A RADIATION MONITORING PROGRAM IN A SOUTH AFRICAN GOLD MINE

A. VAN SCHALKWYK Hons. B.Sc

Mini-dissertation submitted in partial fulfilment of the requirements for the degree Magister Scientae in Physiology at the North-West University

Promotor: Prof. F.C Eloff

November 2005

Potchefstroom Campus

ABSTRACT

Legislation requires the regular monitoring of all employees exposed to radiation in their work environment. The monitoring of α-radiation, which is emitted by radon gas, was the primary concern of this study. Radon comes from the natural decay of uranium, which is a heavy metal found in all rock and soil.

The main objective of this study was to establish a controlled monitoring program through which results could be obtained, captured and studied. The mine monitored in this study had high radiation levels, requiring urgent and effective strategies to reduce employee exposure.

The study included five monitoring cycles which yielded comprehensive results. Insight gained from these results made it possible to identify strategies to reduce the high prevalence of exposure in the mine.

Results were compiled in a database and then used to predict each employee'se annual exposure. The personal history of each employee was also documented in the database. Results revealed that proper administrative and ventilation controls were effective in reducing exposure to radiation in the mining environment. Thus, the hypothesis for this study was proven to be true.

OPSOMMING

Wetgewing vereis dat werknemers wat blootgestel word aan radiasie op 'n gereelde basis gemonitor word. Dit is sodat blootstelling aan radiasie, in hierdie geval α-radiasie, afkomstig vanaf radon gas, gemonitor kan word. Radon gas is 'n natuurlike afbraak-produk van uranium, 'n swaarmetaal wat in alle klip en grond gevind word. Die doel van hierdie studie was om 'n beheerde moniteringsprogram te ontwikkel, waarmee resultate bekom en bestudeer kon word. Die myn wat gemonitor is, was 'n skag met hoë blootstelling aan radiasie. Daar was 'n dringende behoefte aan effektiewe beheermaatreëls om blootstelling van werkers te beperk en te minimaliseer.

Vyf monitering-siklusse is in die studie geïnkorporeer, wat dit moontlik gemaak het om 'n omvattende en akkurate weerspiëeling van werknemers se blootstelling te kry. Die nodige aksieplanne kon bepaal en in plek gestel word na aanleiding van die resultate wat verkry is.

Die resultate wat verkry is, is saamgevat in 'n databasis van waar 'n voorspelling van die dosis waaraan 'n werknemer waarskynlik blootgestel gaan word, bepaal kon word. Die die persoonlike geskiedenis van elke werknemer wat gemonitor is, is ook hierin saamgevat. Die resultate toon dat die maatreëls wat in plek gestel is effektief was om blootstelling te verminder deur behoorlike administrasie en ventilasie beheer-maatreëls. Die hipotese vir hierdie studie is as korrek bewys.

ACKNOWLEDGEMENTS

I would like to thank the following persons for their contributions to this project. Without their help, the project would not have been possible.

- Firstly I want to thank God for the opportunities He gave me, as well as the grace and favour He has placed on my life. I would not be here if it was not for Him.
- Prof FC Eloff, for his guidance and inputs.
- The Management and the SHE Department at Anglogold Ashanti, Vaal River – this study would not have succeeded without your hard work. Thank you for your inputs and eager support.
- Thanks to Mr D Hoffman, Mr T Webb, Mr J van Sittert, Mr E Peterson and Mr G Erasmus for their help with the database and the RGM procedures.
- Special thanks to Mr L du Toit, whose guidance and help is invaluable. Thank you for your patience, example and integrity.
- Mr M le Roux for his time and effort. You made the difference in this project.
- Mrs G Gleason for doing the grammatical checks.
- Finally, all my friends and family. I love you with all my heart – thank you so much for your support and love.

TABLE OF CONTENTS

LIST OF SYMBOLS AND ABBREVIATIONS

SYMBOLS	DESCRIPTION
λ	Wavelength (μm)
γ	Gamma rays
β	Beta rays
α	Alpha rays
RGM	Radon gas monitor
mSv	milli Sievert
mSv/a	milli Sievert per annum
eV	Electron Volts
DU	Depleted uranium

AUTHORS' CONTRIBUTIONS

The contribution of each of the role-players in this study is given in the following table:

Table 1.1: Authors' contribution list

Name	Role in study
Ms A v Schalkwyk	Responsible for literature searches, statistical analysis, collection of data, design and planning of manuscript, interpretation of results and writing of all manuscript.
Prof. F.C. Eloff	Promoter. Supervised the writing of the manuscript, initial planning and design of manuscript.
D.J. Hoffman	Supervised the writing of the manuscript and collection of data.
L du Toit	Supervised the writing of the manuscript and collection of data.

The following is a statement from the co-authors confirming their individual role in each study and giving their permission that the data may form part of this thesis.

I declare that I have approved the above-mentioned manuscript, that my role in the study, as indicated above, is representative of my actual contribution and that I hereby give my consent that they may be published as part of the M.Sc. thesis of Adelle van Schalkwyk.

_____ _____

Prof F.C. Eloff D.J. Hoffman

_____

L. du Toit

LIST OF FIGURES

LIST OF TABLES

CHAPTER 1: INTRODUCTION

1.1 HYPOTHESIS

Radiation exposure underground can be controlled and reduced to well below the allowable dose of 50 mSv per annum (50 mSv/a), if proper administrative and ventilation controls are put in place.

1.2 OBJECTIVES

The following objectives and outcomes were set for this study:

- to write a relevant and reliable radiation monitoring program for the identified mine in order to reduce exposure to below 50 mSv/a.
- to develop a database that contains:
    - the results of personal monitoring for statistical analysis purposes;
    - the results of area sampling via personal monitoring for engineering control purposes;
    - personal history of employee exposures;
- to maintain a continuous and progressive risk assessment of radiation exposure, after the study is completed. And to further reduce exposure below 20 mSv over five consecutive years.

1.3 SCOPE

- Data would be obtained from sampling employees at Anglogold Ashanti, Vaal River;
- Previous data would be gathered and used as reference for future monitoring;
- Data from five monitoring cycles would be used to derive adequate information for statistical purposes;

- The necessary actions would be taken to reduce and control radiation exposure on the mine;
- Monitoring will be done on a regular basis during and after the study, to enable a continuous hazard assessment and evaluation in terms of irradiation.

CHAPTER 2: LITERATURE SURVEY

2.1 INTRODUCTION

2.1.1 OVERVIEW

Radiation occurs naturally in the environment and has always been present on earth. Radiation is a form of energy that travels through space, and exposure to it is referred to as background radiation. Background radiation can be explained as the radiation one is constantly exposed to as a result of natural sources (Hall, 2005). It is similar to background noise, where the ongoing noise in one's immediate surroundings are taken into account, but does not have a direct influence on one's activities.

Exposure to radiation can and must be controlled, since under and over exposure, depending on the type of radiation, can be hazardous to health. The most common form of thermal radiation known to man is sunshine. Without it, no life on earth would be sustainable. Too much sunshine, on the other hand, has adverse health effects on all life forms on earth. Sunshine consists of radiation in a range of wavelengths starting at $10^{-1}\mu m$, which is in the ultraviolet region, up to $10^{2}\mu m$, also known as infrared, as indicated on figure 2.1. Of these, ultraviolet radiation is the most hazardous. Sources of chronic low-dose radiation have become almost omnipresent in our environment as a result of nuclear tests, radiation accidents and diagnostic-, therapeutic- and occupational exposures (Hall, 2005; Kovalchuk *et al.*, 2003; Strauss & Hollander, 1989; Incropera & DeWitt, 1981).

Exposure to radiation is a great concern in some goldmines, due to the constant exposure the employees to radiation, as well as the lack of knowledge concerning the influence of irradiation on the long-term health of the employees. This inefficiency will have major financial implications on the mines, as compensation will have to be paid should employees develop pathology as a result of exposure during their employment.

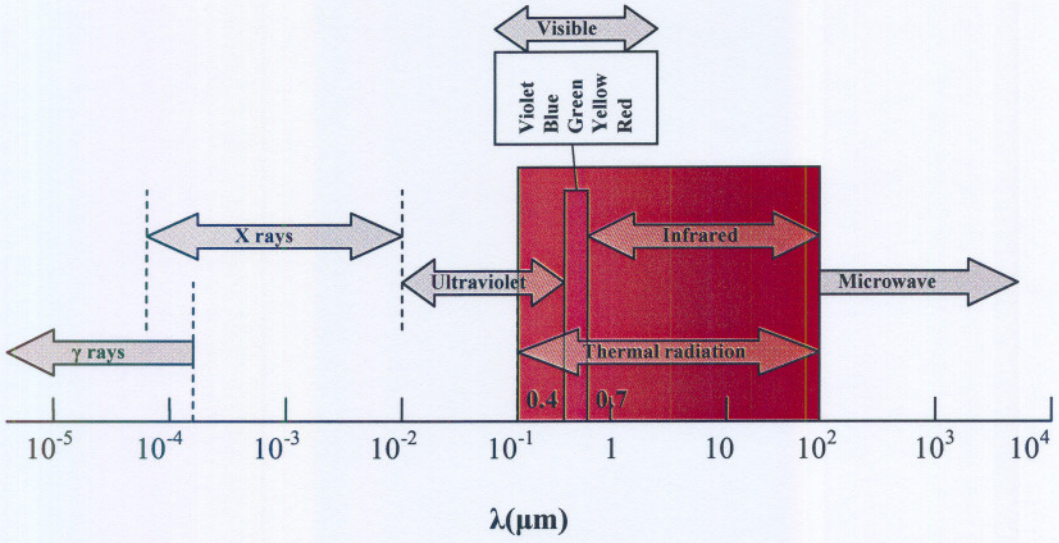

Figure 2.1: The radiation spectrum (adapted from Incropera & DeWitt, 1981).

Figure 2.1 shows the complete radiation spectrum with thermal radiation as indicated. Visible light can also be found within the thermal radiation region, starting at a wavelength of 0.4 μm and ending at 0.7 μm. It consists of violet-, blue-, green-, yellow- and red waves.

2.1.2 Radiation and mining

Mining is an ancient, multi-disciplinary industry, long recognised as being arduous and liable to injury and disease. The industry employs a labour force of several hundred thousand miners, both in South Africa and in the world (Donoghue, 2004; Hnizdo *et al.*, 1997). Working with natural raw materials will always increase exposure to radiation. All rock and soil contains uranium and thorium, which are both radioactive (Uranium isotopes, Ur-238 & Ur-237, and thorium isotope, Th-232) (Gulson *et al.*, 2005; Yamada, 2003).

Most of these radio-nuclides have extremely long half lives, as can be seen in table 2.2. These half lives have a great impact on determining exposure time, since the half-life of

the nuclide influences the risk to become exposed and develop pathology. Exposure to radiation in the mining industry varies greatly, depending for instance, on the uranium concentration in the rock, as well as the presence of radon. Radon (in this case mainly radon-222) is a decay product of uranium, radon gas concentrations will therefore be higher in soil with high levels of uranium. Radon-222 permeates into enclosed areas through the walls and floors (Hall, 2005). The following table is a summary of radiation dose levels and their effects:

Table 2.1 Summary of exposed dosage and effects (adapted from Hall, 2005).

Dose	Effect
0 – 50 mSv	Typical artificial exposure ● *0.05 mSv*: Design target for perimeter fences at nuclear electric generating stations. ± *0.6 mSv*: Most medical exposure doses. ● *3-5 mSv*: Mining background exposure (North-America, Australia, Canada) ± *20 mSv*: Lowest dose that may cause cancer, and highest allowable dose over 5 years consecutively. ± *50 mSv*: Highest allowable annual dose.
›100 mSv	Possibility of cancer.
›1 Sv	Short term dose: Threshold for immediate radiation sickness.
›10 Sv	Short term and whole body dose: Immediate illness and subsequent death.

The above table briefly summarises the doses of radiation used in certain instances, as well as some presumed pathology as a result of exposure. The maximum allowable annual dose is 50 mSv, which is referred to as the 'threshold' or OEL (occupational exposure limit) in this study. This study does not refer to the 20 mSv over

five consecutive years, as the continuous risk assessment that carries on after the study will strive to reach that target.

2.1.3 Protection against radiation

Protection against radiation aims to lower or limit the possible long-term effects of radiation. There are four ways of protecting people against radiation. The first method entails shielding through various barriers. Lead barriers are most commonly used for this purpose. This control measure is typically applied at nuclear installations and radio-therapy institutions. Limiting the time of exposure to sources of radiation is the second method (Hall, 2005). In the mining industry workers are continually exposed to rock with variable uranium concentrations. Many mine workers work shifts that are longer than eight hours a day due to production pressure, bonuses for overtime worked and a lack of self-discipline. Limiting shifts to eight hours a day will have a significant impact on the reduction of exposure levels.

The third method entails the distance between the source and the exposed group. This distance can be enlarged to minimise the effect of radiation when the half-life of radon is taken into account. Unfortunately this solution is not viable in the mining industry, because mine work demands immediate contact with the rock face. Finally, the source can be contained (Hall, 2005). Containing the source of emission in the mining industry is an impossible task, since the labour required involves direct contact with rock. The source can nevertheless be diluted by upgrading ventilation in the areas. Ventilation plays an important role in pro-actively reducing exposure to radiation in the mine. It prevents build-up of radon gas and contaminants in areas that are no longer worked in, as well as areas that are actively mined in. In return, the lower levels of radon gas and contaminants reduce the formation of agglomerated particles that cause internal exposure, as described in section 2.3.

Other factors that might help to improve the situation are engineering and administrative control measures, personal hygiene, personal protective equipment, job rotation etc. Wearing personal protective equipment is impractical on the mine, because the workforce already wears a number of compulsory protective equipment (including a hard hat, overalls, gumboots, ear protection, eye protection and sometimes respirators). Adding the weight of yet another set of protective equipment, especially the kind of equipment used for radiation exposure, cannot be justified. This type of equipment is heavy, expensive and does not allow free movement. The physical workload upon these employees, as well as the environmental conditions underground, also rules out any possibility of this.

2.2 RADIATION AND THE ENVIRONMENT

There are two kinds of radiation: Non-ionising radiation and ionising radiation. Non-ionising radiation includes ultraviolet, visible light, infrared, microwave and radio waves. Ionising radiation consists of radiation that has a higher energy range, and therefore a shorter wavelength than ultraviolet rays. It is used in the medical profession in the form of x-rays and chemotherapy. It is a natural form of energy that originates in low doses from space, as well as small emissions from the earth taking the form of radon, a natural gas which seeps from the earth's crust, in the earth's atmosphere. This kind of radiation can cause damage to matter, and in particular to living tissue, hence the need to control excessive exposure to it (Hall, 2005; ATSDR, 1999; Strauss & Hollander, 1989).

2.2.1 Non-ionising radiation

Non-ionising radiation does not play an active role in the scope of the study, for this reason it will not be discussed in detail and will only be summarised briefly. Non-ionising radiation includes all forms of electromagnetic radiation that has enough energy to heat up biological material, predominantly due to the production of electrically charged particles (ions). This process is also known as ionisation and includes ultrasound and infrasound.

Non-ionising radiation of sound can be categorized as follows (Deonarine, 2005):

ELF *Extremely low frequencies*
VF *Voice frequency*
VLF *Very low frequency*
LF *Low frequency*
MF *Medium frequency*
HF *High frequency*
UHF *Ultra high frequency*
EHF *Extra high frequency*

Infrared rays are generally known as radiation heat, and have thermal effects on the environment. All processes involving heat generation are sources of infrared rays, for example the sun, household appliances, telecommunication, airflow control and electrical circuits (Deonarine, 2005).

2.2.2 Ionising radiation

Ionising radiation can originate from both natural sources and artificial sources, such as accelerators and ortho-voltage machines. In addition, it contributes to the electromagnetic radiation spectrum, in the form of x-rays and gamma-rays (with characteristic short wavelength and high penetration depth capacity). These rays, like non-ionising radiation, cause ionisation in matter and is harmful to both the human body and the environment. Electromagnetic waves have certain characteristics that will differ at different frequencies and wavelengths. These wave characteristics will again determine the kind of reactions the wave has with the matter, for example, penetration depth. The energy an electromagnetic wave carries are called a quantum or a photon (Mouton, 2005).

All matter is composed of atoms. Atoms that are chemically identical, but differ in mass, are called isotopes. Where most atoms are stable, the opposite is true of isotopes. These unstable isotopes are called radio nuclides. Radio nuclides' nuclei will spontaneously

rearrange into stable nuclei, emitting excess energy during the process. The emission of alpha- (α), beta- (β) and gamma- (γ) rays are connected with nuclear reactions. The stable nucleus is called the decay product, and the energy emitted during the reaction can contribute to follow-up ionisation processes (Mouton, 2005).

Radioactive decay occurs when a given amount of radioactive material decreases with time as the nuclei decays. The element, uranium, has no stable isotopes. Note that alpha-, beta-, gamma- and x-radiation do not cause the body to become radioactive; most materials are radio-active up to a certain amount in their natural state already (Hall, 2005). The focus of this study is mainly on alpha rays, but beta- and gamma rays also play an important role in ionising radiation. Alpha rays enter the body and cause internal exposure, leading to all kinds of pathology while gamma and beta rays are effectively diluted via ventilation, before it becomes an exposure hazard.

Alpha rays:

α-disintegration occurs mainly in heavy nuclei and positively charged particles, where a helium nucleus, (2 protons and 2 neutrons) are ejected. The energy ranges from 4 to 5 x 10^6 eV. Intense ionisation is caused because of the large positive charge. The penetration depth ranges between 2 and 10 cm in air (Hall, 2005). α-rays will usually not penetrate the epidermis, as it can be stopped by a sheet of paper (Wymer, 2001). It can however be inhaled through the air in a mining environment (underground), because of the presence of dust particles containing the radio-active atoms. This process is described in section 2.3.

Beta rays:

These rays originate from the emission of negatively charged high-speed electrons from the nucleus. The energy range is between zero and the maximum value of the parent nucleus. The ionisation is reasonably high, but lower than for alpha-rays. Its penetrating depth is more than alpha rays; up to 3 m in air, and 1 – 2 cm in water (Hall, 2005). It can penetrate human tissue up to 5 mm. Beta rays can be

completely absorbed by thin metal (1 – 3 mm) or Perspex (10 mm) (Wymer, 2001).

Gamma rays:

Excess energy from the decaying nucleus is emitted with this type of radiation. It is electromagnetic radiation of very short wavelength, and the energy levels can increase up to 3 x 10^6 eV (Hall, 2005). Again there is a decrease in the amount of ionisation from beta-rays to gamma-rays. Depending on the energy, gamma rays have extreme penetration depths; accordingly thick concrete or a heavy element is needed to absorb the rays. Gamma rays can pass completely through the human body (Wymer, 2001). In mining, the gamma-exposure is mainly an external hazard because of the rock face, stockpiles, localized concentrations and so forth, and is not a risk of major concern in the mining industry.

2.2.3 Uranium

Uranium is a naturally occurring radioactive element that is classified as a heavy metal, with atomic number 92 and an atomic mass of 238.0289 g/mol, and it primarily radiates alpha particles (ATSDR, 1999). It is found in small amounts in rock, soil, surface and underground water, air, plants and animals. The total amount of uranium on the earth (approximately 2 – 4 ppm) remains more or less constant as a result of its long half-life. It can however be moved around by processes like mining (Arfsten *et al.*, 2001; ATSDR, 1999; Veiga *et al.*, 1998; Anon, 1995).

Uranium mining and milling activities have the potential to remobilise radio nuclides and other pollutants and release them into the environment. When rocks are broken, the uranium can become part of the soil (1 – 2 mg/kg), be carried to rivers, lakes (0.01 – 1500 µg/l) and into the air (0.02 – 0.3 ng/m^3). These components have a great contribution to radiation hazards and must therefore be further investigated in order to reduce the radiation risks on the change of other non-radiological risks. Water and vegetable ingestion is the most important pathways to human health risk in this regard

(Arfsten *et al.*, 2001; ATSDR, 1999; Veiga *et al.*, 1998; Anon, 1995). Uranium is usually found in the form of minerals, but can be refined to a very dense, silver-coloured metal. Industrial processes which enrich uranium create a by-product called depleted uranium (DU). The enriched uranium is far more radio-active than DU (ATSDR, 1999).

The external radiation danger of uranium is not great; since the alpha particles do not have enough energy to penetrate the human body to an extent that would cause harm. Furthermore, most absorbed uranium is excreted in the urine within a few days (ATSDR, 1999; ICRP, 1995). However, in the occupational environment these particles become airborne and can be inhaled, ingested and absorbed by the skin, because uranium exists in conjunction with dust in the air (Gulson *et al.*, 2005). Inhalation is dangerous because the particles become lodged in the lungs where it becomes an internal hazard.

Uranium and its compounds are extremely toxic substances, with those compounds soluble in bodily fluids being the most toxic. Fortunately, South African uranium is low in specific radio-activity. The soluble compounds can be inhaled, entering the bloodstream, and be excreted in the urine, or it can remain in the kidneys, which ultimately leads to uranium poisoning (Stanton, 2005). A number of projects have been launched to rehabilitate areas that were subject to uranium mining all over the world. In East Germany the WISMUT Corporation started the WISMUT rehabilitation project, which has since become an international reference project for various mining sites (Schmidt & Regner, 2005).

2.2.4 Radioactive decay

Radioactive decay is the process whereby a radioactive substance spontaneously breaks down into other atoms (or daughters) during a period of time. The length of time, steps involved and type of radiation emitted during decay are well-known, and happens randomly, but with certain characteristics. For example, the type of emission will be characterised, while the actual emissions happen at random. The half-life of atoms in a radioactive substance is the time it takes for half of the atoms to decay, or the time it

takes for the isotope to give off its radiation and become a different element, which can vary greatly (Hall, 2005; ATSDR, 1999). Radon is a by-product of uranium-238 decay, contributing to a great deal of one's natural exposure to radiation.

The decay process of uranium-238 can be summarised as shown in table 2.2. This process will continue until the formation of lead-206, which is a stable element.

Table 2.2: The decay process of uranium-238 to lead-206 (Adapted from Hall, 2005; IRCP, 1993; ATSDR, 1999).

