# THE EFFECT OF AEROSOL EXPOSURE ON FOUNDRY WORKERS AND ARC WELDERS AT A LARGE ENGINEERING PLANT IN SOUTH AFRICA

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**All work and no play makes Jack a dull boy.**

**James Howel**

**to my brother**

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## LIST OF IMPORTANT ABBREVIATIONS

| | |
|---|---|
| AALG | *Ambient Air Level Goal* |
| ACGIH | *American Conference of Governmental Industrial Hygienists* |
| AIER | *Aerosol Inhalation Exposure Risk* |
| DOL | *Department of Labour* |
| FDA | *Food and Drug Administration* |
| HAD | *Health Assessment Document* |
| µm | *Micrometer* |
| $\mu g/m^3$ | *Micrograms per cubic metre* |
| $mg/m^3$ | *Milligrams per cubic metre* |
| mppcf | *Millions of particles per cubic foot* |
| nm | *Nanometre* |
| NAS | *National Academy of Sciences* |
| NIOSH | *National Institute for Occupational Safety and Health* |
| OEL | *Occupational Exposure Level* |
| PEL | *Permissible Exposure Limit (SA)* |
| PIXE | *Particle Induced X-Ray Emmision* |
| ppb | *parts per billion* |
| ppm | *parts per million* |
| REL | Recommended Exposure Limit |
| TLV | *Threshold Limit Value* |
| TLV-C | *Threshold Limit Value Ceiling* |
| TLV-STEL | *Threshold Limit Value Short Term Exposure Limit* |
| TLV-TWA | *Threshold Limit Value-Time Weighted Average* |
| TWA | *Time-weighted Average* |
| USEPA | *United States Environmental Protection Agency* |
| WHO | *World Health Organisation* |

**SUMMARY**

South African data regarding the extent of aerosol exposure and health effects in the workplace are limited. Furthermore, a shortage of industrial hygiene- and epidemiological data from large scale studies exist. Given the increasing concern about the health of industrial metal workers, an inhalation exposure study of South African iron foundry workers and welders at a large engineering plant in Bloemfontein, was undertaken. The aim of the study was to compile a source inventory, identifying and characterising all health related inorganic aerosols to which metal workers of the plant are potentially or actually exposed. In addition, the exposure risk was assessed by the integration of aerosol concentrations and biological data from urine analyses.

Aerosols were sampled by means of time sequence particulate sampling on streaker filter frames and analysed with Particle Induced X-ray Emission (PIXE) on the Tandem van de Graaff accelerator of the University of the Witwatersrand, Johannesburg. The streaker is analysed in 1 mm steps, corresponding to 1 hour of exposure. PIXE analysis yielded concentrations or detection limits of elements Al, Si, P, S, Cl, K, Ca, Ti, Cr, Mn, Fe, Ni, Cu, Zn, Br and Pb.

The urine of selected exposed workers was sampled according to NIOSH method 8310 and analysed using Atomic Absorption (AA) spectrometry for the urine-metal concentrations Zn, Cd, Co, Ni, Mn, Cu, Cr, Al, Fe, Pb, Si and V.

Forced vital capacity (FVC), forced expiratory volume in one second ($FEV_1$), peak expiratory flow (PEF), peak inspiratory flow (PIF), average expiratory flow between 25 % and 75 % of FVC ($FEF_{25-75}$), expiratory flow at 25 % of FVC ($V_{max25}$), expiratory flow at 50 % of FVC ($V_{max50}$), expiratory flow at 75 % of FVC ($V_{max75}$) and forced expiration time (100 % FVC) ($FET_{100}$), was conducted with a Cosmed Pony spirometer. This was done to provide a physical image of the workers' lungs.

A new method for the assessment of aerosol inhalation exposure risk, called AIER, using aerosol concentrations and metal urine concentrations, is proposed for estimation of the inhalation risk. The assumptions and calculation for the new method are presented.

A number of sources or source categories have been identified in the foundry's and welding shop's air by making use of patterns of time variations and elemental ratios. Six sources namely crustal particles, sulphur, Zn-Pb-Cl, two distinct components of different castings and other heavy metals were identified as

sources. The main pollutants and the relative contributions from other sources have been identified for iron foundries and welding shops with recognised air quality problems. Overexposure occurred during specific operations which was also quantified for rectification.

The urine analysis of the foundry workers yielded high concentrations of Cd, Cu, Fe and Si. It is assumed that the Fe and Si concentrations are exposure related. The analysis of urine from the welders yielded high concentrations of Ni, Cr and Fe. Although Ni concentrations in the workroom air were low, the occurrence of all three elements in the urine may be as a result of chronic exposure to welding fumes in their workplace. Except for vanadium, no statistical significant differences ($P > 0.05$) were found between the different metal urine concentrations of the workers of the two localities.

The total exposed foundry population showed a significant decrease in $FEV_1$ and FVC which indicates that the pollution in the workplace contributes to the development of restrictive lung disorders in foundry workers. The dust created during the welding of steel in the welding shop is a contributing agent in the development of obstructive respiratory disorders in the welder population.

Examination of the relationship between elemental variations has allowed identification of several sources and activities contributing to airborne particles. The aerosol profiles did not show similar diurnal time variation patterns in the foundry or the welding shop due to irregularities in the continuation of the processes.

The AIER for the foundry resulted in a maximum value of 92.3 % while the corresponding value for the welding environment was 71.7 %. The results present the worst case scenario during winter conditions and it is expected that conditions will be more healthy during summer time when windows and doors are open. The planning and prioritisation for the improvement of indoor air quality in both workplaces can proceed, using the data on the sources of the pollutants.

It can be concluded that workers exposed to conditions as found during this project, will experience health problems after chronic exposure. The results emphasised the importance of exposure characterisation in order to provide for identification of pollutants, control of sources and the application of industrial hygiene principles for the protection of human health. It is recommended that all industries implement exposure characterisation programmes as a tool in applying good occupational and environmental hygiene.

## OPSOMMING

Suid Afrikaanse data betreffende die omvang van aerosol blootstelling en die effek op gesondheid van werkers in die werkplek, is beperk. Daar bestaan ook 'n tekort aan beroepshigiëne- en epidemiologiese data vanaf grootskaalse studies. As gevolg van die toenemende bekommernis betreffende die gesondheid van industriële metaal werkers, is 'n inasemings blootstelling studie van Suid Afrikaanse yster gietery werkers en sweisers by 'n groot ingenieurs aanleg te Bloemfontein onderneem. Die doel van die studie was om 'n inventaris van die bronne saam te stel en sodoende alle anorganiese aerosols te karakteriseer en te identifiseer waaraan die metaal werkers blootgestel of moontlik blootgestel is. Ter aanvulling, is die blootstellingsrisiko deur die integrasie van aerosol konsentrasies en biologiese data vanaf uriene ontledings bepaal.

Aerosols is deur middel van tyd gekoppelde opeenvolgende partikulêre monsterneming op roterende filter skywe gemonster en met partikel geïnduseerde X-straal emissie (PIXE) op die Tandem van de Graaff versneller van die Universiteit van die Witwatersrand, Johannesburg, geanaliseer. Die skywe is in 1 mm stappe geanaliseer, ooreenkomstig tot 1 uur van blootstelling. PIXE analise verskaf konsentrasies of deteksielimiete van die elemente Al, Si, P, S, Cl, K, Ca, Ti, Cr, Mn, Fe, Ni, Cu, Zn, Br en Pb.

Die urine van geselekteerde blootgestelde werkers is volgens NIOSH metode 8310 gemonster en met behulp van atoomabsorbsie spektrofotometrie (AAS) vir die urine metaal konsentrasies Zn, Cd, Co, Ni, Mn, Cu, Cr, Al, Fe, Pb, Si en V ontleed.

Geforseerde vitale kapasiteit (FVC), geforseerde geëkspireerde volume in een sekonde ($FEV_1$), piek geëkspireerde vloei (PEF), piek geïnspireerde vloei (PIF), gemiddelde geëkspireerde vloei tussen 25 % en 75 % van FVC ($FEF_{25-75}$), geëkspireerde vloei by 25 % van FVC ($V_{max25}$), geëkspireerde vloei by at 50 % van FVC ($V_{maks50}$), geëkspireerde vloei by 75 % van FVC ($V_{maks75}$) en geforseerde geëkspireerde tyd (100 % VK) ($FET_{100}$), is met 'n Cosmed Pony spirometer uitgevoer. Dit is gedoen om 'n fisiese beeld van die werkers se longe te verkry.

'n Nuwe metode vir die bepaling van die blootstelling van aerosol inasemings blootstellingsrisiko, genoem AIBR, deur die toepassing van aerosol- en metaal urine konsentrasies, is vir die die beraming van inasemingsrisiko voorgestel. Die aannames en berekening vir die nuwe metode word verskaf.

'n Aantal bronne of kategorieë van bronne is in die lug van die gietery en die sweiswinkel geïdentifiseer deur van tendense van tydlikse variasies en elementale verhoudings gebruik te maak. Ses bronne naamlik korsagtige partikels, swawel, Zn-Pb-Cl, twee onderskeidbare komponente van verskillende gietstukke en ander swaar metale, is as bronne geïdentifiseer. Die hoof besoedelstowwe en die relatiewe bydraes van ander bronne is vir yster gieterye en sweiswinkels met ooglopende lug kwaliteitsprobleme vasgestel. Oormatige blootstelling het tydens spesifieke operasies voorgekom wat vir regstellende maatreëls gekwantifiseer is.

