no effects on pulmonary function (forced expiratory volume in once second, FEV_1, and forced vital capacity, FVC) or on bronchial reactivity to histamine challenge with exposures of non-asthmatics to H_2S in the region of 2-7 ppm (Jappinen, Vilkka, & Martilla, 1990). A cross-sectional study of US sewer workers employed for up to 20 years suggested reduced pulmonary function when compared to a referent group (Richardson, 1995). However, there were no quantitative measures of H_2S exposure in the work environment (Richardson, 1995). A Japanese study of workers from three viscose-rayon plants showed no evidence for effects on pulmonary function or self-reported respiratory symptoms; workers were exposed to mean concentrations of H_2S of 3 ppm (Higashi et al., 1983).

2.10 Sulphur dioxide

Sulphur dioxide is a colourless gas with a characteristic, irritating, pungent odour that exhibits both acute and chronic health effects. Acute effects include irritation of the eyes and upper respiratory tract, narrowing of the airways, runny nose, choking and coughing. As the concentration increases, these symptoms become intolerable and the individual is forced to leave the area (NIOSH, 2001a). Chronic exposure to sulphur dioxide can lead to permanent pulmonary impairment (NIOSH, 2001a).

Sulphur dioxide in the air primarily results from activities associated with the burning of fossil fuels (coal, oil) such as power plants or from copper smelting. In nature, sulphur dioxide can be released to the air from volcanic eruptions. Its acute health effects have been comprehensively studied (NIOSH, 2001b), and at low concentrations it can irritate the respiratory system and eyes and this can lead to conjunctivitis. At higher concentrations, the ability to smell is lost and, as the concentration increases, exposed people will develop pulmonary oedema leading to cessation of breathing and subsequent death.

2.10.1 Short- term exposure

The health effects of sulphur dioxide has been long established and numerous research results on human exposures have been published over the last half century. Symptoms of acute overexposure include irritation of the eyes and upper respiratory tract (nose, throat, larynx and trachea); runny nose, choking and coughing, which probably result from the action of sulphurous acid formed when the highly soluble gas dissolves (OSHA, n.d.a). Approximately 90% of all sulphur dioxide inhaled is absorbed in the upper respiratory passages, where most effects occur; however, it may produce respiratory paralysis and may also cause pulmonary oedema. Some 10–20% of the healthy young adult population are estimated to be hyper-susceptible to the effects of sulphur dioxide (Zenz, Dickerson & Horvath, 1994). Other factors that can exacerbate the respiratory effects of sulphur dioxide include exposure to cold or dry air and other pollutants such as sulphuric acid, nitrogen dioxide, and ozone. Concurrent exposures to sulphur dioxide, smoke, and particulates have been associated with symptoms including: increased respiratory effects, increased frequencies of respiratory illness, excess mortality, and worsening of existing respiratory disease (WHO, 1979).

Short-term exposure also causes bronchoconstriction measurable as an increase in air flow resistance. The magnitude of the response is dose related. While a concentration of 1 ppm can cause a slight increase in air flow resistance for some ‘healthy’ subjects, a concentration of 5 ppm has been shown to induce an increase in flow resistance of up to 39% when

compared to control subjects. Exposures to 13 ppm has been shown to increase air flow resistance for 72% of subjects (Ellenhorn and Barceloux,1988; WHO, 1979).

2.10.2 Long-term exposures

Long-term exposure to persistent levels of sulphur dioxide can also impact negatively on health. Lung function changes have been observed in some workers exposed to 0.4–3.0 ppm sulphur dioxide for 20 years or more. However, these workers were also exposed to other chemicals, making it difficult to attribute their health effects to sulphur dioxide exposure alone. Furthermore, exercising asthmatics are sensitive to the respiratory effects of low concentrations (0.25 ppm) of sulphur dioxide.

For comparative purposes, typical outdoor concentrations of sulphur dioxide may range from 0 to 1 ppm. Occupational exposures to sulphur dioxide may lawfully range from 0 to 5 ppm as enforced by OSHA (Occupational Safety and Health Administration). During any 8-hour work shift of a 40-hour work week, the average concentration of sulphur dioxide in the workplace may not exceed 2 ppm (NOHSC, 1995a).

Agency	TWA (8 hr)	STEL (15 min)
Safe Work Australia	2 ppm	5 ppm
ACGIH (2012)	No TWA	0.25 ppm
NIOSH (2012)	2 ppm	5 ppm

2.10.3 Inhalation exposure – fatalities

There have been several case reports of human deaths following acute exposure to high concentrations of sulphur dioxide (Rabinovitch et al.., 1989). In most studies, concentrations were not measured. In one study, analysis of gas samples at the time of rescue showed sulphur dioxide concentrations greater than 40 ppm (Rabinovitch et al.., 1989). A sulphur

dioxide level of 150 ppm was measured during the re-enactment of an incident in which a 76-year-old asthmatic woman died of an asthma attack after inhaling vapours from a sulphite-based de-rusting agent used in her dishwasher (Huber and Loving, 1991). Actual sulphur dioxide levels were probably higher since the quantity of de-rusting agent used in the investigation was approximately 7–10 % of the amount originally used by the woman. A concentration of 100 ppm is considered immediately dangerous to life and health (HSDB, 1998).

2.11 Organic compounds within pyritic black shale

Brocks, Summons, Buick, and Logan (2003) discuss the normal concentrations and distribution of aromatic hydrocarbons of shales, which were collected in two giant iron ore deposits at Tom Price and Whaleback. The analysis of the bitumen's from the two mines, by gas chromatography mass spectroscopy (GCMS), revealed the presence of n-alkanes, mid- and end-branched monomethylalkanes, cyclohexylalkanes, acyclic isoprenoids, diamondoids, tri-to pentacyclic terpanes, steranes, aromatic steroids and polyaromatic hydrocarbons (PAHs). Almost all extracts from Tom Price and Whaleback contain very high relative amounts of unsubstituted parent compounds, mainly naphthalene, biphenyl, fluorene, dibenzofuran, dibenzothiophene, phenanthrene and phenylnaphthalenes. Brocks et al. (2003) also state that the bitumens (PBS) from the iron ore mines at Tom Price and Whaleback are unusual. They are characterised by high relative concentrations of parent aromatic hydrocarbons, high relative concentrations of dibenzofuran, dibenzothiophene and phenylnaphthalenes, very low relative concentrations of hydrocarbons with four or more aromatic rings, large variations in the relative concentration of saturated over aromatic hydrocarbons, large variations in absolute bitumen yield, condensate-like n-alkane profiles and the presence of sterane and hopane biomarkers. Many PAHs are well known mutagens and carcinogens (Choudhury &

Bush, 1981), and have been associated with respiratory disease and lung cancer (Fan, Jung, & Lioy, 2006).

The United States Environmental Protection Agency (USEPA) has identified and listed a number of chemical compounds which are known to be important for human health (Carras, Day, Saghafi, Roberts, 2005). Some of these compounds have been classified as air toxics, for which the USEPA intend to introduce air quality standards. Among the compounds included as air toxics are PAHs. The majority of these compounds are produced as a result of the incomplete combustion of coal and organic substances. The carcinogenic effect of some of these compounds has been established; however, not all PAHs are carcinogens. The USEPA has proposed regulations for governing major sources of PAHs and has developed reference doses for some PAHs. In Australia, two types of PAHs, (benzopyrene and chrysene) are specifically referred to in the National and Occupational Health and Safety Commission's National Exposure Database, but no exposure limits have been set. Instead, they recommend that exposure to these compounds be controlled to the lowest practicable limit (Carras et al., 2005).

2.11.1 Naphthalene

The USEPA (2000) reports that acute exposure (short term) to naphthalene by inhalation, ingestion, and dermal contact is associated with haemolytic anaemia, damage to the liver, and neurological damage. Cataracts have also been reported in workers acutely exposed to naphthalene by inhalation and ingestion. Chronic exposure (long term) of workers and rodents to naphthalene has been reported to cause cataracts and damage to the retina. Haemolytic anaemia has been reported in infants born to mothers who 'sniffed' and ingested naphthalene (as mothballs) during pregnancy.

2.11.2 Biphenyl

The USEPA (1995) reports that acute exposure to high levels of biphenyl in humans has been observed to cause eye and skin irritation and toxic effects on the liver, kidneys, and central

and peripheral nervous systems. Kidney effects have been observed in chronically exposed animals. The USEPA has classified biphenyl in Group D, which is not classifiable as to human carcinogenicity.

2.11.3 Dibenzofuran

Exposure to dibenzofuran may occur from inhaling contaminated air, or ingesting contaminated food or drinking water. No information is available on the acute, chronic, reproductive, developmental, and carcinogenic effects of dibenzofuran in humans or animals. Although information on the health effects of the polychlorinated dibenzofurans is available, the USEPA has noted that the biological activity of various chlorinated dibenzofurans varies greatly; therefore, risk assessment by analogy to any of these more widely studied compounds would not be recommended. The USEPA has classified dibenzofuran as a Group D, not classifiable as to human carcinogenicity (USEPA, 1992).

2.11.4 Benzene

Benzene is a Group 1 known human carcinogen with sufficient evidence that chronic benzene exposure causes acute myeloid leukaemia (IARC, 2010). The main route of exposure to benzene is by inhalation but can also be absorbed through the skin. Acute exposure to high concentrations of benzene vapours can result in irritation of the skin, eyes and respiratory system and in central nervous system depression and arrhythmias (IARC, 1987).

2.12 Bitumens

The International Agency for Research on Cancer (IARC) has classified bitumen vapour in group 2B (possibly carcinogenic to humans) (IARC, 2011), whereas the American Conference of Industrial Hygienists (ACGIH) has classified bitumen in group 4 (not classifiable as a human carcinogen) (Campo et al., 2006). In contrast, a recent European epidemiological study on cancer mortality of asphalt workers has concluded that workers

employed in road paving, asphalt mixing, and other jobs related to bitumen vapour exposure may experience an increase in mortality due to lung cancer (Campo et al., 2006).

2.13 Occupational exposure

PAHs are generally not produced commercially except as research chemicals. However, PAHs are found in coal, coal tar, and in creosote oils, oil mists and pitches formed from the distillation of coal tars. The National Institute for Occupational Safety and Health (NIOSH) concluded that occupational exposure to coal products can increase the risk of lung and skin cancers in workers. NIOSH established a recommended occupational exposure limit time-weighted average (REL-TWA) for coal tar products of 0.1 mg/m³ for an 8-hour workday, within a 40-hour workweek (NIOSH, 1978). The ACGIH recommends an occupational exposure limit for coal tar products of 0.2 mg/m³ averaged over an 8-hour exposure (OSHA, 2012).

Carras et al. (2005) measured the concentrations and exposures of PAHs that might be experienced by mine workers due to selected organic air toxics arising from emissions associated with spontaneous combustion in open-cut coal mining. This was achieved by fitting field samplers within a D11 bulldozer cabin and several stationary locations in close proximity to spontaneous combustion fires. Results obtained from within the bulldozer cabin were lower than those collected from stationary locations. For each sample taken within the bulldozer, the total concentration of PAHs was low and similar to what might be expected for urban air. The air samples taken from the outdoor stationary locations were higher, and in some cases much higher, than the cabin air. The data suggest that people working in close proximity to spontaneous combustion fires may experience exposures that are higher than the recommended occupational health and safety values.

2.14 Dusts associated with iron ore mining

Dust is a general term used to describe particles that are suspended in the air we breathe. Scientists and regulators use the term “particulate matter” (PM) followed by a number to

describe the size of dust particles, for environmental health purposes. PM10 for instance, refers to all microscopic dust particles measuring 10 micrometres in aerodynamic diameter and less. Dust comes in a wide range of sizes and from a wide variety of sources, including soil, vegetation, microorganisms (pollens, fungi, bacteria) sea salt, fossil fuel combustion, bush fires, and industrial activities. In addition, common atmospheric gases such as sulphur dioxide and nitrogen oxides may react in the atmosphere over time to form particles.

A wide range of mining-related activities generate dust in the Pilbara, including the removal of vegetation, transport and loading activities and wind action on industry stockpiles and exposed areas. The combustion of gasoline and diesel also contributes to levels of air particle pollution. Different mining activities generate different amounts of dust. Climatic factors, such as rainfall, temperature and winds also significantly influence dust levels which makes the dry windy climate of the Pilbara an ideal environment for disturbing and distributing dust (Western Australian Department of Health, 2010).

2.14.1 Health effects of dust

The size and composition of the dust particles, concentration of dust and duration of exposure are all factors that influence how dust affects health and there is on-going research investigating the relative contribution of these factors on health.

Dust irritates the lungs and can trigger hay-fever type reactions, as well as asthma attacks. In people who already have these problems these attacks can be serious and cause breathing problems. Dust can cause coughing, wheezing and runny noses in most people but the elderly, babies and children are more sensitive to these affects. People with existing breathing and heart conditions and smokers may not be aware they are being affected and are at greater risk of developing chronic disorders and future disorders (WHO, 2007).

The composition of dust plays an important role in its overall toxicity. Studies have shown fine urban dust particles, rich in combustion materials such as vehicle exhaust, are more

hazardous to human health than dust particles rich in crustal material such as soil (US EPA, 1986).

The health effects of exposure to coalmine dust have been studied in more than 50,000 coal miners in the UK, with detailed concurrent measurements and analyses of dust over more than 25 years (e.g. Hurley, Cherrie, Donaldson, Seaton & Tran, 1987). The strongest early evidence linking long-term exposure to ambient particles with reductions in life expectancy (Pope et al., 1995) was based on an American Cancer Society study of more than 500,000 adults in about 150 US cities, followed up for several years. In each case, it was necessary also to take account of other possible causal factors such as smoking habits.

These major research programmes have shown clear relationships between workplace dusts, ambient pollution and various adverse health effects, especially on the cardio-respiratory systems. There are, however, still many uncertainties and controversies, and ongoing research has the aim of reducing or resolving them. While such studies only show statistical associations, and quantify the likelihood that those associations might have occurred by chance, they are the most informative in deciding whether a given population is at measurable risk. They have been used extensively in estimating risks to workers in dusty industries and determining appropriate exposure standards to prevent disease occurring. These are also the types of study on which information on the health effects of urban pollution have been based (Hurley et al., 2003).

2.14.2 What factors influence the risks of disease?

Together, toxicological and exposure research, along with epidemiology and human experimental studies have helped establish what aspects of exposure to particles influence the risks of disease. There are uncertainties but the main factors that need to be taken into account are given below.