Type of radiation	**Nuclide**	**Half-life**
α	Uranium-238	4.5 billion years
β	Thorium-234	24.5 days
β	Protactinium-234	1.17 minutes
α	Uranium-234	269 000 years
α	Thorium-230	83 000 years
α	Radium-226	1 600 years
α	Radon-222	3.823 days
α	Polonium-218	3.05 minutes
α	Lead-214	26.8 minutes
β	Bismuth-214	19.7 minutes
β	Polonium-214	0.000 146 seconds
α	Lead-210	22.3 years
β	Bismuth-210	5.01 days
β	Polonium-210	138.4 days
α	Lead-206	Stable

The decay process can be further described as follow:

- The uranium-238 atom has 92 protons and 146 neutrons, and a half-life of 4.5 billion years. With decay, it emits an alpha particle, leaving behind thorium-234.
- Thorium-234 has 90 protons and 144 neutrons, with a half-life of 24.5 days. A beta particle and a gamma ray are emitted on decay, and it leaves behind a protactinium-234 atom.
- This atom has 91 protons and 143 neutrons. Uranium-234 has a half life of 269 000 years. When protactinium-234 decays, it emits a beta particle and a gamma ray, leaving behind thorium-230.
- Thorium-230 has 90 protons and 140 neutrons, and a half life of 83 000 years. An alpha particle and a gamma ray are emitted during decay, and it leaves behind radon-222 (Hall, 2005).

2.3 PHYSIOLOGICAL EFFECTS OF RADIATION

2.3.1 Overview

Epidemiological studies have shown a correlation between a prolonged exposure to ionising radiation and definite and measurable increases in the occurrence of cancers, such as lung cancer, and leukaemia (blood cancer) (Hall, 2005; Kovalchuk *et al.*, 2003; Hnizdo *et al.*, 1997; Amandus & Costello, 1991).

According to McDiarmid (2001), there is insufficient evidence to determine a correlation between uranium exposure and lymphatic- and bone cancer, but the possibility cannot be ruled out. Lipsztein *et al.* (2001) also suggest that exposure to radiation in a gold mine does not justify close monitoring, although there has been previous correlation between silica dust, smoking and radiation regarding the risk of lung cancer (Hnizdo *et al.*, 1997; Amandus & Costello, 1991).

The prevalence of lung cancer seems to be higher in silicotics than nonsilicotics, as determined by Amandus and Costello (1991), regardless of the smoking habits of mine workers who were exposed to low radon levels over extended periods of time. The results were inconclusive though, because of the large amount of variables that play a role in the onset of cancer.

It can be assumed that any dose, whether small or large, pose a health risk. Responses to low doses of radiation appears to depend on genetic and environmental factors, type of cells, proximity of the cells to one another, the functional state and demands of the affected organs (to name only a few), and not solely on the dose received (Dawson *et al.*, 2005; Mothersill *et al.*, 2004; Verschaeve, 2004).

The lungs, for example, are able to tolerate high doses of exposure, in small volumes, but cannot tolerate low doses in large volumes, while the spinal cord cannot handle a high dose exposure at low volumes (Stone *et al.*, 2003). Environmental factors play a definite role in the onset of cancer as result of radiation exposure, which complicates the verification of the outcome (responses and symptoms), because not all contributing factors, including those at cellular level, are known (Mothersill *et al.*, 2004).

It takes a couple of years, up to 20, for carcinogens to form, and lifestyle must be taken into consideration as well. There are different kinds of cancer, under which carcinomas (85 %), which start in the epithelium of the body, sarcomas (6 %), which form in the connective tissue of the body (muscle, bone, fatty tissue), leukaemia or lymphomas (5 %), occurs where the white blood cells are formed (bone marrow, lymphatic system) and then other types, for example brain tumours (4 %) which are rare (Anon, 2004a).

Smoking, strong sunlight (especially in South Africa), environmental-, dietary-, health- and genetic factors all play a role in the production of carcinogens (Verschaeve, 2004; Wymer, 2001; Anon, 1995). The body has defence mechanisms in place against the damage done by radiation, since we are being bombarded by background radiation, which constantly affects approximately 10 million cells per minute. On the other hand, radiation

is widely used (in a controlled, direct manner) to kill cancerous cells in a tumour, often saving lives, as well as to kill bacteria in food and sterilize medical equipment (Wymer, 2001; Anon, 1995).

There are no scientific back-up for the determined occupational exposure limits' (OEL's)/threshold level of safety. It seems that the lower the dose and the rate (at least 10 mSv/a), the greater possibility that there will either be a fifty percent beneficial or adverse effect following exposure. It can also be assumed that genetic mutations occur after extensive exposure, affecting future generations. Currently however there is not any evidence of radiation induced mutation in humans, as the studies were done on animals and plants. At very high doses, exposure can cause sickness and death within weeks (Hall, 2005; ICRP, 1993).

The degree of damage depends on many factors, including dose, type of radiation, age, health etc. Radiation injury can be defined as acute, consequential and late effects of radiation exposure, depending on the latent time between exposure and the first signs of symptoms (Hall, 2005; Stone *et al.*, 2003; ICRP, 1993).

Damage to cells because of radiation will have adverse health effects if not given sufficient time to repair. These effects are known as deterministic and stochastic effects. Deterministic effects are found when the dose exceeds the threshold, and the cell cannot survive or reproduce. Excessive damage can result in loss of tissue function. When the dose is above the threshold, the harm caused rises steeply with increased dosage. This effect is uncommon under normal mining conditions and usually restricted to accidents at nuclear installations (Wymer, 2001).

Underground radon-222 exposure comes through the ore or underground water. The radiated particles are not retained in the respiratory system, because these radon daughters are metallic ions that attach themselves to water molecules and atmospheric gasses, forming small particles that again attach themselves to airborne particulates (with a diameter of 0.3 mm). It is these agglomerated particles that are then inhaled and become

logged in the airways and lungs. The radiation particles are deposited onto the tissue before it can be removed naturally, and this is called the stochastic effect (Wymer, 2001). Thus, proper ventilation plays a very important role in the prevention of over-exposure to radiation in the mining environment.

When uranium dust is inhaled, some of the particles become lodged in the lungs, while others are exhaled. This mainly depends on the size of the particles. The larger particles become lodged in the nose and throat where they are blown out, sneezed out or swallowed. When the particles lodge in the lungs and cannot be effectively removed within a short period of time, it will remain in the lungs and cause internal exposure to radiation. The uranium particles can also dissolve in the blood, via the oxygen that is taken up into the blood in the lungs, carrying the particles throughout the whole body. Most of the particles are excreted within a few days, but some stay behind in the kidneys and bones. Animal studies show a tendency toward kidney diseases after prolonged exposure to large doses of uranium (ATSDR, 1999). These diseases are not prevalent in mine workers as a result of uranium exposure, since they are only subject to small amounts of exposure.

Some animal studies also show decreased reproduction when exposed to uranium and depleted uranium. Many deformities have also been observed in human foetuses, babies and children as a result of high doses of radiation exposure (Mirkarimi, 1992). In their research on genome response to acute and chronic low-dose irradiation, Kovalchuk *et al.* (2004) found that male and female mice reacted differently to genotoxic stress, or irradiation. This is an area that has not been researched before. Further studies into this field might yield invaluable new information about the process and affects of irradiation.

Low-dose ionising radiation has become an area of great concern, even though there is not sufficient evidence or data available to substantiate findings and assumptions (Donoghue, 2004; Goldberg *et al.*, 2004). Radiation dose-response relationships can be determined with the linear-no-threshold model that has been adopted, but is limited, insufficient and too simplistic for scientific research.

The linear no-threshold model is a model of the damage caused by ionising radiation, and particularly the increased risk of cancer. It assumes that the response is linear and that this linear relationship continues to very small doses. In other words, there is no threshold of exposure below which the responses cease to be linear. A practical example is that if a particular dose of radiation is found to result in one extra case of cancer in every thousand people exposed, the model predicts that one thousandth of this dose will produce one extra case in every million people equally exposed, and that one millionth of this dose will produce one extra case in every billion people exposed. This plain model has been in use for a long period of time (Goldberg *et al.*, 2004).

The effects resulting from exposure can be acute, where symptoms are immediately recognised. This happens mostly in tissue with proliferating cells (cells that are able to multiply themselves) such as the epithelial surface of the skin. The effect is mainly deterministic, because the functional cell damage occurs at the stem-cell compartment, and the cell cannot be replaced. Compensatory proliferation occurs in the skin and gastrointestinal tract, since these cells are more tolerant to irradiation than others (Stone *et al.*, 2003).

Vital cellular components are damaged as a result of ionisation, including the free radicals that are produced with radiation exposure, which leads to DNA damage, and, ultimately, cell death. Unrepaired chromosome damage also causes cell death. The maintenance of genomic stability depends on the ability of cells to sense and recognise damaged DNA and then to either repair or induce an exit, through apoptosis or cell differentiation. During the latent phase, lymphocytes, spermatogonia and serous cells (salivary gland), undergo apoptosis (Stone *et al.*, 2003; Offer *et al.*, 2002; Wright, 2002; Schwartz & Rotter, 1998). Apoptosis can be defined as programmed cell death, brought on by a genetic process where cells destroy themselves by the fragmentation of nuclear DNA. This is activated by the presence or absence of stimuli and is a normal physiological process of removing unwanted and damaged DNA cells from the body. Uncontrolled cell growth and tumour formation, similar to genetic mutation, might result when this process is blocked (MWOD, 2005; MWOT, 2005).

It is suggested that p53, a tumour suppressor gene that is activated following genotoxic stress, may trigger the onset of DNA-repair leading to the completion of the cell cycle. It may also induce apoptosis or terminal differentiation, leading to exit from the cell cycle. It was found that this DNA repair was enhanced at high doses of γ-irradiation and when p53-protein levels were reduced (Offer *et al.*, 2002; Schwartz & Rotter, 1998). Responses to irradiation include fibroblast responses, which results in excess collagen deposition and fibrosis, activation various cellular signalling pathways involved with the onset of oedema and inflammatory conditions (Stone *et al.*, 2003).

Late effects have extended latent periods (as with the onset of carcinogens) and symptoms appear spontaneously and in a number of ways. The tissues involved are tissue with slow turnover, such as subcutaneous-, fatty-, muscle-, brain-, kidney- and liver tissue, as well as the intestine wall - resulting in injuries like fibrosis, necrosis, atrophy and vascular damage. The responses to these injuries include cytokine production, which leads to adaptive responses in the surrounding tissue and cells (much like wound healing responses). Cytokines are a class of immunoregulatory substances (similar to lymphokines) that are secreted by cells of the immune system. Consequential effects target mainly the skin, mucosa, urinary and intestinal systems and are the result of chronic injury/lesions (Stone *et al.*, 2003).

2.3.2 Pathology

Radiation therapy, usually conducted with gamma-rays, has advanced technologically in the last couple of years. It has only been recently that dose can be administrated and distributed more accurately, ensuring less tissue damage, reduced toxicity-levels, increased quality of life and a subsequent increase in the likelihood of tumour control. It is not only the targeted cells that respond to radiation, but also the bystander cells, or those cells in close proximity to the targeted cells that is influenced by irradiation (Dawson *et al.*, 2005; Goldberg *et al.*, 2004; Little, 2004; Azzam *et al.*, 2003; Somosy *et al.*, 2002; Wright, 2002).

There are a number of limitations to radiation therapy research. These include methods of analysis, limitations of the cumulative dose-volume histogram that is used, uncertainty concerning normal tissue complication probabilities and unethical as well as unplanned exposure of individuals. Dose-volume applications will remain subjective until these variables are better understood. The most studied organs up to date are the lungs, liver and parotid glands (Dawson *et al.*, 2005; Goldberg *et al.*, 2004; Verschaeve, 2004; Somosy *et al.*, 2002).

An understanding of the possible effects of radio-frequency exposure on the genetic material of cells are important, since damage to DNA can lead to all kinds of pathology in different cell types. There is little evidence that exposure is directly mutagenic, although there is some indirect effects on DNA replication/transcription of genes under controlled exposures (Verschaeve, 2004). Accumulation of dense extra cellular matrix (collagen and glycosaminoglycans), or fibrosis, plays a large role in irradiation processes, especially in the submucosa, muscular propria and subserosa of the lung, skin, muscle, liver and gastro-intestinal tract (Somosy *et al.*, 2002). Changes in cellular organelles include:

- cellular swelling;
- mitochondrial swelling;
- irregular shaping of the cell membrane;
- degranulation and vesicularisation of the endoplasmic reticulum;
- enlargement of the Golgi complex;
- rearrangement of the cytoplasmic actin and cytokine filaments;
- protein degradation;
- increase in the cytoplasmic volume of lysosome-like vacuoles in the enterocytes
- and increased activity of lysosomal hydrolysis (Azzam *et al.*, 2003; Somosy *et al.*, 2002).

Irradiated cells have abnormal projections, altering the cells' interaction with one another, as well as normal homeostasis in the cellular environment. Tight junctions in the epithelial and endothelial cells are very important for transport processes in as well as

between cells and have a great impact on the pathology of the organs. Gap junctions are junctions that consist of a complete cell-to-cell channel that spans two plasma membranes. They result from the association of two connexons (an extensive family of proteins), contributed separately by each of the two participating cells. Homeostatic control of normal cell growth pathways is known to be strongly dependent on oxidants. A disruption of the balance between oxidant production and antioxidant defence leads to a state of oxidative stress that can induce several pathological conditions. The endogenous targets of oxidants are diverse and include nucleic acids, proteins and lipids (Azzam *et al.*, 2003; Somosy *et al.*, 2002).

Research done on cyclic AMP (cAMP) and the adenylate cyclase system which is involved in intracellular communication, found that functions of both these systems was altered with irradiation; cAMP concentrations increased, and adenylate cyclase activity via VIP (vasointestinal peptide) stimulation also increased. Gap junctions in the cells are selective, and these second messengers (cAMP and cGMP) might be discriminated by or favoured when damage is done. Calcium can also influence intracellular homeostasis in the small intestine (Azzam *et al.*, 2003; Somosy *et al.*, 2002).

Direct or indirect altering of intracellular signalling pathways may lead to pathophysiological changes in different tissues (inflammatory reactions, fibrosis, tumour formation, modification of immune responses of cells, increased proliferation etc). The cells may be affected via membrane signals or altered activation of certain receptors like gene expression and secretion of certain cytokines, such as interleukins, TNFα, EGFα, TGFα and TGFβ (Azzam *et al.*, 2003; Somosy *et al.*, 2002).

2.3.2.1 Lungs

The lungs are the most frequently exposed and the most radio-sensitive organs in the body. Symptoms of exposure vary from congestion, cough, dyspnoea, fever, pneumonitis to breathing difficulties (Stone *et al.*, 2003). Tuberculosis might also result from exposure, but mostly because of the dust particles itself.

On cellular level, the concentration of type II pneumocytes and alveolar macrophages increase, parenchymal cells and surfactant concentrations decrease, and hyaline membranes tend to develop. Studies on cancer patients undergoing radiotherapy revealed that those with high plasma concentrations of the cytokines interleukin 1 and/or interleukin 6, before or during therapy, have a higher risk of developing pneumonitis. Those with increased TGFβ have a higher risk of radiation induced lung injury. TGFβ is a group of polypeptides that are secreted by a variety of cells, like monocytes, T cells, or blood platelets, and have diverse effects on the division and activity of cells. These effects include induced angiogenesis, stimulating fibroblast proliferation, or inhibiting T cell proliferation. (Okunieff *et al.*, 2005; MWOD, 2005; MWOT, 2005; Stone *et al.*, 2003). The renin-angiotensin system, associated with the development of radiation nephropathy, is also involved in the development of pulmonary injury after radiation exposure (Stone *et al.*, 2003).

Lung cancer is the most common form of cancer diagnosed in the United States and a major cause of death. Lung cancer accounts for 28 % of all cancer related deaths; with cigarette smoking contributing to 87 % of all lung cancer deaths to date (Anon, 2000). Lung cancer, as a result of radon exposure, is the second leading cause of lung cancer in the U.S. Scientists believe that radon induced lung cancer is responsible for 15 000 to 22 000 deaths per year.

Researchers first associated radon exposure with the prevalence of lung cancer when it became obvious that a large population of underground mine workers suffered from lung cancer (Archer *et al.*, 2004; Anon, 2004a; Anon, 2004b; Anon, 2000). ATSDR (1999) disagree with the theory that uranium exposure contributes to cancer, saying that no human cancer has ever resulted because of uranium, although uranium decay products might contribute after prolonged exposure, and supports the view of Dondon (2005), Mcdiarmid (2001) and Lipsztein *et al.*, (2001).

These contradictions necessitate an investigation into the effects of irradiation on workers in the mining environment, stressing the importance of obtaining data, which is why this study was conducted. This will allow proactive and progressive intervention in order to minimise any possibility of occupational disease in this regard.

Cells in the body generally reproduce and divide in order to repair damaged tissue and bring about growth (proliferation). With cancer, the cells multiply at uncontrollable rates, forming tissue masses that are called tumours. These tumours can either be malignant (cancerous) or benign (non-cancerous). Benign tumours usually stay localized in the area it first appeared and are in general not life threatening. Malignant tumours can spread throughout the body and damage healthy tissue. Lung cancer will generally spread through the whole body until it reaches the lymphatic system. From there it moves toward any organ in the body. Secondary tumours, also called metastic tumours, are formed and primarily found in the brain, liver and bone (including bone marrow) (Anon, 2000).

2.3.2.2 Upper extremities

Studies found that the skin, mucosa, subcutaneous tissues, bone and salivary glands are the most affected areas when patients undergo radiotherapy for head and neck cancer (Stone *et al.*, 2003). The studies conducted in Sweden showed that 60 % of all soft tissue sarcomas are located in the extremities, with two thirds of these occurring in the lower limbs, and the others in the head, neck, trunk and lungs.

Soft tissue sarcomas represent a heterogeneous group of tumours with wide variations in presentation, histological appearance and prognosis. They are labelled and grouped by their cell of origin. The origin however, might be unknown sometimes. Histological grade, tumour size and depth have become well-established markers for local control and disease-free survival. There are two possible therapies (in Sweden) for these kind of sarcoma, which is EBRT (external beam radiation therapy) and BRT (brachytherapy) (Strander *et al.*, 2003)

The skin exhibits symptoms like erythema (abnormal redness of the skin due to abnormal capillary congestion), dry and moist scaling of cells, pruritis (itching), hypersensitivity, pain, dermatitis and mucositis. Both the skin and mucosa are subject to pathology like hyperemia, vascular congestion, vasodilatation, plasma leakage, epithelial denudation, hair loss, pigmentation changes, telangiectasia (abnormal capillary dilatation), atrophy, retraction, fibrosis and ulceration. These responses are enhanced by cytokine-mediated reactions. Oedema and lymph oedema might also occur due to vascular and connective tissue damage (Stone *et al.*, 2003).

The salivary glands are composed of the parotid, submandibular, sublingual and buccal glands. The parotid glands contain serous cells, which secrete ptyalin (Guyton & Hall, 2005). These cells die by apoptosis (programmed cell death) when exposed to radiation. The submandibular and sublingual glands on the other hand, are more resistant to radiotherapy (Stone *et al.*, 2003) and contain both serous and mucous cells (mucous cells secrete mucin) (Guyton & Hall, 2000).

Damage occurs mainly at the parenchyma of the salivary gland. The severity of the damage is greatly increased by inflammation, vascular changes and oedema that also results from irradiation. The primary symptom is xerostomia, an abnormal dryness of the mouth as result of the saliva that becomes viscous and sticky, which can lead to dental pathology (Stone *et al.*, 2003).

Treatment of prostate and cervical cancer primarily affects the rectum, but the gastrointestinal tract is also sensitive to irradiation. The main symptoms are diarrhoea, as a result of decreased epithelium function and increased mucous secretion.

Other symptoms include:

- tissue oedema;
- hyperemia;
- increased stool frequency;
- spotting of blood;
- partial incontinence;
- fluid and electrolyte imbalance;
- bacteraemia;
- endotoxemia and
- impaired hemopoiesis.

Hemopoiesis is the ongoing process by which the cellular components of blood are replenished as and when needed (MWOD, 2005; MWOT, 2005).

A study done on mice revealed that damage to the hemopoietic system can result in death within a period of one month. Mice that survived longer than one month suffered severe damage to their oral mucosa (including the associated glandular and lymphatic elements) and other hematological tissues, as well as other tissue and organs (Potten, 1996).

This is mainly due to fibrosis and ischemia that occur in the submucosa and muscularis. Telangiectasia and vascular abnormalities, mucosa congestion, increased collagen deposition, abnormal fibroblasts, cell necrosis, crypt abscesses and structural changes, villi damage, loss of Paneth cells, decreased mitoses, loss of the margination of lymphocytes and stem cell depopulation in slowly dividing tissue also occurred (Goldberg *et al.*, 2005; Stone *et al.*, 2003; Rubio & Jalnas, 1996).

Molecular processes are not fully understood or explored yet, but decreases in endothelial thrombomodulin have been observed. This leads to increased fibrin deposition, fibrinic cytokines and mRNA concentrations for $TGH\beta_1$ and $TNF\alpha$ in mice. After irradiation the mucosa is lined with abnormal epithelial cells (omega cells), racket-shaped cells

(luminal side), giant cells and clear cells. (Goldberg *et al.*, 2005; Storm *et al.*, 2003; Rubio & Jalnas, 1996).

Growth factors show protective tendencies as a result of the rapid apoptic responses of the epithelium, after irradiation. These growth factors increase Akt phosphorylation, while at the same time preventing apoptosis of non-proliferating epithelium. The cell cycle is also influenced by growth factors that cause the cells to accumulate in radio resistant phases (Goldberg *et al.*, 2005). Administration of interleukin 11 before and after radio-therapy significantly increases the survival rate of crypts in the intestinal tract of mice (Potten, 1996).

As mentioned earlier, the gastro-intestinal (GI) tract is very sensitive to irradiation. A lot of research has been done on the biochemical and histochemical components in the small intestine cells. There are mainly two types of effects that occur; prodromal or early effects, and acute or late effects. Prodromal effects result because of damage to the central processes of the autonomous nervous system. Main symptoms include nausea and vomiting, temporal incapacitation, and immune system damage. An increase in prostaglandin and neurotensin levels has also been noted. Acute symptoms entail radiation enteritis and radiation-induced enteropathy (Somosy *et al.*, 2002; Potten, 1996).

Stem cells in the crypts of the GI tract respond to radiation quickly, both with cell loss and repopulation. The loss of these stem cells is a result of apoptosis, mitotic inhibition (because of a G2 block) and reproductive sterilisation. About 70 – 80 % of all the crypts are destroyed when a dose of 12 Gy of radiation is administered. This dose also caused the death of half the mice subjected to testing within a week after administration. Alterations in the number of lymphocytes, macrophages and granulocytes have also been reported after irradiation. GI syndrome only occurs after high dose whole body x-radiation. Irradiation of cells have an influence on enzyme activity of the microvilli, such as lactase, sucrase maltase, leucine amino peptidase, alkaline phosphatase, Na^+- and K^+-ATPase (Somosy *et al.*, 2002; Potten, 1996).

2.3.2.3 Radiation sickness

A single, high dose of radiation can be lethal. Uranium poisoning has adverse effects on the kidneys and the body's defence mechanisms (Anon, 2000(a)). This is generally as a consequence of accidents at nuclear installations or similar incidents and is not something commonly encountered (Wymer, 2001).