Die analise van urine van die gietery werkers het hoë konsentrasies Cd, Cu, Fe and Si getoon. Dit word aanvaar dat die Fe en Si konsentrasies blootstelling verwant is. Die analise van die sweisers se urine het hoë konsentrasies Ni, Cr en Fe getoon. Alhoewel Ni konsentrasies in die werksomgewing laag was, kan die voorkoms van al drie die elemente in die urine as gevolg van chroniese blootstelling aan sweisdampe wees. Behalwe vir vanadium, is geen betekenisvolle statistiese verskille ($P > 0.05$) tussen die verskillende urine metaal konsentrasies van die werkers van die twee verskillende lokaliteite gevind nie.

Die totale blootgestelde gietery populasie het 'n betekenisvolle afname in $FEV_1$ en FVC getoon wat daarop dui dat die besoedeling in die werkplek tot die ontwikkeling van restriktiewe long abnormaliteite kan bydra. Die damp wat tydens die sweis van staal in die sweiswinkel gegenereer is, is 'n bydraende faktor tot die ontwikkeling van obstruktiewe respiratoriese abnormaliteite in die sweiser populasie.

Eksaminering van die verwantskap tussen elementale variasies het die identifikasie van verskeie bronne en aktiwiteite bydraend tot luggedrae partikels moontlik gemaak. Die aerosol profiele het nie dieselfde daaglikse tyd variasie patroon in die gietery of die sweiswinkel getoon nie. Dit is as gevolg van die onreëlmatigheid in die deurlopendheid van die prosesse.

Die AIBR vir die gietery het 'n maksimum waarde van 92.3 % getoon terwyl die ooreenstemmende waarde vir die sweis omgewing 71.7 % was. Die resultate bied die swaks moontlike toestand tydens winter toestande aan en dit word verwag dat die toestande meer bevorderlik vir gesondheid gedurende die somer maande sal wees wanneer die deure en vensters oop gehou word. Die beplanning en prioritisering vir die verbetering van die binneshuise lug kwaliteit in beide werkplekke kan deur die gebruik van die data afkomstig van die besoedelstowwe, uitgevoer word.

Dit kan verwag word dat werkers wat aan toestande blootgestel word soos dit gedurende hierdie projek gevind is, gesondheidsprobleme na chroniese blootstelling sal ondervind. Die resultate beklemtoon die belangrikheid van blootstellings karakterisering ten einde vir die identifikasie vir besoedelstowwe, beheer van bronne en die toepassing van beroepshigiëne beginsels vir die beskerming van menslike gesondheid voorsiening te maak. Dit word aanbeveel dat alle industrieë blootstelling karakterisering programme as 'n werktuig in die toepassing van goeie beroeps- en omgewingshigiëne implementeer.

# 1. Introduction

Over the past decade there has been a major effort in many countries to develop, what are now called, air toxics programs. In order to initiate such programs in South Africa, Dr D Rees, Director of the National Centre for Occupational Health (NCOH) started to organise workshops with researchers to identify key occupational health issues and develop different exposure programs (NCOH, 1996). The need for environmental monitoring programs (EMP's) was also expressed by the South African Institute of Mining and Metallurgy (Von Sittert, 1993). These programs are essentially designed to address health concerns resulting from exposure to airborne contaminants. The need for regulatory action to control toxic air pollutants has been based on a number of arguments. Most notably is the occurrence of higher incidences of lung cancer in the urban environment and ambient air monitoring studies, especially in urban areas, showing a large number of contaminants, which also includes carcinogens.

The attribution of the higher incidence of lung cancer in urban settings to air pollution is highly controversial given the multifactorial nature of complex diseases such as cancer. Factors such as dietary differences, stress, smoking patterns, indoor air pollutant levels and other important independent variables, that may vary between urban and rural areas, contribute to uncertainties in seeking a consensus on the role of the urban factor in lung cancer. Biostatistical and epidemiological approaches of different investigators have reflected the lack of consensus on this point. Karch and Schneiderman (1981) have estimated that at least 11 %, and possibly as much as 21 %, of lung cancer may be attributable to air pollution, after correction for smoking and occupational effects, whereas Doll and Peto (1981) estimate that the contribution of all forms of pollution (air, water and food) to cancer incidence is 2 %, with a range of 1 to 5 %.

Despite the unresolved issue over the causes of air pollution, there is little dispute over the observation that workroom air of industries contains numerous pollutants, many of which are mutagens and carcinogens in animal systems. The question however is, whether the levels observed pose significant health risks to the occupational population. In recent years, the need to quantify the health risks associated with exposure to environmental and occupational toxicants has generated a new interdisciplinary methodology referred to as risk assessment. For given conditions of exposure, risk assessment provides (Hallenbeck and Cunningham, 1987):

- characterisation of the types of health effects expected;
- an estimate of the probability (risk) of occurrence of these health effects;
- an estimate of the number of cases of these health effects; and
- suggested acceptable concentrations of the toxicant in air, water and food.

The outputs of risk assessment are necessary for informed regulatory decisions regarding worker exposures, plant emissions and effluents, ambient air and water exposures, chemical residues in foods, waste disposal sites, consumer products, and naturally occurring contaminants.

South African data regarding the extent of dust exposure and health effects in the workplace are limited. While some industries such as mining have good exposure monitoring records and compensation registers on pneumoconiosis amongst other lung diseases, little information is available for industries such as manufacturing and construction (Nurminen *et al.*, 1992). Therefore an approach to national risk assessment is needed to support and supplement existing monitoring and health effects data. Other health outcomes such as lung cancer or chronic obstructive airway disease have also not been assessed for the labour force occupationally exposed to elevated concentrations of airborne particles.

Risk estimates are developed based upon the exposure of the group at risk. The following should be considered for aerosol exposure: who will be exposed, the concentration-, route-, duration- and the nature of exposure to any other toxic materials which are generated as a result of the release of a toxicant. This process can be relatively straightforward for agents of known toxicity (e.g. asbestos in an asbestos textile plant), but extremely complicated in industries where exposures vary greatly in type, intensity and toxicity of substances. The latter, alone or in combination, is often poorly understood, such as in the iron foundry and welding shop, where different materials and processes are used daily. Thus methods used to characterise exposure vary considerably depending on the circumstances surrounding exposure, i.e., the exposure scenario and the accuracy with which exposure is characterised, is a primary determinant of the ultimate validity of a risk assessment. In some situations, workers' reports of symptoms and illness may help to identify at least classes of potentially toxic exposures, but in this study such information is non-existent.

The aim of this project is to compile a source inventory, identifying and characterising all health-related agents regarding inorganic aerosols to which metal workers of the engineering plant are potentially exposed. Furthermore, to

estimate the risks of the different hazardous airborne substances with exposure at the current concentration levels in South Africa or, at the most, at a threshold limit value as recommended by European countries and the American Conference of Governmental Industrial Hygienists (ACGIH).

The project also provides information on biological parameters for comparison with known limits. Finally, it contributes to the estimation of the workers health risk by the integration of clinical, epidemiological and industrial hygiene data.

The project was a case study involving a large metal works. The properties of the aerosols in a foundry and welding shop were measured using 1 hour time resolution sampling and analysed for elemental content. Conventional Time weighted average (TWA) gravimetric sampling was used for determination of ratio's between the streaker sampler and individual work stations. Samples of urine were collected from selected workers and analysed for metallic content. From these data a risk assessment was performed, combining the environmental and biological indices. Additionally, spirometry was done to provide information on the physical image of the worker's lungs.

The scope of the project covered only the one metal works, but included 124 foundry workers and 68 welders. It is typical of industrial foundry and welding in South Africa.

## 1.1 Occupational epidemiology

A primary goal of occupational epidemiology is to determine which specific workplace exposure factors result in disease or injury. This involves the estimation of dose-response relationships that are ultimately used to predict effects in populations other than those studied, forming the base of occupational and non-occupational exposure guidelines. The estimability of dose-response relationships depends largely on the quality and quantity of available historical exposure data. In practice, complete and accurate historical exposure data are often non-existent or unavailable and therefore must be estimated (Rom, 1992).

The toxic effect varies with the dose-response relationship and depends on the individual contaminant. According to Klaassen (1986) certain assumptions can be made, namely that the response is due to the chemical administered, or that the response is related to the dosage, or that there exist both a quantifiable method of

measuring and a precise means of expressing toxicity. If the response is related to the dosage, the chemical interacts with the molecular or receptor site to produce the response, the response is related to the concentration of the chemical at the reactive site and the concentration at the site is related to the dose.

For a chemical to cause injury, there must be contact. Inhalation is the principal route by which toxicants gain entry to the body (Rom, 1992). For this reason, plus the potential for deposition onto skin, this study pays particular attention to contaminants that are, or become, airborne. Substances which enter the body are distributed by the bloodstream. Once a toxicant is in the body, the concern is about the damage it causes, the duration in the body and the removal thereof. Such information would be of great benefit to link exposure data to individual workers for future research on the protection of workers' health.

Substances enter the lungs via the nose and nasal cavity, although some workers are constant mouth breathers while other workers with respiratory congestion, as from a cold, become temporary mouth breathers. Inhaled particles are trapped by the mucous lining of the nasal cavity. The bronchi are lined with cells which have hair-like structures or cilia. The cilia are constantly in motion in a wavelike manner which assists in moving impacted particulate matter and secretions out of the lung upwards toward the trachea and pharynx, from where it is coughed up and expectorated, or swallowed. From the oral and nasal cavities it can be swallowed, creating the possibility of absorption from the gastrointestinal tract or removal from the body with the faeces. However, compounds from the inhaled particles may enter the blood. This is most likely to occur if the particles reach the alveoli. Phagocytic cells whose function is to engulf foreign substances and bacteria, normally cleanse the contact area in the lungs, then migrate from the alveoli to the bronchi to be moved out of the lungs on the mucous. Exposure to dust stimulates the expansion of this cell population. Some substances like cigarette smoke, narcotise these cells resulting in that both the particulate and the cells accumulate in the alveoli. For the above mentioned reason, spirometry was conducted during this study to determine the lung function of exposed workers.