(a) *How much exposure* to dust do people at risk experience in the short term and/or in the long term, e.g. over a working life. In considering this, long-term exposure of individuals is usually separated into two components:

- *duration of exposure,* i.e. amount of time spent exposed to the dust of interest; and
- *intensity of exposure,* i.e. the concentration of dust to which a person is exposed for any given period of time.

(b) *The toxicity of the dust per unit exposure*. On best current thinking, this toxicity is determined by various physico-chemical characteristics of the dust itself, such as:

- its size distribution, and in some circumstances the shape of the particles;
- the composition of the dust, including its solubility; and
- various surface properties of the dust.

(c) *The susceptibility of the exposed population* – the risks of disease depend not only on how much and what kinds of dust people are exposed to, but also on the vulnerability of those who are exposed; for example, the extent of pre-existing heart or lung disease (IOM, 2003).

2.14.3 Inhalability of dust

The same physical principles that determine the rate at which particles in the air fall to the ground determine where they can lodge in the lung (Donaldson et al., 2002). It is usual for scientists in this field to refer to inhalable and respirable dust. The former includes all particles that, when breathed in, pass beyond the larynx (voice box). The latter are those that reach the delicate structures in the furthest part of the lung where exchange of gases occurs, the acinus. They differ in their sizes, however, and this determines what proportion upon reaching the acinus is actually deposited (Volckens & Leith, 2002). Of the smallest particles, below about 100 nm, about half are deposited. Above that size, up to about 7μm, about a third are deposited. Particle size refers not to actual diameter but to a relative diameter in relation to that of a particle of the density of water that determines the rate at which the particle falls, this is referred to as the aerodynamic diameter. Thus a particle of iron may have an actual

diameter of 1µm but would have the falling speed of a much larger particle and would thus be less likely to reach the acinus as it would be more likely to settle in the airways on the way down the lung (Hurley, et al 2003).

2.14.4 Deposition of inhaled particles

The lung comprises a system of ever-narrowing tubes that terminate in the alveolar region where gas exchange occurs. Conventionally the respiratory system is divided into three compartments: the upper airways (nose, throat and larynx), the tracheo-bronchial region (windpipe and bronchial tubes) and the acinar region, which are the terminal airways and their associated alveoli (the tiny tubes and the fine air spaces into which they lead). Inhaled particles, depending on their size and density, may deposit in the upper airways, the tracheo-bronchial region or the central parts of the acini (centri-acinar region of the lung), the terminal bronchioles, alveolar ducts and proximal alveoli (Donaldson et al., 2002). If they deposit in the upper airways or the airways of the bronchial tubes, then clearance by the 'mucociliary escalator' is relatively rapid, with most of the particles being swept upwards and out of the lungs to be swallowed, within 24 hours (Donaldson et al, 2002).

Particles that are aerodynamically small enough continue deeper into the lungs before depositing and a proportion of them settle in the acinus. Here clearance is mediated by macrophage cells which ingest the particles and move them back up the respiratory system to be removed from the lungs by the mucociliary escalator. Approximately 65 days is needed before half of the particles are removed in rats, this process takes longer in humans with unimpaired clearance (Tran, Miller & Soutar, 2005). This process takes even longer in humans whose clearance mechanism has become impaired by lung disease. There is therefore a special research interest in the behaviour of particles and cells beyond the airways towards the centri-acinar region of the lung, since particles deposited there remain in the lung longer and are in contact with an especially delicate region of the lung that is prone to injury. Particles that deposit in this region are the major concern for long-term health effects caused by poorly soluble particles (Brody et al., 1984).

The primary function of the lung is to allow the transfer of oxygen from air into the blood stream and of carbon dioxide in the opposite direction and this necessitates that the blood and the air are brought into close proximity, a potentially dangerous situation. The mammalian lung has therefore evolved robust systems to defend the acinar structures, which are constantly threatened from the time of birth by deposition of inhaled particles, particularly dust, bacteria and viruses. As indicated above, larger particles are removed from the airways by their capture in airway mucus. Smaller particles are engulfed by defensive macrophage cells that can both remove the particles and also summon the assistance of other cells by generating an inflammatory reaction. The ability of particles to generate such a reaction does not necessarily mean that lung damage will occur, but it indicates that they are capable of provoking a defensive reaction. However, protracted (chronic) low level inflammation or large scale (acute) inflammation can result in permanent change in lung structure such as fibrosis.

These defences may be overcome in a number of ways. Firstly, particles such as quartz, asbestos and some microbes may simply prove too toxic for macrophages to cope with (Brody et al., 1984). Secondly, the sheer number of particles may be too large for the macrophages to clear. Thirdly, the size of the particles may be so small that they evade phagocytosis and interact with the epithelial cells in prolonged fashion even passing through the epithelial cell layer to set up inflammation, not in the airspaces but in the internal (interstitial) tissue of the lung itself (Brody et al, 1984).

This process of ‘interstitialisation’ is one factor that has been evoked to explain the harmful effects of the ultrafine particles characteristic of urban air (Seaton et al., 1995; Donaldson, Stone, Seaton, & MacNee, 2001). Removal of particles from this interstitial space is slower and depends on transport through the lung’s internal system of lymph vessels to glands (lymph nodes) where the immune defences are concentrated. Finally, even if inflammation leads to lung damage, there are systems for repair by regeneration of the lining cells of the air

spaces. Thus most individuals are able to cope with inhalation of large numbers of particles, some potentially very toxic, without showing any measurable damage to their lungs.

2.14.5 Particle characteristics and mechanisms of toxicity

Two cell types can be seen as being especially important in the lung response to particles:

(ii) the epithelial cells, which line the lung acinus, and

(iii) the alveolar macrophages that protect the acinus by scavenging.

As particles are deposited in the lungs they initially come into contact with a very thin layer of lipid-rich fluid that covers the epithelial cells. This fluid may be important in the iron ore mining industry as it may trigger chemical reactions upon contact with iron-rich particles.

Deposited particles then encounter the epithelial cell layer and contact between particles and epithelial cells occurs at the particle surface therefore the outcome of the interaction depends on the surface characteristics of the particle.

Macrophages then migrate to the particles and phagocytose ('swallow') them. The particle may affect the macrophage during phagocytosis and during residence inside it (Onodera, Suzuki, Matsuno, Kaneda, Takagi and Nishihira, 1997).

2.14.6 Low surface reactivity particles

Particles affect cells directly through contact with them and so the larger the aggregated surface area of the deposited particles, the larger the contact area between particles and cells and consequently the greater the effects of particles on the cells (compared with similar particles of lesser surface area). There is a clear relationship between surface area and pro-inflammatory effects *in vivo* (Duffin et al., 2002) and *in vitro* (Faux et al., 2003). Contact between particles and epithelial cells triggers the release of chemotactic proteins called chemokines (such as IL-8) (Gilmour et al., 2003) which attract leukocytes to the site of deposition and cause inflammation. Particles may also activate soluble chemotactic components in the lung lining fluid (Warheit et al., 1985).

2.14.7 Reactive particles

Some particles have much greater, or smaller, effects than others. For a given surface area, for example, quartz has a much more intense pro-inflammatory effect than does titanium dioxide (TiO_2) (Duffin et al., 2002). These effects are considered to be a consequence of the reactions between the quartz surface an cell membranes, resulting from the generation of free radicals at the quartz surface (Duffin et al, 2002).

2.14.8 Particle surface

As noted earlier, the mass of particulate matter per unit volume of air (expressed as mg/m^3 or $\mu g/m^3$) is the most common exposure metric used for regulatory purposes. Clearly, however, particle mass is not the metric of choice if the component that mediates the response is the total surface area, the reactive surface area, or a metal component released from the surface. Therefore, in terms of quantifying risk, the mass dose metric may be limited in its usefulness. This central role of the surface of particles means that better characterisation of the surface is one key component of future improved risk assessment (Donaldson & Borm, 2007).

2.14.9 Hygiene surveys and risk

The occupational hygiene survey is designed to evaluate employee exposures to hazardous substances and physical agents in the work place environment. The survey should identify potential sources of employee exposures and be based on the most cost effective monitoring strategy. Once the exposure is identified and measured it can be compared to the relevant OEL to determine conformity. The risk assessment should be based on the exposure and type of substance being assessed.

2.14.10 Conclusions from literature survey

The likelihood of exposures from occupational hazards such as dust, vapour and other organic compounds whilst undertaking mining activities in black shale areas at Tom Price is possible. The nature of the mining process liberates dust, gas and vapours during drill and blast,

sampling and haulage tasks. Exposures to these contaminants are known to have adverse side effects dependant on dose. Legislation dictates that exposures need to be kept to as low as reasonably practicable which requires the exposure to be identified, evaluated and then controlled which is the aim of this study.

3. RESEARCH AND EXPERIMENTAL DESIGN

3.1 Experimental design

This investigation used a longitudinal survey design and traditional occupational hygiene sampling methodology to collect and analyse samples of PBS, and to determine risks for workers by comparing measured exposure levels to exposure standards.

The aims of the study were to:

- Measure worker personal exposure to dust, fume and gases generated by mining PBS.
- Compare exposures with current OELs to identify and remediate potential health risk through the application of controls.
- Undertake analysis of PBS samples in lab to characterise gas/vapour hazards.
- Undertake static monitoring for gas/vapour at the mine site to identify potential hazard to workers.

Workers were grouped together into three SEGs dealing with:

1. drilling and blasting operations (drill and blast);
2. the analysis and sampling of the ore material (geologists and samplers); and
3. the movement of ore material to the process plant (heavy machinery operators).

3.2 Occupational study groups

3.2.1 Geologists and samplers

Geologists working in the pit coordinate the logging and sampling of shot holes, geological mapping, interaction with other pit personnel and inspection/clearance of shots. Office-based duties relate to pit designs, project work and administration (Adelsmayr, S, personal

communication, 2007). Geologists generally work a 48-hour week. Geologists perform an administrative role in relation to samplers within the pit environment, and geologists generally spend 50 % less time in the pit than samplers. Samplers working in the pit under the direction of the geologists are responsible for logging and collection of shot samples. Samplers generally work a 40-hour week and both groups work weekdays (Monday to Friday).

3.2.2 Heavy mobile equipment operators

This group includes workers who operate mobile heavy equipment within the pit including dump truck, excavator, water truck, grader and dozer operators. They work a 56-hour week on a roster of six days on and six days off (days on can also be nightshift). For this study, dump truck and excavator operators were selected to be sampled as they work in pits containing black shale.

3.2.3 Drill and blast team

The drill and blast team includes both the driller operators and their offsiders who drill the holes for areas proposed to be mined, and a blast crew team including shot firers who administer the explosives and detonation procedures. They generally work a 12-hour day on a roster of six days on and six days off (days on can also be nightshift).

Employees selected for participation in the study were conducting work in the pit environment and were therefore deemed most likely to be exposed to PBS. These groups were established prospectively (before the study commenced). Dust and toxic gas data collected from 2006 to 2009 were used to assess exposures. Due to the dynamic nature of mining operations, work in black shale pits occurred intermittently during the study period, thus limiting opportunities to conduct personal VOC monitoring. Laboratory testing of PBS was conducted to obtain additional data relating to the composition of the PBS and its associated emissions.

The exposure data collected for shift lengths greater than 8 hours were converted to an equivalent 8-hour time-weighted average as recommended by the Western Australia Department of Consumer Protection and Employment (Department of Mines and Petroleum, 1999). Substances with chronic health effects were adjusted using the ratio of average hours worked per week to the standard work week of 40 hours.

Statistical analysis indicates that some monitoring data do not conform to a lognormal distribution. However, for those similar exposure groups indicated, this may be the result of low sample numbers rather than a true deviation from the expected lognormal distribution. This being the case, the geometric mean and the geometric standard deviation were used to assess the SEGs with exposure levels above the occupational exposure limit (OEL) and the variability of the data, respectively. The arithmetic mean is used on a site basis to identify average exposures and for assessing control measures.

The data for each SEG and each contaminant were then analysed using the IHSTAT spreadsheet for statistical analysis of hygiene data (Mulhausen & Damiano, 1998), and the results were compared to the 8-hour OEL adopted by Brief and Scala and Safe Work Australia. Where it is likely that the 95 % upper confidence interval for the time-weighted average (TWA) mean concentration for total inhalable dust, respirable dust, or respirable crystalline silica, exceeded the relevant OEL, a SEG was deemed to be at risk and managed as such.

3.3 Dust sampling

Pall DM Metricel 0.8 µm 25 mm filter membranes were used for sample collection as they are hydrophobic and well suited for use under the humid conditions that can occur on site. Inhalable dust samples were collected in accordance with methods prescribed by Australian Standard AS 3640 (Standards Australia, 2004b). Respirable dust samples were collected in accordance with AS Australian Standard 2985 (Standards Australia, 2004a). All membranes were weighed prior to and after sampling at Edith Cowan University using a calibrated

laboratory balance. Some of the inhalable dust, respirable dust and respirable crystalline silica samples used in this study were collected by the site Occupational Health Technician and were analysed by MPL laboratories in Perth.

There is a time-weighted average (TWA) exposure standard for many particulates commonly found in workplace air and these vary with toxicity (Safe Work Australia, 2012). The TWA exposure standard is the concentration of a substance that most people can be exposed to over a normal 8-hour working day, for a 40-hour working week over 40 years. It is often expressed as milligrams per cubic metre of air sampled (mg/m^3), or as parts per million (ppm), or as fibres per millilitre of air sampled (f/mL). As examples, the following TWA values have been adopted by Rio Tinto:

- total inhalable dust not otherwise classified is 10 mg/m^3
- respirable dust is 3 mg/m^3
- free silica (as quartz) is 0.1 mg/m^3.

These OELs have been based on Safe Work Australia's adopted national exposure standards for atmospheric contaminants in the occupational environment (NOHSC, 1995b) and adjustment of exposure standards for extended work shifts follows the Western Australian Department of Minerals and Energy method (Department of Minerals and Energy, 1999)

3.4 Toxic gas measurements

The Rio Tinto guidance note for PBS (Rio Tinto, 2007) states that when undertaking activities in areas where there is potential for exposure to hazardous gases from PBS, personal electrochemical gas detectors (e.g. a Draeger Miniwarn and Multiwarn) must be worn by employees. The gas detector provides an audible warning alarm at the OEL for the following:

- sulphur dioxide (SO_2) at 5 ppm (STEL) and 1 ppm (TWA 12 hour)
- hydrogen sulphide (H_2S) at 15 ppm (STEL) and 5 ppm (TWA 12 hour)

- carbon dioxide (CO_2) at 0.5 % volume
- carbon monoxide (CO) at 15 ppm (TWA 12 hour); and
- oxygen (O_2) below 19.5 % volume.