2.4 STANDARDS AND REGULATIONS

Occupational exposure to radiation in the mining industry in South Africa is regulated by two laws. There is the Mine Health and Safety Act, 1996 (SA, 1996:967), as well as the National Nuclear Regulator Act, 1999 (SA, 1999:1537). The maximum doses of exposure to radiation are outlined beneath.

The controversial "linear no threshold" (LNT) hypothesis is recommended for radiation purposes, despite several inadequacies. The International Commission on Radiological Protection (ICRP) has three basic principles, which has become the international standards for radiation protection: justification of practice, optimisation of protection and individual dose and risk limits.

According to the abovementioned laws the following dose limits have been proposed:

- An effective dose of 20 mSv/a, over 5 consecutive years;
- A maximum effective dose of 50 mSv/a (Ellis, 2004).

2.5 RADIATION MEASUREMENT

The amount of radioactive material is measured in Becquerel (Bq), which is a measure that enables one to compare the radioactivity of materials with one another. One Becquerel equals one atomic decay per second. The former unit of radioactivity was Curie (Mouton, 2005).

The decay of radon results in a decay chain with the short-living radioactive nuclides Po-218, Pb-214, Bi-214 and Po-214. In general Po-214 is not specified. Because of its short half-life, the concentration of Po-214 is in practice equal to the concentration of Bi-214. The decay chain looks as follow:

$$\text{Rn-222} \rightarrow \text{Po-218} \rightarrow \text{Pb214} \rightarrow \text{Bi-214} \rightarrow \text{Po-214} \rightarrow \text{Pb-210}$$

The complete process is summarized in table 2.2. Decay product concentration is often described with a special unit: The potential alpha energy concentration (PAEC). This unit originates from uranium ore mining and quantifies the biological influence of a decay product mixture. It describes the resulting α-energy of all decay products in a distinct volume until their total decay. The common unit is MeV/l (Tracerlab, 2004).

$$1 \text{ Bq} = 27 \times 10^{-12} \text{ curies}$$

The amount of radiation, or 'dose' that a person is exposed to, are expressed in gray (Gy) (Mouton, 2005).

$$1 \text{ Gy} = 1 \text{ Joule/kg}$$

It is difficult to express different types of exposure in the same unit, because one gray of alpha radiation will have a greater effect than one gray of beta radiation. Therefore, radiation effects are referred to as effective dose, which is a unit called Sievert (Sv). In

most cases the the dose is expressed in milli Sievert (mSv). The effective dose reflects the biological effects, not the specific dose (Mouton, 2005).

$$1 \text{ Sv} = 1000 \text{ mSv}$$

2.5.1 Available methods

Radiation cannot be detected through human sense, accordingly a variety of instruments have been developed to measure radiation. These instruments are both as accurate and reliable as is possible.

2.5.1.1 Passive measuring methods

Table 2.3: Summary of passive measuring methods (Tracerlab, 2004).

Method	**Description**
Charcoal Absorption method	After a sampling period ranging between hours to three days, charcoal and absorbed radon, as well as decay products is measured with liquid scintillation or gamma-spectroscopy. Various influences like atmospheric pressure, humidity, the type of charcoal used and so forth, must be taken into account.
Nucleus tracing	A synthetic detector foil is placed in a measuring chamber with an inlet filter. Nuclei from radio-active decays inside the chamber cause damage the foil. By corroding procedures this damage results in tiny, visible holes - where the number of holes is proportional to the radon concentration.
Lucas-method	Radon decays cause scintillation effects in a chamber with activated (ZnS) walls. These scintillations are counted by use of a photo-multiplier.

The above table shows a summary of the methods. These methods do not need any power supply during sampling and a great number of equipment can be used at minimal costs. The impact of laboratory work on expenses to analyse results must be taken into account.

2.5.1.2 Active measuring methods

Table 2.4: Summary of active measuring methods (Tracerlab, 2004).

Method	Description
Double-filter method	Air is pumped through a measuring chamber, with a specific volume of air passing through an inlet filter. New decay products generated in the chamber are sampled and measured on an outlet filter.
Diffusion-chamber method	This method corresponds to the nucleus-trace method. In this method, however an, electronic detector and additional concentration systems are used instead of foil. Decay products deposit on the walls and the detector. An electrical field between the detector and chamber is often used for higher deposition and measurement efficiency.
Ionisation-chamber method	Charge pulses from decays in a ray sensible volume are counted. At high radon levels a direct current can be measured instead of single pulses.
Filter method	Air is sucked through a filter while decay products deposit on it. A detector measures the radioactivity on the filter. Various methods exist with different designs, flow rates, detectors, sampling and measuring intervals and calculation algorhythms. The use of filter ribbons and automatic transport systems allow long-term operation.

These methods generally use pumps and electronic detectors for automatic operation. The double-filter method, diffusion-chamber or pulse-ionisation chamber are preferred methods to detect and analyse irradiation from radon gas, as can be seen from table 2.4.

2.5.1.3 Area monitoring

(a) GRS-2000 MultiSpec gamma-ray spectrometer

This instrument can be used for a variety of tasks for surface and borehole surveying. It is a compact water-proof probe, equipped with a scintillator and sophisticated analysis applications. It was designed for geophysical and environmental surveying, geological prospecting, industrial monitoring of radioactivity, and laboratory analysis. Both single and profile measurements can be taken and results are displayed in a graph on downloading. One of the disadvantages is that it only measures gamma-rays. Accordingly it will not be effective in mines where alpha-rays are the main concern. The instrument is also very expensive (Gisco, 2005).

(b) PGR portable scintillation meter

The PGR meter is accurate, sensitive and reliable. It has the same disadvantages as the GRS-2000 MultiSpec gamma-ray spectrometer, namely that it only measures gamma-rays and it is relatively expensive. It can be used in a lot of different spectrums, including mining and geology. It is fairly easy to use and data can be downloaded onto a computer. The fact that it can function in very rugged and robust conditions is a major advantage (Gisco, 2005).

(c) GRM-260

The GRM-260 is designed for field assays of rocks and for dose rate measurements. Unfortunately its application is also limited to measuring gamma-rays, and it is therefore also not suitable for the purposes of this study. It can be operated from a computer and is easy to operate in the field. The GRM is quite expensive (Gisco, 2005).

(d) Electra

The Electra is a digital, microprocessor-based rate meter and is compatible with most survey probes. Readings are displayed numerically and/or as bar graphs. It is robust, sturdy, balanced and easy and comfortable to use over long periods of time. This instrument is currently used in the mine.

(e) Tracerlab IRGprobe

This instrument is an AC-line/battery operating Radon gas monitor. It is a self contained portable system based on air diffusion and system-control by integrated display/keyboard (Tracerlab, 2004).

The impracticality of area sampling makes it necessary to use personal sampling methods to monitor radiation in the mine. Most area sampling instruments are expensive and produce a representative reading of doses in specific areas. The instruments are either stationary, subject to blasting fumes and not representing the whole area, or must be carried around by a designated person, which influences exposure time and reliability. These instruments are however effective when spot readings are required for planning purposes.

2.5.1.4 Personal analysis

(a) LCD-BWLM-PLUS

This is an AC-line/battery operation radon monitor, which is a self contained light-weight portable system with a removable sampler. It is compatible with most computers (Tracerlab, 2004).

(b) BWLM-PLUS

This is also an AC-line/battery operating radon-monitor. It is self contained, portable simple to operate and can also be used to determine free fraction (Tracerlab, 2004).

(c) WLM-PLUS/ASF-200

This AC-line/battery operating radon monitor is also self contained and portable (Tracerlab, 2004).

(d) RGM/ARDM-PLUS

This is the instrument that is in use at the mines. It is an AC-line/battery operating radon-, thoron-daughter working level monitor with a fixed-filter. This self contained system has a protection-cap for installation under rough conditions (Tracerlab, 2004).

RGM badges are used because it is the most cost effective method available. The large amount of employees required to be monitored on a regular basis makes this the best option. The size of the badges makes it logistically flexible and it has the added advantage of not influencing the mine workers performance. It is also the best source of area monitoring, as radon gas concentrations fluctuate over time. These instruments accompany the workers everywhere in the workplace. A representative reading of the radon gas concentrations for that workplace over a given period of time is acquired in effect.

CHAPTER 3: EXPERIMENTAL PROCEDURE

3.1 INTRODUCTION

Underground radiation gas monitoring is conducted to determine present exposure levels of the workforce to ensure that exposure levels comply with the requirements of the National Nuclear Regulator and to continuously monitor and assess risks as required by the forementioned Council. Monitoring is done in accordance with the RPP (Radiation Protection Procedures) guidelines for radiation gas monitoring in terms of National Nuclear Safety Act (Act no 47 of 1996), as well as the Mine Health and Safety Act (Act no 29 of 1999).

The Anglogold Ashanti (Vaal River) requirements were followed during the program, according to which each business unit has to conduct a radiation monitoring program to monitor the entire shaft quarterly. Each cycle consists of three months, during which RGM's (radon gas monitors) are issued, retrieved and analysed.

The shaft is divided into ventilation districts, or adjacent groups of working areas. These ventilation districts share common intake and return airways. On the shaft monitored, there are two ventilation districts, namely Kop 1 and Kop 2. The districts are further divided into workplaces (for example K1-2, K2-3, K2-6) and within these workplaces, into stopes, crosscuts and raises. A haulage refers to a main access facility for tramming, transport, travelling and air, for the purpose of this study. The haulage forms part of the main infrastructure and is necessary for the life of the mine. A crosscut is the actual connection between the haulage and the reef horizon/workplace. Every workplace has its own crosscut, and this crosscut, which is part of the sub-infrastructure of the mine, is necessary for the life of the workplace. Crosscuts are sealed off in order to prevent unwanted air movement, build-up of flammable gasses and travelling of people once a workplace is depleted. A raise is the main service pathway that connects the workplace with the two levels above and below.

The RGM's for the normal four-cycle monitoring program are issued so that at least three employees per workplace are monitored for a representative sample of that specific workplace. One employee is monitored at the bottom of the workplace, where the fresh air enters, one in the middle of the workplace, and one at the top, where the air leaves the workplace. This ensures that fresh air from the intake airways, as well as the contaminated air that comes from the workplaces, are included in the sampling population. Areas are investigated to determine the amount of radiation in that workplace, and action plans are set in place to lower the exposure of the workers when high readings are obtained. The following figure is a summary of the procedure followed during sampling cycles:

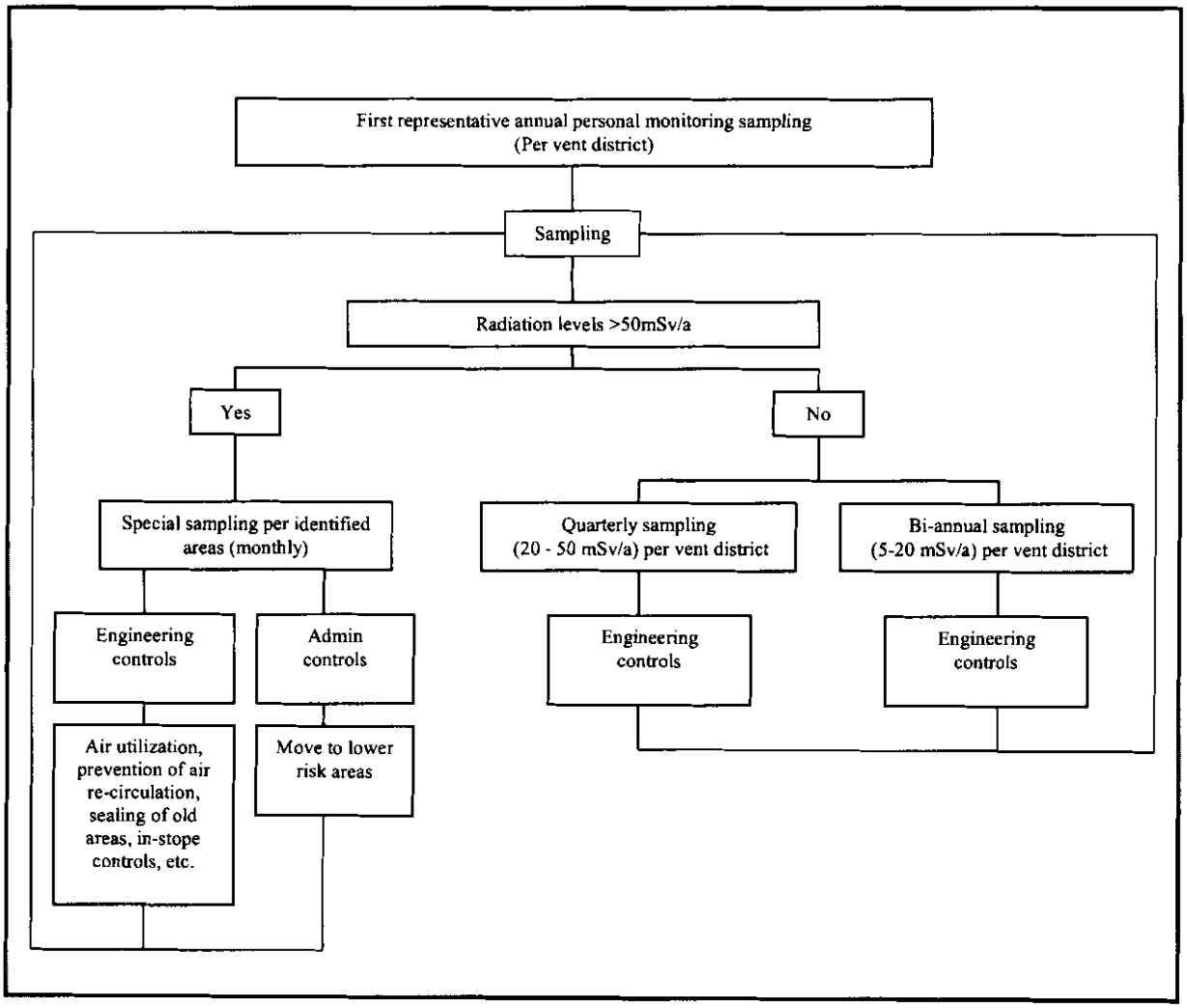

Figure 3.1: Diagram of the radiation sampling procedure (adapted from Ellis, 2004).

Figure 3.1 shows that this is a progressive study, where data is obtained and analysed, actions and controls are implemented and the cycle repeated to reassess the situation. It also shows how sampling cycles are determined. If a person is exposed to 20 - 50 mSv/a, they are sampled quarterly. Bi-annual sampling commences when readings are lower than 20 mSv/a. Special sampling are conducted on a monthly base and includes all the employees that are exposed to more than 50 mSv/a.

3.2 INSTRUMENTS

RGM's are small, cap-like objects that are attached with cable-ties to lamps assigned to selected employees. The cable ties are effective in keeping the RGM attached to employees' lamps. It is shown in Figure 3.2 and Figure 3.3.

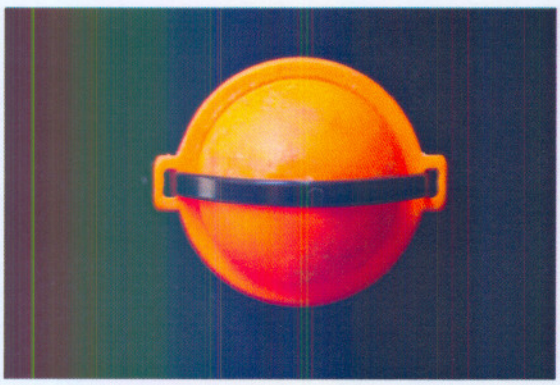

Figure 3.2: The RGM (top)

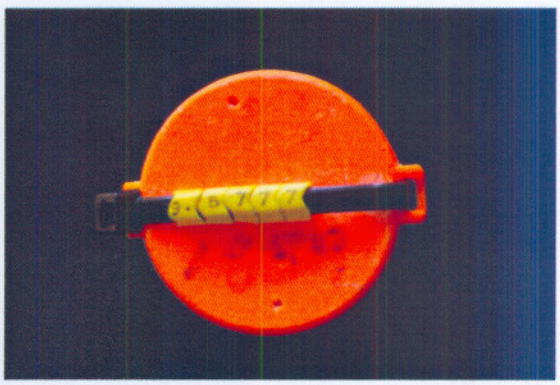

Figure 3.3: The RGM (bottom)

For reference purposes each RGM has a serial number engraved on both the outside and inside of the casing. The lamp issued to each employee also has a number, which is linked to the number assigned to an employee at engagement. The number on the RGM is recorded and also linked to the lamp number of the employee. These numbers are entered into the developed system and matched to results from the laboratory upon return. When the RGM's are retrieved, they are sealed in containers, and transported to the lab within two days. It takes around one month before the results are available.

3.3 SAMPLING

Special case areas are identified and further monitored based on the results. This is a difficult task, since the workforce changes and moves regularly. Keeping tract of these changes are almost impossible. One of the aims of the study was to create a database that is easily accessible, complete and accurate, with the option of inputting data on a monthly basis to keep the system updated making it possible to locate the employees and their workplaces underground. The database is therefore trustworthy and reliable, and allows for proactive steps to minimize the health hazards associated with radiation.

Another aim was to clarify and refine the current monitoring program, focussing on the issuing and retrieval of RGM's, as well as the strategy of sampling. Each RGM has a number, which makes it possible to allocate a dose to a specific employee and workplace. In the past the issue and retrieval of RGM's were unstructured, rendering results unreliable. One of the objectives was to successfully refine this process.

Firstly, areas of concern were identified based on to the previous cycle's results. The workers in those areas were traced using the bonus-system as reference point, since this system is the most reliable source of workforce information available to date. The bonus system closely tracks the movement of employees underground as bonuses are based on a gang's performance during the month.

These gangs are referred to as power teams and there are three to four power teams in a working area. A power team consist of approximately 10 – 14 employees. An area that continues to deliver high radon readings is classified as a special case area. Every person in that area is issued with a RGM in the following cycle. Specials (RGM's in the special case areas) are issued monthly, in comparison with normals (the normal RGM cycle) which are only issued quarterly. Special case monitoring continues for at least three months, or at least until consistent satisfactory readings are obtained well below the OEL (Occupational Exposure Limit).

The shaft was categorized as a special case mine during the November 2004 cycle, which means that radiation exposure is a great concern in this area of the mine. Working places that had to be monitored every month instead of once per cycle were identified. These areas were K1-2, K1-3, K1-6, K2-3, K2-5 and K2-6. An in depth study commenced that brought the database up to standard, located all the employees that had to be sampled and issued RGM's.

3.4 DATA ANALYSIS

The data were collected and recorded into an MS-Excel worksheet (figure 3.5) created specifically for the purpose of calculation and reference. It consists of the following information:

Company number of the employee
Initial
Surname
Occupation
Workplace
Ventilation district
Section
Lamp number
RGM number
RGM reading
Exposure time
Dose

This format allows for monitoring of each employee's exposure levels, and the determination of the annual dose (figure 3.6), which in turn permits the determination of the necessary steps to reduce risks. Retrieved RGM's where matched to the data obtained with the issue of RGM's at the beginning of the cycle as a control measurement. The time of exposure is calculated by multiplying the amount of days the RGM's were exposed

with the amount of hours worked per shift (e.g. 30 days x 9.5 hours) and sent to the lab for analysis. This is explained in figure 3.5 and figure 3.6. The results and dose are calculated accordingly. The average working shift is 9.5 hours long, and is used as such in the aforementioned calculation. Five background RGM's were placed as references within the lamp room, to determine the normal background radiation exposure. The following table shows the format in which results returned from the laboratory:

Table 3.1: RGM result format

RGM number	RGM reading (Bq)
62133	1.47E+05
62156	2.01E+05
62276	2.07E+05
62180	2.07E+05
62316	2.12E+05
62147	2.18E+05
62303	2.23E+05
62234	2.34E+05
62383	2.34E+05
62084	2.39E+05
62235	2.39E+05
62224	2.45E+05
62361	2.45E+05
62216	2.50E+05
62260	2.50E+05
62281	2.50E+05
62141	2.50E+05
62133	1.47E+05
62156	2.01E+05

The results are in scientific notation, as can also be seen from table 3.1. These results were analysed, using the following formulas:

$$BackgroundRGM = \left(\frac{AverageBackground}{Dose} \right) \times f1 \quad (3.1)$$

$$PlusBackground = \left(\frac{Dose}{ExposureTime} \right) \times f1 \quad (3.2)$$

$$LessBackground = PlusBackground - BackgroundRGM \quad (3.3)$$

$$Dose = LessBackground \times f2 \quad (3.4)$$

$f1 = 0.0063651$
$f2 = 2.0979$

The results were imported into a MS Excel datasheet and analysed as shown in figure 3.5. The abovementioned equations were used, as can be seen in the figure below.

LAMPROOM RGM's		EXPOSURE TIME (h)
RGM Nr.	Bq/m3	
6187	3.31E+05	323
6188	4.21E+05	323
6189	3.87E+05	323
6190	3.88E+05	323
6191	3.45E+05	323
		BG DOSE (mSv/a)
Average	3.74E+05	7.4

ICRP-65 Conversion factor:
0.0063651
with an F-factor of 0.4 and exposure of 2 000 hours

This is the total exposure time of the background RGM's in the lamproom

Total exposure time for RGM's underground X 9.5 hours X number of days exposed

Data received from lab

CO NR	INI	SURNAME	OCC	W/PLACE	VD	SECTION	LAMP	RGM	RGM2	RGM READ	EXP TIME	TIME	PLUS BG	LESS BG	DOSE
XXX	X	XXX	T/LEADER	44BW3	K1-3	12	XXX	96370	96163	96370	5.65E+05	323	11.1	3.7	3.7
XXX	X	XXX	DRILLER	44BW3	K1-3	14	XXX	96345	96413	86345	6.47E+05	323	12.7	5.4	5.4

CALCULATION:
Dose = [[RGM reading (Bq/m3.h) / Exposure time (h)] x 0.0063651] - [[Background reading (Bq/m3.h) / Background exposure time (h)] x 0.006365

Figure 3.4: Example of a datasheet used for analytical purposes
(adapted from Ellis *et al.*,2004)

The whole analysis process is repeated and documented separately with every sampling cycle. Each result from the laboratory is matched according to the RGM number that was assigned to the employee at the start of the sampling cycle. Background RGM's are also matched and an average background dose is determined. The dose is calculated by using the formula in figure 3.5. The data is then sorted according to ventilation district, working-place and dose, and compiled into a report that is sent to all the managers and supervisors responsible for that area. The graphs, statistical distributions, averages, maximum potential exposures and 95^{th} percentiles for each ventilation district, are also calculated.

The developed program is able to calculate the required information without using any external programs. Area classifications are made with the 95^{th} percentile on an annual basis and the average exposure for a ventilation district is allocated to all workers in that section for the period monitored.