The ultimate objective of industrial toxicology is the prevention of health impairment that may result from exposure to chemicals at workplaces. This implies the definition of permissible levels of exposure, that is, levels that according to the present status of knowledge are estimated to cause no adverse health effects during the lifetime of the workers, and the regular assessment of the possible

health risk associated with exposure by comparing the current or the integrated exposure with the permissible exposure limits (Lauwerys and Hoet, 1993a).

Biological monitoring of exposure to industrial chemicals accesses the health risk through the evaluation of the internal exposure of the organism (i.e. the internal dose) by a biological method. Biological monitoring of exposure is complementary to other monitoring programs which are carried out to evaluate the health risk associated with occupational pollutants, namely ambient monitoring and health surveillance or biological monitoring of early effects (Lauwerys, 1984). The basis of these monitoring programs is defined by following up the fate of a chemical exerting systemic biological effects from the environment to the target molecules in the organism.

Once absorbed and present in the circulation, the chemical is distributed to different compartments of the body. It may be eliminated unchanged in urine or in expired air. The chemical or its metabolites may bind reversibly or irreversibly to sites on the target molecules. Binding to noncritical sites induces nonadverse effects that may or may not be reversible. Binding to critical sites may give rise to adverse health effects at least when the amount bound has reached a certain level and the repair mechanisms are inadequate or insufficient. This leads to pre-clinical lesions, at an early stage, and to clinical lesions at a more advanced stage of intoxication.

The main pathway for removal of toxicants from the body is to transfer them from the blood to the urine via the kidneys. Body fluids are filtered in the kidneys into the glomerules, leaving behind blood cells and proteins, which are very large molecules. The filtrate passes down a tube, the walls of which include selective transport structures that bind desirable compounds and move them back into the blood. By the time the end of the tube is reached, the glucose, amino acids, sodium ion's and most of the water have been scavenged. Unfortunately, as the concentration increases due to the removal of water, nonpolar molecules move through the walls of the tube and return to the blood without the help of a transport structure. Although other adjustments of less importance have occurred, waste products and polar foreign substances are concentrated in the urine and sent to the bladder. During this study, biological monitoring by means of urine sampling was carried out to determine the concentrations of toxicants excreted by the kidneys to the urine.

## 1.2 Exposure characterisation

Measurement of airborne dust with personal samplers is a commonly accepted means of monitoring worker exposure to hazardous dust in the work place. Normally, industrial hygienists would determine the time weighted average (TWA) exposure and by calculating the average concentration, the exposure level of a worker can be determined. The time weighted average concentration is then compared to the time weighted threshold limit (TWA.TLV), which is usually expressed in milligrams of contaminant per cubic meter of air ($mg/m^3$). While routine measurements provide a direct measurement of the average exposure over a complete shift, they do not provide information on variations of concentrations in time and space as the worker proceeds with his duties.

In practice it is found that the concentration of any airborne contaminant may vary considerably in the course of one 8 hour working day. Most sampling methods only show the total exposure expressed as a TWA exposure and since contaminant concentrations may vary during the shift, it is not known if any high exposures occurred during the sampling. Furthermore, if any high exposures did occur during the sampling, it is not known what the source or the time interval of such excursion was, because the processes are not carried out at the same time each day. Such exposures can only be identified and characterised by time sequence sampling and characterisation.

Time weighted average threshold limit values (TWA.TLV), may be exceeded for short periods of time, provided that the average concentration for the 8 hour shift does not exceed the TWA and that no ceiling concentration (C.TLV) has been set. According to Schoeman and Schröder (1994), the amount by which a TWA may be exceeded without injury to health, depends on factors such as:

- nature of the contaminant;
- whether high concentrations, even for short periods, will cause acute poisoning;
- whether the effects are cumulative;
- the frequency with which high concentrations occur; and
- the duration of such periods.

No data exist for time sequence intermittent weekly exposures of welders and foundry workers and the dust concentrations which vary temporally and spatially in such workplaces, are not characterised. Due to the lack of such exposure data, the emissions from processes are controlled in general and are not focused on excursions from processes polluting the work environment at specific times.

Time sequence sampling in 1 hour increments show the pattern of exposure necessary to identify the sources of exposure. Identification is carried out by keeping a logsheet of all activities in the foundry at particular times. With such exposure data, emissions can be identified, characterised and controlled, either specifically at the source or only at the time of emission. A streaker sampler was used to sample dust in 1 hour increments for periods of 24 hours during a week.

The streaker sampler was placed in the centre of the activities to obtain air representative of the work environment. Personal sampling was simultaneously carried out on workers at fixed work stations to calculate the exposure ratio between the streakers' sampling point and those of the workers. The sampling was conducted during winter months to get the worst-case scenario during which doors and windows are closed and fires being made inside the workplace, to heat the environment.

## 1.3 Spirometry

Measurement of lung volumes provides fundamental information that makes possible categorisation and staging of lung diseases. However, lung volumes provide a static picture and do not measure dynamic performance. Analysis of volume-time or flow-volume relationships provides an assessment of the ventilatory apparatus "in action" (Garay, 1992). Spirometry is the measurement of volume change achieved by various breathing manoeuvres. Volume-time analysis utilises the "simple" or "classic" spirogram, whereas the flow-volume relationships are described by the maximum expiratory flow-volume curve. The same basic manoeuvre, the forced vital capacity (FVC) manoeuvre, generates data for both analyses.

Pulmonary function testing attempts to detect and quantitate abnormal lung function. However these studies usually do not give a precise, specific anatomic or pathologic diagnosis. The detection of abnormal lung function by such studies helps assess the severity and progression of a disease process as well as response to therapy (Garay, 1992).

## 1.4 Foundries

Since risk assessment can be extremely complicated in industries where exposures vary greatly, it was decided to evaluate foundries and welding shops where different materials and processes are used on a daily basis.

Foundry work comprises of making the pattern and the mould; assembling the mould; pouring the metal into the mould; and finally, removing the finished casting (fettling). Founding consists of pouring molten metal into a mould which is made to the outside shape of a pattern of the article required and contains, in some cases, a core which will determine the dimensions of any internal cavity.

Developments in recent years have widened the scope of metals and materials considerably, so that cast metals and alloys may now contain aluminium (Al), titanium (Ti), chromium (Cr), nickel (Ni) and magnesium (Mg) and even toxic metals such as beryllium (Be), cadmium (Cd) and thorium (Th) (Parmeggiani, 1985). Moulds are made from silica (Si) and bound with clay and oils. Many of these substances when heated above melting point, may be dispersed into the work environment and can be inhaled by workers - thus producing health risks.

It is well known that pneumoconiosis is a classic occupational disease in foundry workers. In most foundries, moulds are made from sand mixed with clay and resins and therefore, airborne crystalline silica is virtually ubiquitous in plants that use silica sand. Although all foundries melt and cast metals, they differ from foundry to foundry as the amounts and concentrations of materials differ. The toxic properties of various alloys differ widely and moulding methods and materials are also diverse. Decomposition of products released into the foundry air depend upon the pouring temperature and the organic materials present in the binders and additives.

Silicosis and other related respiratory abnormalities have decades ago been reported to be major health hazards for foundry workers. In considering this disease, attention must be given to the type of sand used in the foundry. If the sand contains free silica, as is the case in most foundries, the free silica occurs in flint and quartz which is extremely dangerous. Furthermore, the most dangerous forms of silica are those produced by heating where tridymite is formed after heating at temperatures of 650-1000 °C. During normal founding operations silica becomes heated to such temperatures by the molten metal and dust is formed, especially during fettling.

Workers exposed to respirable quartz may experience pulmonary diseases after exposure, depending on the duration and concentration of exposure and their health status. Over and above that, most South African foundry workers have a very low socio-economic lifestyle and live in poorly ventilated houses. A recent

study showed that African people living in poor conditions are also exposed to high total suspended particulate concentrations and gases as a result of fossil fuel burning in houses for heat production and cooking (Terblanche, Nel and Golding, 1994). The workers examined in this study are subjected to such conditions of living and working for 8 hours per day in a typical iron foundry.

## 1.5 Welding

Full-time welding is relatively a recent phenomenon, dating from the time when welding largely replaced riveting as the superior method for joining metal surfaces during and after World War II. Among those who habitually perform welding, a large proportion may experience some transient or reversible effect on the respiratory system. Chronic respiratory effects, however, may also be found as a result of environmental exposure.

During welding, the output from one side of a low-voltage, high-current power supply is connected to the workpiece by a flexible cable. The other side of the output circuits are connected by another cable to an insulated clamp that holds a consumable wire electrode. When the electrode touches the workpiece, it completes the circuit. The resulting arc is hot enough to melt the electrode and fuse the workpiece. The workers in this study joined metals by using either gas tungsten arc welding (GTAW) or gas metal arc welding (GMAW). These processes were formerly called TIG (tungsten inert gas) and MIG (metal inert gas) welding (Rekus, 1991). Both GTAW and GMAW use inert gases such as argon, carbon dioxide ($CO_2$)or helium (He) in place of the flux on stick electrodes.