Two Draeger Multiwarn gas detectors ARSE 1794 and ARKD 0002 with data-logging capabilities are used by operations to monitor toxic gases whilst employees/contractors are working in black shale pits. All of the historical data from 2006-2007 were collated. The Multiwarns were set up to sample for oxygen, hydrogen sulphide, sulphur dioxide, carbon dioxide and methane.

3.5 Organic vapours

Geotech Pty Ltd geotechnical services provided tenax carbosieve sorbent tubes for organic vapour sampling. Organic vapours were sampled in terms TO17 (USEPA, 1999). Samples were analysed by Geotech Pty Ltd, which is a National Association of Testing Authorities (NATA) approved laboratory, in order to identify specific components such as PAHs and VOCs.

Geotech Pty Ltd was provided with 20 kg of cold black shale in order to conduct a preliminary trial. Three samples of the black shale were crushed and combined. A sub sample of this combined shale was placed in a quartz furnace tube which was heated to 160 ºC with a flow of nitrogen gas at a rate of 50 ml/min. The gas was collected on a tenax carbosieve sorbent tube and analysed by thermal desorption gas chromatography mass spectrometry (TD-GCMS) at 300°C and 30 mL/min to determine the amount of VOCs within the shale according to WIENV 31, based on the analytical section of TO17 (USEPA, 1999). This experiment was repeated in triplicate. The solid material was also analysed by LECO for carbon and sulphur content.

A second laboratory trial was devised to test if there was a difference between cold black shale and hot reactive shale. Seven reactive black shale samples were collected on 16/11/07. The samples were crushed and milled at the Tom Price laboratory and 1 g of hot black shale

was placed into several quartz sample tubes. The samples were placed into an iced portable cooler for transport to the analytical laboratory in Perth, and then refrigerated until delivery to Geotech. The quartz tubes were placed individually in a furnace and heated to 240 ºC and then 500 ºC with a flow of nitrogen gas at a rate of 50 ml/min. The gas was collected on a sorbent tube at both of these temperatures and analysed by thermal desorption gas chromatography mass spectrometry to determine the amount of VOCs within the shale according to WIENV 31, based on the analytical section TO17 (USEPA, 1999).

Three positional samples from the NTD2 waste dump were taken using tenax carbosieve tubes (Figures 5 & 6). The tenax carbosieve sorbent tubes were analysed to determine the amount of VOCs according to WIENV 31, based on the analytical section of TO17. The results are tabulated and presented in Chapter 4; compounds present were quantified using an internal standard (d8 toluene).

Figure 5. Photo of sampling equipment in waste dump NTD2 sampling vapour from reactive hot black shale.

Figure 6. Photo of sampling equipment and vapour being expulsed hot black shale at waste dump NTD2.

3.6 Limitations

There were some limitations of the sampling program that should be noted;

- Samples were transported back to Perth by air for analysis. Some of the sampled dust could have become displaced from the filter papers during transit.
- Vapour tubes were sealed immediately after sampling and were also transported by air. Tubes were kept cool on ice and provided to the laboratory the next day. The time lapse between sample collection and analysis could have resulted in some of the vapour off gassing.
- Weather conditions varied each day when sampling was conducted which could also result in some variability.

4. RESULTS

4.1 Dust results

Table 1 provides a summary of total dust levels collected for all SEGs (all results have been corrected to an 8-hour shift). As can be seen in Table 1, 70 inhalable dust samples were collected over the study period for the three SEGs. None of these samples were above the OEL of 10.0 mg/m³. A total of 80 respirable dust samples were obtained over the study period. Of these samples, three (6 %) were found to be above the Western Australian OEL of 3 mg/m³. The exceedences for respirable dust were recorded for three drill rig operators drilling blast patterns.

The 95th percentile values and the 95 % upper confidence intervals for respirable and inhalable dusts were below the OELs for all SEGs. However, the geometric standard deviations for both were above 2.5 for drill and blast, and heavy machinery operators, indicating high variability and the possibility of heterogeneous data sets within these SEGs.

A total of 80 respirable crystalline silica samples were collected over the study period. Of those samples, six (17 %) were found to be above the OEL of 0.1 mg/m³. The six exceedences for respirable crystalline silica were recorded for three excavator operators, and three drill rig operators drilling blast holes. The 95 % upper confidence interval was below the OEL for all SEGs. The 95th percentile value was below the OEL while the values for drill and blast, and heavy machinery operators, exceeded the OEL.

The geometric mean and minimum variance unbiased estimator (MVUE) data for all SEGs were below the OEL; however, the geometric standard deviations exceeded 2.5 for drill and blast, and heavy machinery operators respectively, indicating that there may be more than one exposure group within each of these data sets.

Table 1. Total dust summary for all SEGs (all results have been corrected to an 8-hour shift)

Inhalable Dust

SEG	number of samples	Samples >OEL numbers	Samples >OEL %	Range	Max	Min	Median	Mean	SD	Geom. Mean	Geom. SD	MVUE	95th %ile	Mean 95% UCL
Geologists/Samplers	5	0	0	1.443	2.140	0.700	1.645	1.478	0.575	1.373	1.567	1.487	2.876	2.841
Drill and Blast	50	0	0	4.950	5.000	0.050	1.400	1.801	1.296	1.309	2.597	2.038	6.292	2.805
Heavy Machinery Operators	15	0	0	3.750	3.800	0.050	0.200	0.644	1.020	0.245	4.231	0.615	2.632	2.634
Total	70	0	0%											

Respirable Dust

SEG	number of samples	Samples >OEL numbers	Samples >OEL %	Range	Max	Min	Median	Mean	SD	Geom. Mean	Geom. SD	MVUE	95th %ile	Mean 95% UCL
Geologists/Samplers	6	0	0	0.480	0.120	0.600	0.434	0.416	0.159	0.375	1.773	0.429	0.962	0.908
Drill and Blast	50	3	6	4.298	4.350	0.050	0.435	0.733	0.977	0.376	3.353	0.764	2.754	1.210
Heavy Machinery Operators	24	0	0	2.550	2.600	0.050	0.400	0.400	0.670	0.328	3.694	0.725	2.815	1.692
Total	80	3	6%											

Respirable Crystalline Silica

SEG	number of samples	Samples >OEL numbers	Samples >OEL %	Range	Max	Min	Median	Mean	SD	Geom. Mean	Geom. SD	MVUE	95th %ile	Mean 95% UCL
Geologists/Samplers	6	0	0	0.013	0.020	0.007	0.010	0.012	0.006	0.011	1.607	0.012	0.025	0.022
Drill and Blast	50	3	16	0.452	0.450	0.005	0.005	0.046	0.092	0.013	4.210	0.035	0.140	0.065
Heavy Machinery Operators	24	3	17	0.155	0.160	0.005	0.020	0.042	0.052	0.020	3.604	0.043	0.164	0.097
Total	80	6	17%											

4.2 Cold black shale VOCs

A summary of the quantitative results of the thermal desorption gas chromatography mass spectrometry of the tenax carbosieve tubes used for collection of the combined cold black shale heated to 160 ºC is shown in Table 2. The available OELs are also shown for comparison. Compounds detected as atmospheric contaminants included hexane, benzene, cyclohexene, heptane, toluene, ethylbenzene and xylenes. Hexanoic acid had the greatest concentration (0.756 ppm) but the concentrations of individual compounds do not approach any exposure standard levels.

Table 2. Volatile organic compound results of black shale heated to 160 ºC

Tentative Identification	**Test method**	*Sample ID* *Date sampled* **Exposure Standard (ppm)**	10190707.D 15/10/2007 **ppm**
Acetic acid	WIENV 31	10 (NIOSH)	0.048
Hexane	WIENV 31	20 (Safe Work Aust)	0.036
Benzene	WIENV 31	1 (Safe Work Aust)	0.081
Cyclohexene	WIENV 31	300 (NIOSH)	0.015
Heptane	WIENV 31	400 (Safe Work Aust)	0.018
2-Hexene, 2,3-dimethyl-	WIENV 31	Not Listed	0.007
Toluene	WIENV 31	20 (Safe Work Aust)	0.255
1-Octene	WIENV 31	Not Listed	0.012
Hexanoic acid	WIENV 31	Not Listed	0.756
Ethylbenzene	WIENV 31	100 (NIOSH)	0.013
1,3-Xylene	WIENV 31	80 (Safe Work Aust)	0.018
1,2- Xylene	WIENV 31	80 (Safe Work Aust)	0.013
Heptanoic acid	WIENV 31	Not Listed	0.040
Nonanal	WIENV 31	Not Listed	0.012
Octanoic acid	WIENV 31	Not Listed	0.088
Benzoic acid	WIENV 31	Not Listed	0.148
Nonanoic acid	WIENV 31	Not Listed	0.379
Tridecane	WIENV 31	Not Listed	0.012
2-nonenoic acid	WIENV 31	Not Listed	0.024
Decanoic acid	WIENV 31	Not Listed	0.106
Tetradecane	WIENV 31	Not Listed	0.010
Undecanoic acid	WIENV 31	Not Listed	0.058
Pentadecane	WIENV 31	Not Listed	0.008
Dodecanoic acid	WIENV 31	Not Listed	0.319
Tridecanoic acid	WIENV 31	Not Listed	0.280
Tetradecanoic acid	WIENV 31	Not Listed	0.105
Pentadecanoic acid	WIENV 31	Not Listed	0.017
Tetradecene	WIENV 31	Not Listed	0.011
Hexadecanoic acid	WIENV 31	Not Listed	0.122

4.3 Positional samples

The positional samples taken from the reactive hot black shale waste dump at NTD2 onto tenax carbosieve tubes and analysed by TD-GCMS are shown in Table 3. The exposure standard values are also shown for comparison. Compounds detected as atmospheric contaminants included xylene, heptane, limonene, phenol, toluene and a number of acids (acetic acid, benzoic acid, formic acid). Acetic acid had the greatest concentration (0.873 mg/m^3) but the concentrations of individual compounds do not approach any exposure standard levels.

Table 3. Volatile organic compound results of positional samples at a reactive hot black shale waste dump (NTD2) (volume 15 L)

Tentative Identification	Test method	*Sample ID* *Date sampled* **Exposure standard (mg/m3)**	11300709.D 29/11/2007 **Tube 1** **ppm**	11300708.D 29/11/2007 **Tube 2** **ppm**	11300710.D 29/11/2007 **Tube 3** **ppm**	11300707.D 29/11/2007 **Tube 4** **ppm**
1,2-xylene	WIENV 31	350 (Safe Work Aust)		0.0014		ND
1,4-Dioxane	WIENV 31	3.6 (NIOSH)	0.061		0.015	ND
1H-Pyrrole	WIENV 31	Not Listed	0.005			ND
1-Tetradecene	WIENV 31	Not Listed	0.003			ND
2-Pentanone	WIENV 31	530 (NIOSH)	0.005	0.0009	0.001	ND
6-Octadecenoic acid, methyl ester	WIENV 31	Not Listed			0.003	ND
Acetic acid	WIENV 31	25 (NIOSH)	0.34	0.22		ND
Acetic acid, methyl ester	WIENV 31	Not Listed		0.05	0.04	ND
Benzoic acid	WIENV 31	Not Listed	0.13	0.05	0.062	ND
Benzoic acid, methyl ester	WIENV 31	Not Listed		0.02	0.004	ND
decanoic acid	WIENV 31	Not Listed	0.005	0.003	0.005	ND
Disulfide, dimethyl	WIENV 31	Not Listed			0.003	ND
Dodecanal	WIENV 31	Not Listed	0.003			ND
Dodecane	WIENV 31	Not Listed	0.002			ND
dodecanoic acid	WIENV 31	Not Listed	0.007	0.007	0.005	ND
Eicosane	WIENV 31	Not Listed	0.009			ND
Formic acid	WIENV 31	9 (NIOSH)		0.02	0.014	ND
Geranyl acetone	WIENV 31	Not Listed	0.004			ND
Heptane	WIENV 31	1640 (Safe Work Aust)	0.006			ND

Hexadecane	WIENV 31	Not Listed	0.008			ND
Hexadecanoic acid	WIENV 31	Not Listed	0.01	0.017	0.02	ND
Hexadecanoic acid, methyl ester	WIENV 31	Not Listed			0.036	ND
Hexanoic acid	WIENV 31	Not Listed		0.001		ND
Hexanoic acid, 2-ethyl-	WIENV 31	Not Listed	0.005			ND
Limonene	WIENV 31	Not Listed	0.005			ND
N-Hexanal	WIENV 31	Not Listed	0.003			ND
Nonanoic acid	WIENV 31	Not Listed	0.015	0.004		ND
Octadecanoic acid, methyl ester	WIENV 31	Not Listed			0.009	ND
Octanoic acid	WIENV 31	Not Listed	0.006			ND
Pentadecane	WIENV 31	Not Listed	0.003			ND
Phenol	WIENV 31	4 (Safe Work Aust)		0.007		ND
Phenol, nonyl-	WIENV 31	Not Listed			0.003	ND
Silane, methoxytrimethyl-	WIENV 31	Not Listed			0.03	ND
Silanol, trimethyl-	WIENV 31	Not Listed			0.14	ND
Tetradecane	WIENV 31	Not Listed	0.003			ND
Tetradecanoic acid	WIENV 31	Not Listed	0.014	0.005	0.006	ND
Tetradecanoic acid, methyl ester	WIENV 31	Not Listed			0.0006	ND
Thiophene	WIENV 31	Not Listed	0.007	0.002		ND
Toluene	WIENV 31	75 (Safe Work Aust)	0.015			ND
tridecanal	WIENV 31	Not Listed	0.002			ND
Tridecane	WIENV 31	Not Listed	0.004			ND
Tridecanoic acid	WIENV 31	Not Listed		0.008		ND

4.3.1 Hot black shale samples heated to 340 °C and subsequently 500 °C

Results of several hot black shale samples from a waste dump heated to 340 °C are shown in Table 4. The OELs available are also shown for comparison, although it must be recognised that these concentrations cannot be directly linked to their OELs as they are not personal samples and do not relate to actual concentrations in the pit. Benzene and carbon disulfide had the highest concentrations. Other compounds detected as atmospheric contaminants included: 1-octene; 1-hexene; acetamide xylene; heptanes; limonene; butanal; cyclohexene; hexane; pentene, 2-methyl; phenol; toluene; and a number of acids.