Figure 3.6 illustrates the statistical process of analysis of the data in this study. The dose measured is selected and copied into another sheet for calculation purposes. Dose distributions are calculated by using the available data from the MS Excel program. The frequency and cumulative percentages are calculated and shown on the graph. The average, minimum, maximum and 95^{th} percentile values can also be calculated for comparison with previous results. This allows the tracking of trends within ventilation districts.

Each month's results are pulled into another database that tracks each employee's personal exposure. The employee's dose is inserted into the MS Excel sheet according to the month the sample was taken. This enables the determination of actual exposure to date, as well as projected doses for the year for each employee. An employee who is projected to be over exposed within the year can be removed from the high risk area into a low risk area, or be assigned another task until the risk has been eliminated or reduced.

Thus, there are two databases that run simultaneously. One monitors the areas in which the employees are sampled, while the other monitors personal sampling. Here each individual is monitored continuously.

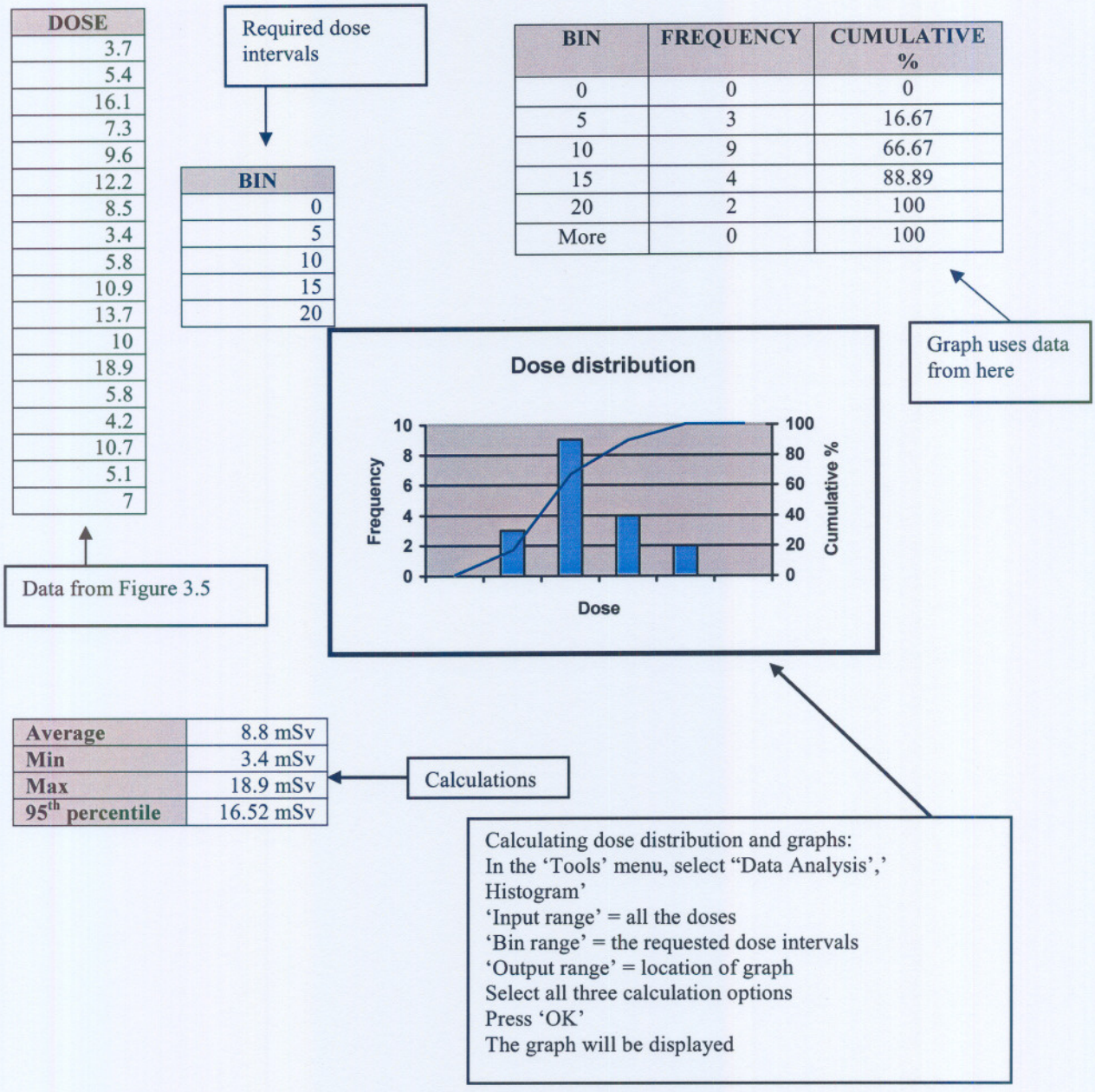

Figure 3.5: Statistical analysis of data (adapted from Ellis *et al.*, 2004)

All the graphs obtained from the analysis, as shown in the above figure, are compiled into a report that is sent to the appropriate supervisors on a monthly basis. The system is regulated by administrative controls that were established. All the areas on the shaft were noted as well as information regarding the amount of people in that working place, their occupation, and personal history and so forth.

The method can be summarised as follow:

- issue RGM's to the identified employees, whether specials or normals;
- retrieve RGM's after the sampling period is over;
- send RGM's to the laboratory for analysis;
- process the results in MS Excel;
- update the personal history and projected dose datasheets.

CHAPTER 4: RESULTS AND DISCUSSION

4.1 RESULTS

The following figures are a summary of the data retrieved during the study. There are two graphs for each ventilation district. The first graph shows the progressive annual dose projected for each ventilation district for the year based on the actual exposure to date. These figures help to predict problem areas and enable preventative action. The maximum levels shown in the figures portrayed in the progressive monthly dose figures, vary greatly in some cases and will be explained per graph.

An increase can be seen in the dose during April in four of the six areas included in this study. It can be assumed that the large earthquake that occurred at a shaft in the local area might be responsible for this rise. This seismic event occurred on the 9th of March 2005 at 12:15:31pm and reached 5.3 on the Richter scale. Changes in conditions underground are almost inevitable in response to the large amount of energy released into the system by such an event.

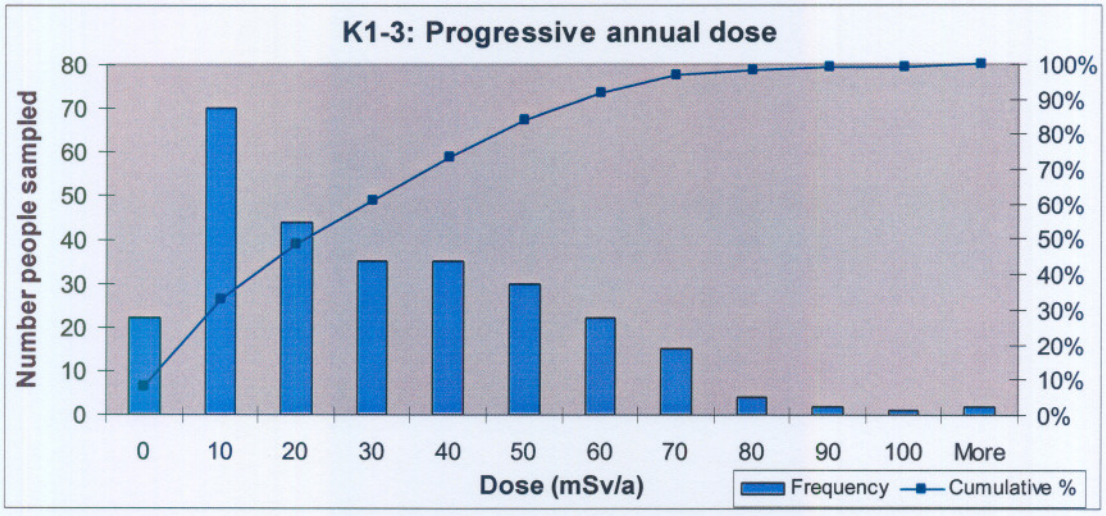

Figure 4.1: Projected annual dose for K1-3 for 2005.

In the figure above, the cumulative percentage represents the progressive frequency of employees exposed to a range of radiation within the ventilation district. The number of people exposed to higher dosage levels is very low, even though there is an even distribution over the whole dose range. It is estimated that 85 % of the employees monitored in this area are exposed to less than 50 mSv/a. The following figure shows the monthly exposures for this area.

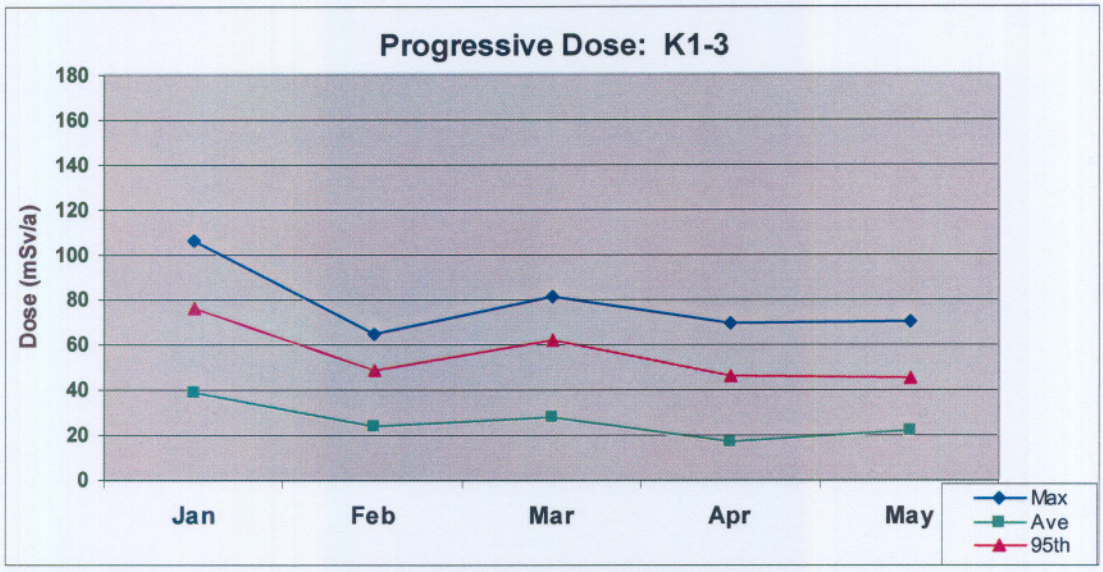

Figure 4.2: Progressive dose exposure in K1-3 for the period January to May 2005.

The figure clearly shows a steady decrease from January through to May, with the average reading of 38.5 mSv during January declining to 21.5 mSv in May, which is well below the threshold. These results were satisfactory as there was a 55.7 % decrease in average exposure. The maximum readings and 95th percentile confidence level correlates with the average readings for this area. Monitoring will continue until the area is no longer a concern. The slight increase during March may be a result of seasonal changes, which influences barometric pressure and temperature, and therefore gas flow and release underground.

The results of K1-6, the next monitored area, can be seen in the two graphs below. In Figure 4.3, it can be seen that all the employees are predicted to be exposed to dosage levels between 0 mSv/a and 35 mSv/a. This is far below the maximum allowable dosage of 50 mSv/a.

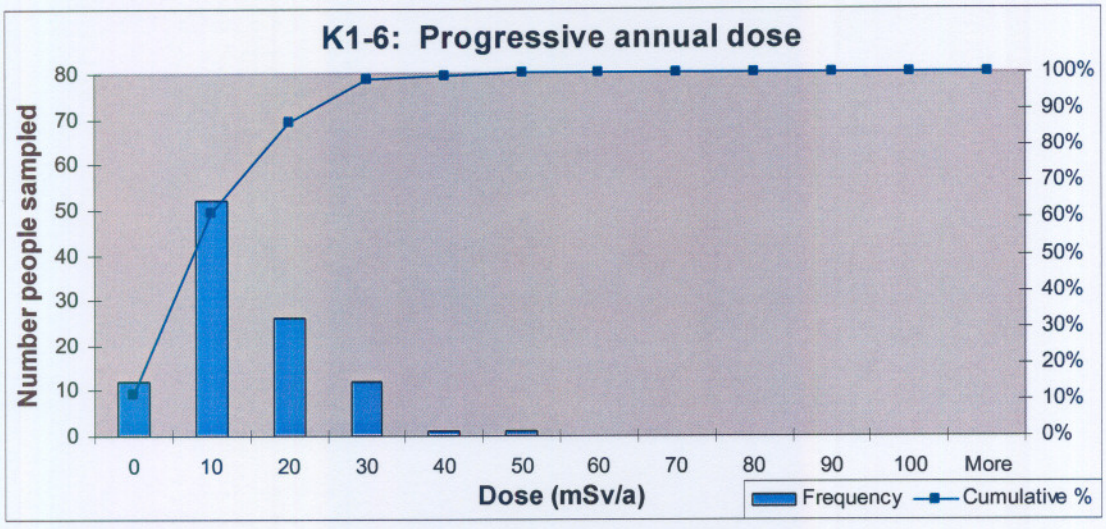

Figure 4.3: Projected annual dose for K1-6 for 2005.

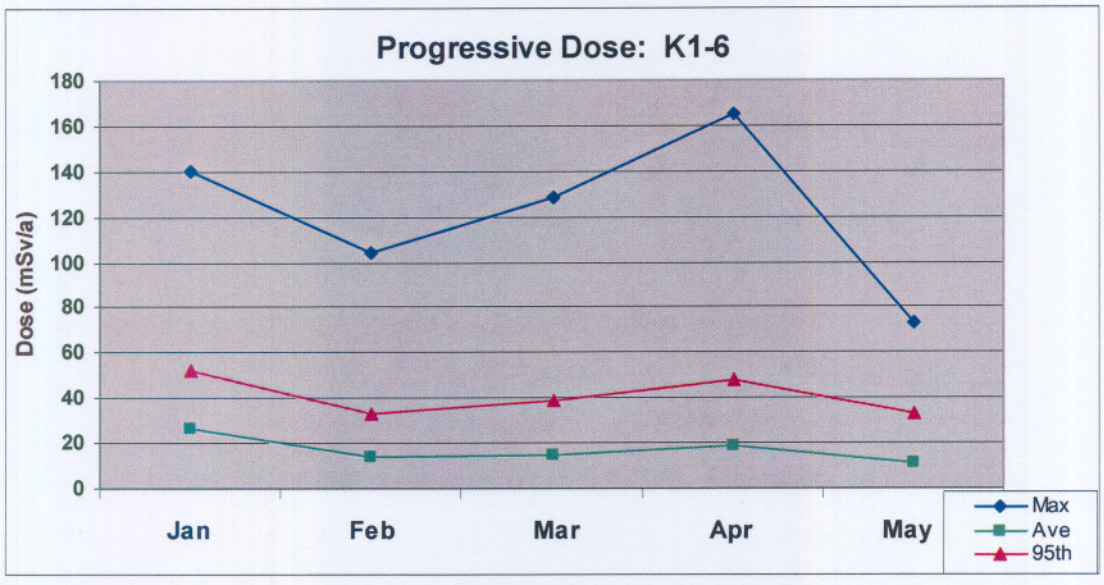

Figure 4.4: Progressive dose exposure in K1-6 for the period January to May 2005.

Following the 'hot spot' area classification in December, remedial actions were implemented, as described in section 4.2, leading to a decrease in January. Figure 4.4 reveals a steady decline in the average and 95th percentile values over the monitoring period, despite variation in maximum values. Average exposure declined with 15.2 mSv, from 25.8 mSv to 10.6 mSv representing a 41.1 % decline in exposure. The rise in April, from 13.9 mSv to 18.1 mSv, might be as a result of the seismic event that took place on 9 March 2005, and reached 5.3 on the Richter scale. The large amount of energy that is released as a result of an earthquake is bound to have an environmental impact. Again, seasonal changes may also contribute to deviations in the maximum values during March and April.

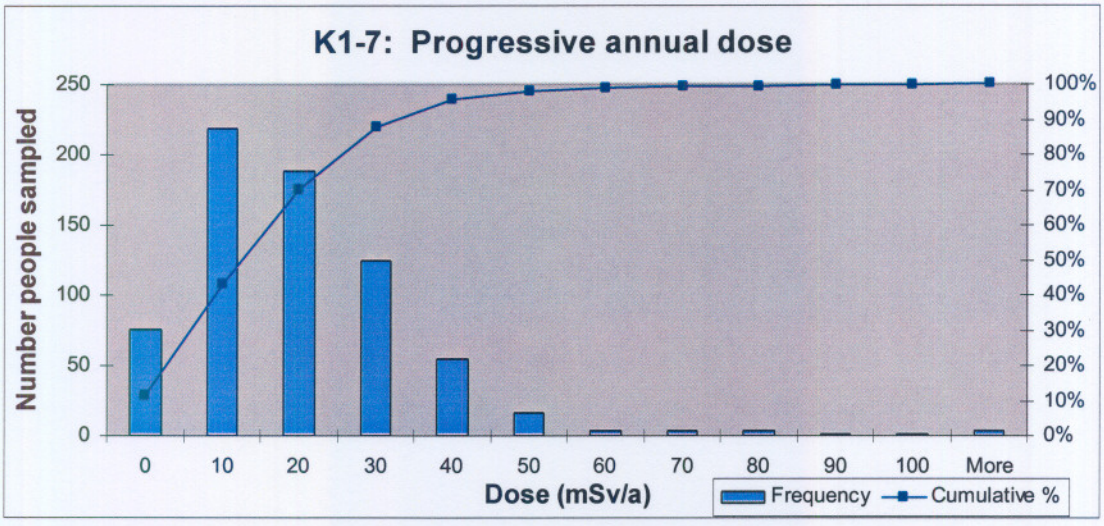

Figure 4.5: Projected annual dose for K1-7 for 2005.

Currently, the projected annual dose for the greater number of people whose exposure was measured in K1-7, lies below the 50 mSv/a limit with most exposed employees falling between 10 – 20 mSv/a. The distribution in this figure reduces with increased dosage. Around 95 % of all the employees in this area are at no or low risk of being exposed to radiation levels higher than the threshold. Exposure for the bulk of the employees, about 425 of those monitored, is projected to be below 30 mSv/a by December 2005.

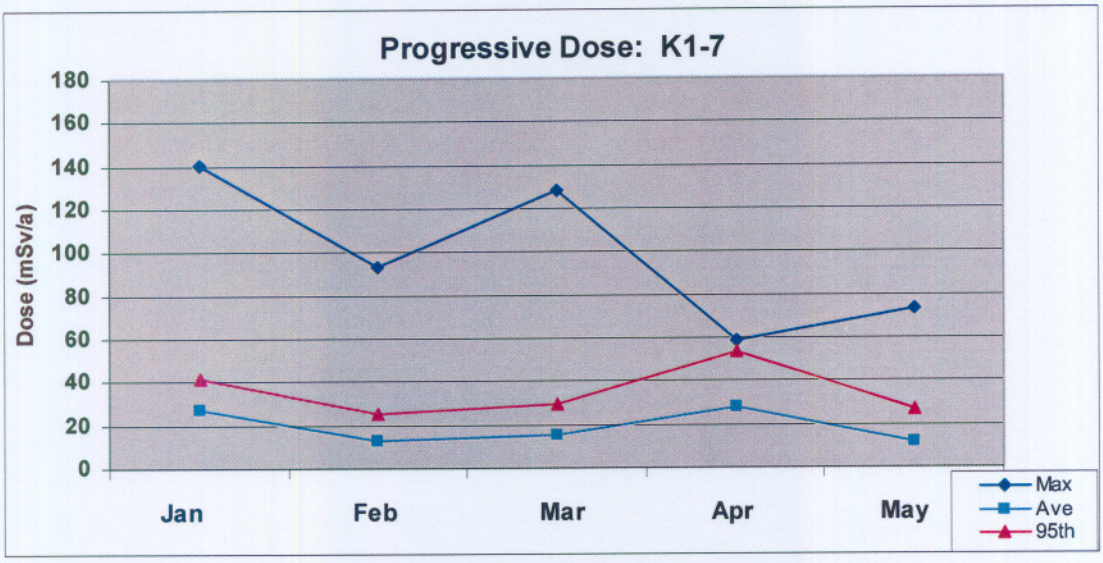

Figure 4.6: Progressive dose exposure in K1-7 for the period January to May 2005.

The increase in April corresponds with the abovementioned areas' increase in the same month, which can again be contributed to the seismic event. The general trend in this area remained relatively stable, only decreasing slightly in February and May. The maximum dose readings varied greatly, but decreased drastically in February and again in April. Both these declines were followed by an increase in the next cycle, although the May results are much lower than in January.

This maximum value represents only one reading that is not representative of the whole area. It varies mostly because of the inconsistent levels of radon gas underground. The average declined with 41.3 %, from 26.9 mSv in January, to 11.1 mSv in May. The readings continued to drop in the following cycle, of which the results are not shown as it falls outside the scope of this study. Most of the areas continue to be closely monitored.

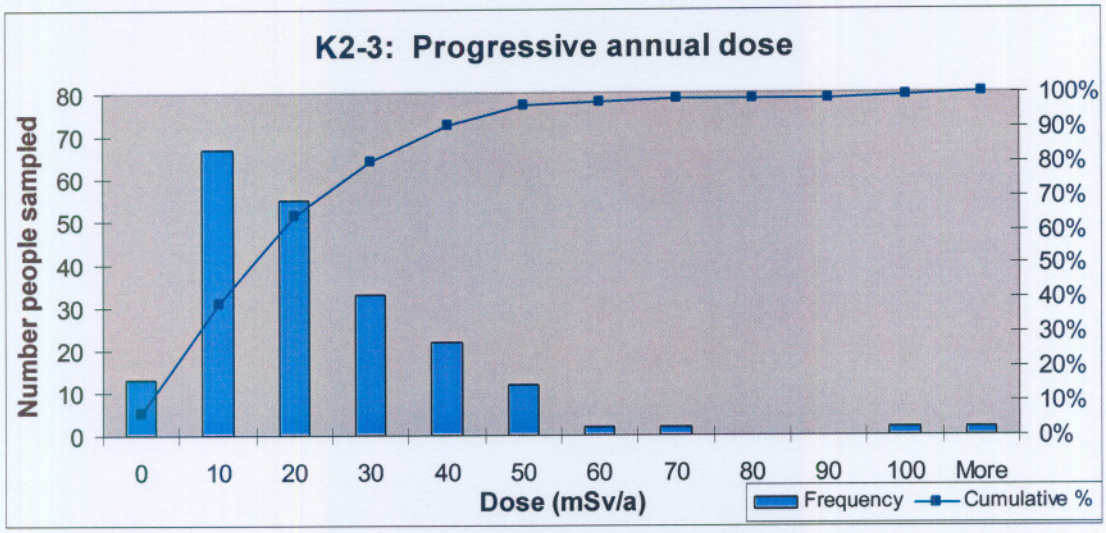

Figure 4.7: Projected annual dose for K2-3 for 2005.