In GTAW welding, the arc is formed between the workpiece and a hand-held torch that houses a non-consumable tungsten electrode. The electrode is surrounded by a ceramic tube through which inert gas flows to the surface of the newly formed weld. Extra filler metal may be added as needed by means of a manually applied rod. This type of welding is used to a limited extent during the welding operations.

GMAW welding is similar to GTAW except that the tungsten is replaced by a consumable wire electrode. The wire is similar in composition to the base metal and is continuously fed to the tip of the torch by a mechanically driven dispensing reel. GMAW is used mostly in the welding process. All welding processes produce fumes whereof the chemical composition is determined by the base metal, the electrode or filler rod used and any preservative coatings that may be on the base metal. Welding fumes are solid particles generated by condensation from the

gaseous state, generally after volatilisation from molten metals. In the case of metals this process is mostly coupled with a process of oxidation, so that the metallic fumes present in the air are partly in the form of an oxide.

Welding fume particles are almost always spherical in shape and are typically about 1 µm in diameter. This particle size allows them to penetrate the body's natural defences in the nose and upper respiratory passages and eventually make their way into the deep lung passages (alveoli sacs). Once there, the macrophages will engulf the foreign particles. Macrophages, having ingested the foreign matter, will move up into the bronchioles and are either coughed up or swallowed. During the International Pneumoconiosis Conference in Johannesburg in 1959, a respirable sampling curve was constructed in view of the differential depositing of dust particles. The curve was constructed on the basis that 100% of dust particles with an equivalent diameter of 1 µm such as welding fumes, are deposited in the lungs (Schoeman and Schröder, 1994). Once there, they may pass through the lungs and be absorbed into the bloodstream, where they can be carried to all parts of the body (Rekus, 1991).

The aerosols and gases generated in welding processes are considered to be among the more harmful of the many exposures of welders. Welders may suffer from systemic toxicity from welding related substances absorbed through the lungs. Some welders are also exposed to hazards not directly produced by the welding process (such as silica dust), which may cause both respiratory and non-respiratory effects. In considering the respiratory health of welders, emphasis is placed on specific substances associated with respiratory effects and uptake in the human body. Although it is useful to characterise the airborne concentration of particulate or fume materials generated during the welding process, the actual dose delivered to the respiratory system is of prime interest in considering the health effects of welding fumes (Sferlazza and Beckett, 1991).

## 1.6 Hypothesis

Given the nature of the industrial process and the chemical agents, as well as the likelihood of toxic human exposures at present or in the past, this study considers the plausible adverse health effects (acute, subacute, chronic) for which employees may be at risk. A null hypothesis regarding the presence of toxicants in the mentioned workplaces is formulated as follows:

$H_o$: The workers of both workplaces are exposed to concentrations of air toxicants which will affect their health after chronic exposure.

The alternate hypothesis is:

$H_a$: The workers of both workplaces are exposed to concentrations of air contaminants which will not affect their health after chronic exposure. The $H_o$ can be accepted if it is within the 95 % confidence interval.

# 2. Methods

## 2.1 Experimental Set-up

### 2.1.1 Iron Foundry

A large iron foundry in the Free State, South Africa was chosen as a single plant for this survey as it is representative of most iron foundries in the province and most workers live and work in similar conditions. The foundry has been in operation for the past 35 years. Operations proceed during two 8 hour shifts for 6 days a week. Most of the workers have been employed at the foundry since it was established. Approximately 125 workers with an average age of 42.3 years are working in the foundry.

The plant produces metal balls for stone crushing as well as brake blocks for trains. All the moulds are made of a sand and carbon mixture. Dust is mostly generated at the fettling process, as well as from normal operations. All workers have access to local ventilation systems, but no dust masks or any other personal respiratory protective devices are worn. The exposed group of 65 people had an average age of 38.4 years.

### 2.1.2 Welding shop

The welding shop is approximately 700 meters away from the iron foundry and houses an assembly line for train trucks. This welding operation was chosen because it is the largest welding process in the province and is representative of most welding situations. The welding shop has been in operation for the past 30 years. Operations proceed during one 8 hour shift a day for a 5 day work week and in special cases, also on Saturdays and Sundays. Some of the workers have been at the plant since it was established. However, most of them only have a few years of working experience.

The plant assembles all train trucks for the national railway company. Metal sheets are cut, moulded and prepared for assembly in the shop. The sheets are placed in jigs (frames) where they are welded together.

### 2.1.3 Preliminary air sampling and analyses

Preliminary air sampling was carried out at both localities for determination of metal fume concentrations, in order to establish which contaminants are present and pose a health risk. Self compensating Gilian Sc de Luxe dust sampling pumps (Gilian Instrument Corporation) were calibrated and used according to the method

of the suppliers. The pumps were calibrated at a flow-rate of 1.9 ℓ/min with loaded in-line filter cassettes by means of the absolute volume displacement method. After sampling, each complete apparatus' flow-rate was checked with a Gilibrator (Gilian Instrument Corp.). The flow-rate of all three pumps was within the permissible standard deviation of ±5 % from the pre-set flow-rate of 1.9 ℓ/min. The analyses of the samples for the determination of silica dust and other toxic elements were conducted by South African Bureau of Standards (SABS) laboratories.

Twelve 8 hour samples of the air were collected in triplicate on 37 mm cellulose nitrate filters (Nuclepore, Costar Corporation) with a pore size of 0.8 µm. The samples were collected in the breathing zones of the workers. The workshift time-weighted average concentration of total dust was determined gravimetrically for all samples. Samples were analysed for Cr, Cu, Fe, Mn, Pb, Zn, Ni, Cd, Al, Ca, K, V, Ti and Mg. All element concentrations in the air, except for Pb and $Cr^{6+}$ were analysed by using inductively coupled plasma (ICP) emission spectrometry after sample digestion in hydrofluoric acid (NIOSH, 1994a). Pb and $Cr^{6+}$ concentrations were determined with atomic absorption spectrometry (NIOSH, 1994b; 1994c). Crystalline silica were sampled and analysed according to NIOSH (1994d). No gas sampling was carried out during the survey.

## 2.2 Metal urine concentrations

### 2.2.1 Selection of experimental and control group

For the experimental group, three workers at each locality were selected on the basis of being healthy, absence of disease and a long working history in the specific plant. The workers were of African origin with corresponding dietary patterns and a mean body weight of 71.2 ±4.6 kg for foundry workers and 69.1 ± 4.9 kg for welders. The obtained experimental data was compared to international biological exposure indices (BEI) for industrial workers.

### 2.2.2 Urine sampling

Three 12 hour urine samples were collected quarterly from 3 healthy workers at both localities. Pre-cleaned high density polyethylene bottles with a 2 ℓ capacity were used. For cleaning, the bottles were soaked in a 20 % (volume/volume) solution of nitric acid for 24 hours, washed out with distilled water and stored with 2 M hydrochloric acid (HCl). Before sampling, the acid was discarded and the bottles rinsed three times with distilled water. After sampling, the bottles were

sealed, put in a sample case with a temperature of ± 10 °C and transported to the laboratory.

### 2.2.3 Urine analysis

Samples for analyses of Al, Cd, Cr, Cu, Fe, Mn, Ni, Ti and Zn were prepared and analysed according to NIOSH method 8310 (NIOSH, 1994e). The urine samples were further analysed for the other elements by means of atomic absorption (AA) spectrometry (Que Hee and Boyle, 1988; Tietz, 1990). Statistical significance was calculated with the Sigma Plot version 5.0 computer programme. However, the sample size (n = 3) is too small to make positive conclusions from the statistics.

## 2.3 Spirometry

### 2.3.1 Selection of experimental and control group

**Foundry:** A group of 78 iron foundry workers, exposed to dust and fumes during a normal 8 hour workday, were examined. The exposed subjects were aged 23 - 56 years (43.38 ± 9.07) with a mean height of 1.72 ± 0.067 m. The mean mass of the exposed group was 71.7 ± 12.7 kg and they had an average of 19.25 ± 9.59 years of service at the specific foundry. The study group consisted of 47 smokers and 31 non-smokers. The smokers were aged 23 - 56 years (43.4 ± 9.0) and the non-smokers 25 - 55 years (43.2 ± 9.3). The average exposure of the smokers was 19.0 ± 9.4 years compared to the 19.6 ± 9.8 years of the non-smokers.

**Welding:** The welders in the welding shop consisted of 36 people exposed to welding fumes during a normal 8 hour workday. The exposed subjects were aged 23 - 52 years (37.2 ± 8.8) with a mean height of 1.77 ± 0.078 m. Their mean mass was 74.8 ± 13.8 kg and they had an average of 14.5 ± 8.5 years of service at the welding shop. The group consisted of 20 smokers, aged 24 - 52 years (38.3 ± 8.1) and 16 non-smokers, aged 23 - 52 years (35.2 ± 9.5). The smokers had an average exposure of 15.8 ± 8.7 years compared to the 13.0 ± 8.3 years of the non-smokers.

**Control group:** The study included a control group of 32 workers employed in various jobs during which they were not exposed to dust and fumes in their work atmosphere. This group matched the exposed group by sex, height, smoking habits, place of residence and social status. The control group subjects were aged 23 - 65 years (33.9 ± 10.2).

### 2.3.2 Lung function testing

Subjects were interviewed and tested before starting work in the morning (6:45 - 8:30). The interview consisted of obtaining a medical and occupational history and information on personal data, exposure duration and smoking history. No subjects were tested if any symptoms of illness or complaints regarding any disease or discomfort were found.