The tubes that were heated to 340 °C were further heated to 500 °C with the resultant gas collected onto a new set of tenax carbosieve tubes and analysed by TD-GCMS. Results are shown in Table 5. Common compounds detected as atmospheric contaminants included 1,3-cyclopentadiene; 1,4-cyclohexadiene; acetamide, N,N-dimethyl; 1-hexene; 1-pentene; 1-octene; acetic acid; benzene; benzene, 1,2-dichloro; butanal; hexane; phenol; and toluene.

Table 4. Volatile organic compound results of black shale heated to 340 ºC collected on a sorbent tube and analysed by TD-GCMS

		Sample ID	11300703.D	11210717.D	11280714.D	11280718.D	11290722.D	11290725.D	11300705.D
Tentative Identification	**Test method**	*Date sampled* **Exposure standard (PPM)**	**18/11/2007 Tube 1 ppm**	**18/11/2007 Tube 2 ppm**	**18/11/2007 Tube 3 ppm**	**18/11/2007 Tube 4 ppm**	**18/11/2007 Tube 5 ppm**	**18/11/2007 Tube 6 ppm**	**18/11/2007 Tube 7 ppm**
1,2-xylene	WIENV 31	80 (Worksafe Aust)						0.036	0.017
1,3/1,4-xylene	WIENV 31	80 (Worksafe Aust)	0.035			0.016			
1,3-Butadiene, 2-methyl-	WIENV 31	Not Listed	0.106						0.070
1,3-Butanediamine	WIENV 31	Not Listed							0.172
1,3-Cyclopentadiene	WIENV 31	Not Listed		0.293					
1,3-Cyclopentadiene, 5-methyl-	WIENV 31	Not Listed							0.036
1,3-Pentadiene	WIENV 31	Not Listed						0.061	
1,4-Cyclohexadiene	WIENV 31	Not Listed							0.043
1,4-Pentadiene	WIENV 31	Not Listed		0.139					
1-Butanol	WIENV 31	50 (NIOSH)					0.088		
1-Butene	WIENV 31	Not Listed	0.213						0.391
1-Butene, 2,3-dimethyl-	WIENV 31	Not Listed			0.147				0.070
1-Butene, 2,3-dimethyl-	WIENV 31	Not Listed							
1-Butene, 2-methyl-	WIENV 31	Not Listed		0.508					
1-Heptene	WIENV 31	Not Listed		0.147		0.067		0.143	0.125
1-Hexene	WIENV 31	Not Listed	0.215	0.384	0.206	0.036	0.180		
1-Hexene, 2-methyl-	WIENV 31	Not Listed		0.024					
1H-Tetrazole, 5-methyl-	WIENV 31	Not Listed	0.024						
1-Octene	WIENV 31	Not Listed		0.096	0.057	0.076	0.044	0.094	0.064
1-Pentene, 4-methyl-	WIENV 31	Not Listed						0.117	0.287
1-Propene	WIENV 31	Not Listed		0.095					
1-Propene, 2-methyl-	WIENV 31	Not Listed	0.737	1.716	0.123		0.044		0.123
1-Tetradecene	WIENV 31	Not Listed			0.036				
2-Butenal	WIENV 31	Not Listed						0.130	
2-Butenal, (E)-	WIENV 31	Not Listed							0.146
2-Butene	WIENV 31	Not Listed		0.487					
2-Butene, 2,3-dimethyl-	WIENV 31	Not Listed	0.039				0.058		
2-Butene, 2-methyl-	WIENV 31	Not Listed			0.234			0.052	
2-Butenoic acid, methyl ester	WIENV 31	Not Listed				0.018			
2-Heptene	WIENV 31	Not Listed		0.057					0.018
2-Hexene	WIENV 31	Not Listed	0.286	0.292			0.058	0.047	0.185
2-Hexene, 2-methyl-	WIENV 31	Not Listed	0.029	0.073			0.076		0.045
2-Hexene, 3-methyl-	WIENV 31	Not Listed		0.029					
2-Methyl-2-hexene	WIENV 31	Not Listed			0.070				
2-Oxetanone, 4,4-dimethyl-	WIENV 31	Not Listed			0.412				
2-Pentanone	WIENV 31	Not Listed	0.047						
2-Pentene	WIENV 31	Not Listed		0.161					
2-Pentene, 2-methyl-	WIENV 31	Not Listed	0.143						
2-Pentene, 3-methyl-	WIENV 31	Not Listed	0.091	0.164	0.048				0.125
2-Pentene, 4-methyl-	WIENV 31	Not Listed	0.221	0.159					
2-Propanone	WIENV 31	Not Listed		2.340		0.280			
2-Propenal, 2-methyl-	WIENV 31	Not Listed						0.056	
2-Propenoic acid, 2-methyl-, methyl e	WIENV 31	Not Listed						0.026	
3-Hexene	WIENV 31	Not Listed		0.080					
3-Methyl-trans-2-pentene	WIENV 31	Not Listed	0.136				0.040		

Table 4. Volatile organic compound results of black shale heated to 340 ºC collected on a sorbent tube and analysed by TD-GCMS

		Sample ID	11300703.D	11210717.D	11280714.D	11280718.D	11290722.D	11290725.D	11300705.D
Tentative Identification	**Test method**	*Date sampled* **Exposure standard (PPM)**	**18/11/2007 Tube 1 ppm**	**18/11/2007 Tube 2 ppm**	**18/11/2007 Tube 3 ppm**	**18/11/2007 Tube 4 ppm**	**18/11/2007 Tube 5 ppm**	**18/11/2007 Tube 6 ppm**	**18/11/2007 Tube 7 ppm**
Acetaldehyde	WIENV 31	200 (OSHA)				0.207		0.225	
Acetamide, N,N-dimethyl-	WIENV 31	Not Listed	0.272	0.536	0.680	0.550	0.639	0.894	0.266
Acetic acid	WIENV 31	10 (NIOSH)	0.413			0.132	0.222	0.726	0.415
Benzaldehyde	WIENV 31	Not Listed		0.037					
Benzene	WIENV 31	1 (Worksafe Aust)	0.842	**1.387**	**1.023**	0.502	**1.288**	**1.314**	**1.205**
Benzene, 1,2,4-trimethyl-	WIENV 31	Not Listed		0.032					
Benzene, 1,3-dichloro-	WIENV 31	50 (NIOSH)		0.026	0.026				
Benzene, 1,3-dimethyl-	WIENV 31	Not Listed		0.026					
Benzene, 1,4-dichloro-	WIENV 31	50 (NIOSH)				0.046			
Benzene, nitro-	WIENV 31	1 (NIOSH)					0.290		
Benzothiazole	WIENV 31	Not Listed				0.022			
Butanal	WIENV 31	Not Listed		0.187	0.129	0.014	0.084		0.152
Butanal, 3-methyl-	WIENV 31	Not Listed					0.109		0.029
Butane, 2-methyl-	WIENV 31	Not Listed	0.156	0.104	0.172				0.052
Carbon disulfide	WIENV 31	1 (NIOSH)				**1.707**			
Cycloheptane	WIENV 31	Not Listed			0.140				
Cyclohexadecane	WIENV 31	Not Listed						0.052	
Cyclohexane	WIENV 31	100 (Worksafe Aust)							0.055
Cyclohexane, methyl-	WIENV 31	Not Listed		0.030					
Cyclohexene	WIENV 31	300 (NIOSH)	0.115	0.072			0.026	0.028	0.047
Cyclopentane, methyl-	WIENV 31	Not Listed			0.069				
Cyclopentane, methylene-	WIENV 31	Not Listed					0.077		
Cyclopentene	WIENV 31	Not Listed	0.213	0.109					0.164
Cyclopentene, 1-methyl-	WIENV 31	Not Listed	0.128						
Cyclopentene, 4-methyl-	WIENV 31	Not Listed		0.113	0.090				0.087
Cyclopropane, 1,2-dimethyl-, cis-	WIENV 31	Not Listed	0.819	0.377	0.114		0.159		0.243
Cyclotetradecane	WIENV 31	Not Listed				0.055			0.073
Decanal	WIENV 31	Not Listed		0.031					
Decane	WIENV 31	Not Listed		0.038		0.028	0.096		
Decane, 2-methyl-	WIENV 31	Not Listed						0.018	
Dodecane	WIENV 31	Not Listed		0.024		0.021			
Dodecanoic acid	WIENV 31	Not Listed						0.043	
Eicosane	WIENV 31	Not Listed						0.061	
Ethanol	WIENV 31	1000 (NIOSH)				0.026			0.076
Furan, 2-methyl-	WIENV 31	Not Listed						0.085	
Heptane	WIENV 31	400 (Worksafe Aust)		0.301				0.092	
Heptane, 2-methyl-	WIENV 31	Not Listed		0.020					
Hexadecane	WIENV 31	Not Listed		0.018					
Hexadecene	WIENV 31	Not Listed		0.090					
Hexane	WIENV 31	20 (Worksafe Aust)	0.628	0.423	0.146	0.029	0.155	0.236	0.552
Hexane, 2,4-dimethyl-	WIENV 31	500 (Worksafe Aust)		0.026					
Hexane, 2-methyl-	WIENV 31	500 (Worksafe Aust)		0.069					
Hexane, 3-methyl-	WIENV 31	500 (Worksafe Aust)		0.033					0.044

Table 4. Volatile organic compound results of black shale heated to 340 ºC collected on a sorbent tube and analysed by TD-GCMS

		Sample ID	11300703.D	11210717.D	11280714.D	11280718.D	11290722.D	11290725.D	11300705.D
Tentative Identification	**Test method**	*Date sampled* **Exposure standard (PPM)**	**18/11/2007 Tube 1 ppm**	**18/11/2007 Tube 2 ppm**	**18/11/2007 Tube 3 ppm**	**18/11/2007 Tube 4 ppm**	**18/11/2007 Tube 5 ppm**	**18/11/2007 Tube 6 ppm**	**18/11/2007 Tube 7 ppm**
Isoprene	WIENV 31	Not Listed						0.027	
Limonene	WIENV 31	Not Listed				0.031			
l-Limonene	WIENV 31	Not Listed			0.023				
Methane, chloro-	WIENV 31	Not Listed						0.039	
Methyllaurate	WIENV 31	Not Listed				0.014			
Naphthalene	WIENV 31	0.005 (NIOSH)		0.021					
N-Hexanal	WIENV 31	Not Listed				0.044			
Nonanal	WIENV 31	Not Listed		0.037		0.032			
Nonane	WIENV 31	Not Listed				0.025	0.054		
Nonanoic acid	WIENV 31	Not Listed		0.026				0.028	
Octane	WIENV 31	75 (NIOSH)		0.057					
Octane, 4-methyl-	WIENV 31	Not Listed		0.035					
pentadecane	WIENV 31	Not Listed		0.025	0.057				
Pentanal	WIENV 31	50 (NIOSH)			0.130	0.104			
Pentane	WIENV 31	120 (NIOSH)							2.707
Pentane, 2,2,4-trimethyl-	WIENV 31	Not Listed	0.046					0.025	0.086
Pentane, 2-methyl-	WIENV 31	Not Listed	0.109	0.079	0.085		0.030		0.123
Pentane, 3-methyl-	WIENV 31	Not Listed	0.063	0.030					0.102
Phenol	WIENV 31	1 (Worksafe Aust)	0.462	0.508	0.531	0.706	0.766	0.951	0.411
Phenol, 4-nonyl-	WIENV 31	Not Listed		0.013					
Propanal, 2-methyl-	WIENV 31	Not Listed		0.024					0.085
Propane	WIENV 31	1000 (NIOSH)					0.240		0.388
Propanoic acid, methyl ester	WIENV 31	Not Listed						0.047	
Tetradecane	WIENV 31	Not Listed		0.022	0.065	0.012			
Thiophene	WIENV 31	Not Listed	0.052	0.079					
Toluene	WIENV 31	100 (NIOSH)	0.141	0.296	0.145	0.176	0.256	0.120	0.161
Tridecanal	WIENV 31	Not Listed		0.014					
Tridecane	WIENV 31	Not Listed		0.030	0.037	0.015			
Undecane	WIENV 31	Not Listed		0.047		0.022			

Table 5. Volatile organic compound results of black shale heated to 500 ºC collected on a sorbent tube and analysed by TD-GCMS

Tentative Identification	Test method	*Sample ID* *Date sampled* Exposure standard (PPM)	11300704.D 18/11/2007 Tube 1 ppm	11210718.D 18/11/2007 Tube 2 ppm	11280716.D 18/11/2007 Tube 3 ppm	11280720.D 18/11/2007 Tube 4 ppm	11290724.D 18/11/2007 Tube 5 ppm	11290727.D 18/11/2007 Tube 6 ppm	11300706.D 18/11/2007 Tube 7 ppm
(E)-3-Methyl-2-hexene	WIENV 31	Not Listed	0.019						
1,2-Pentadiene	WIENV 31	Not Listed			0.046		0.044		
1,2-xylene	WIENV 31	80 (Worksafe Aust)							0.032
1,3,5-Heptatriene	WIENV 31	Not Listed							0.034
1,3/1,4-xylene	WIENV 31	80 (Worksafe Aust)	0.031				0.016		0.020
1,3/1,4-xylene	WIENV 31	80 (Worksafe Aust)							
1,3-Cyclohexadiene	WIENV 31	Not Listed				0.031			
1,3-Cyclopentadiene	WIENV 31	Not Listed		0.210	0.138	0.136	0.093	0.174	0.352
1,3-Cyclopentadiene, 5-methyl-	WIENV 31	Not Listed						0.049	0.051
1,3-Cyclopentadiene, methyl-	WIENV 31	Not Listed		0.027	0.031		0.022		
1,3-Pentadiene	WIENV 31	Not Listed		0.050				0.069	0.089
1,3-Pentadiene, (Z)-	WIENV 31	Not Listed				0.041			
1,4-Cyclohexadiene	WIENV 31	Not Listed			0.043	0.046	0.031	0.071	0.079
1-Butanol	WIENV 31	50 (NIOSH)		0.054		0.093		0.137	
1-Butene, 2-methyl-	WIENV 31	Not Listed						0.016	
1-Heptene	WIENV 31	Not Listed		0.101	0.118		0.101	0.160	0.133
1-Hexanol, 2-ethyl-	WIENV 31	Not Listed					0.024		
1-Hexen-3-yne, 2-methyl-	WIENV 31	Not Listed						0.045	
1-Hexene	WIENV 31	Not Listed	0.043	0.167	0.144	0.101	0.100	0.184	0.183
1-Octene	WIENV 31	Not Listed		0.116	0.057	0.070	0.078	0.087	
1-Pentene	WIENV 31	Not Listed		0.079	0.097	0.052	0.046	0.101	0.132
1-Propene, 2-methyl-	WIENV 31	Not Listed			0.110	0.067		0.114	0.168
1-Tetradecene	WIENV 31	Not Listed				0.030			
2,4-Hexadiene	WIENV 31	Not Listed			0.029		0.018	0.038	
2-Butenal	WIENV 31	2 (NIOSH)		0.050	0.065		0.053		
2-Butene, 2-methyl-	WIENV 31	Not Listed	0.031						
2-Heptene	WIENV 31	Not Listed	0.076						
2-Hexene	WIENV 31	Not Listed	0.147					0.034	0.077
2-Hexene, 2-methyl-	WIENV 31	Not Listed	0.066						
2-Pentanone	WIENV 31	Not Listed		0.059					
2-Pentene, (E)-	WIENV 31	Not Listed	0.028						
2-Pentene, 3-methyl-, (E)-	WIENV 31	Not Listed	0.017						
2-Pentene, 4,4-dimethyl-	WIENV 31	Not Listed	0.045						
2-Propanone	WIENV 31	Not Listed		0.526					
2-Propenoic acid, 2-methyl-, methyl ester	WIENV 31	Not Listed					0.023		
3-Heptene	WIENV 31	Not Listed	0.081						
Acetaldehyde	WIENV 31	200 (OSHA)	0.062						
Acetamide, N,N-dimethyl-	WIENV 31	Not Listed	0.332		1.143	0.375	0.913	1.256	0.247
Acetic acid	WIENV 31	10 (NIOSH)	0.443	0.108	0.107	0.494	0.435	0.901	0.319
Benzene	WIENV 31	1 (Worksafe Aust)	0.871	**1.881**	0.939	**1.224**	0.862	**1.337**	**1.163**
Benzene, 1,2-dichloro-	WIENV 31	75 (OSHA)		0.016	0.019	0.048			
Benzoic acid	WIENV 31	Not Listed					0.031		
Benzoic acid, phenyl ester	WIENV 31	Not Listed					0.372		
Butanal	WIENV 31	Not Listed		0.063	0.042	0.029	0.027	0.031	0.042
Butanal, 2-methyl-	WIENV 31	Not Listed		0.185					
Butanal, 3-methyl-	WIENV 31	Not Listed			0.097	0.096	0.087		
Butane, 2-methyl-	WIENV 31	Not Listed						0.077	