In figure 4.7 it can be seen that the bulk percentage of the population is below 50 mSv, with most of the workers projected to be exposed between 10 to 20 mSv/a, and to a lesser extent between 30 to 40 mSv/a. It is estimated that the exposure of 95 % of the people are below 45 mSv/a. The overall distribution of the population looks satisfactory.

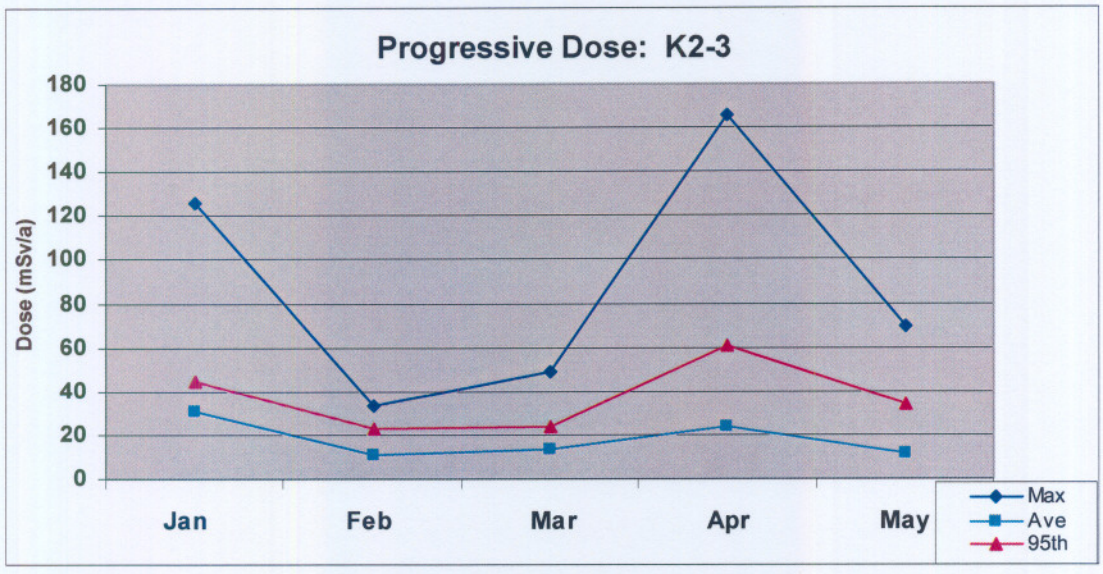

Figure 4.8: Progressive dose exposure in K2-3 for the period January 2005 to May 2005.

The figure above also shows the rise in dose during April, while the general trend is a decline in dose values. In K2-3, as can be seen from figure 4.8, the average decreased from 30.5 mSv during January to 11.1 mSv in May. This is a decrease of 36.4 %. The high maximum value represents only one reading and is not representative of the average maximum levels found in this area. When the 3 maximum readings are discarded, the average drops from 23.7 mSv to 19.1 mSv.

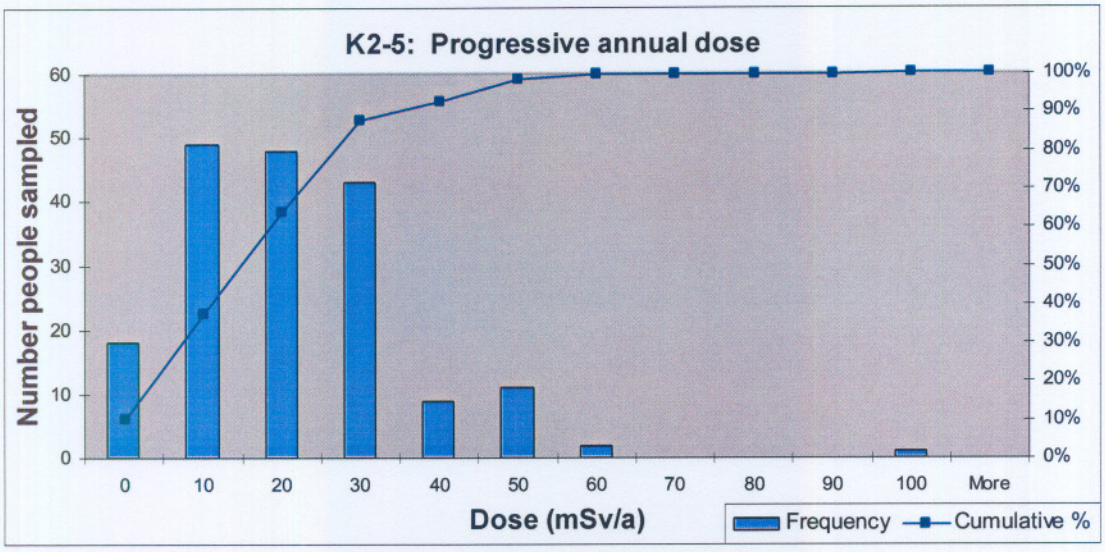

Figure 4.9: Projected annual dose for K2-5 for 2005.

In K2-5, of which the results can be seen in figure 4.9 and figure 4.10, it can be seen that exposure decreased. In figure 4.9, it can be seen that 90 % of the people will be exposed between 10 to 30 mSv/a. There is a probability that around 10 people will be exposed to 50 mSv/a, and 3 people will be exposed to levels above 50 mSv/a. Preventative measures can and has been put in place as described in section 4.2.

The following figure, figure 4.10, shows actual exposure for January through to May.

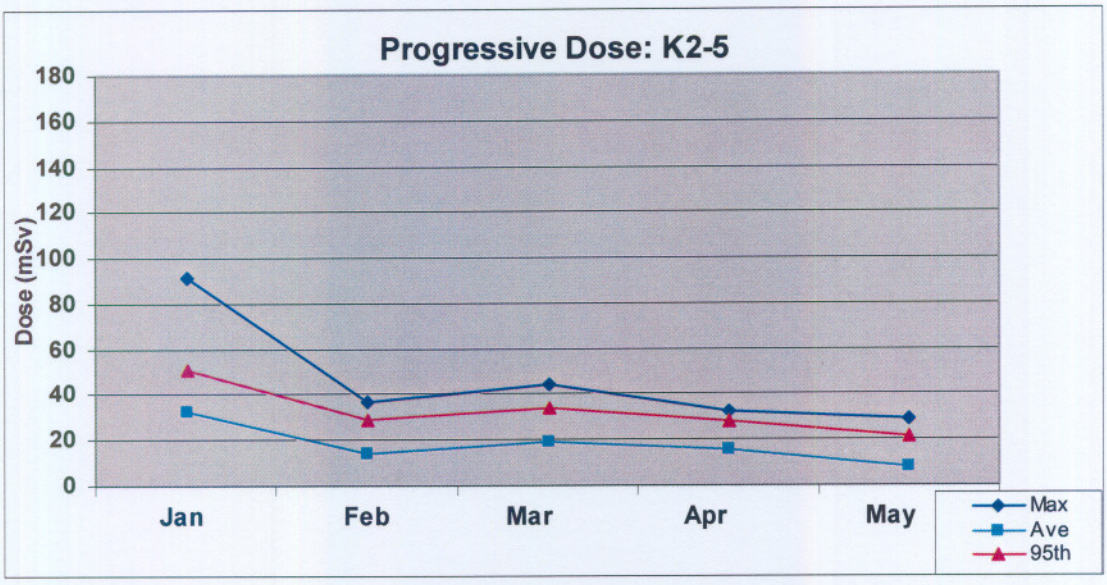

Figure 4.10: Progressive dose exposure in K2-5 for the period January to May 2005.

A dramatic decrease in dose readings in this area can be seen from the above figure. Exposure fell with 24,7% from 31.6 mSv in January to 7.8 mSv in May. Pro-active steps that were taken, as described in section 4.2, were effective in improving ventilation and decreasing exposure to radon gas. Here both the average and maximum values are below 10 mSv/a and 30 mSv/a respectively, which is well below the annual threshold value of 50 mSv/a.

90 % of all the projected readings in K2-6, as portrayed in figure 4.11, lies below 20 mSv/a. Less than 10 employees are at risk of over-exposure and there is a 70 % chance that these employees will not be exposed above the threshold. This percentage was calculated at a 95 % confidence level.

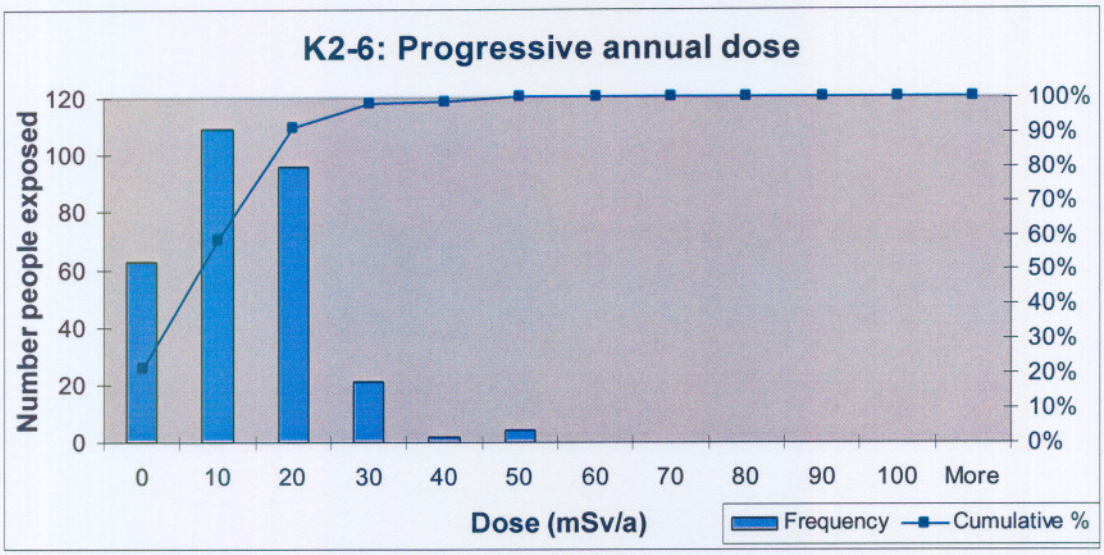

Figure 4.11: Projected annual dose for K2-6 for 2005.

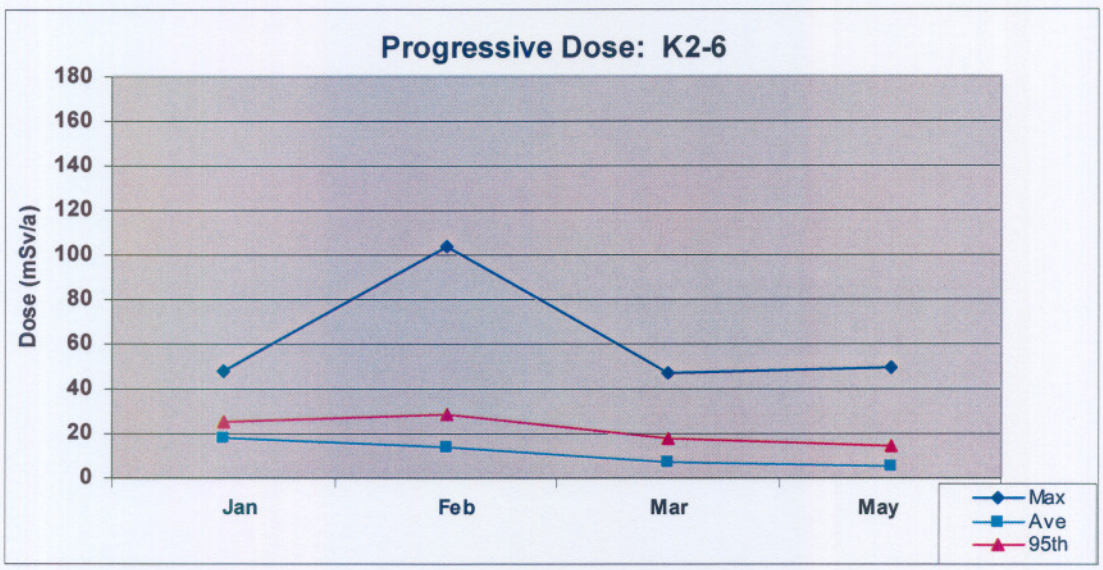

Figure 4.12: Progressive dose exposure in K2-6 for the period January 2005 to May 2005.

Figure 4.12 shows that the area K2-6 does not have readings as high as most of the other areas monitored. As in the other areas a general decrease can be seen here from 17.1 mSv in January to 5 mSv in May. The maximum value in this area for February was very high, but this was a single exposure and does not correspond with the rest of the readings for

that month. For both March and May, the average and 95th percentile values are below 20 mSv/a. There were no samples taken in April due to administrative reasons.

In conclusion, figure 4.13 shows the total distribution for the whole of the shaft, as monitored from January 2005. The downward trend in dosage can be clearly seen.

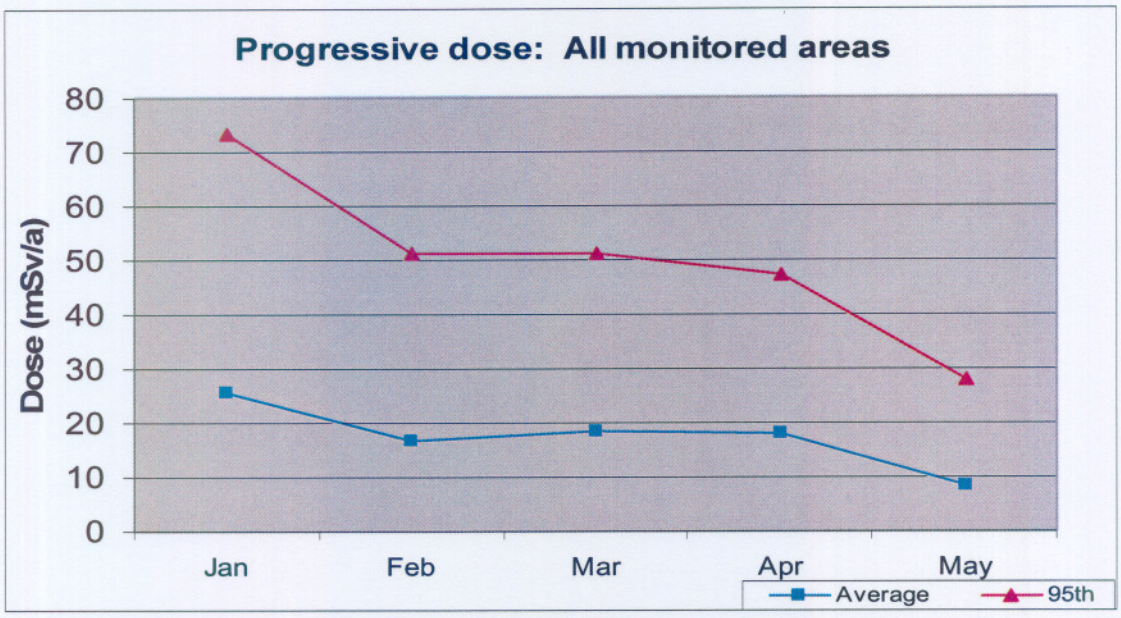

Figure 4.13: Progressive dose exposure for the whole shaft for the period January 2005 to May 2005.

4.2. DISCUSSION

All but two areas, K1-3 and K1-7 were declassified as red areas. During the December cycle the readings were unusually high, contributing to concern over ionising radiation underground. Kop 1 was an area of major concern, since K1-3 had an average of 54 mSv/a, with 58 % of the readings above the 50 mSv/a limit. K1-6 did not pose any problems, since it had an average reading of 17.6 mSv/a and no employees were exposed above the recommended limit. This was a decrease in the readings obtained from the December cycle, which was an average of 35.8 mSv/a.

In Kop 2 the area K2-5 had one very high dose reading, 74.4 mSv/a, with an average reading of 26 mSv/a. K2-6 had an average of 13.6 mSv/a and no readings above 33 mSv/a. The table below shows the average readings that were higher than the allowable threshold of 50 mSv/a in each ventilation district, for each cycle that was monitored.

Table 4.1: % Readings > 50 mSv/a per ventilation district

	% > 50 mSv/a				
VENTILATION DISTRICT	**JAN**	**FEB**	**MAR**	**APR**	**MAY**
K1-3	39.20	4.60	18.20	3.80	5.13
K1-6	0.00	0.00	0.00	0.00	0.00
K1-7	3.10	1.20	2.70	10.00	1.23
K2-3	4.40	0.00	0.00	8.90	0.00
K2-5	12.50	0.00	0.00	0.00	0.00
K2-6	0.00	2.90	1.00	-	0.00

The problematic workplaces (for example 73 AE 29 raise) were identified, and action plans were implemented based on investigation findings concerning these workplaces. Approximately 85 % of the remedial actions were taken during January, and the last 15 % during February.

73AE 29 Raise

The ventilation door was sealed at the 30S crosscut where the two haulages connect. This strategy was aimed at reducing polluted air, as a result of air moving through old, radio-active areas, which was pulled from another shaft. The amount of contaminated air that enters the shaft was reduced from $5m^3/s$ to only a drift through the sealing of the door.

44BW3 15S Development

A ventilation hole was drilled to allow access of clean air. There was also a leakage at 47BW1 16N crosscut access 1 that was sealed off. All the box holes were checked and sealed to prevent unwanted air from entering the area. An additional ventilation wall was also constructed at 44BW3 16A crosscut.

47BW1 16A Raise

This area has been depleted. All equipment was therefore removed when production stopped. A wall was built and sealed at 47BW1 16B crosscut access 1. An open 570 mm pipe and a steel door were installed at 47BW1 16B access 2. A wall was also constructed at 44BW3 16A crosscut to seal the area.

44BW3 16C Raise

44BW3 16A was sealed, increasing the quantity of air to this raise line. Axial fans were removed and a regulator installed at 47BW1 16B access 2 to improve ventilation. Leakage of air at 47BW1 16N crosscut access 1 was sealed off. All the box holes were checked and sealed in 47BW1 16N crosscut. At 44BW3 16A crosscut an additional wall was constructed.

44BW3 16BN Raise

47BW1 16B raise 1 was sealed off. The air leak at 47BW1 16N crosscut access 1 was also sealed. All the box holes were checked and sealed off in 47BW1 16N crosscut and a wall was constructed at 44BW3 16A crosscut to control air flow.

47BW 15N Raise

Vamping was in progress and walls were built at the required places.

44BW1 9S Raise

The holing of 47BW 14N raise and subsequent changeover to upcast 9 raise will reduce the airflow through old areas before becoming available for 9S raise. It will now take air in directly from the main 47BW haulage. The construction of a ventilation hole was proposed at 47BW 8N crosscut.

44BW1 10N Raise

Walls were built at appointed places in the 47BW area to reduce airflow in old areas before entering 10N raise. This raise is now worked out, and vamping is in progress.

70SW1 14N Raise

Doors were constructed at the appointed places, fire doors were closed and air was properly regulated in the 68SW1 area. The down dip panels were ventilated with fans to ensure airflow. This area is now also depleted.

44BW1 14N Raise

A hole was made in the wall at 44BW1 15N access 1 to improve airflow and a second 45kW fan was started at 42 incline to improving ventilation conditions.

62 SW3 22 Raise 8, 9

Air Utilization (AU's) had to be maintained at optimal levels to reduce air travelling time. Instruction was given to ensure that the return airway (RAW) ventilation hole at raise 9 remained open at all times.

62 SW4 22 Raise2, 3

AU's were maintained at optimal levels to reduce air travelling time to at least $4m^2$. RAW restriction at number 3 intake airway raise 2 was opened to at least $4m^2$. Only single sided mining can take place in this areas as only $12m^3/s$ of air is available to ventilate the raise.

In Kop 2 the averages in K2-3 fluctuated, first declining until February, then increasing steadily increasing up until April and then dropping suddenly to an average of 8 mSv/a. Even so, the amount of people exposed to doses above the 50 mSv/a limit remained low. K2-4 was investigated in April and May, but showed no reason for concern. Exposure levels in K2-5 declined steadily after the action plans employed. Exposure in this area currently averages at 5.32 mSv/a and none of the employees are exposed above the limit. K2-6 showed a similar pattern, and is currently on a 2.8 mSv/a average with nobody over the dose limit.

During April and May, K1-2 was sampled in addition to the rest, having an average reading of 20.6 mSv/a and 29.7 mSv/a respectively. During April, only 10.5 % of K1-2's readings were above the 50 mSv/a threshold. K1-3 showed a steady decline in readings during the cycles up to May, and had an average of 18.6 mSv/a and only 5 % readings above the threshold to date. K1-4 was sampled only as routine measurement during April, but there was no indication of radiation problems warranting further action.. The readings in K1-6 also declined up to an average of 3.5 mSv/a, with no readings exceeding the limit. In K1-7 the readings declined, but showed an interesting incline in April, from an average 16 mSv/a to an average of 27.5 mSv/a in April, and then down again in May to 8.18 mSv/a. This could be a result of seasonal changes around that time of year or uranium rich ore that was mined.

The following additional action plans were implemented, before the next monitoring cycle started:

62 SW3 22 raise 8, 9

Air Utilization Indexes (AUI's) was maintained at optimal levels to reduce air travelling time and to in effect lower the amount of contaminated air. The return airway (RAW) ventilation hole at raise 9 was to remain open at all times.

62 SW4 22 raise 2, 3

The AUI's must be maintained at optimal levels to reduce air travelling time to at least $4m^2$. This will help to lower the amount of contaminated are in the area. The RAW restriction at 3 intake airway, raise 2 was opened to at least $4m^2$. Only single sided mining was allowed as only $12m^3/s$ of air is available to ventilate the raise.

The second special sampling cycle, during February 2005, had very satisfactory results. Kop 1 showed major improvement, with an average reading of 23.1 mSv/a, and only 4.6 % readings above 50 mSv/a. K1-6 again showed no problems with an average of 9 mSv/a and no readings above 50 mSv/a. K1-7's average was 12 mSv/a and only 1.2 % of the readings were above 50 mSv/a. Kop 2 also improved drastically, while K2-3 had an average of 11.2 mSv/a with no readings above 50 mSv/a. K2-5's average was 14 mSv/a with no readings above 50 mSv/a. K2-6 had an average of 13.3 %, with 2.8 % of the readings above 50 mSv/a.