### 2.3.3 Spirometry analysis

Measurements of lung function were made using a Cosmed Pony Spirometer (Cosmed Instruments Inc.). Forced vital capacity (FVC), and forced expiratory volume in one second ($FEV_1$) were measured on three efforts and the average of the best two efforts was used. The spirometer was calibrated with a 1 $\ell$ gas pump before commencement of the tests and the calibration checked regularly throughout the study. Other parameters measured with the spirometer were peak expiratory flow (PEF), peak inspiratory flow (PIF), average expiratory flow between 25 % and 75 % of FVC ($FEF_{25-75}$ ), expiratory flow at 25 % of FVC ($V_{max25}$), expiratory flow at 50 % of FVC ($V_{max50}$), expiratory flow at 75 % of FVC ($V_{max75}$) and forced expiration time (100 % FVC) ($FET_{100}$). The instrument was used according to the manufacturer's specifications. The results of the measurements of the ventilatory parameters were analysed by using the paired two-tailed *t*-test (Sigma Plot, Version 5.0) and statistical significance was calculated at the 0.05 level.

## 2.4 Aerosol concentrations

### 2.4.1 Sampling

Sampling was conducted in the foundry and welding shop of a railway truck factory in Bloemfontein, as described in Section 2.1. Samples were collected with a PIXE International Incorporated circular streaker sampler (PIXE International Corporation) (Annegarn *et al.*, 1990) as depicted in Figure 1.

The streaker sampler is 12.6 cm high with a diameter of 11.5 cm and weighs 1.4 kg. This air sampling device draws air through a 0.4 µm pore size Nuclepore membrane on which particulate matter is deposited as a 7 mm wide streak. The sampler has a rectangular orifice through which the air is drawn at a rate of 1 $\ell$/min. The membrane is mounted on a circular streaker frame, which is placed onto the sampler motor and rotated at 1 revolution per week. An impactor stage at the inlet of the streaker has $d_{50}$ = 10 µmad, restricting the sampled particles to the

equivalent of a PM10 sampler (Kneen *et al.*, 1994). The sampling train was equipped with an in-line gas meter for the measurement of flow and the calibration was checked with a Gilibrator (Gilian Instrument Corp.).

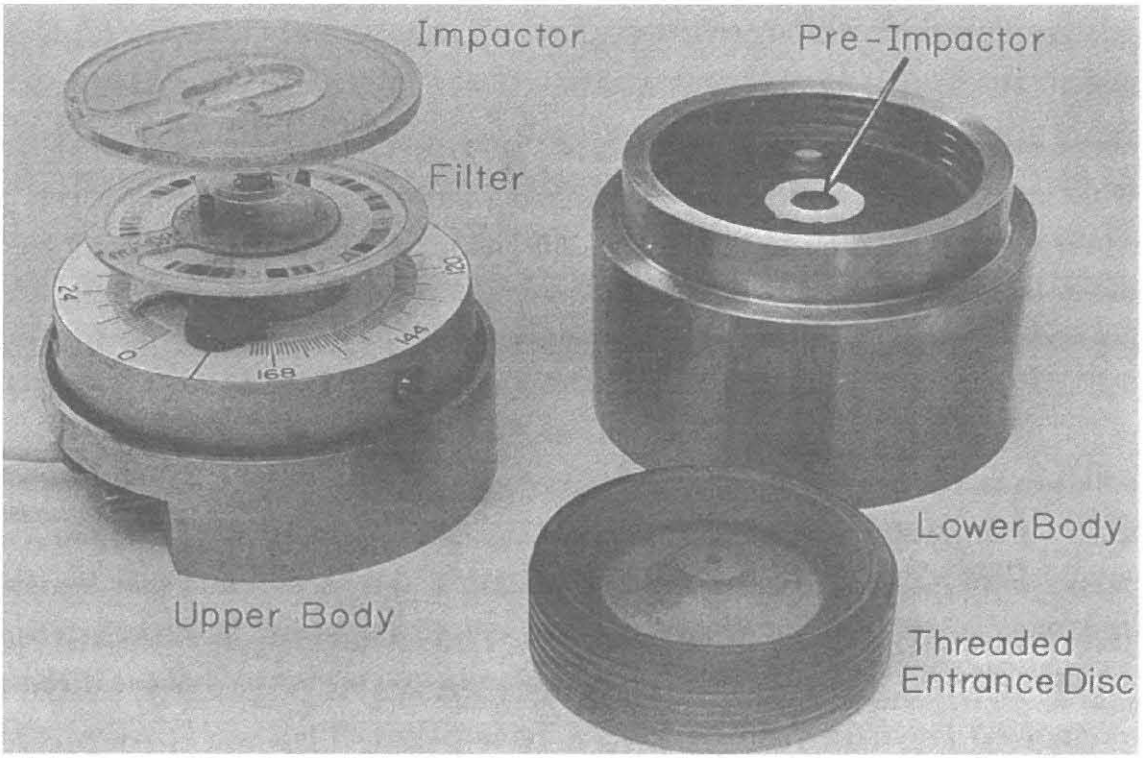


**Figure 1:** Streaker sampler

### 2.4.2 Aerosol properties

Aerosols (aerodispersed systems) consist of a gas (dispersion medium) in which liquids or solid substances (dust, smoke, haze and fume) may be dispersed. On the basis of the state (liquid or solid) of the dispersed substance, dispersion and condensation aerosols can be differentiated (Spurny, 1986). Dispersion aerosols are formed by atomisation of solids and liquids and describe the type of aerosols found in foundries. Condensation aerosols are formed when supersaturated vapours condense and describe the type of aerosols found in welding shops.

Dispersion aerosols are mostly coarser than condensation aerosols. The smallest aerosol particles are about the size of gaseous ions, in the range of 1 nm, and the molecular aggregates comprising a charged molecule with neutral gas molecules as a cluster. The upper limit of particle size in aerodispersed systems may be 1 mm (Spurny, 1986). Aerosols therefore involve a very wide size range.

The size of welding fumes normally ranges between 0.01 and 10 µm while the size of dust particlulates in foundries ranges between 0.01 and 150 µm (Schoeman en Schröder, 1994; Schröder, 1989).

### 2.4.3 PIXE analysis

Particle Induced X-ray Emission (PIXE) analysis of aerosol particles were among the first applications of the method (Johansson, Akselsson and Johansson, 1970). Since then, several reviews regarding the capabilities of PIXE for aerosol studies have been published (Koltay, 1990; Maenhaut, 1992). Currently, aerosol related programs form approximately 20 % of all PIXE applications (Cahill, Miranda and Morales, 1991).

Analysis of aerosol samples were conducted according to the method as described by Annegarn *et al.* (1988). PIXE analysis is performed by bombarding aerosol particles with proton beams of energies typically between 2 and 4 MeV. X-rays from excitation of the target by the beam are detected by a solid state Si(Li) detector, similarly to conventional secondary X-ray fluorescence (XRF) analysis.

Irradiations of aerosol samples last from 15 seconds to a few minutes, with the sample either in vacuum, a helium atmosphere or, sometimes in air. To reduce complexities in quantitative reduction of the X-ray spectra, targets must satisfy a thin-target criterion, determined by the energy loss of protons in the target, and self absorption of secondary X-rays in the target. This effectively limits the areal density of the sample deposit to less than 5 mg/cm$^2$. As typical proton beam areas are 1 cm$^2$ or less, this imposes an upper limit on the useful amount of material needed for PIXE analysis to a few tens or hundreds of micrograms. Typical detection limits for aerosol samples analysed with PIXE, ranged from 36 ng/m$^3$ for Si to 2 ng/m$^3$ for Zn (Annegarn *et al.*, 1988).

Analysis of the streaker frames was performed by using PIXE analysis on the Tandem van de Graaff accelerator of the University of the Witwatersrand, Johannesburg, using a 10 nA beam of 3.2 MeV protons. Each streaker frame was analysed in 1 mm steps, corresponding to 1 hour of exposure. PIXE analysis yielded concentrations or detection limits of elements Al, Si, P, S, Cl, K, Ca, Ti, V, Cr, Mn, Fe, Ni, Cu, Zn, Br and Pb.

The concentrations (µg/m$^3$) for toxic compounds such as $SiO_2$, were calculated from the PIXE determined elemental concentration as follows:

*Total Compound [ ] ( µg / m$^3$ ) = Measured element [ ]$^1$ + Binding element [ ]$^2$*

### 2.4.4 Time plots

A single stage circular streaker was set up in the foundry and the welding shop, respectively, in such a way that the sampled air was representative of the indoor air to which the workers were exposed during the shift. Air at this point contained an integrated sample of all dust and fume sources within the building.

The sampler and PIXE analyses were configured for 1 hour increments, yielding 168 sample steps per filter frame, thus producing hourly time plots or concentrations over a week period. Such time plots give a descriptive picture of the fluctuating exposure over the week.

After a few streaker samples were obtained, it was decided to select one sample from each batch by visual inspection for analysis. The samples in Table 1 were analysed for exposure characterisation.

**TABLE 1:** Streaker samples analysed with PIXE

| Sample # | Locality | Dates sampled |
|---|---|---|
| BLO95c | Foundry | 8-14 May 1995 |
| BLO95b | Welding shop | 2-5 May 1995 |

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*(1)* *Concentration determined with PIXE analysis in µg/m$^3$*

*Binding element [ ] =*

*(2)* $$\frac{\text{Measured Element [ ]} \times \text{Relative atomic mass of binding element}^3 \times \text{Number of binding atoms}}{\text{Relative atomic mass of compound}^4}$$

*(3)* *Relative atomic mass of the element that binds with the measured element*

*(4)* *Relative atomic mass of the compound containing the element analysed with PIXE.*

### 2.4.5 Source apportionment

Source apportionment was carried out according to the method of Annegarn *et al.* (1992). Iron (Fe) was used as tracer element in determining the ratios for both sampling localities.