Table 5. Volatile organic compound results of black shale heated to 500 ºC collected on a sorbent tube and analysed by TD-GCMS

Tentative Identification	Test method	*Sample ID* *Date sampled* Exposure standard (PPM)	11300704.D 18/11/2007 Tube 1 ppm	11210718.D 18/11/2007 Tube 2 ppm	11280716.D 18/11/2007 Tube 3 ppm	11280720.D 18/11/2007 Tube 4 ppm	11290724.D 18/11/2007 Tube 5 ppm	11290727.D 18/11/2007 Tube 6 ppm	11300706.D 18/11/2007 Tube 7 ppm
Carbon disulfide	WIENV 31	1 (NIOSH)	0.185						
Cycloheptene	WIENV 31	Not Listed						0.034	0.016
Cyclohexane	WIENV 31	100 (Worksafe Aust)					0.108		
Cyclohexane, methyl-	WIENV 31	Not Listed	0.016	0.028					
Cyclohexane, methyl-	WIENV 31	Not Listed							
Cyclohexane, methylene-	WIENV 31	Not Listed						0.030	
Cyclohexene	WIENV 31	300 (NIOSH)	0.033	0.021	0.026		0.042	0.039	0.031
Cyclohexene, 1-methyl-	WIENV 31	Not Listed	0.024						
Cyclohexene, 1-methyl-	WIENV 31	Not Listed							0.024
Cyclohexene, 4-methyl-	WIENV 31	Not Listed	0.025						
Cyclopentene	WIENV 31	Not Listed		0.026	0.039		0.029	0.074	
Cyclopentene, 4-methyl-	WIENV 31	Not Listed				0.055		0.089	0.073
Cyclopropane, 1,1-dimethyl-	WIENV 31	Not Listed							0.059
Cyclopropane, 1,2-dimethyl-, cis-	WIENV 31	Not Listed						0.032	0.026
Cyclopropane, 1,2-dimethyl-, trans-	WIENV 31	Not Listed					0.024		
Decane	WIENV 31	Not Listed		0.022	0.035	0.067			
Didodecyl phthalate	WIENV 31	Not Listed		0.017					
Dodecanal	WIENV 31	Not Listed		0.017					
Dodecane	WIENV 31	Not Listed		0.020	0.014	0.047		0.014	
Dodecane, 2-methyl-	WIENV 31	Not Listed	0.066						
Dodecanoic acid	WIENV 31	Not Listed					0.055		
Ethanol	WIENV 31	1000 (NIOSH)			0.060				0.052
Ethene, fluoro-	WIENV 31	Not Listed						0.042	
Heneicosane	WIENV 31	Not Listed		0.059					
Heptane	WIENV 31	400 (Worksafe Aust)		0.221				0.130	0.140
Hexanal	WIENV 31	Not Listed			0.070				
Hexane	WIENV 31	20 (Worksafe Aust)	0.198		0.106	0.072	0.108	0.151	0.305
Hexane, 3-methyl-	WIENV 31	500 (Worksafe Aust)	0.024						
Limonene	WIENV 31	Not Listed			0.020	0.033			
l-Limonene	WIENV 31	Not Listed						0.044	
Methane, iodo-	WIENV 31	Not Listed		0.037					
N-Hexanal	WIENV 31	Not Listed				0.056	0.056	0.063	0.048
Nonanal	WIENV 31	Not Listed			0.023	0.033		0.020	0.012
Nonanoic acid	WIENV 31	Not Listed		0.035			0.052		
Nonyl aldehyde	WIENV 31	Not Listed					0.016		
Octadecane	WIENV 31	Not Listed		0.037					
Octane, 4-methyl-	WIENV 31	Not Listed		0.053	0.022				
Pentane	WIENV 31	120 (NIOSH)	0.224				0.336		0.413
Pentane, 2,2,4-trimethyl-	WIENV 31	Not Listed	0.151						
Phenol	WIENV 31	1 (Worksafe Aust)	0.487	0.689	**1.050**	0.397	**1.367**	0.873	0.407
Propyl acetate	WIENV 31	Not Listed							0.009
p-Xylene	WIENV 31	80 (Worksafe Aust)						0.011	
Tetradecane	WIENV 31	Not Listed		0.014		0.039			
Thiophene	WIENV 31	Not Listed	0.063						0.113
Toluene	WIENV 31	100 (NIOSH)	0.232	0.247	0.154	0.254	0.205	0.264	0.168
Tridecane	WIENV 31	Not Listed		0.018		0.055			
Undecane	WIENV 31	Not Listed		0.044		0.033			
Undecane, 4,7-dimethyl-	WIENV 31	Not Listed		0.013					

4.4 Toxic gas

Data for toxic gases are summarised in Figures 7–12. Figures 7–9 show all the data from May 2006 to July 2007 compiled into 15, 30 and 60-minute rolling averages, plotted over a 24-hour period, while Figures 10–12 display the same rolling average times plotted by day over the course of the study.

Figures 7–9 show that sulphur dioxide exposure was above the OEL between 8 am and 1 pm on several days; the STEL was also exceeded during this period. The field data sheets show that a dozer operator and six different excavator operators were working during this time. Hydrogen sulphide exposure was above the OEL at noon, although the STEL was not reached. An excavator operator was working during this time (the sulphur dioxide levels were also low).

Figures 10–12 show that sulphur dioxide exposure was above the OEL on eight days, occurring in the months of January, February, March, April and September; the STEL was reached on four of these days in January, February, April and September. The field data sheets show that excavator operators were working in hot black shale pits on two of these days. Hydrogen sulphide exposure was above the OEL on only one day in the month of April, and STEL was not reached.