CHAPTER 5: CONCLUSIONS AND RECOMMENDATIONS

5.1 CONCLUSIONS

The database is complete and up to date. Reports were drawn up, statistical analysis was done and proper action plans were implemented to ensure a permanent improvement in the underground radiation conditions. The ongoing monitoring program will be able to run smoothly seeing that the basis is broad and correct, and only new sampling data needs to be added to the developed system. The five cycles that were closely monitored revealed an effective reduction in radiation problems and no problems are expected in the future (not taking into account geographical earth movement and occasional high uranium concentrations encountered in the rock). The hypothesis for this study was thus proven true.

It is possible that radon-gas exposure and dust inhalation have an accumulative effect to induce silicoses, tuberculosis and pneumoconiosis in mine workers. This is an area that has not been studied sufficiently and would surely deliver interesting and worthwhile results for future remedial action. The availability of this database can significantly contribute to further studies in these areas. The doses are much lower than it was at the start of this study and below the first target of 50 mSv/a. Future remedial action includes reducing the current exposure further to below 20 mSv/a over 5 consecutive years.

5.2 RECOMMENDATIONS

Following the study and analysis of the results, it is recommended that:

- the situation be monitored closely;
- the data basis be kept up to date;
- the areas checked regularly, and
- a progressive and continuous risk assessment be done,

This will ensure that the implemented changes that were brought on are will stay intact and will function properly on a continuous base. It will also ensure that a safe and healthy working environment will be maintained as far as possible.

CHAPTER 6: ARTICLE

6.1 JOURNAL OF THE MINE VENTILATION SOCIETY: AUTHOR GUIDELINES

Preparing a two column paper with MS Word for Windows

A.B. Author & C. Author
University of SA, South Africa

B. Unknown
Second Institute, Japan

ABSTRACT: Authors of papers to proceedings have to type these in a form suitable for direct photographic reproduction by the publisher. In order to ensure uniform style throughout the volume, all the papers have to be prepared strictly according to the instructions set below. A laser printer should be used to print the text. The publisher will reduce the camera-ready copy to 75% and print it in black only. For the convenience of the authors template files for MS Word 6.0 (and higher) are provided.

1 GENERAL INSTRUCTIONS

1.1 *Type area*

The text should fit exactly into the type area of 187 × 272 mm (7.36" × 10.71"). For correct settings of margins in the Page Setup dialog box (File menu) see Table 1.

1.2 *Typefont, typesize and spacing*

Use Times New Roman 12 point size and 13 point line spacing (Standard;text tag). Use roman type except for the headings (Heading tags), parameters in mathematics (not for log, sin, cos, ln, max., d (in d*x*), etc), Latin names of species and genera in botany and zoology and the titles of journals and books which should all be in italics. Never use bold, except to denote vectors in mathematics. Never underline any text. Use the small font (10 points on 11 points) for tables (Table tags), figure captions (Figure caption tag) and the references (Reference text tag).

Never use letterspacing and never use more than one space after each other.

2 GETTING STARTED

2.1 *Preparing the new file with the correct template*

Copy the template file MVSpapera4.dot (if you print on A4 size paper) to the template directory. This directory can be found by selecting the Tools menu, Options and then by tabbing the File Locations. When the Word programme has been started open the File menu and choose New. Now select the template MVSpapera4.dot (see above). Start by renaming the document by clicking Save As in the menu Files. Give a file name of your paper in *.doc format. Now you can type your paper, or copy the old version of your paper onto this new formated file.

2.2 *Copying old text onto new file*

Open your old file and the new file. Switch between these two with the Window menu. Select all text of the old file (excluding title, authors, affiliations and abstract) and paste onto bottom of new file, after having deleted the word INTRODUCTION (see also section 2.5). Check the margin setting (Page Setup dialog box in File menu) and column settings (see Table 1 for correct settings). After this copy the texts which have to be placed in the frames (see sections 2.3 and 2.4). In order to avoid disruption of the text and frames, copy these texts paragraph by paragraph without including the first word (which includes the old tag). It is best to first retype the first words manually and then to paste the correct text behind. When the new file contains all the text, the old tags in the text should be replaced by the new Balkema

Table 1. Margin settings for A4 size paper and letter size paper.

Setting	A4 size paper		Letter size paper	
	cm	inches	cm	inches
Top	1.2	0.47"	0.32	0.13"
Bottom	1.3	0.51"	0.42	0.17"
Left	1.15	0.45"	1.45	0.57"
Right	1.15	0.45"	1.45	0.57"
All other	0.0	0.0"	0.0	0.0"
Column width*	9.0	3.54"	9.0	3.54"
Column spacing*	0.7	0.28"	0.7	0.28"

* Column dialog box in Format menu.

tags (see section 3). Before doing this apply automatic formatting (AutoFormat in Format menu).

2.3 *Title, author and affiliation frame*

Place the cursor on the T of Title at the top of your newly named file and type the title of the paper in lower case (no caps except for proper names). The title should not be longer than 75 characters). Delete the word Title (do not delete the paragraph end). Place the cursor on the A of A.B.Author(s) and type the name of the first author (first the initials and then the last name). If any of the co-authors have the same affiliation as the first author, add his name after an & (or a comma if more names follow). Delete the words A.B. Author etc. and place the cursor on the A of Affiliation. Type the correct affiliation (Name of the institute, City, State/Province, Country). Now delete the word Affiliation. If there are authors linked to other institutes, place the cursor at the end of the affiliation line just typed and give a return. Now type the name(s) of the author(s) and after a return the affiliation. Repeat this procedure until all affiliations have been typed.

All these texts fit in a frame which should not be changed (Width: Exactly 187 mm (7.36"); Height: Exactly 73 mm (2.87") from top margin; Lock anchor).

2.4 *Abstract frame*

If there are no further authors place the cursor one space behind the word ABSTRACT: and type your abstract of not more than 150 words. The top of the first line of the abstract will be 73 mm (2.87") from the top of the type area. The complete abstract will fall in the abstract frame, the settings of which should also not be changed (Width: Exactly 187 mm (7.36"); Height: Automatic; Vertical 73 mm (2.87") from margin; Lock anchor).

2.5 *First line of text or heading*

If your text starts with a heading, place the cursor on the I of INTRODUCTION and type the correct text for the heading. Now delete the word INTRODUCTION and start with the text after a return. This text should have the tag First paragraph.

If your text starts without a heading you should place the cursor on the I of INTRODUCTION, change the tag to First paragraph and type your text after deleting the word INTRODUCTION.

3 LAYOUT OF TEXT

3.1 *Text and indenting*

Text is set in two columns of 9 cm (3.54") width each with 7 mm (0.28") spacing between the columns. All text should be typed in Times New Roman, 12 pt on 13 pt line spacing except for the paper title (18 pt on 20 pt), author(s) (14 pt on 16 pt), and the small text in tables, captions and references (10 pt on 11 pt). All line spacing is exact. Never add any space between lines or paragraphs. When a column has blank lines at the bottom of the page, add space above and below headings (see opposite column).

First lines of paragraphs are indented 5 mm (0.2") except for paragraphs after a heading or a blank line (First paragraph tag).

3.2 *Headings*

Type primary headings in capital letters roman (Heading 1 tag) and secondary and tertiary headings in lower case italics (Headings 2 and 3 tags). Headings are set flush against the left margin. The tag will give two blank lines (26 pt) above and one (13 pt) beneath the primary headings, 1½ blank lines (20 pt) above and a ½ blank line (6 pt) beneath the secondary headings and one blank line (13 pt) above the tertiary headings. Headings are not indented and neither are the first lines of text following the heading indented. If a primary heading is directly followed by a secondary heading, only a ½ blank line should be set between the two headings. In the Word programme this has to be done manually as follows: Place the cursor on the primary heading, select Paragraph in the Format menu, and change the setting for spacing after, from 13 pt to 0 pt. In the same way the setting in the secondary heading for spacing before should be changed from 20 pt to 7 pt.

3.3 *Listing and numbering*

When listing facts use either the style tag List signs or the style tag List numbers.

3.4 *Equations*

Use the equation editor of the selected word processing programme. Equations are not indented (Formula tag). Number equations consecutively and place the number with the tab key at the end of the line, between parantheses. Refer to equations by these numbers. See for example Equation 1 below:

From the above we note that $\sin\theta = (x + y)z$ or:

$$K_I = \left(1 - \frac{R^2\tau}{c_a + \nu \tan\delta}\right)^4 k_1 \qquad (1)$$

where c_a = interface adhesion; δ = friction angle at interface; and k_1 = shear stiffness number.

For simple equations in the text always use superscript and subscript (select Font in the Format menu). Do not use the equation editor between text on same line.

3.5 *Tables*

Locate tables close to the first reference to them in the text and number them consecutively. Avoid abbreviations in column headings. Indicate units in the line immediately below the heading. Explanations should be given at the foot of the table, not within the table itself. Use only horizontal rules: One above and one below the column headings and one at the foot of the table (Table rule tag: Use the Shift-minus key to actually type the rule exactly where you want it). For simple tables use the tab key and not the table option. Type all text in tables in small type: 10 on 11 points (Table text tag). Align all headings to the left of their column and start these headings with an initial capital. Type the caption above the table to the same width as the table (Table caption tag). See for example Table 1.

3.6 *Figure captions*

Always use the Figure caption style tag (10 points size on 11 points line space). Place the caption underneath the figure (see Section 5). Type as follows: 'Figure 1. Caption.' Leave about two lines of space between the figure caption and the text of the paper.

3.7 *References*

In the text, place the authors' last names (without initials) and the date of publication in parentheses (see examples in Section 5). At the end of the paper, list all references in alphabetical order underneath the heading REFERENCES (Reference heading tag). The references should be typed in small text (10 pt on 11 pt) and second and further lines should be indented 5.0 mm (0.2") (Reference text tag). If several works by the same author are cited, entries should be chronological:

Larch, A.A. 1996a. Development ...
Larch, A.A. 1996b. Facilities ...
Larch, A.A. 1997. Computer ...
Larch, A.A. & Jensen, M.C. 1996. Effects of ...
Larch, A.A. & Smith, B.P. 1993. Alpine ...

3.7.1 *Typography for references*

Last name, First name or Initials (ed.) year. *Book title*. City: Publisher.

Last name, First name or Initials year. Title of article. *Title of Journal* (series number if necessary) volume number (issue number if necessary): page numbers.

3.7.2 *Examples*

Grove, A.T. 1980. Geomorphic evolution of the Sahara and the Nile. In M.A.J. Williams & H. Faure (eds), *The Sahara and the Nile*: 21-35. Rotterdam: Balkema.

Jappelli, R. & Marconi, N. 1997. Recommendations and prejudices in the realm of foundation engineering in Italy: A historical review. In Carlo Viggiani (ed.), *Geotechnical engineering for the preservation of monuments and historical sites; Proc. intern. symp., Napoli, 3-4 October 1996*. Rotterdam: Balkema.

Johnson, H.L. 1965. Artistic development in autistic children. *Child Development* 65(1): 13-16.

Polhill, R.M. 1982. *Crotalaria in Africa and Madagascar*. Rotterdam: Balkema.

3.8 *Notes*

These should be avoided. Insert the information in the text. In tables the following reference marks should be used: *, **, etc. and the actual footnotes set directly underneath the table.

3.9 *Conclusions*

Conclusions should state concisely the most important propositions of the paper as well as the author's views of the practical implications of the results.

4 PHOTOGRAPHS AND FIGURES

Number figures consecutively in the order in which reference is made to them in the text, making no distinction between diagrams and photographs. Figures should fit within the column width of 90 mm (3.54") or within the type area width of 187 mm (7.36").

Figures, photographs, etc. should be in black only. Paste copies of the same size onto the typescript where you want them to appear in the text. Do not place them sideways on a page; however if this cannot be avoided, no other text (except the figure caption) should appear on that page. Figures, etc. should not be centered, but placed against the left margin. Leave about two lines of space between the actual text and figure (including caption). Never

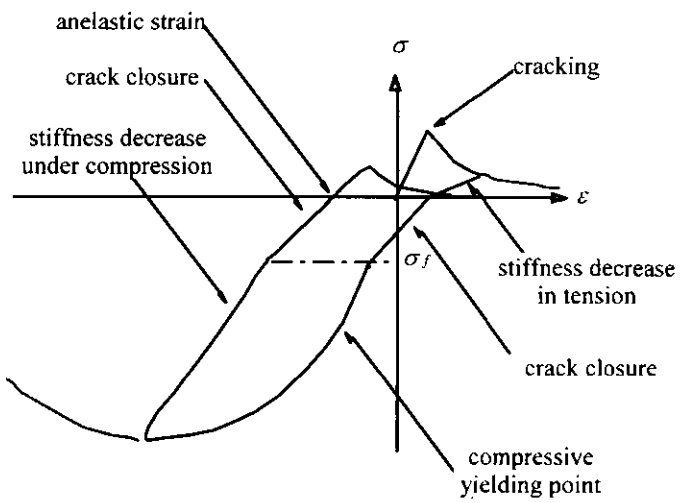

Figure 1. Caption of a typical figure. Photographs will be scanned by the printer. Always supply original photographs.

place any text next to a figure. Leave this space blank. The most convenient place for placing figures is at the top or bottom of the page. Avoid placing text between figures as readers might not notice the text. Line drawings (as well as photographic reproductions of these) should be in black (not grey) on white. Keep in mind that everything will be reduced to 75%. Therefore, 9 point should be the minimum size of the lettering. Lines should preferably be 0.2 mm (0.1") thick. Keep figures as simple as possible. Avoid excessive notes and designations.

Photographs should be black and white, with good contrast and on glossy paper. Photographic reproductions cut from books or journals, photocopies of photographs and screened photographs are unacceptable. The proceedings will be printed in black only. For this reason avoid the use of colour in figures and photographs. Colour is also nearly always unnecessary for scientific work.

5 PREFERENCES, SYMBOLS AND UNITS

Consistency of style is very important. Note the spacing, punctuation and caps in all the examples below.

- *References in the text:* Figure 1, Figures 2-4, 6, 8a, b (not abbreviated)
- *References between parentheses:* (Fig. 1), (Figs 2-4, 6, 8a, b) (abbreviated)
- USA / UK / Netherlands / the Netherlands *instead of* U.S.A. / U.K. / The Netherlands
- Author & Author (1989) *instead of* Author and Author (1989)
- (Author 1989a, b, Author & Author 1987) *instead of* (Author, 1989a,b; Author and Author, 1987)
- (Author et al. 1989) *instead of* (Author, Author & Author 1989)
- *Use the following style:* (Author, in press); (Author, in prep.); (Author, unpubl.); (Author, pers. comm.)

Always use the official SI notations:

- kg / m / kJ / mm *instead of* kg. (Kg) / m. / kJ. (KJ) / mm.;
- 20°16′32″SW *instead of* 20° 16′ 32″ SW
- 0.50 *instead of* 0,50 (*used in French text*); 9000 *instead of* 9,000 *but if more than* 10,000: 10,000 *instead of* 10000
- ^{14}C *instead of* C^{14} / C-14 *and* BP / BC / AD *instead of* B.P. / B.C. / A.D.
- × 20 *instead of* ×20 / X20 / x 20; 4 + 5 > 7 *instead of* 4+5>7 *but* –8 / +8 *instead of* – 8 / + 8
- e.g. / i.e. *instead of* e.g., / i.e.,

6 SUBMISSION OF MATERIAL TO THE EDITOR

The camera-ready copy of the complete paper printed on a high resolution printer on one side of the paper as well as two copies of the paper should be sent to the editor. Include the original photographs. Check whether the paper looks the same as this sample: Title at top of first page in 18 points, authors in 14 points and all other text in 12 points on 13 points line space, except for the small text (10 point on 11 point line space) used in tables, captions and references. Also check if the type width is 187 mm (7.36"), the column width 90 mm (3.54"), the page length is 272 mm (10.71") and that the space above the Abstact is exactly as in the sample. Write your name and the shortened title of the paper in pencil in the bottom margin of each page and number the pages correctly.

7 DEADLINE

The above material should be with the editor before the deadline for submission. Any material received too late will not be published. Send the material by airmail or by courier well packed and in time. Be sure that all pages are included in the parcel.

6.2 JOURNAL OF THE MINE VENTILATION SOCIETY: ARTICLE

A radiation monitoring program in a South African gold mine

A. van Schalkwyk & F.C. Eloff
North-West University, South Africa

L. du Toit
Anglogold Ashanti, Vaal River

Legislation requires the monitoring of mine employees for radiation exposure. Alpha-radiation, emitted as result of radon gas, which is a natural decay product of uranium (a heavy metal found in all rock and soil), is the main focus of this study. The objective of the study was to establish a controlled monitoring program through which results could be obtained, captured and studied. Five monitoring cycles were included in the study and comprehensive insight was obtained from the results. This made it possible to identify appropriate measures to reduce the high prevalence of exposure on this mine. Results were compiled in a database and then used to predict each employee's annual exposure. The personal history of each employee was also documented in the database for easy traceability. The results revealed that implemented measures were effective in reducing exposure to radiation in the mining environment.

1 INTRODUCTION

1.1 *Overview*

Radiation occurs naturally in the environment and will always be present on the earth. In addition, it can be seen as a form of energy that travels through space. Exposure to radiation can and must be controlled, since over or under exposure will have adverse health effects. Exposure to this natural radiation is referred to as background radiation (Hall, 2005). Thermal radiation, in the form of sunshine, is the type of radiation most known to man. Without it, life on earth would not be sustainable. Too much sunshine, on the other hand, has adverse health effects.

Low doses of radiaton derives naturally from space. Small emissions also originates from the earth. From here radiation enters the atmosphere in the form of radon, a natural gas which seeps from the earth's crust. This kind of radiation can cause damage to matter and in particular to living tissue. Hence the need to control excessive exposure to it.

This study primarily focuses on ionising radiation and more specifically alpha rays. Beta- and gamma rays also play an important role in ionising radiation, but alpha rays enter the body and cause internal exposure that leads to all kinds of pathology. Gamma and beta rays in contrast with alpha rays, are effectively diluted via ventilation before it becomes an exposure hazard.

Alpha rays, or α-disintegration, mainly occur in heavy nuclei and positively charged particles, where a helium nucleus (2 protons and 2 neutrons) is ejected. The energy range is between 4 and 5 x 10^6 eV. This large positive charge causes intense ionisation. The penetration depth ranges between 2 and 10 cm in air (Hall, 2005). α-rays will in general not penetrate the epidermis, as it can be stopped by a sheet of paper (Wymer, 2001). It can, however be inhaled through the air in an underground mining environment, because of dust particles contains the radio-active atoms. Beta rays originate from the emission of negatively charged high-speed electrons from the nucleus. The energy range is between zero and the maximum value of the parent nucleus. The ionisation is reasonably high, but lower than that of alpha-rays. Its penetrating depth is more than alpha rays; up to 3 m in air, and between 1 and 2 cm in water (Hall, 2005). It can penetrate human tissue up to 5 mm. Beta rays can be completely absorbed by thin metal (1 – 3 mm) or Perspex (10 mm) (Wymer, 2001).

Gamma rays revolve around excess energy emitted from the decaying nucleus. It consists of electromagnetic radiation of very short wavelength, and the energy levels can increase up to 3 x 10^6 eV (Hall, 2005). Again there is a decrease in the amount of ionisation from beta-rays to gamma-rays. Depending on the energy, gamma rays have extreme penetration depths. Thick concrete, or a heavy element, is therefore needed to absorb these rays. Gamma rays can pass completely through the human body (Wymer, 2001). Gamma-exposure in the mining industry is nevertheless primarily an external hazard due to the

rock face, stockpiles, localized concentrations and so forth, but not a great risk in the underground environment as such.

1.2 *Radiation and mining*

Mining is an ancient, multi-disciplinary industry, which requires arduous labour. Exposure to radiation is a major concern in this environment and it holds major financial implications for the industry. The concern for the most part is due to a lack of knowledge concerning the effects of radiation and constant exposure of employees to radiation.

Working with natural raw materials will always increase exposure to radiation, since all rock and soil contain uranium and thorium, which are both radioactive (Yamada, 2003, Gulson et al., 2005). Exposure to underground radiation fluctuates to a large extent, depending on the uranium concentration in the rock as well as the presence of radon gas. Exposure to this gas, radon-222, a decay product of uranium, is the main hazard (Hall, 2005).

Radon gas results in a natural process from the decay of uranium:

- The uranium-238 atom has 92 protons and 146 neutrons, and a half-life of 4.5 billion years. With decay, it emits an alpha particle, leaving behind thorium-234.
- Thorium-234 has 90 protons and 144 neutrons. It has a half-life of 24.5 days. A beta particle and a gamma ray are emitted on decay, leaving behind a protactinium-234 atom.
- The protactinium-234 atom has 91 protons and 143 neutrons. Its half life is 269 000 years. During decay it emits a beta particle and a gamma ray, leaving behind thorium-230.
- Thorium-230 has 90 protons and 140 neutrons, and a half life of 83 000 years. An alpha particle and a gamma ray are emitted during decay leaving behind radon-222 (Hall, 2005).

2 RADIATION AND THE ENVIRONMENT

There are principally two types of radiation, namely non-ionising radiation and ionising radiation.

2.1 *Non-ionising radiation*

The range of non-ionising radiation consists of ultraviolet, visible light, infrared, microwave and radio waves. It includes all forms of electromagnetic radiation that has enough energy to heat up biological material, predominantly due to the production of electrically charged particles, also called ions (Deonarine, 2005). This type of radiation will not be discussed in detail as it is not relevant in this study.

2.2 *Ionising radiation*

Ionising radiation can originate from both natural sources and artificial sources. It also contributes to the electromagnetic radiation spectrum in the form of x-rays and gamma-rays. These rays cause ionisation in matter and have harmful effects on both the human body and the environment (Mouton, 2005).

All matter is composed of atoms. Atoms that are chemically identical, but differ in mass are called isotopes. These unstable isotopes are referred to as radio nuclides. Radio nuclides' nuclei spontaneously rearrange into stable nuclei emitting excess energy in the process. Energy emitted during the reaction can take part in follow-up ionisation processes, consisting of alpha, gamma and beta rays that are emitted during the decay process. The stable nucleus is called the decay product. Radio-active decay occurs when a given amount of radioactive material decreases with time as the nuclei decays (Mouton, 2005, Hall, 2005).

2.3 *Uranium*

Uranium is a natural occurring radio-active element that is classified as a heavy metal, with atomic number 92 and atomic mass of 238.0289 g/mol. It primarily radiates alpha particles. The total amount of uranium on the earth remains relatively constant as a result of its long half-life. Uranium can however be moved around by processes like mining (Arfstein *et al.*, 2001, ATSDR, 1999). There is not much risk involved in the external radiation of uranium, since alpha particles do not have sufficient energy to penetrate the human body to an extent that would be harmful. This is because most of the absorbed uranium is excreted in the urine within a few days (ATSDR, 1999). In the occupational environment, however, these particles become airborne and can be inhaled, ingested and absorbed by the skin, because uranium exists in conjunction with dust in the air (Gulson *et al.*, 2005). Inhalation of the particles is dangerous, because it becomes lodged in the lungs turning it into an internal hazard.