### 2.4.6 Weather data

Relevant weather data were obtained from the local weather station of the Department of Environmental Affairs, Bloemfontein. The weather station is approximately 5 km north of the sampling localities. The data provides additional information for the explanation of exposure patterns.

## 2.5 Risk assessment

### 2.5.1 Threshold limit values and workplace air monitoring

Workplace air monitoring consists of assessment of inhalation exposure to chemicals in the workplace through measurement of the chemical concentration in the ambient air. Threshold limit values (TLV's) serve as reference values and refer to airborne concentrations of substances representing conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse health effects. Because of the wide variation in individual susceptibility, a small percentage of workers however, may experience discomfort from some substances at concentrations at or below the threshold limit; a smaller percentage may be affected more seriously by aggravation of a pre-existing condition or by development of an occupational illness (ACGIH, 1996).

Threshold limit values are based on the best available information from industrial experience, from experimental human and animal studies, and, when possible, from a combination of the three. Health impairments considered include those that shorten life expectancy, compromise physiological function, impair the capability for resisting other toxic substances or disease process or adversely affect reproductive function or developmental process. These limits are not fine lines between safe and dangerous concentrations nor are they a relative index of toxicity. The limits are intended for use in the practice of industrial hygiene as guidelines or recommendations in the control of potential health hazards (ACGIH, 1996) and can therefore be used in the risk assessment of occupational exposure.

### 2.5.2 Biological exposure indices and biological monitoring

Biological monitoring provides occupational health personnel with a tool for assessing a worker's exposure to chemicals. It consists of an assessment of overall exposure to chemicals that are present in the workplace through measurement of the appropriate determinant(s) in biological specimens collected from the worker at the specified time.

Biological exposure indices (BEI's) are reference values intended as guidelines for the evaluation of potential health hazards and represent the levels of determinants that are most likely to be observed in specimens collected from a healthy worker who has been exposed to chemicals to the same extent as a worker with inhalation exposure to the TLV. BEI's do not indicate a sharp distinction between hazardous and nonhazardous exposures. Due to biological variability, it is possible for an individual's measurement to exceed the BEI without incurring an increased health risk. If, however, measurements in specimens obtained from a worker on different occasions persistently exceed the BEI, or if the majority of measurements in specimens obtained from a group of workers at the same workplace exceed the BEI, the cause of the excessive values must be investigated and proper action taken to reduce the exposure (ACGIH, 1996). The BEI's determined by the ACGIH (1996) were used for risk assessment in this study.

Each BEI consists of available information on absorption, elimination and metabolism of chemicals and on the correlation between exposure intensity and biological effect in workers. The BEI is based either on the relationship between intensity of exposure and biological levels of the determinant or on the relationship between biological levels and health effects. Human data from controlled and field studies are used to find such relationships (ACGIH, 1996).

### 2.5.3 Risk assessment calculation

Various formulae exist for the assessment of health risk, but are complex and do not provide a rapid answer on the occupational risk of the exposed workers. Due to the lack of such methodology, a formula is proposed for the calculation of the occupational health risk of workers exposed to inorganic aerosols in their work place by using measured ambient aerosol concentrations and metal urine concentrations. Inclusion of biological specimen concentrations not only represent occupational exposures, but also nonoccupational exposures such as food and water. The formula therefore combines clinical, epidemiological and industrial hygiene data, including the dose-effect relationship described by Schoeman and Schröder (1994). Such inclusion is relevant in the developing industrial set-up of

South Africa, taking into consideration the primary health care programs that form part of overall occupational health programs.

Industrial working conditions are likely to vary considerably from day to day as well as within the shift due to the fluctuation of the exposure concentration. Inhalation exposure and consequently uptake, are not constant. Variable factors such as variation in exposure level, route of exposure, physical form of the chemical physical workload, rates of absorption, distribution, metabolism and excretion were not included in the risk assessment formula because it would make the calculation too complex for rapid everyday use in industrial hygiene. The formula for calculating aerosol inhalation exposure risk for workers with a light work load (metabolic rate = 875 kJ/h) and working with both arms, was compiled as follows:

*Aerosol Inhalation Exposure Risk (AIER) (%) =*

$$\frac{1}{2}\left(\frac{TWA\ [\ ]\ of\ measured\ element^{1}}{TWA.TLV^{2}} + \frac{Metal\ urine\ [\ ]\ of\ biological\ specimen^{3}}{BEI^{4}}\right) \times 100$$

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*(1) Time weighted average concentration of the measured element ($mg/m^3$)*

*(2) Time weighted average threshold limit value for the measured element ($mg/m^3$)*

*(3) Metal urine concentration of the determinant in the biological specimen (µg/g creatinine)*

*(4) Biological exposure indice for the determinant (µg/g creatinine)*

# 3. Results

## 3.1 Metal urine concentrations

For the purpose of the results of this study, the different chemicals sampled were grouped according to their oxidation state on the periodic table of elements.

### 3.1.1 Foundry

**TABLE 2:** Results of urine analyses from foundry workers (n = 3)

| Element | Mean Concen-tration | Std. Dev. | Unit | 95 % Confidence limit | 99 % Confidence limit |
|---|---|---|---|---|---|
| **Oxidation state 2** | | | | | |
| **Zn** | 24.0 | 11.0 | µmol/ℓ | 27.2 | 61.9 |
| **Cd** | 2.9 | 2.0 | µg/g Creat. | 7.3 | 16.5 |
| **Oxidation-state 2,3** | | | | | |
| **Co** | 3.7 | 1.5 | µg/g Creat. | 3.6 | 8.2 |
| **Ni** | 3.2 | 1.7 | µg/ℓ | 4.1 | 9.5 |
| **Mn** | 1.2 | 0.2 | µg/ℓ | 0.6 | 1.4 |
| **Cu** | 2.9 | 2.0 | µmol/ℓ | 5.0 | 11.6 |
| **Oxidation-state 3** | | | | | |
| **Cr** | 1.9 | 0.6 | µg/g Creat. | 1.4 | 3.2 |
| **Al** | 12.0 | 7.1 | µg/g Creat. | 17.5 | 40.0 |
| **Fe** | 84.3 | 50.6 | µg/ℓ | 125.0 | 285.0 |
| **Oxidation-state 4** | | | | | |
| **Pb** | 24.2 | 8.8 | µg/ℓ | 21.8 | 50.0 |
| **Si** | 17.3 | 1.7 | mg/ℓ | 4.3 | 10.0 |
| **Oxidation-state 5** | | | | | |
| **V** | 2.1 | 0.5 | µg/g Creat. | 1.3 | 2.9 |

### 3.1.2 Welding shop

**TABLE 3:** Results of urine analyses from welders (n = 3)

| Element | Mean Concentration | Std. Dev. | Unit | 95 % Confidence limit | 99 % Confidence limit |
|---|---|---|---|---|---|
| **Oxidation state 2** | | | | | |
| **Zn** | 15.5 | 3.0 | µmol/ ℓ | 1.7 | 7.5 |
| **Cd** | 5.8 | 3.1 | µg/g Creat. | 7.9 | 18.0 |
| **Oxidation-state 2,3** | | | | | |
| **Co** | 3.6 | 0.7 | µg/g Creat. | 1.7 | 3.8 |
| **Ni** | 12.4 | 9.2 | µg/ ℓ | 22.8 | 52.0 |
| **Mn** | 1.7 | 0.4 | µg/ ℓ | 0.9 | 2.0 |
| **Cu** | 1.8 | 0.1 | µmol/ ℓ | 7.5 | 17.0 |
| **Oxidation-state 3** | | | | | |
| **Cr** | 9.3 | 9.7 | µg/g Creat. | 24.1 | 54.9 |
| **Al** | 8.6 | 3.5 | µg/g Creat. | 8.6 | 19.7 |
| **Fe** | 52.7 | 19.0 | µg/ ℓ | 47.4 | 108.0 |
| **Oxidation-state 4** | | | | | |
| **Pb** | 31.8 | 9.5 | µg/ ℓ | 23.6 | 53.8 |
| **Si** | 13.4 | 0.5 | mg/ ℓ | 1.3 | 2.9 |
| **Oxidation-state 5** | | | | | |
| **V** | 1.6 | 0.5 | µg/g Creat. | 1.1 | 2.6 |

## 3.2 Spirometry

### 3.2.1 Foundry

The results of spirometry measurements for the exposed subjects of the foundry are compared with the results of the control group in Table 4.