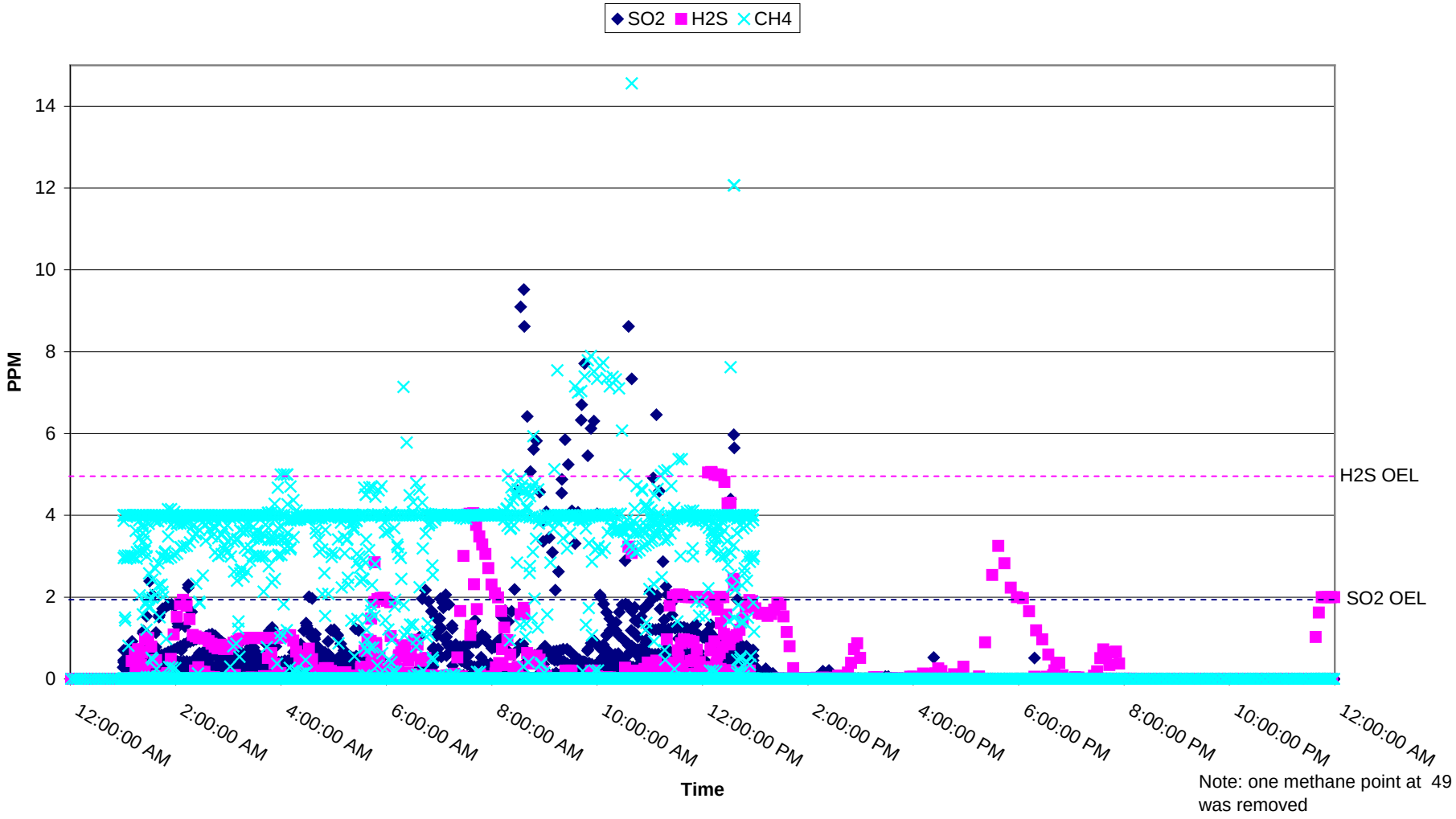

Figure 7. Toxic gas 15m rolling average over 24 hours

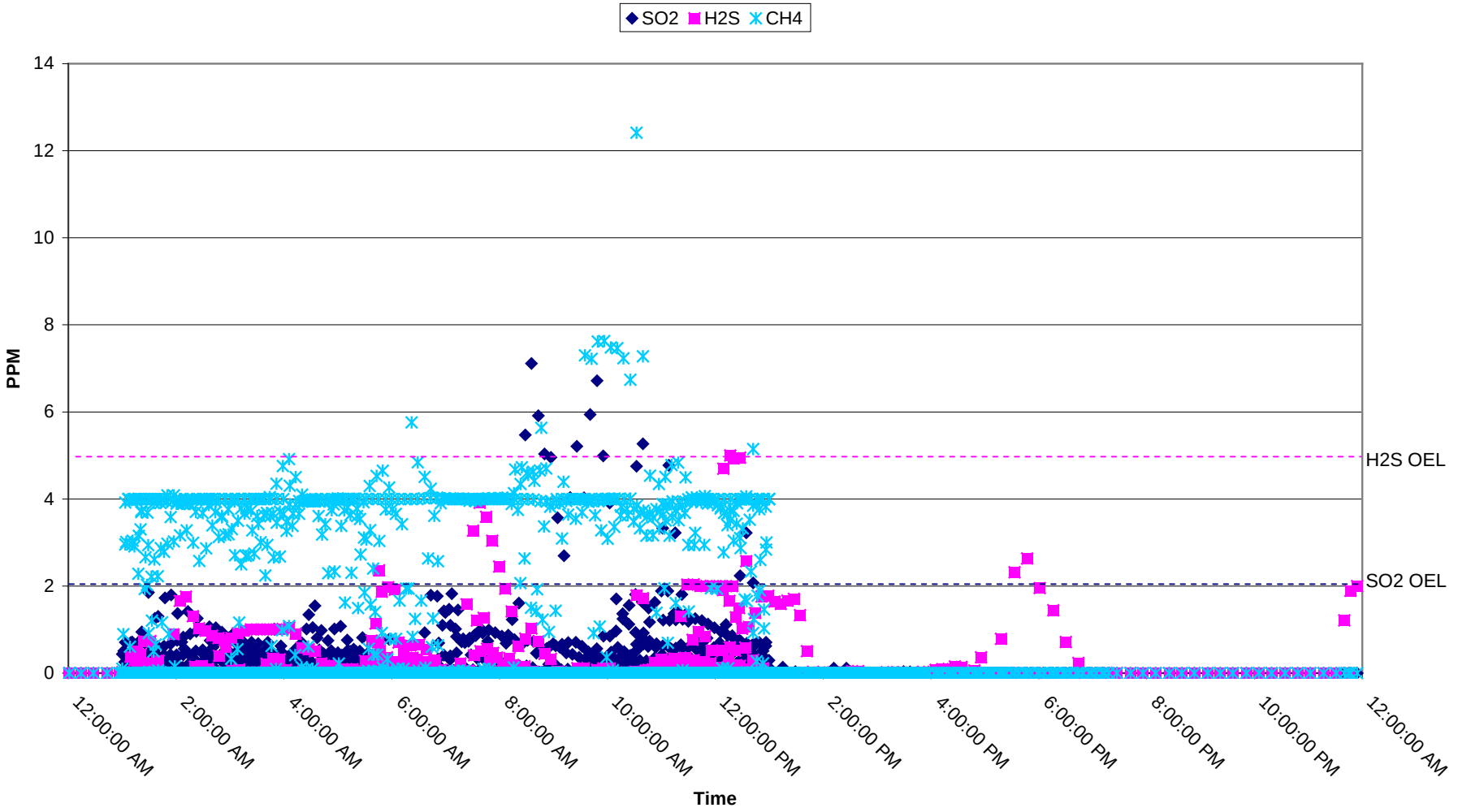

Figure 8. Toxic gas 30m rolling average over 24 hours

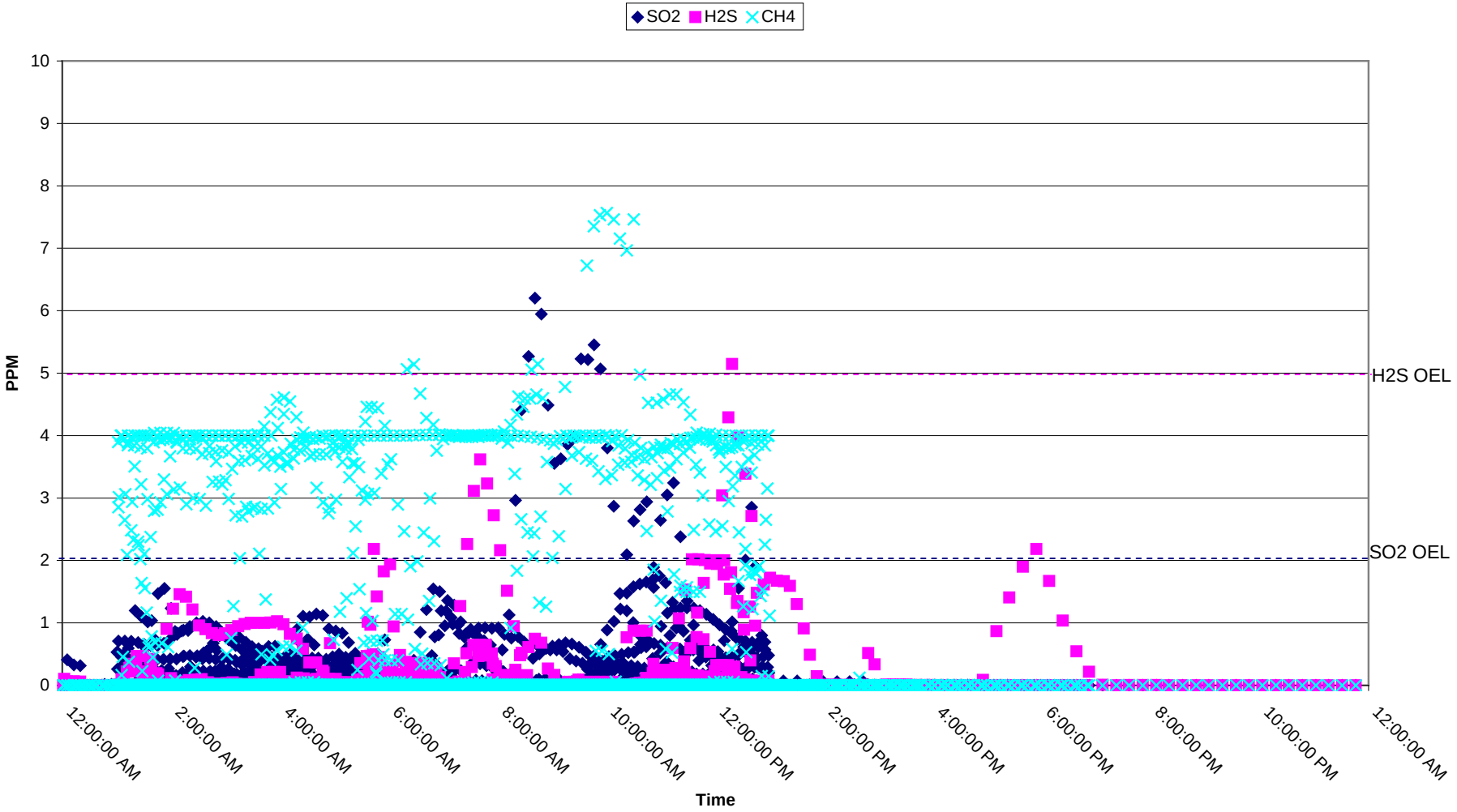

Figure 9. Toxic gas 60m rolling average over 24 hours

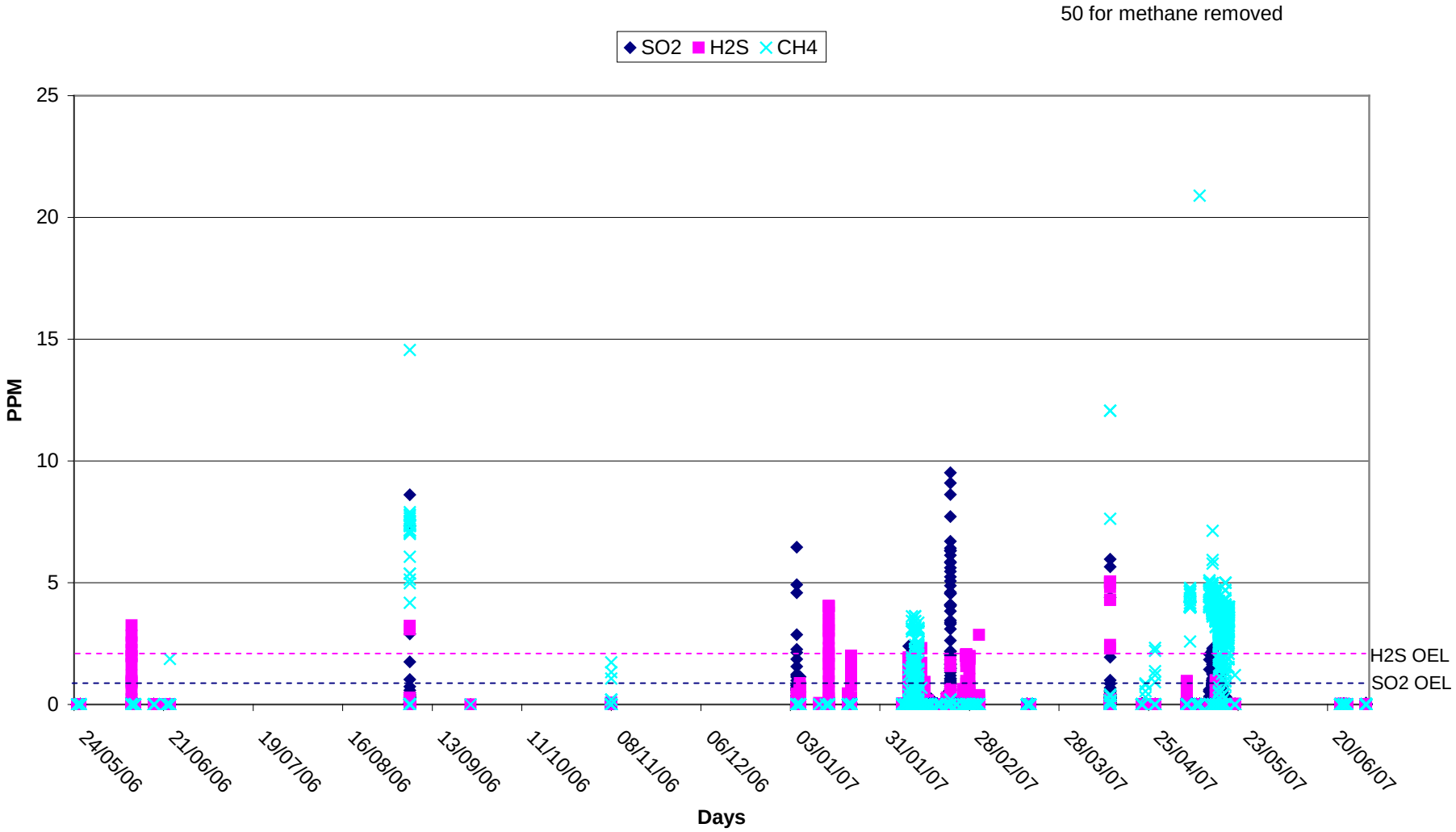

Figure 10. Toxic gas 15m rolling averages by date

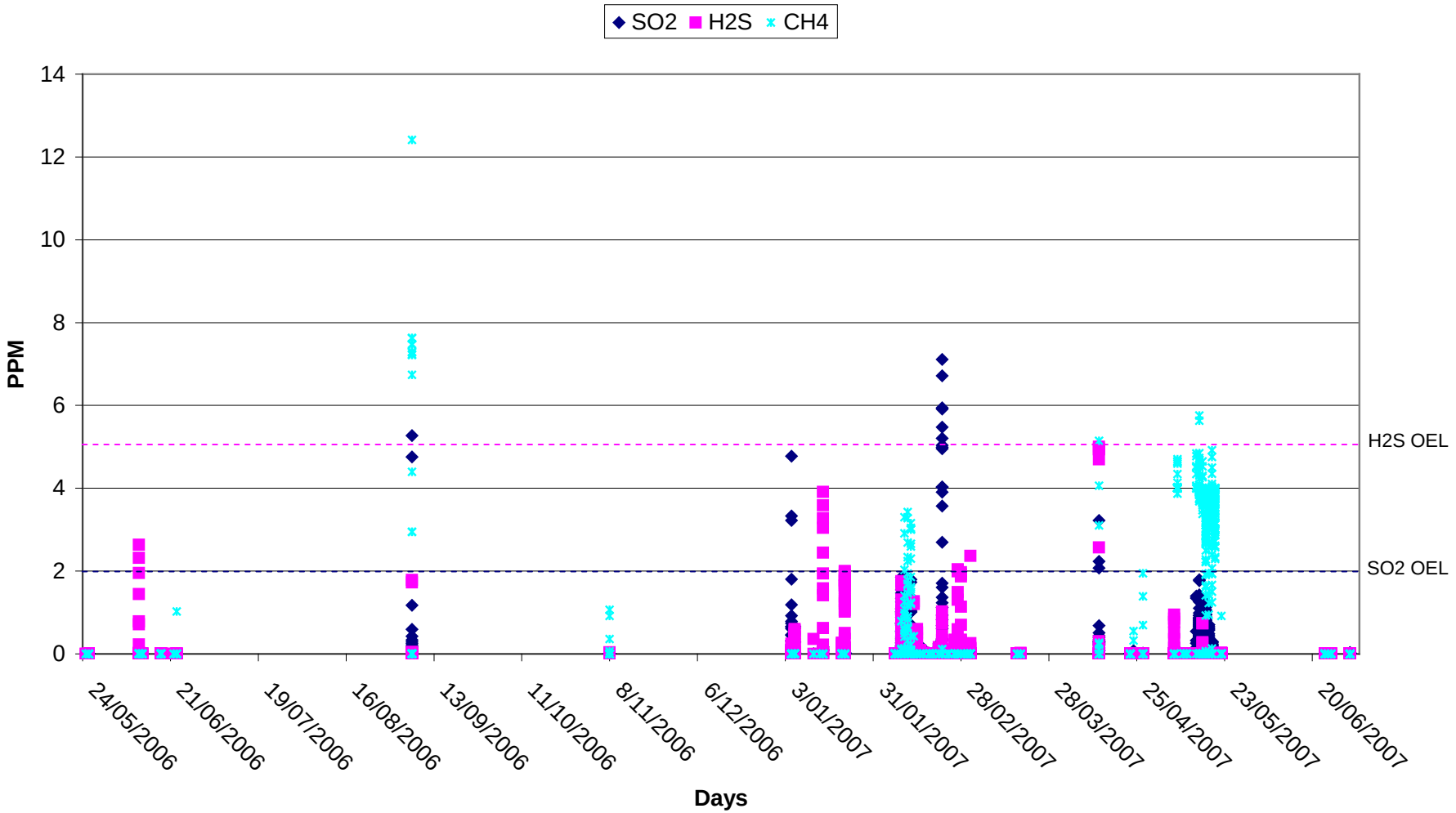

Figure 11. Toxic gas 30m rolling averages by date

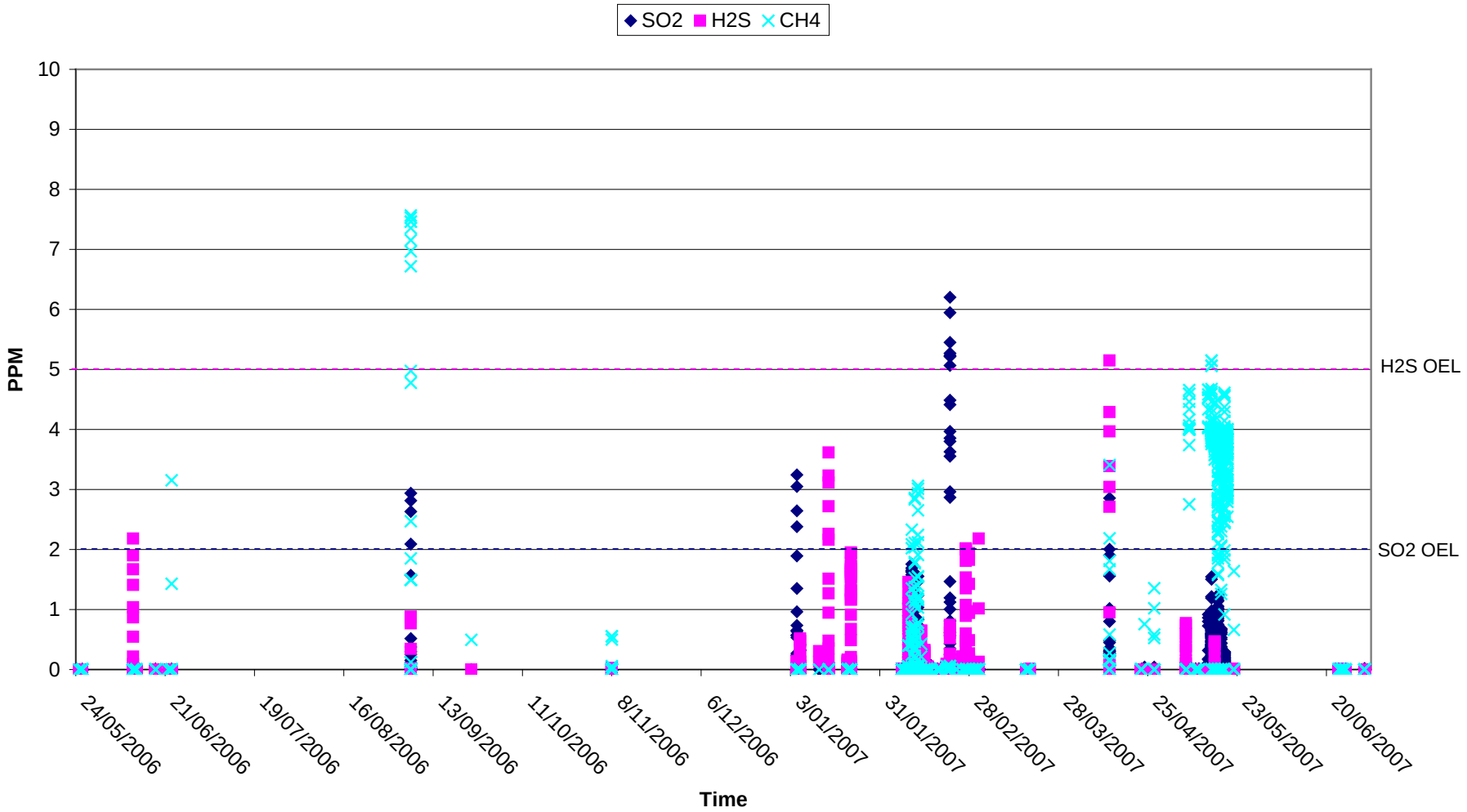

Figure 12. Toxic gas 60m rolling averages by date

5. DISCUSSION

5.1 Inhalable and respirable dust

In scientific terminology, an aerosol is defined as a system of particles suspended in a gaseous medium, usually air in the context of occupational hygiene. Aerosols may exist in the form of airborne dusts, sprays, mists, smokes and fume. In the occupational setting, all these forms may be important because they relate to a wide range of occupational diseases. Airborne dusts are of particular concern because they are well known to be associated with classical widespread occupational lung diseases such as the pneumoconioses, and systemic intoxications such as lead poisoning. However, in the modern age there is also increasing interest in other dust-related diseases, such as asthma, allergic alveolitis, and irritation as well as a whole range of non-respiratory illnesses which may occur at much lower exposure levels (WHO, 1999).

Health implications associated with particulate exposures have been subjected to increasing scrutiny by health regulators in recent times, due to their potential to cause adverse effects in local communities as well as the workforce. This scrutiny has the potential to translate into a range of operational constraints, such as legal liabilities and financial risks associated with adverse health effect litigation. Some examples of diseases linked to exposure include occupational asthma, silicosis and asbestosis (Firth, 2004).

In all Australian States, the UK, USA and many other countries, it is management's legal responsibility to identify and control workplace particulate exposure issues in order to ensure that exposure concentrations are safe and without risk to the health and safety of employees 'as far as practicable'(Occupational Safety and Health Act Western Australia, 1984).

The hazard posed by dust, mist or fume is often a function of particle size. The International Organization for Standardization (ISO, 1995) and the European Standardization Committee (CEN, 1993) have defined three criteria for biologically-relevant size-selective aerosol

sampling, primarily for studies of workers exposed to hazardous dust in industrial situations: respirable dust, thoracic dust, and total inhalable dust.

Not all of the particulate that is inhaled will lodge in the lungs. Figure 13 shows how particle size effects deposition during inhalation. Larger particles are filtered out in the nose and the tubes leading to the lungs (the bronchi and bronchioles). These particles are coughed up, spat out or swallowed. Much of the particulate that gets into the lungs is cleared out by the lung's own defence system. Macrophage cells encapsulate particulates and then dissolve them for excretion through the lymphatic system. Proteins in the lungs can also 'neutralise' some particulates. This mechanism can be overwhelmed by large amounts of dust and some dusts – crystalline silica, for instance – are lethal to macrophages (Guidotti and Koehncke, 1998). The dust particles that are trapped in the upper airways are termed 'inhalable' dust. Only the finest particles reach the lungs. These fine particles (normally too fine to see) that can enter deep into the lungs are called 'respirable' and 'thoracic' dust (Figure 14). The thoracic fraction is the mass fraction that penetrates beyond the larynx. The respirable fraction (4.2 micron median cut point) is the mass of inhaled particles penetrating to the non-ciliated, smallest, airways of the lung (the gas exchange region) (Martonen, Katz, Fults and Hickey, 1992).

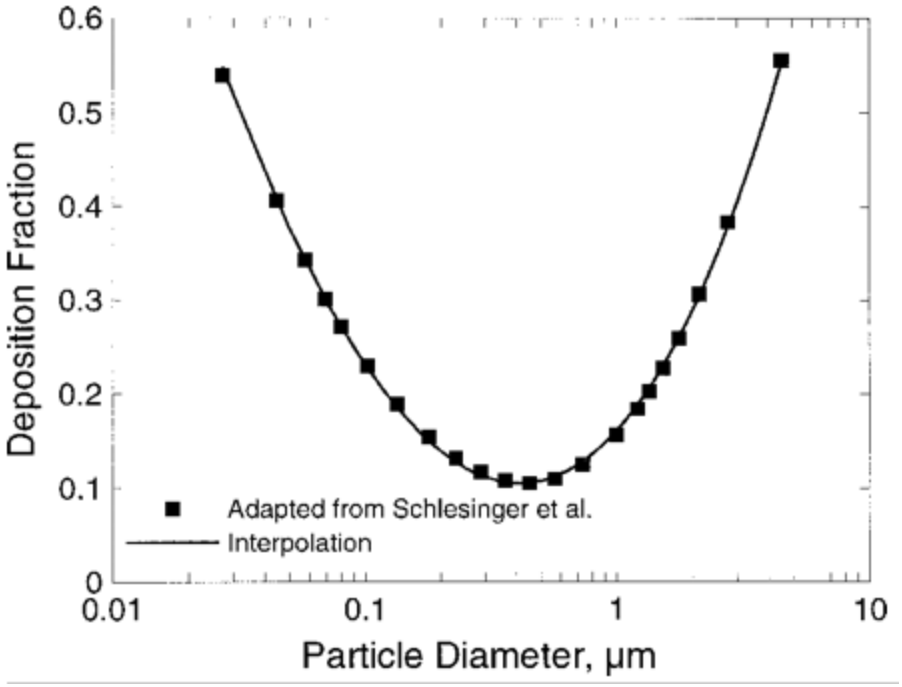

Figure 13. Interpolation of the inhaled particle deposition curve (Volckens & Leith, 2002)

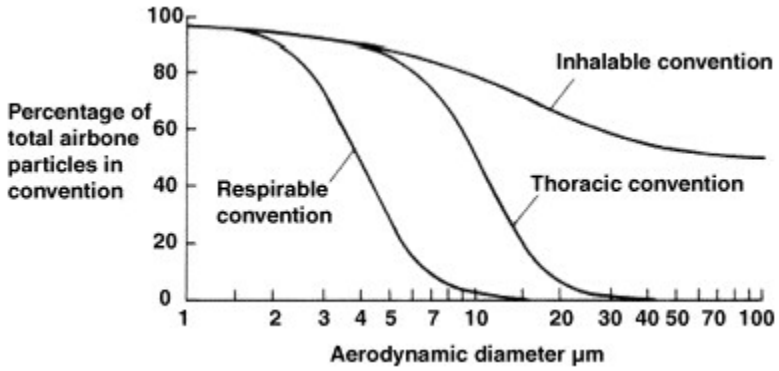

Figure 14. The inhalable, thoracic and respirable convention as percentages of total airborne particles (Petavratzi, Kingman & Lowndes, 2005)

The concentration of particulates in the workplace air should be measured at regular intervals and whenever there are changes in engineering controls, production methods or materials used. The nature and degree of exposure to particulates can be determined by monitoring airborne concentrations in both the workplace (static) and employee (personal). Static sampling provides information about contamination from fixed sources and assesses the effectiveness of controls but results cannot be compared to personal exposure or be compared

to hygiene exposure standards. This is why personal sampling is the preferred method as per AS 3640- 2004 and AS 2985-2009, although real-time monitoring may also be an alternative if sampling equipment can be validated to the Australian Standards.

5.1.1 Inhalable dust results

The 10 mg/m^3 exposure standard is the recognised upper level standard for all unclassified dusts (i.e. particles less than 10 µm and greater than 2.5 µm in size and those that have not been given a lower standard due to health effects) (NOHSC, 1995a). An exposure standard of 10 mg/m^3 was instituted in the 1970s on the basis of potential for reduced visibility, eye, nose and skin irritation and what was deemed reasonably practicable. Compliance with an exposure standard can be considered as an outcome of a system of work that is in control. Non-compliance should be used as a driver to review the effectiveness of existing controls (NSW Government, 2008).