2.4 *Radio-active decay*

This is the process through which a radioactive substance spontaneously breaks down into other atoms. Radioactive decay occurs when a given amount of radioactive material decreases with time as the nuclei decays. Note that alpha-, beta-, gamma- and x-radiation do not cause the body to become radioactive; most materials are radioactive up to a certain extent in their natural state (Hall, 2005). The element, uranium, for example, has no stable isotopes.

The half-life of an atom in a radio-active substance is the time it takes for half of the atoms to de-

cay, or the time it takes for the isotope to give off its radiation and become a different element. Uranium (Ur-238) will continue to decay until it becomes lead-206, which is a stable element (Hall, 2005, ATSDR, 1999).

3 PHYSIOLOGICAL EFFECTS OF RADIATION

3.1 *Overview*

Epidemiological studies have shown a correlation between prolonged exposure to ionising radiation and the occurrence of cancers, such as lung cancer. There is a great deal of controversy surrounding the effects of radiation in the mining industry, as well as the pathology resulting from irradiation (Kovalchuk *et al.*, 2003, McDiarmid, 2001, Lipsztein, 2001, Hnizdo *et al.*, 1997, Amandus & Costello, 1991). The prevalence of lung cancer seems to be higher in silicotics than nonsilicotics, regardless of the smoking habits of the mine workers (Amandus & Costello, 2001). Researchers first started to associate radon exposure with lung cancer when it became evident that a large population of underground mine workers suffered from lung cancer (Archer *et al.*, 2004, Anon, 2000).

Any dose, whether small or large, pose a health risk. Responses to low doses of radiation depend on a multiple number of factors, such as genetic and environmental influences, types of cells, proximity of cells to one another as well as the functional state and demands of affected organs. (Dawson *et al.*, 2005, Mothersill *et al.*, 2004, Verschaeve, 2004).

Exposure to radon gas underground is caused by ore or underground water. The radon daughters are metallic ions that attach themselves to airborne particulates (dust) and become inhaled. These particles become lodged in the lungs and cause internal radiation. The particles are transported from the lungs through the rest of the body and then excreted. Most of the particles are excreted within a few days, but some stay behind in the kidneys and bones (ATSDR, 1999).

Apoptosis, or programmed cell death, occurs as a result of ionisation. This is a consequence of DNA damage, genomic instability and damage to vital cellular components (Stone *et al.*, 2003, Offer *et al.*, 2002).

3.2 *Pathology*

Fibrosis plays a major role in the irradiation process, especially in the submucosa, muscular propria and subserosa of the lung, skin, muscle, liver and gastrointestinal tract. Changes in the cellular organelles include cellular and mitochondrial swelling, irregular shaping of the cell membrane, degranulation and vesicularisation of the endoplasmic reticulum and protein degradation. (Azzam *et al.*, 2003, Somosy *et al.*, 2002).

Irradiated cells have abnormal projections that alter the cells' interaction with one another resulting in abnormal cell homeostasis. Direct or indirect altering of intracellular signalling pathways leads to pathophysiological changes in different tissues (Azzam *et al.*, 2003, Somosy *et al.*, 2002).

The lungs are the most frequently exposed and most sensitive organs to irradiation. Symptoms vary, and can include congestion, cough, dyspnoea, fever, pneumonitis and breathing difficulties (Stone *et al.*, 2002).

Studies found that the skin, mucosa, subcutaneous tissue, bone and salivary glands are areas most affected when patients undergo radiotherapy. The skin exhibits symptoms like erythema, scaling, pruritis, hypersensitivity, pain, dermatitis and mucositis. Vasodilatation, plasma leakage and hyperemia also occur (Stone *et al.*, 2003).

The gastro-intestinal tract is prominent in terms of pathology. Symptoms include diarrhoea, tissue oedema, hyperaemia as well as fluid and electrolyte imbalances. Molecular processes are not fully understood or explored yet, but it has been established that the mucosa is lined with abnormal epithelial cells after irradiation (Goldberg et al., 2005, Stone *et al.*, 2003, Rubio & Jalnas, 1996).

4 STANDARDS AND REGULATIONS

There are two laws that govern radiation exposure: The Mine Health and Safety Act, (Act nr 29 of 1996) and the National Nuclear Regulator Act (Act nr 47 of 1999). The dose limits that must be maintained are:

- an effective dose of 20 mSv/annum, over 5 consecutive years
- a maximum effective dose of 50 mSv/annum.

5 EXPERIMENTAL PROCEDURE

RGM (radon gas monitor) badges were used in this study, because it is the most cost effective method available due to the large amount of employees that need to be monitored on a regular basis. The badges are flexible logistically, because of their sizes, and do not have any effect on job performance. It is also the best method for area monitoring, as radon gas concentrations continuously fluctuate. These instruments accompany the workers everywhere in the workplace, thus obtaining a reliable reading of the radon gas concentrations at the workplace. Each monitoring cycle consists of three months in which the RGMs are issued, retrieved and analysed.

The shaft is divided into ventilation districts or adjacent groups of working areas that share intake

and return airways. On the shaft monitored, there are two ventilation districts, namely K1 and K2. Each ventilation district is further divided, identifying stopes, crosscuts, raises and also workplaces (ex. K1-2, K2-3). The RGMs for the normal four-cycle monitoring program are issued so at least three employees per workplace are monitored for a representative sample. When high readings are obtained, the areas are investigated to determine the possible causes of excessive radiation and action plans are then set in place to lower the exposure to workers. The following diagram shows the process that is followed:

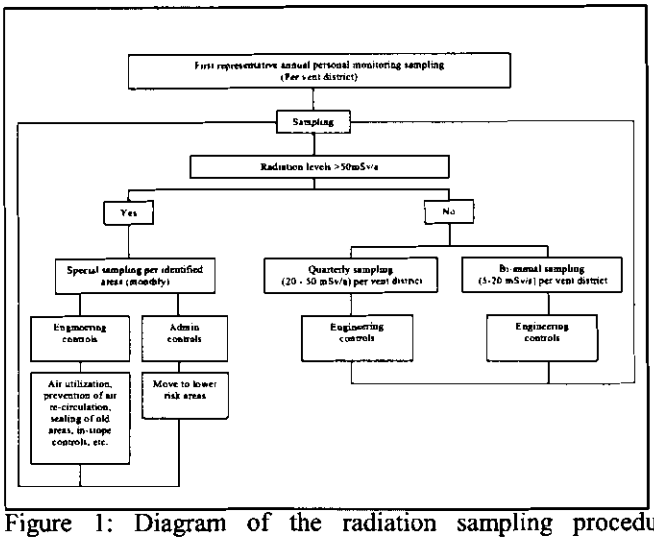

Figure 1: Diagram of the radiation sampling procedure (adapted from Ellis, 2004)

Special case areas are identified and further monitored according to the results. This is a very difficult task given that the workforce change and move regularly. To keep up with these changes is almost impossible.

The first aim of the study was to create a database that is easily accessible, complete and accurate, with the option of adding data on a monthly basis to keep the system updated. This makes it possible to locate the employees and their workplaces underground. The database is therefore trustworthy and reliable, allowing for proactive steps to minimize the health hazards associated with radiation. The second aim was to clarify and refine the current monitoring program specifically with regard to the issuing and retrieval of the RGMs. Each RGM has a number, which makes it possible to allocate a dose to a specific employee and workplace. In the past the issue and retrieval of RGMs were not structured, making the results unreliable. One of the objectives was to successfully refine this process.

Areas of concern were first identified based on the previous cycle's results. The workers in these areas were traced, using the bonus-system as reference point, since this system is the most reliable source of workforce information available at this stage. The employees' bonuses are determined by this system according to the gang's performance during the month.

These gangs are divided into three or four power-teams, which consist of approximately ten to fourteen employees per working area. If an area continues to deliver high radon readings, it is classified as a special case area. Every person in that area is issued with an RGM during the next cycle. Specials (RGMs in the special case areas) are issued monthly, in compassion with normals (the normal RGM cycle), which are issued quarterly. Special case monitoring continues for at least three months or at least until consistent satisfactory readings, well below the OEL (Occupational Exposure Limit) are obtained.

The special areas that were identified for this study were K1-2, K1-3, K1-6, K2-3, K2-5 and K2-6.

Data were collected and recorded into an MS-Excel worksheet created specifically for this purpose (calculation and reference). It consists of the following information: company number, initial, surname, occupation, workplace, ventilation district, section, lamp number, RGM number, RGM reading, exposure time and dose.

This allowed for the monitoring of each employee's exposure, the determination of the projected annual dose and the identification of the necessary measures to reduce the risks. The RGMs retrieved were matched to the data obtained from those that were issued, as a control measurement. The time of exposure is calculated by multiplying the amount of days the RGMs were exposed to radiation with the amount of hours worked per shift (ex. 30 days x 9.5 hours). RGMs were sent to the lab for analysis and the results and dose calculated. The average working shift is 9.5 hours long and is used as such in the abovementioned calculation. As reference, five background RGMs are placed within the lamp room, to determine the normal background radiation exposure. The method can be summarised as follow:

- issue RGMs to the identified employees - whether specials or normals;
- retrieve RGMs after the sampling period is over;
- send RGMs to the laboratory for analysis;
- process the results in MS Excel;
- update the personal history and projected dose datasheets.

6 RESULTS AND DISCUSSION

The general trend in the areas monitored shows a decrease from January to May. Average exposure decreased from 25.4 mSv to 7.8 mSv in May. There are two graphs for each area monitored. The first shows the projected dose for the employees in that area, while the second graphs shows the actual dose for that area. An increase in the dose was observed during April in four of the six areas included in the study. It can be assumed that the large earthquake

that occurred in the local area might be responsible for this increase. This seismic event occurred on the 9th of March 2005 and reached 5.3 on the Richter scale. Changes in the conditions underground are almost inevitable in response to the large amount of energy released into the system by such an event. The following figure shows the progressive decrease in dosage as measure in all areas for the period of the study:

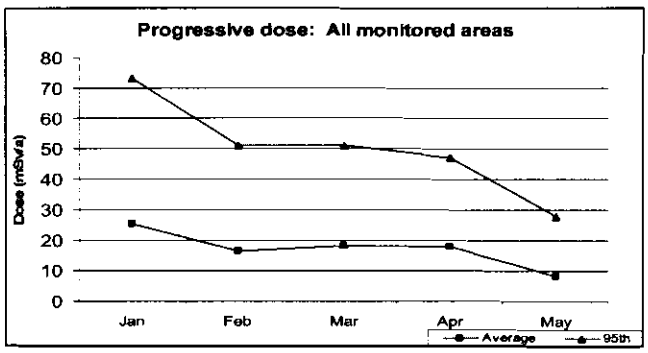

Figure 2. Progressive dose: All monitored areas

The detailed results per area monitored will be discussed below. Note that the maximum values shown in the figures, only represents the single highest reading of one RGM for that period. In figure 3, the cumulative percentage represents the progressive frequency of employees exposed to a range of radiation within the ventilation district. Although there is an even distribution over the whole dose range, the number of people exposed to higher dosage levels are very few. It is estimated that 85 % of the employees monitored in this area are exposed to less than 50 mSv/a. The following figure shows the monthly exposures for this area.

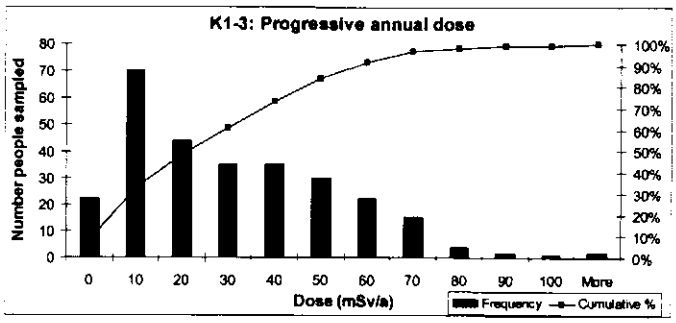

Figure 3. Projected annual dose for K1-3 for 2005.

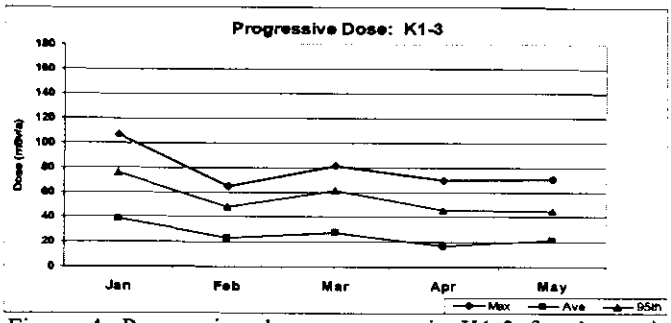

Figure 4. Progressive dose exposure in K1-3 for the period January to May 2005.

Figure 4 clearly shows a steady decrease from January through to May, with the average reading of 38.5 mSv during January declining to 21.5 mSv in May, which is well below the threshold. These results are very satisfactory, as there was a 55.7 % decrease in average exposure. The maximum readings and 95th percentile confidence level correlates with the average readings for this area. Monitoring will continue until the area is no longer a concern. The slight increase during March may be a result of seasonal changes, which influences barometric pressure, temperature and therefore gas flow and release underground.

The results of K1-6, the next monitored area, can be seen in the two graphs below. In figure 5, it can be seen that all the employees are predicted to be exposed to dosage levels between 0 mSv/a and 35 mSv/a. This is far below the maximum allowable dosage of 50 mSv/a.

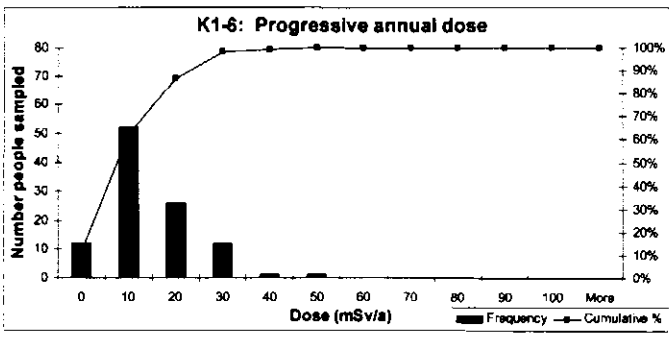

Figure 5. Projected annual dose for K1-6 for 2005.

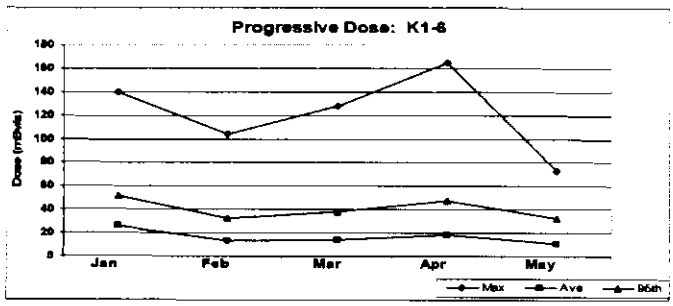

Figure 6. Progressive dose exposure in K1-6 for the period January to May 2005.

Following the 'hot spot' area classification in December, remedial actions were implemented leading to a decrease in January. The figure reveals that there was a steady decline in the average and 95th percentile values over the monitoring period despite variation in maximum values. Average exposure declined from 25.8 mSv to 10.6 mSv. This is a significant 41.1% decrease. The rise in April, from 13.9 mSv to 18.1 mSv, could be contributed to the aforementioned seismic event. Again, seasonal changes might also be a contributing factor.

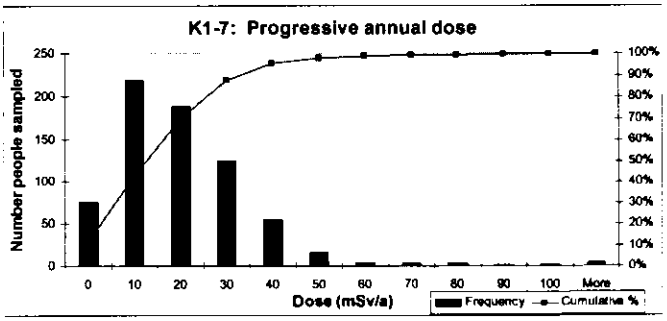

Figure 7. Projected annual dose for K1-7 for 2005.

Currently, the projected annual dose for the greater number of people whose exposure was measured in K1-7, lies below the 50 mSv/a limit with most exposed employees falling between 10 – 20 mSv/a. The distribution in this figure shows a reduction in number of employees sampled with increased dosage. Around 95 % of all the employees in this area are at no or low risk of being exposed to radiation levels higher than the threshold. Exposure for the bulk of the employees, about 425 of those monitored, is projected to be below 30 mSv/a by December 2005.

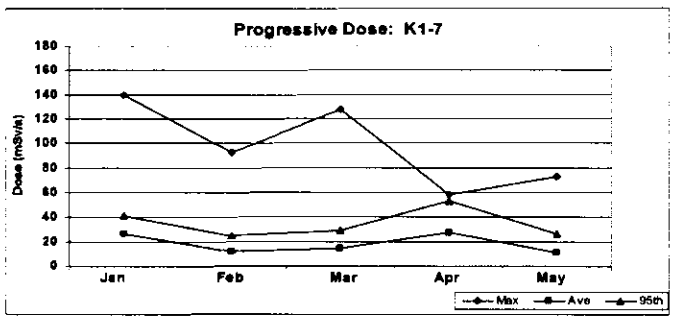

Figure 8. Progressive dose exposure in K1-7 for the period January to May 2005.

The increase in April corresponds with the above-mentioned areas' increase in the same month, which can again be contributed to the seismic event. The general trend in this area stayed relatively constant, only decreasing slightly in February and May. The maximum dose readings varied greatly, but decreased drastically in February, and again in April. Both these declines were followed by an increase in the next cycle, although the May results are much lower than in January.

This maximum value represents only one reading that is not representative of the whole area. It varies mostly because of the inconsistent levels of radon gas underground. The average declined with 41.3 %, from 26.9 mSv in January, to 11.1 mSv in May. The readings continued to drop in the following cycle, of which the results are not shown as it falls outside the scope of this study. Most of the areas continue to be closely monitored.

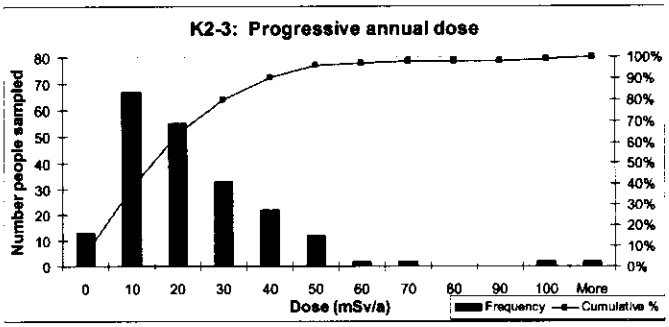

Figure 9. Projected annual dose for K2-3 for 2005.

In figure 9 it can be seen that the bulk percentage of the population is below 50 mSv, with most of the workers projected to be exposed between 10 to20 mSv/a, and to a lesser extent between 30 to40 mSv/a. It is projected that the exposure of 95 % of the people are below 45 mSv/a. The overall distribution of the population looks satisfactory.

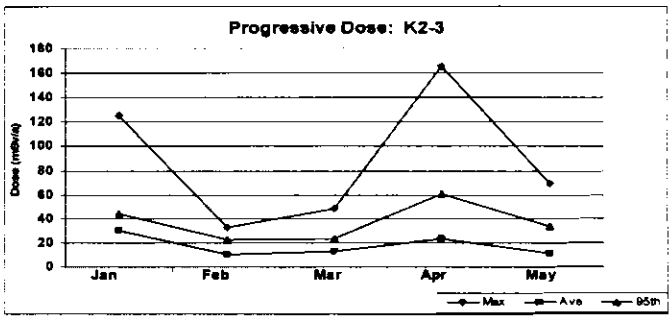

Figure 10. Progressive dose exposure in K2-3 for the period January 2005 to May 2005.

The figure above also shows a rise in April, while the general trend is a decline in dose values. In K2-3, (figure 8), the average decreased from 30.5 mSv during January, to 11.1 mSv in May. This is a decrease of 36.4 %. The high maximum value represents only one reading, and is not representative of the average maximum levels found in this area.

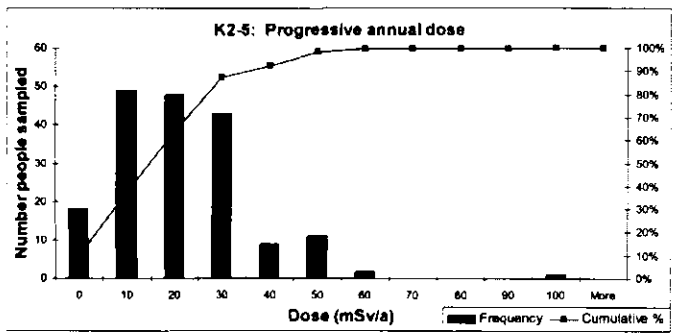

Figure 11. Projected annual dose for K2-5 for 2005.

In K2-5, of which the results can be seen in figure 11 and figure 12, it can be seen that exposure decreased. . In figure 4.9, it can be seen that 90 % of the people will be exposed to 10 to 30 mSv/a. It also shows that there is a probability that around 10 people will be exposed to 50 mSv/a, and 3 people will be exposed to doses above 50 mSv/a. Preventative measures can

and has been implemented figure 10, shows actual exposure for January through to May.

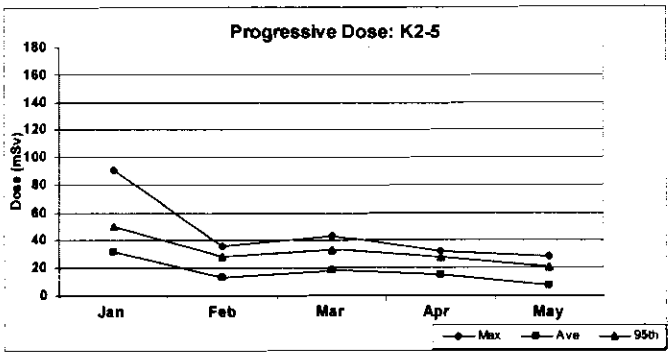

Figure 12. Progressive dose exposure in K2-5 for the period January to May 2005.

A dramatic decrease in dose readings in this area can be seen from the above figure. The decrease in exposed dosage, which started at 31.6 mSv in January to 7.8 mSv in May, was a 24.7 % decline. Pro-active steps that were taken, as described in later on, were effective in improving ventilation and decreasing exposure to radon gas. Here both the average and maximum values are below 10 mSv/a and 30 mSv/a respectively, which is well below the annual threshold value of 50 mSv/a.