**TABLE 4:** Respiratory functions measured for iron foundry workers

| Respiratory Functions | Total Experiment n = 78 | Total Control n = 33 | Smokers Experiment n = 47 | Smokers Control n = 22 | Non smoke Experiment n = 31 | Non smoke Control n = 11 |
|---|---|---|---|---|---|---|
| FVC | 3.44±0.71 | 3.69±0.85 | 3.45±0.72 | 3.76±0.86 | 3.42±0.70 | 3.55±0.84 |
| $FEV_1$ | 3.00±0.64 | 3.21±0.71 | 2.98±0.64 | 3.29±0.74 | 3.02±0.66 | 3.06±0.63 |
| PEF | 7.51±1.81 | 8.09±2.27 | 7.36±1.70 | 8.01±2.31 | 7.74±1.98 | 8.27±2.30 |
| PIF | 5.77±2.05 | 6.42±2.16 | 5.36±2.01 | 5.87±2.44 | 6.38±1.98 | 7.01±1.80 |
| $FEF_{25-75}$ | 3.56±1.13 | 3.93±1.07 | 3.47±1.12 | 4.01±1.18 | 3.71±1.14 | 3.75±0.80 |
| $V_{MAX25}$ | 6.54±1.77 | 7.17±2.07 | 6.38±1.67 | 7.04±2.17 | 6.79±1.90 | 7.45±1.93 |
| $V_{MAX50}$ | 4.03±1.28 | 4.61±1.22 | 3.95±1.28 | 4.59±1.38 | 4.13±1.29 | 4.66±0.85 |
| $V_{MAX75}$ | 1.84±2.09 | 2.06±0.81 | 1.76±0.75 | 2.03±0.67 | 1.95±0.71 | 2.12±1.08 |
| $FET_{100}$ | 2.09±0.89 | 3.50±2.12 | 2.16±0.99 | 3.18±1.78 | 1.98±0.71 | 4.16±1.69 |
| $FEV_1$/FVC % | 87.4±7.1 | 87.4±5.7 | 86.72±7.85 | 87.62±5.17 | 88.4±5.75 | 87.05±7.02 |

### 3.2.2 Welding shop

The results of spirometry measurements for the exposed subjects of the welding shop are compared with the results of the control group in Table 5.

**TABLE 5:** Respiratory functions measured for welders

| Respiratory Functions | Total Experiment n = 36 | Total Control n = 33 | Smokers Experiment n = 20 | Smokers Control n = 22 | Non-smoke Experiment n = 16 | Non smoke Control n = 11 |
|---|---|---|---|---|---|---|
| FVC | 3.99±1.21 | 3.69±0.85 | 3.83±1.23 | 3.76±0.86 | 4.20±1.18 | 3.55±0.84 |
| $FEV_1$ | 3.39±0.92 | 3.21±0.71 | 3.18±0.89 | 3.29±0.74 | 3.67±0.91 | 3.06±0.63 |
| PEF | 8.11±2.08 | 8.09±2.27 | 7.36±2.19 | 8.01±2.31 | 9.12±1.44 | 8.27±2.30 |
| PIF | 5.70±3.56 | 6.42±2.16 | 5.19±3.55 | 5.87±2.44 | 6.56±3.57 | 7.01±1.80 |
| $FEF_{25-75}$ | 3.87±1.24 | 3.93±1.07 | 3.47±1.27 | 4.01±1.18 | 4.41±1.00 | 3.75±0.80 |
| $V_{MAX25}$ | 6.77±1.84 | 7.17±2.07 | 6.02±1.76 | 7.04±2.17 | 7.77±1.48 | 7.45±1.93 |
| $V_{MAX50}$ | 4.30±1.31 | 4.61±1.22 | 3.91±1.37 | 4.59±1.38 | 4.81±1.05 | 4.66±0.85 |
| $V_{MAX75}$ | 2.11±0.83 | 2.06±0.81 | 1.89±0.82 | 2.03±0.67 | 2.41±0.77 | 2.12±1.08 |
| $FET_{100}$ | 2.21±1.07 | 3.50±2.12 | 2.45±1.28 | 3.18±1.78 | 1.89±0.63 | 4.16±1.69 |
| $FEV_1$/FVC % | 86.3±9.3 | 87.4±5.7 | 84.48±10.79 | 87.62±5.17 | 88.7±6.66 | 87.05±7.02 |

## 3.3 Aerosol concentrations

### 3.3.1 Foundry

The results obtained after analyses of the streaker samples in the foundry are depicted in Figures 2 to 9. For the purpose of exposure characterisation, a daily logsheet (Table 6) of the activities inside the foundry was compiled. The workers have the following daily routine:

**TABLE 6:** Daily routine of foundry workers

| Time | Activity |
|---|---|
| 06:50 | Safety talk |
| 07:00 | Start working in foundry |
| 08:50 | Tea time. Process stops |
| 09:00 | Resume duties |
| 11:55 | Lunch break. Process stops |
| 12:30 | Resume duties |
| 14:50 | Tea time. Process stops |
| 15:00 | Resume duties |
| 16:20 | Shut off systems if work is finished |
| | Workers leave for home |
| 18:00 | Start of night shift |
| 02:00 | Shut off systems |
| | Workers leave for home |

All the alloys are tested before the molten metal is poured into the moulds. The results of the alloy composition were obtained from the in-house laboratory which analyses metal samples for the foundry. The composition of the different products are shown in Table 7.

**TABLE 7:** Chemical composition ( %) of products manufactured

| Brake Blocks | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Date** | **P** | **S** | **Al** | **C** | **Cr** | **Fe** | **Si** | **Mn** | **Sn** | **V** | **Ni** |
| **8 May** | 0.11 | 0.09 | | 3.30 | 0.05 | 94.10 | 1.55 | 0.70 | 0.02 | | |
| **9 May** | 0.12 | 0.10 | | 3.16 | 0.05 | 94.30 | 1.53 | 0.72 | 0.02 | | |
| **10 May** | 0.09 | 0.14 | | 3.00 | 0.06 | 94.60 | 1.20 | 0.86 | 0.02 | | |
| **11 May** | 0.09 | 0.10 | | 3.60 | 0.05 | 94.00 | 1.28 | 0.77 | 0.02 | | |
| **12 May** | 0.11 | 0.10 | | 3.20 | 0.06 | 94.10 | 1.51 | 0.82 | 0.02 | | |
| **13 May** | 0.14 | 0.09 | | 3.32 | 0.06 | 94.20 | 1.51 | 0.63 | 0.02 | | |
| **Crusher Balls** | | | | | | | | | | | |
| **Date** | **P** | **S** | **Al** | **C** | **Cr** | **Fe** | **Si** | **Mn** | **Sn** | **V** | **Ni** |
| **8 May** | 0.02 | 0.02 | 0.03 | 1.45 | 0.56 | 95.99 | 0.54 | 1.15 | | 0.02 | 0.05 |
| **9 May** | 0.03 | 0.03 | 0.03 | 1.61 | 0.61 | 95.48 | 0.70 | 1.27 | | 0.02 | 0.05 |
| **10 May** | 0.04 | 0.03 | 0.03 | 1.51 | 0.54 | 95.99 | 0.49 | 1.13 | | 0.02 | 0.04 |
| **11 May** | 0.04 | 0.05 | 0.03 | 1.64 | 0.58 | 95.34 | 0.58 | 1.27 | | 0.01 | 0.04 |
| **12 May** | 0.03 | 0.03 | 0.03 | 1.64 | 0.63 | 95.85 | 0.55 | 1.00 | | 0.01 | 0.04 |
| **13 May** | 0.03 | 0.03 | 0.03 | 1.47 | 0.54 | 96.15 | 0.54 | 1.00 | | 0.01 | 0.04 |

**Total inorganic fumes:** The exposure is shown in Figure 2. Although peaks appear to be higher during normal working hours, no specific pattern of exposure is shown. The results showed a time weighted average (TWA) concentration of 211.0 $\pm$203.3 µg/m$^3$ with a maximum value of 871.9 and a minimum value of 0.1 µg/m$^3$. A 95 % confidence limit of 30.6 µg/m$^3$ and a 99 % confidence limit of 40.4 µg/m$^3$ were computed from the values.

**Chromium:** The measured chromium concentrations with a TWA concentration of 0.2 $\pm$0.1 µg/m$^3$ are shown in Figure 3A. The maximum concentration was 1.0 µg/m$^3$ and the minimum concentration 0 µg/m$^3$. The corresponding $CrO_3$ concentration was 1.9 µg/m$^3$. Although peaks appear to be higher during normal working hours, no specific pattern of exposure is shown.

**Copper:** The copper concentrations measured reached a maximum concentration of 0.8 µg/m$^3$ with a minimum concentration of 0 µg/m$^3$. The results are shown in Figure 3B. The measured values showed a TWA concentration of 0.1 $\pm$ 0.1 µg/m$^3$. Once again, the peaks appear to be higher during normal working hours, but no specific pattern of exposure could be established. The maximum CuO concentration was 1.0 µg/m$^3$.