Several factors need to be considered when interpreting the results of this study. Firstly, the personal dust samples taken were from workers operating predominantly in PBS pits, although they also worked within other pits of the mine without PBS. The heavy mobile equipment and geologists/samplers SEGs showed a lognormal distribution whilst the drill and blast SEG was not lognormal. There were 70 samples collected for inhalable dust, which all resulted in exposures below the occupational exposure limit (OEL) of 10.0 mg/m^3. The 95th percentile values and the 95% upper confidence intervals for inhalable dust were below the OEL for all SEGs. Since all of the minimum variance unbiased estimators (MVUE) were found to be below the 95% UCL, it can be assumed with 95% confidence that the exposure profile for inhalable dust for all SEGs is below the OEL. The highest MVUE recorded for all the SEGs was 2.03 mg/m^3 (drill and blast). The geometric means for all SEGs were below the OEL; however, the geometric standard deviations for inhalable dust for drill and blast was 2.59 mg/m^3 and for heavy mobile equipment operators it was 4.21 mg/m^3 indicating high variability and the possibility of heterogeneous data sets within these SEGs. This indicates

that there may in fact be more than one exposure group within each of these data sets and therefore the SEG may need to be split for future sampling.

5.1.2 Respirable dust results

A total of 80 respirable dust samples were taken over the study period. Of these samples 3 (4 %) were found to be above the Mine Safety Inspection Regulations 1995 OEL of 3 mg/m³. The exceedences for respirable dust were recorded for three drill rig operators drilling blast patterns. All the SEGs had a normal distribution for respirable dust. The 95th percentile values and the 95% upper confidence intervals for respirable dust were below the OELs for all SEGs. All of the MVUEs were below the 95% UCL, and it can be assumed with 95% confidence that the exposure profile for respirable dust for all SEGs is below the OEL. The MVUEs ranged from 0.429 mg/m^3 (geologists/samplers) to 0.764 mg/m^3 (drill and blast).

The geometric means for all SEGs were found to be lower than the OEL; however, the geometric standard deviations for respirable dust were above the OEL for drill and blast, and heavy mobile equipment operators were above the OEL, indicating high variability and heterogeneous data sets within these two SEGs. In addition, this indicates that there may be more than one exposure group within each of these two data sets and in future sampling these SEGs may need to be split.

5.2 Respirable crystalline silica results

The importance of identifying and quantifying respirable crystalline silica exposures is related to the fact that this airborne contaminant has been identified as a carcinogen (AIOH, 2009) and Rio Tinto recognises this. Pyritic black shale has been identified as having the potential to contain silica in the form of fibrous quartz found on the rims of pyrite nodules (Spodniewski, 2003). The percentage of airborne respirable crystalline silica in a measured dust sample depends on the process the bulk material is subjected to. Currently, there is no available and reliable method for determining the respirable fraction of crystalline silica in bulk materials. A suggested method involves measuring the crystalline silica content in the bulk fraction

below 10 μm. This approach would be a simple way to roughly assess the level of risk, but is generally not favoured (see BS 2006, *Method EN 15051: Workplace atmospheres. Measurement of the dustiness of bulk materials).* Requirements and reference test methods could, however, be used to extrapolate the risk. Although respirable crystalline silica exposures may not have been quantified specifically for PBS pits in the past, regular dust monitoring has occurred on an ongoing basis, stratified by SEGs.

A total of 80 respirable crystalline silica samples were collected over the study period. Of those samples 6 (12%) were found to be above the OEL of 0.1 mg/m³. These exceedences for respirable crystalline silica were evenly split between heavy mobile equipment operators (specifically excavators), and three drill rig operators drilling blast holes. Because respirable crystalline silica is an indirect carcinogen Rio Tinto recognises it is important to adhere to good control strategies so as to reduce exposures to as low as reasonably practicable (ALARP).

The geologists/samplers data are log-normally distributed whilst the drill and blast and heavy mobile equipment operators had normal distributions. The 95th percentile values were above the OEL for drill and blast and heavy mobile equipment operators and below the OEL for geologists/samplers. The 95% upper confidence intervals were below the OEL for all SEGs. Since all of the MVUEs were below the 95% UCL, it can be assumed with 95% confidence that the exposure profile for respirable crystalline silica for all SEGs is below the OEL. The MVUEs ranged from 0.02 to 0.097.

The geometric means for all SEGs were below the OEL; however, the geometric standard deviations were all above the OEL. The geometric standard deviations for drill and blast (4.210) and heavy mobile equipment operators (3.604) indicate high variability and the possibility of heterogeneous data sets within these SEGs. This confirms the finding that there may be more than one exposure group within each of these data sets and for future monitoring the SEG should be split.

These results are supported by a technical report for Hamersley Iron (Firth, 2004) which stated that respirable crystalline silica exposures exceeding 0.1 mg/m^3 only occur in mine-based jobs such as heavy mobile equipment operation. In this study, heavy mobile equipment operators were exposed to an estimated arithmetic mean respirable crystalline silica level of 0.043 mg/m³, estimated 95th percentile 0.164 mg/m³ as compared to the OEL of 0.1 mg/m³ (NOHSC, 1995a). Surveys of industries in both the USA and the UK have shown that occupational respirable crystalline silica exposures were below 0.1 mg/m^3 for 49–52% of the occupations sampled (Rio Tinto, 2010).