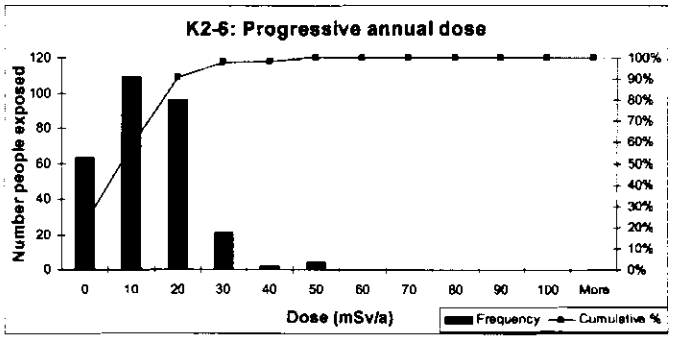

Figure 13. Projected annual dose for K2-6 for 2005.

90 % of all the projected readings in K2-6, as portrayed in figure 13, lies below 20 mSv/a. Less than 10 employees at risk of over-exposure and there is a 70 % chance that these employees will not be exposed above the threshold. This percentage was calculated with a 95 % confidence level.

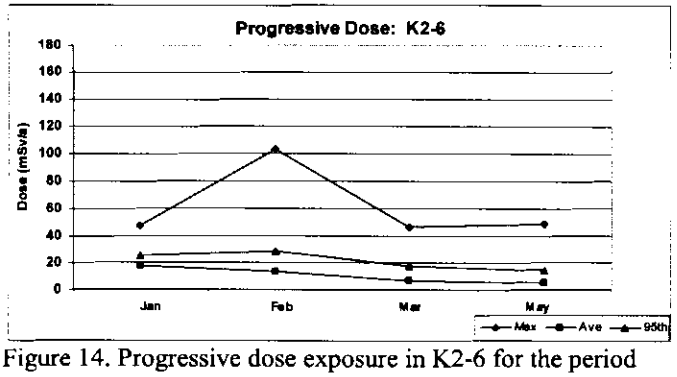

Figure 14. Progressive dose exposure in K2-6 for the period December 2004 to May 2005.

Figure 14 shows that the area K2-6, does not have readings as high as most of the other areas monitored. As in the other areas a general decrease can be seen here from 17.1 mSv in January, to 5 mSv during May. The maximum value in this area for February was very high, but this was a single exposure and does not correspond with the rest of the readings for that month. For March and May, the average and 95th percentile values are below 20 mSv/a. There were no samples taken in April due to administrative reasons.

When a workplace is depleted, the crosscut is sealed off in order to prevent unwanted air movement and unauthorised access of people. A raise is the main service pathway that connects the workplace with the two levels above and below.

All but two areas, K1-3 and K1-7 were declassified as special areas. The table below shows the average readings that were higher than the allowable threshold, which is 50 mSv/a in each ventilation district, for each cycle that was monitored.

Table 1. Percentage readings > 50 mSv/a per ventilation district.

	% > 50 mSv/a				
VENTILATION DISTRICT	JAN	FEB	MAR	APR	MAY
K1-3	39.2	4.6	18.2	3.8	5.13
K1-6	0	0	0	0	0
K1-7	3.1	1.2	2.7	10	1.23
K2-3	4.4	0	0	8.9	0
K2-5	12.5	0	0	0	0
K2-6	0	2.9	1	-	0

Problematic workplaces were identified based on investigation findings in these areas Action plans were implemented as follow:

- Ventilation doors were sealed off to prevent airflow from old contaminated areas
- Box holes were checked and sealed
- Ventilation walls were built at appointed places
- Regulators and fans were installed
- Change-overs of air were implemented, and
- the AUI's (air utilisation indexes) were maintained at maximum percentage.

In K2, the averages in K2-3 fluctuated a little, first declining until February and then rising steadily up until April, and then suddenly dropping to an average of 8 mSv/a. The amount of people exposed above the 50 mSv/a limit nonetheless remained constantly low. K2-4 was investigated in April and May, but showed no reason for concern. Exposure levels in K2-5 declined steadily after the action plans were employed The current average is 5.32 mSv/a and nobody was exposed above the limit. K2-6 showed a similar pattern and is currently on a 2.8 mSv/a average with no people over the dose limit.

During April and May, K1-2 was sampled additionaly and had an average reading of 20.6 mSv/an and 29.7 mSv/a respectively. During April, only 10.5 % of K1-2's readings were above the 50 mSv/a threshold. K1-3 showed a steady decline in readings during the cycles up to May, and had an average of 18.6 mSv/a and only 5 % readings above the threshold to date. K1-4 was sampled only as routine measurement during April, but there was no indication of radiation problems warranting further action. The readings in K1-6 also declined to an average of 3.5 mSv/a, with no readings exceeding the limit. In K1-7 the readings declined, but showed an interesting incline in April, from 16 mSv/a average, to 27.5 mSv/a in April, and then down again in May to 8.18 mSv/a. This could be a result of seasonal changes around that time of year or uranium rich ore that was mined.

The third special sampling cycle, during February 2005, had very satisfactory results. K1 showed major improvement, with an average reading of 23.1 mSv/a, and only 4.6 % readings above 50 mSv/a. K1-6 again showed no problems, having an average of 9 mSv/a, and no readings above 50 mSv/a. K2 also improved measureably, and K2-3 had an average of 11.2 mSv/a with no readings above 50 mSv/a. K2-5's average was 14 mSv/a, and no readings above 50 mSv/a. In K1, K1-7's average was 12 mSv/a, and only 1.2 % of the readings were above 50 mSv/a. K2-6 had an average of 13.3 %, with 2.8 % of the readings above 50 mSv/a.

7 CONCLUSION

The data basis is complete and up to date. Reports were drawn up, statistical analysis was done and proper action plans were implemented to ensure a permanent improvement in the underground radiation conditions. The ongoing monitoring program will be able to run smoothly, seeing that the basis is broad and correct. The five cycles that were closely monitored revealed an effective reduction in radiation problems and no problems are expected in the future (not taking into account geographical earth movements and occasional high uranium concentrations encountered in the rock).

It is possible that radon-gas exposure and dust inhalation have an accumulative effect to induce silicoses, tuberculosis and pneumoconiosis in mine workers. This is an area that has not been studied sufficiently and would surely deliver interesting and worthwhile results for future remedial action. Now that a reliable database is in place, this correlation can be studied.

Following the study and analysis of the results, it is recommended that the situation is monitored closely, the data basis kept up to date, the areas checked regularly, and a progressive and continuous risk assessment is done. This will ensure that the changes that were brought on will stay intact and will function properly on a continuous basis. It will also ensure the maintenance of a safe and healthy working environment and include future remedial actions to reduce the radiation exposure to below 20 mSv over 5 years consecutively.

8 REFERENCES

Amandus, H. & Costello, J. 1991. Silicosis and lung cancer in U.S. metal miners. *Archives of EnvironmentalHealth*, 46(2):82-89, March/April.

Anon. 2000. American Lung Association. Lung Cancer. [Web:] http://www.alahv.org/bookfiles4/lung_cancer.htmlhttp://www.uic.com.au/ral.htm [Date of access: 11 Jan. 2005].

Archer, V.E., Coons, T., Saccomanno, G. & Hong, D.Y. 2004. Latency and the lung cancer epidemic among United States uranium miners. *Health Physics*, 87(5):480-489.

Arfsten, D.P., Still, K.R. & Ritchie G.D. 2001. A review of the effects of uranium and depleted uranium exposure on reproduction and foetal development. *Toxicology and Industrial Health*, 17:180-191.

ATSDR. 1999. Toxicological profile for uranium (an update). Atlanta, GA: Agency for toxic substances and disease registry. Report TP-90-29.

Azzam, E.I., De Toledo, S.M. & Little, J.B. 2003. Oxidative metabolism, gap junctions and the ionising radiation-induced bystander effect. *Oncogene*, 22:7050-7057.

Dawson, L.A., Biersack, M., Lockwood, G., Math, M., Eisbruch, A., Lawrence, T.S., & Ten Haken, R.K. 2005. Use of principal component analysis to evaluate the partial organ tolerance of normal tissues to radiation. *International Journal of Radiation Oncology & Biological Physics*, 62(3):829-837.

Deonarine, D.C. 2005. Non-ionizing radiation. (*In* Schoeman, J.J., van den Heever, D.J. & Stanton, D.W. *ed.* Occupational Hygiene for Mining & Other Industries. 6(4):1–20.

Ellis, J. 2004. Anglogold Ashanti - Radiation protection. Controlled documents (Revision 0). September [CD-ROM].

Goldberg, Z., Schwietert, C.W., Lehnert, B., Stern, R., & Nami, I.O., B.A. 2004. Effects of low-dose ionising radiation on gene expression in human skin biopsies. *International Journal of Radiation Oncology & Biological Physics*, 58(2):567-574.

Gulson, B.L., Mozon, K.J., Dickson, B.L. & Korsch, M.J. 2005. The effect of exposure to employees from mining and milling operations in a uranium mine on lead isotopes – a pilot study. *Science of the Total Environment*, 339:267-272.

Hall, E.J. 2005. Radiation and Life. [Web:] http://www.uic.com.au/ral.htm [Date of access: 11 Jan. 2005].

Hnizdo, E., Murray, J & Klempman, S. 1997. Lung cancer in relation to exposure to silica dust, silicosis and uranium production in South African gold miners. *Thorax*, 52:271-275.

Kovalchuk, O., Ponton, A., Filkowski, J. & Kovalchuk, I. 2003. Dissimilar genome response to acute and chronic low-dose radiation in male and female mice. *Mutation Research*, 550:59-72.

Lipsztein, J.L., Dias Da Cunha, K.M., Azeredo, A.M.G., Julião, L., Santos, M., Melo, D.R. & Simões Filho, F.F.L. 2001. Expo-

sure of workers in mineral processing industries in Brazil. *Journal of Environmental Radioactivity*, 54:189-199.
Mcdiarmid, M.A. 2001. Depleted uranium and public health. BMJ, 322:123-124.
Mothersill, C. & Seymour, C. 2004. Radiation-induced bystander effects and adaptive responses – the yin and yang of low dose radiobiology? *Mutation Research,* 68:122-128.
Mouton, C.J. 2005. Ionizing radiation. (*In* Schoeman, J.J., *et al., ed.* Occupational Hygiene for Mining & Other Industries. 6(6):1–18.)
Offer, H., Erez, N., Zurer, I., Tang, X., Milyavsky, M., Goldfinger, N. & Rotter, V. 2002. The onset of p53-dependent DNA repair or apoptosis is determined by the level of accumulated damaged DNA. Carcinogenesis, 23(6):1025-1032.
Rubio, C.A. & Jalnas, M. 1996. Dose-time-dependent histological changes following irradiation of the small intestine of rats. *Digestive Diseases and Sciences*, 41(2):392-401.
Somosy, Z., Horvath, G., Telbisz, A., Rez, G. & Palfia, Z. 2002. Morphological aspects of ionising radiation response of small intestine. *Micron,*33:167-178.
Stone, H.B., Coleman, C.N., Anscher, M.S. & Mcbride, W.H. 2003. Effects of radiation on normal tissue: consequences and mechanisms. *Lancet Oncology,* 4:529-536.
Verschaeve, L. 2004. Genetic effects of radiofrequency radiation. (*In* Toxicological applications in pharmacology: Paper at the Tenth International Congress of Toxicology help in Pampere, Finland on 11-15 July 2004. Tampere)
Wymer, D. 2001. Ionising radiation. (*In* SIMRAC, Guild, R., Ehrlich, R.R., Johnston, J.R. & Ross, M.H.., *ed.* Handbook of Occupational Health Practice in the South African Mining Industry. Braamfontein: SIMRAC. p. 295-314.)
Yamada, Y. 2003. Radon exposure and its health effects. *Journal of Health Science,* 49(6):417-422.

REFERENCES

AMANDUS, H. & COSTELLO, J. 1991. Silicosis and lung cancer in U.S. metal miners. *Archives of environmental health*, 46(2):82-89, March/April.

ANON. 1995. An insidious hazard, Western Deep Levels – a clean bill of health. *SA mining, coal, gold & base minerals,* June, p15-16.

ANON. 2000. American Lung Association. Lung Cancer. [Web:] http://www.alahv.org/bookfiles4/lung_cancer.html [Date of access: 11 Jan. 2005].

ANON. 2000(a). Environmental diseases from A to Z. Health source – consumer edition. p1-7.

ANON. 2004(a). Cancerbacup. Types of cancer. [Web:] http://www.cancerbacup.org.uk/Aboutcancer/Wahtiscancer/Typesofcancer [Date of access: 11 Jan. 2005].

ANON. 2004(b). Radon and Cancer: Questions and Answers. National Cancer Institute. [Web:] http://cis.nci.nih.gov/fact/3_52.htm [Date of access: 11 Jan. 2005].

ANON. 2005. What is radon? [Web:] http://science.howstuffworks.com/radon1.htm [Date of access: 11 Jan. 2005].

ARCHER, V.E., COONS, T., SACCOMANNO, G. & HONG, D.Y. 2004. Latency and the lung cancer epidemic among United States uranium miners. *Health physics*, 87(5):480-489.

ARFSTEN, D.P., STILL, K.R. & RITCHIE G.D. 2001. A review of the effects of uranium and depleted uranium exposure on reproduction and foetal development. *Toxicology and industrial health*, 17:180-191.

ATSDR. 1999. Toxicological profile for uranium (an update). Atlanta, GA: Agency for toxic substances end disease registry. Report TP-90-29.

AZZAM, E.I., DE TOLEDO, S.M. & LITTLE, J.B. 2003. Oxidative metabolism, gap junctions and the ionising radiation-induced bystander effect. *Oncogene*, 22:7050-7057.

CHAUDHARY, U.B., TAKSEY, J.D. JOHNSON, R.D. & LEWIN, D.N. 2003. Small-cell cancers, and an unusual reaction to chemotherapy. *Journal of clinical oncology*, 21(12):2441-2442.

DAWSON, L.A., BIERSACK, M., B.S.E., LOCKWOOD, G., MATH, M., EISBRUCH, A. M.D., LAWRENCE, T.S., M.D., PH.D. & TEN HAKEN, R.K. 2005. Use of principal component analysis to evaluate the partial organ tolerance of normal tissues to radiation. *International journal of radiation oncology & biological physics*, 62(3):829-837.

DEONARINE, D.C. 2005. Non-ionising radiation. (*In* Schoeman, J.J., van den Heever, D.J. & Stanton, D.W. *ed.* Occupational hygiene for mining & other industries. 6(4):1–20.

DOLINSKY, C. 2002. Lung cancer: the basics. University of Pennsylvania, Medical School. Posting date: 31 May.

DONOGHUE, A.M. 2004. Occupational health hazards in mining: an overview. *Occupational Medicine*, 54:283-289.

DONDON, M.G., DE VATHAIRE, F., QUENEL, P. & FIERY, N. 2005. Cancer mortality during the 1968-1994 period in a mining area in France. *European journal of cancer prevention*, 14(3):297-301.

ELLIS, J. 2004. Anglogold Ashanti - Radiation protection. Controlled documents (Revision 0): Kopanang Mine, September [CD-ROM].

GISCO, 2005. Gisco sales and services of geophysical instruments. [Web:] http://www.giscogeo.com/pages/giscorad.html [Data of access: 15 Jun. 2005].

GOLDBERG, Z., M.D., F.R.C.P.C., SCHWIETERT, C.W., M.S., LEHNERT, B., PH.D., STERN, R., PH.D. & NAMI, IO., B.A. 2004. Effects of low-dose ionising radiation on gene expression in human skin biopsies. *International journal of radiation oncology & biological physics*, 58(2):567-574.

GULSON, B.L., MIZON, K.J., DICKSON, B.L. & KORSCH, M.J. 2005. The effect of exposure to employees from mining and milling operations in a uranium mine on lead isotopes – a pilot study. *Science of the total environment*, 339:267-272.

GUYTON, A.C. & HALL, J.E. 2000. Medical physiology. 10th ed. Philadelphia: WB Saunders. p740-741.

HALL, E.J. 2005. Radiation and Life. [Web:] http://www.uic.com.au/ral.htm [Date of access: 11 Jan. 2005].

HNIZDO, E., MURRAY, J & KLEMPMAN, S. 1997. Lung cancer in relation to exposure to silica dust, silicosis and uranium production in South African gold miners. *Thorax*, 52:271-275.

IRCP (International Commission on Radiological Protection). 1993. Protection against Radon-222 at home and at work. Report of a task group of the International Commission on Radiological Protection. Great Britain: BPCC 45 p. (ICRP Publication 65)

ICRP (International Commission on Radiological Protection). 1995. Age dependent doses to the members of the public from intake of radio nucleotides: Part 3. *Ann ICRP* 25:57-74 (ICRP Publication 72)

INCROPERA, F.P. & DEWITT, D.P. 1981. Fundamentals of heat and mass transfer. 4th ed. New York: John Wiley & Sons. p634-715.

KOVALCHUK, O., PONTON, A., FILKOWSKI, J. & KOVALCHUK, I. 2003. Dissimilar genome response to acute and chronic low-dose radiation in male and female mice. *Mutation research,* 550:59-72.

LIPSZTEIN, J.L., DIAS DA CUNHA, K.M., AZEREDO, A.M.G., JULIãO, L., SANTOS, M., MELO, D.R. & SIMõES FILHO, F.F.L. 2001. Exposure of workers in mineral processing industries in Brazil. *Journal of environmental radioactivity*, 54:189-199.

LITTLE, M.P. 2004. The bystander effect model of Brenner and Sachs fitted to lung cancer data in 11 cohorts of underground miners, and equivalence of fit of a linear relative risk model with adjustment for attained age and age at exposure. *Journal of radiological protection,* 24(3):243-255.

MCDIARMID, M.A. 2001. Depleted uranium and public health. BMJ, 322:123-124.

MIRKARIMI, R.B. 1992. The environmental and human health impacts of the gulf region with special reference to Iraq. [Web:] http://www.xs4all.nl/%Estgvisie/VISIE/extremedeformities.html [Date of access: 9 May 2005]

MWOD (Merriam-Webster Online Dictionary) 2005. [Web:] http://www.m-w.com/

MWOT (Merriam-Webster Online Thesaurus) 2005. [Web:] http://www.m-w.com/

MOTHERSILL, C. & SEYMOUR, C. 2004. Radiation-induced bystander effects and adaptive responses – the yin and yang of low dose radiobiology? *Mutation research,* 68:122-128.

MOUTON, C.J. 2005. Ionising radiation. (*In* Schoeman, J.J., *et al., ed.* Occupational hygiene for mining & other industries. 6(6):1–18.)

OFFER, H., EREZ, N., ZURER, I., TANG, X., MILYAVSKY, M., GOLDFINGER, N. & ROTTER, V. 2002. The onset of p53-dependent DNA repair or apoptosis is determined by the level of accumulated damaged DNA. Carcinogenesis, 23(6):1025-1032.

OKUNIEFF, P., M.D., CORNELISON, T, M.D., PH.D., MESTER, M., M.D., LIU, W., M.D., DING, I., M.D., CHEN, Y., M.D., PH.D., ZHANG, H., M.D., PH.D., WILLIAMS, J.P., PH.D. & FINKELSTEIN, J., PH.D. 2005. Mechanism and modification of gastrointestinal soft tissue response to radiation: role of growth factors. *International journal of radiation oncology & biological physics*, 62(1):273-278.

POTTEN, C.S. 1996. Protection of the small intestinal clonogenic stem cells from radiation-induced damage by pre-treatment with interleukin 11 also increases murine survival time. *Stem cells*, 14(4):452-459.

RUBIO, C.A. & JALNAS, M. 1996. Dose-time-dependent histological changes following irradiation of the small intestine of rats. *Digestive diseases and sciences*, 41(2):392-401.

SCHMIDT, P. & REGNER, J. 2005. Improvement of the radon situation at former uranium mining and milling sites in East Germany. *International congress series* 1276:238-239.

SCHWARTZ, D. & ROTTER, V. 1998. p53-dependent cell cycle control: response to genotoxic stress. [In Process Citation] *Seminars in Cancer Biology*, 8:325-336.

SHARMA, S. & MAYCHER, B. 2005. Lung cancer, non-small cell. [Web:] http://www.emedicine.com/radio/topic406.htm

SOMOSY, Z., HORVATH, G., TELBISZ, A., REZ, G. & PALFIA, Z. 2002. Morphological aspects of ionising radiation response of small intestine. *Micron,* 33:167-178.

SOUTH AFRICA. 1996. Mine Health and Safety Act (Act nr. 29 of 1996). *Government Gazette*, 7242:967, Jun. 14.

SOUTH AFRICA. 1999. National Nuclear Regulator Act (Act 47 of 1999). *Government Gazette*, 20760:1537, Dec. 23.

STANTON, D.W. 2005. Hazardous chemical substances. (*In* Schoeman, J.J., van den Heever, D.J. & Stanton, D.W., *ed.* Occupational hygiene for mining & other industries. 5(9):36-37.)

STONE, H.B., COLEMAN, C.N., ANSCHER, M.S. & MCBRIDE, W.H. 2003. Effects of radiation on normal tissue: consequences and mechanisms. *Lancet Oncology,* 4:529-536.

STRANDER, H., TURESSON, I. & CAVALLIN-STAHL, E. 2003. A systematic overview of radiation therapy effects in soft tissue sarcomas. *Anta Oncologica,* 42:516-531.

STRAUSS, J. & HOLLANDER, W.J. 1989. Nuclear chemistry in our world. *Spectrum,* 27:15-17.

TOBIN, M.J. 2003. Tuberculosis, Lung infections, interstitial lung disease, and Journalology in AJRCCM 2002. Information research: *American Journal of Respiratory and Critical Care Medicine*, Vol 167. P. 345-355. [Date of access: 4 Apr. 2005]

TRACERLAB. 2004. Tracerlab Catalogue: Radiation measurement, sampling systems, sample presentation. (In Tracerlab GmbH) [CD-ROM.]

VERSCHAEVE, L. 2004. Genetic effects of radio frequency radiation. (*In* Toxicological applications in pharmacology: Paper at the Tenth International Congress of Toxicology help in Tampere, Finland on 11-15 July 2004. Tampere)

WRIGHT, E.G. 2002. An overview of radiation-induced genomic instability. (*In*_Consultative Exercise on radiation risks of internal emitters (CERRIE)., *eds.* 2002 4th MeetingAshdown House, London. Paper 4/14.

WYMER, D. 2001. Ionising radiation. (*In* SIMRAC, Guild, R., Ehrlich, R.R., Johnston, J.R. & Ross, M.H.., *ed.* Handbook of occupational health practice in the South African mining industry. Braamfontein: SIMRAC. p. 295-314.)

YAMADA, Y. 2003. Radon exposure and its health effects. *Journal of Health Science*, 49(6):417-422.