5.3 Toxic gases

Rumball (1984) states that the production of hydrogen sulphide gas is a likely product of pyrite oxidation. Numerous geological studies describe the presence of pyrite and sulfur in PBS (Davies, 2002; Davies and Takos, 2003; Zhang, 2003, Spodniewski, 2003). Hence exposures to sulphur dioxide and hydrogen sulphide are likely when disturbing PBS. The direct reading equipment used in this study also showed a weak correlation with time of day; it was found that sulphur dioxide exposure was most likely to occur in the morning. This could be due to increased relative humidity in the morning causing the PBS to oxidise more readily hence expelling more gas. Takos (2004) describes in a laboratory trial, analysing PBS from Tom Price, that humidity may play a catalytic role in the oxidation reaction, increasing the intensity of the reaction as humidity increases. In addition, Figures 10, 11 and 12 illustrate that sulphur dioxide emissions showed a weak trend to occur in the hottest summer months (January–April). Bureau of Meteorology (BOM) data also show that average humidity and rainfall for Tom Price is highest in this period. Yearly analysis of meteorological data show above average rainfall in 2006 from January through to April and again in 2007 from March and April. Above-average cyclonic activity occurred in 2006 and may have influenced these data. In April 1983, a wall of pyritic black shale was reported as ‘turning white’ after heavy rain; this was due to the formation of a thin coat of a white efflorescence, possibly potassium sulphate (Spodniewski, 2003).

The various gases sampled during this study include a number of chemicals for which OELs exist in Australia, due to the acute and chronic health effects associated with exposures. Data for toxic gases are summarised in Chapter 4, Figures 7–12. Figures 7–9 show all the data compiled into 15, 30 and 60 minute rolling averages, plotted over a 24-hour period. Figures 10–12 display the same rolling average times plotted by day over the course of the study.

Figures 7–9 represent toxic gas data from many days plotted over a 24-hour period. The results show that sulphur dioxide exposure was above the OEL between 8 am and 1 pm, and the STEL was also exceeded during this period. The field data sheets show that a dozer operator and six different excavator operators were working during this time. Hydrogen sulphide exposure was above the OEL at noon, although the STEL was not reached. An excavator operator was working during this time (the sulphur dioxide levels were also low).

Figures 10–12 represent toxic gas data plotted against sample time. The results show that sulphur dioxide exposure was above the OEL on 8 days occurring in the months of January, February, March, April and September, and the STEL was reached on four of these days in January, February, April and September. The field data sheets show that excavator operators were working in hot black shale pits on two of these days. Hydrogen sulphide exposure was above the OEL only on one day in the month of April and the STEL was not exceeded. The results show that there is a potential for being exposed to both sulphur dioxide and hydrogen sulphide during the summer.

Sulphur dioxide was detected with direct reading equipment among heavy mobile equipment operators (excavators) working in the black shale pits, with higher levels being recorded in the morning and in the summer.

5.4 PAH results

The laboratory trial findings show that when PBS is heated, chemicals such as benzene, naphthalene and phenol are released. These findings are supported by geological studies that

identify the presence of elevated pyrite, carbon, bitumens, PAHs and VOCs concentrations (Brocks et al., 2003).

Heating PBS to 160 °C prior to the initiation of the TD-GCMS sampling was based on the work of Davies (2002) who found that detectable sulphur dioxide emissions do not commence until a temperature of 160 °C is attained. The furnace at the laboratory had an upper safe working limit of ~ 500°degrees (personal communication, J. Menegazzo) hence data may not be entirely indicative of oxidation of PBS in the field, since Rumball (1984) reported that reaction temperatures as high as 672 ºC have been detected under field conditions. The detection of organic vapours such as benzene, toluene, heptane, cyclohexene, phenol, acetic acid, carbon disulfide and hexane indicates the possibility for adverse health impacts that may not have been previously considered. The sustained exposure to large quantities of these substances has the potential to produce chronic health effects in the form of cancers and nervous system damage (NIOSH, 1987). Benzene is classified as a known carcinogen based on occupational studies in adults that demonstrated increased incidence of several types of leukemia in exposed adults (US EPA 2006). Benzene has also been shown to be genotoxic (cause damage to DNA) in experimental animal studies with the primary targets of benzene exposure in humans are the hematopoietic (blood cell-forming) system and the immune system (US EPA, 2006).

Carras et al. (2005) undertook a study in 1999 at a coal mine in the Hunter valley region of New South Wales, where bulldozer operators' exposure to PAHs working in the vicinity of spontaneous combustion was measured. They found that the PAHs measured within the cabin were below the values expressed in the occupational health and safety guidelines. In the outside air and in close proximity to spontaneous combustion fires, the PAH levels may, however, have been higher, thus exposure to VOCs/PAHs is likely to be greater for SEGs in direct contact with burning PBS (e.g. field-based personnel such as drill and blast teams and geologists/samplers) compared to heavy mobile equipment operators who have supplied filtrated air inside their machinery. Although personal sampling of VOCs/PAHs did not occur

in this study the detection of organic vapours in the laboratory trial indicates the need for personal exposure to be further investigated and quantified.

5.4.1 Control of particulate exposures, RCS and PAH

The most effective means of restricting particulate and vapour health effects is by eliminating or controlling exposure. The implementation of 'as low as reasonably practicable' (ALARP) principles has led to improvements in engineering controls such as the design of heavy mobile equipment in-cab vehicle ventilation filtration systems including the routine planned maintenance of these systems, which assist in promoting low exposure levels in vehicles. Area supervisors and employees should develop and implement work practices, through standard work procedures, which limit the production of dust. However, if excessive exposure occurs then the removal of affected workers from exposure needs to be considered. PPE such as respiratory protection should be a last resort in addition to regular sampling.

5.4.1.1 Elimination & substitution controls

Recent innovative technology has resulted in automated mobile equipment such as haul trucks and drill rigs which can be operated remotely. The advantage of this control is that it removes the operator from the exposure in addition to noise and vibration hazards. It does introduce some safety impacts on the mine site regarding light vehicle interactions with unmanned mobile plant. This control has been implemented at some Rio Tinto sites in the Pilbara such as Yandicoogina, West Angeles, Hope Downs 4 and Brockman 4 but not at Tom Price.

5.4.1.2 Engineering controls

Engineering controls should be used as the primary method to eliminate potential hazardous exposures in the workplace. Achieving and maintaining reduced concentrations of hazardous exposures in the workplace depend on the implementation of engineering control measures such as properly constructed and maintained closed-system operations and exhaust ventilation

with appropriate safety designs. Covering hot black shale pit faces with inert waste material will inhibit oxidation and limit exposure to PAH and toxic gas.

If feasible, workers should be isolated from direct contact with the work environment by the use of automated equipment operated from a closed control booth or room. The control room should be maintained at a greater air pressure than that surrounding the process equipment so that air flows out of the room rather than into it. This control can be implemented in relation to mobile equipment and drill rigs. Ventilation equipment should be checked at intervals that will ensure adequate performance. System effectiveness should also be checked when there are any changes in production, process, or control that might result in increased exposure to airborne contaminants. This type of control will not protect workers who must perform process checks, adjustments, maintenance, and related operations outside of the room/cabin of the equipment. Thus special precautions are often necessary to prevent or limit worker exposure in these situations, and they frequently involve the use of personal protective equipment.

5.4.1.3 Containment /suppression

The use of water as a mist/spray to reduce dust is widely practised on haul roads, at dump points and on drills. This method of control is regularly used at Tom Price. Products such as "Rainstorm" can facilitate the efficacy of water as a dust suppressant. Argyle Diamonds has developed the innovative use of mixing locally available molasses with water as a relatively inexpensive road-dust suppressant. The use of water mist, sometimes with detergent, to suppress dust on drills is not the most effective means of dust control. Local exhaust ventilation should also be considered for drills.

5.4.1.4 Ventilation

Work areas with activities likely to generate harmful particulate-in-air concentrations should be enclosed if practicable and well ventilated (exhaust ventilation). An alternative to source containment and ventilation is worker enclosure (e.g. provide cabs on vehicles/drill rigs with filtered air-conditioned air supply). Where ventilation systems are installed, these systems

should be maintained in good working order and should be operated in the correct manner to provide optimum protection from dust exposure. Mobile equipment at Tom Price undergoes regular maintenance and inspection of in-cab filtration systems.

5.4.1.5 Administrative controls

A reduction of level and duration of exposure of employees to particulates may be achieved by periodic rotation of employees both through and in areas with potentially harmful particulate exposures. However, this should be restricted to trained employees. There should also be a limit on the amount of overtime an individual can do to ensure they are not over exposed.

Regular inspection and maintenance routines for engineering controls and periodic formal review of the practicality of these controls are essential administrative controls.

5.4.1.6 Housekeeping

The maintenance of a high standard of housekeeping will minimise exposure to dusts and fumes. Methods of wet cleaning inside mobile equipment (e.g. hosing, mopping or sweeping with wet sawdust) should be used where practicable. Where suitable, vacuum cleaners fitted with HEPA final filters can be considered. Mobile equipment at Tom Price regularly undergoes inspections and cleaning of filters and inside the operators' cabins.

5.4.1.7 Personal hygiene

Good personal hygiene can assist in reducing particulate inhalation, although individuals can be very clean and be placed in a dusty environment exposing them to particulates. Employees should be required to:

- shave daily if they need to wear respiratory protection;
- wear clean clothes/overalls each day;
- wash hands and face before eating or smoking; and
- not eat or smoke in areas or jobs with potentially harmful particulate exposures.

5.4.1.8 Personal protection

All personal protective equipment (PPE) should comply with Australian safety standards. PPE should be used as a last resort, where other control measures have been unsuccessful or not reasonable to implement. Respirators should be selected with regard to the appropriate dust exposure level and particulate size, their comfort and their compatibility with other safety equipment. Training specific to the PPE as well as fit testing is required for identifying the correct PPE to use, to ensure the correct fit and to maintain the PPE in good working order.

5.4.1.9 Worker education

Employers are to establish a worker education program for all workers exposed to hazardous substances. Training should be provided at the time of initial assignment and revalidated at regular intervals. This training should be designed to inform the worker about the hazards to which they are exposed, the potential health risks from exposure to these materials, the proper use of personal protective equipment and clothing and other methods for control, and proper work practice procedures.

5.4.1.10 Medical surveillance

Employers should establish a medical surveillance program to evaluate both the acute and chronic effects of exposure to PAH and RCS. The physician should be given information concerning the adverse effects of exposure to organic solvents and an estimate of the worker's potential exposure to them. This information should include any available results from workplace sampling and a description of any protective devices or equipment the worker may be required to use. A medical and work history should be taken initially and updated periodically. Workers who are currently exposed or who may be exposed to PAH should have pre-placement and periodic evaluations focusing on their histories of previous exposure to PAH and other agents, particularly those associated with neurotoxic effects. The examining physician should direct particular attention to the nervous, respiratory, reproductive, and

cardiovascular systems, and to the skin, eyes, liver, blood, kidneys, and gastrointestinal tract, as these are the most likely targets for the adverse effects of PAH.

5.5 Control measures for toxic gases

The most effective means of restricting adverse toxic gas exposures is by regular inspection and sampling of pits that have previously had toxic gas detected or identifying pits through geological survey whereby pyritic black shale may be encountered. Pit design, and immediately covering pyritic black shale with inert material when transporting as waste to prevent chemical reactions that produce toxic gas, are necessary. Area supervisors and employees should abide by work practices which limit the likelihood of producing toxic gas. Areas where toxic gas is likely to be generated should be signposted and access restricted. Education of workers as to the hazards associated with working with toxic gas is essential, as are evacuation procedures and an emergency response plan in the event of encountered dangerous concentrations.

6. CONCLUSIONS & RECOMMENDATIONS

Operations involved in surface mining often lead to increased occupational exposure to dust, and potentially fume and gases associated with the processing of raw materials. This study identified organic vapours such as benzene, toluene, heptane, cyclohexene, phenol, acetic acid, carbon disulfide and hexane in PBS pits.

Static vapour samples of smoking PBS waste were obtained in the pit. Furthermore PBS samples were heated under controlled conditions within a laboratory environment and desorbed vapour was analysed through GCMS. These organic vapours have not been previously recognised in iron ore mining in the Pilbara region. Although the sampling conducted was not personal and hence can't be compared to exposure standards, the identification of these organic vapours needs to be further quantified to ensure exposure is as low as reasonably practicable. Exposure to organic vapours indicates the possibility for adverse health impacts not previously considered. Sustained exposure to large quantities of these substances has the potential to produce chronic health effects in the form of cancers and nervous system damage, therefore the implementation of specific work procedures in the PBS pits are required to protect the health of employees.

Specific recommendations for PBS work are provided below.

1. Implement a personal exposure monitoring program for workers operating in PBS pits – specifically, investigating PAH exposure. As appoint of departure, at least six personal samples should be collected for each SEG working in PBS pits using active sampling methods.

2. Mobile equipment operators, drill rig operators and in-pit maintenance staff working in PBS pits should be provided with respiratory protection that provides protection against VOCs and thermally generated particulate (e.g. fume) as well as toxic gases.

3. Routine inspection and replacement of gas and vapour filters based on the preventative maintenance order system within the mobile plant/drill rigs is required to provide adequate protection. These should be set up weekly until determined that the frequency can be extended.

4. Carbon monoxide (CO), although not measured, is likely to be present as a by-product of PBS oxidation. Carbon monoxide is produced from the incomplete oxidation of carbon-containing compounds and forms when there is not enough oxygen to produce carbon dioxide. Carbon monoxide exposure should be quantified in future sampling/studies by use of electrochemical detection.

5. PBS has been identified as having the potential to contain relatively high levels of respirable crystalline silica. Heavy mobile equipment operators and drill and blast personnel should also wear a minimum of P2 respiratory protection due to exposures detected above the OEL.

6. Increased toxic gas exposures are associated with high humidity and rainfall events when increased oxidation of pyrite occurs due to contact with water. These events tend to be more prevalent during the summer months when there is increased cyclonic activity. Entry to PBS areas should be avoided during periods of high humidity and directly after rainfall events to ensure toxic gas exposure is as low as reasonably practicable (ALARP).

7. The study did not consider the additive or synergistic effects of dust/fume/gas exposure in combination; further study into the combined effects of these chemicals should be undertaken and exposure verified by monitoring.

This research has identified potential exposures not previously considered when mining iron ore in Western Australia specific to PBS. These data will assist Rio Tinto and other mining companies who operate in the Pilbara and encounter PBS in their operations, in the

identification of potential occupational exposures not previously considered. Inclusion of these agents in risks assessments and monitoring programs will further enhance the health and safety of employees working in PBS environments and will assist in the development of a variety of controls that will protect workers.

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