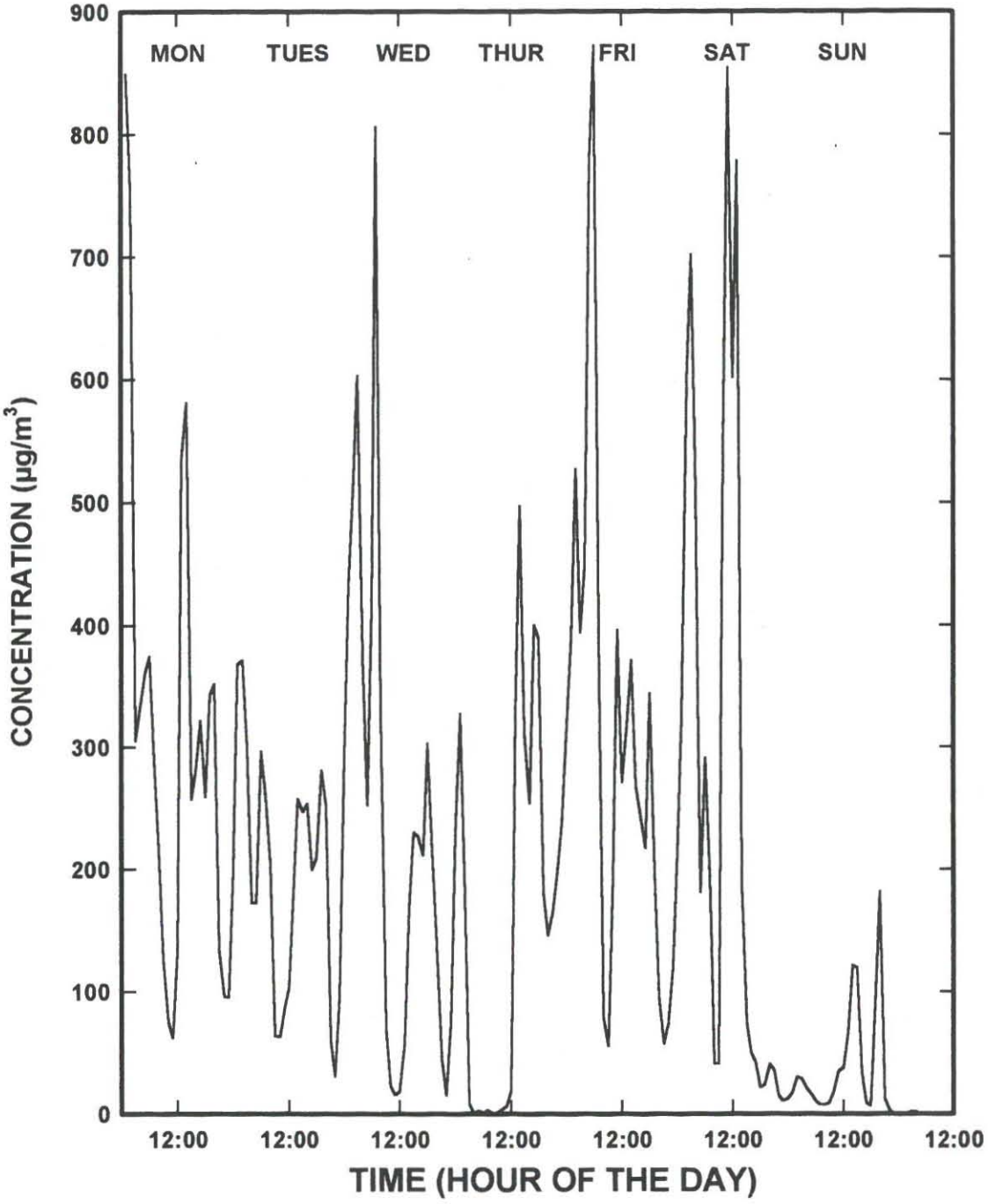


**FIGURE 2: Total respirable inorganic dust concentrations (µg/m$^3$) measured in foundry air.**

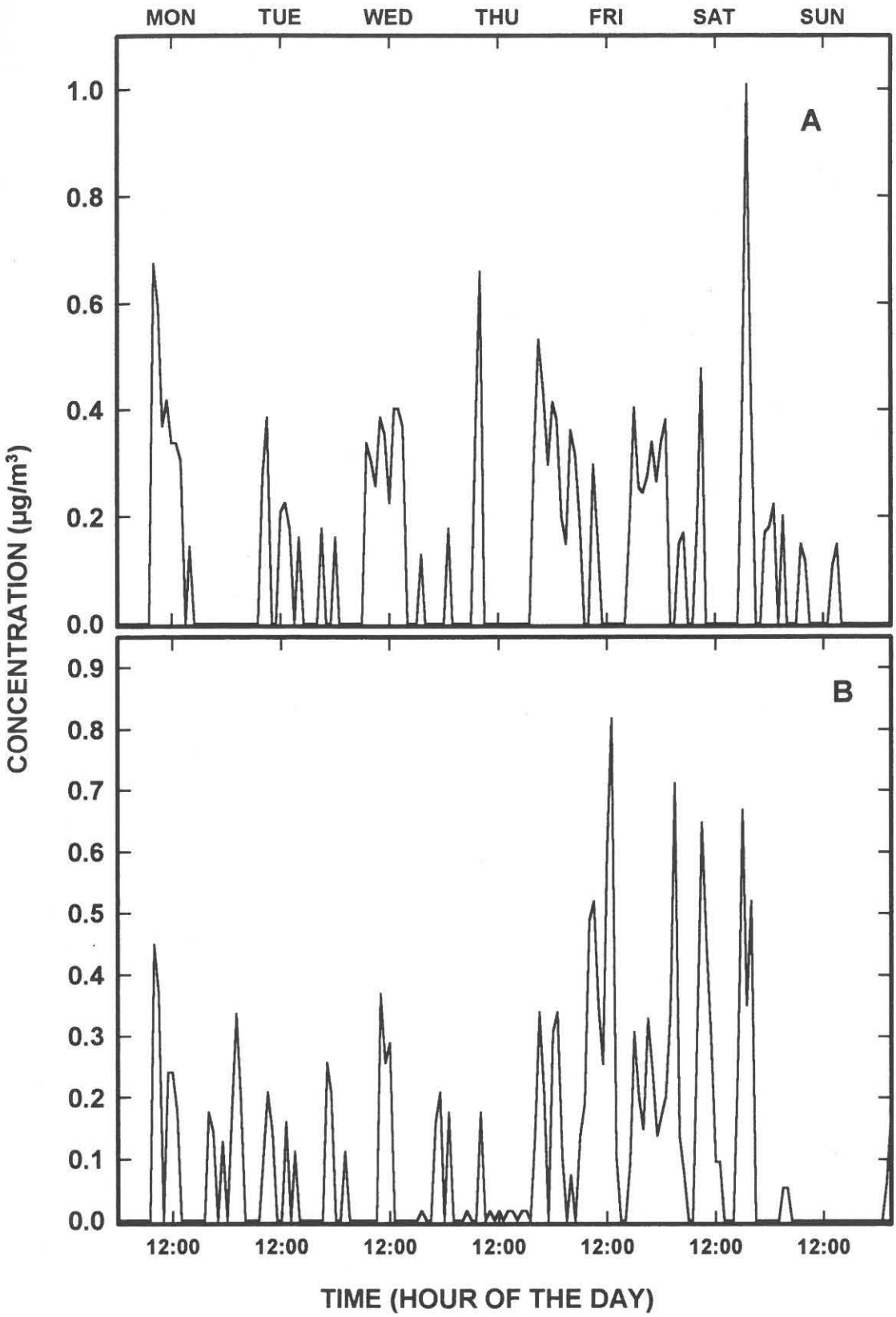


**FIGURE 3: A: Respirable dust Cr-concentrations (µg/m$^3$) measured in foundry air.**
**B: Respirable dust Cu-concentrations (µg/m$^3$) measured in foundry air.**

**Iron:** Iron concentrations were the second highest concentrations measured with a TWA concentration of 51.8 ± 50.6 µg/m$^3$. It had a maximum concentration of 216.2 µg/m$^3$ with a minimum value of 0 µg/m$^3$. The iron exposure is depicted in Figure 4A. The highest $Fe_2O_3$ concentration was 309.7 µg/m$^3$.

**Manganese:** Manganese concentrations are shown in Figure 4B. Manganese concentrations ranged between 0 and 78.5 µg/m$^3$ with a TWA concentration of 12.2 ± 15.1 µg/m$^3$. The corresponding maximum value for $MnO_2$ was 124.3 µg/m$^3$.

**Chlorine:** The chlorine concentrations measured are shown in Figure 5A. It showed a TWA concentration of 2.3 ± 2.8 µg/m$^3$ with a maximum value of 16.0 µg/m$^3$. The concentration dropped to 0 µg/m$^3$ during some events.

**Zinc:** Measured Zn-concentrations are shown in Figure 5B. It ranged from 0 to 32.6 µg/m$^3$ and a mean concentration of 5.2 ± 6.5 µg/m$^3$ was calculated. The equivalent TWA concentration ZnO was 8.0 µg/m$^3$. ZnO exhibited a maximum concentration of 40.6 µg/m$^3$.

**Lead:** Lead-concentrations varied between 0 and 6.8 µg/m$^3$ with a TWA concentration of 0.9 ± 1.2 µg/m$^3$. The Pb-concentrations are shown in Figure 5C. The highest PbO exposure was 7.0 µg/m$^3$.

**Bromine:** Bromine concentrations in comparison with the measured Pb concentrations, are shown in Figure 6. This substance, normally used as a petrol additive showed a TWA concentration of 0.2 ± 0.4 µg/m$^3$ and a maximum concentration of 4.2 µg/m$^3$. The concentration dropped to 0 µg/m$^3$ at times.

**Aluminium:** Aluminium concentrations are shown in Figure 7A. The concentration ranged between 0 and 162.5 µg/m$^3$ with a TWA concentration of 30.2 ± 33.7 µg/m$^3$. $Al_2O_3$ concentrations ranged from 0 to 306.9 µg/m$^3$.

**Magnesium:** The Mg-exposure showed a TWA concentration of 7.5 ± 11.8 µg/m$^3$ with a maximum value of 104.6 µg/m$^3$. The Mg-concentrations are depicted in Figure 7B.

**Silicon:** Silicon was the element with the highest concentrations measured during the sampling period. It ranged from 0 to 456.6 µg/m$^3$ with a TWA concentration of 89.0 ± 96.3 µg/m$^3$. The exposure is shown in Figure 7C. The corresponding maximum value for $SiO_2$ was 978.4 µg/m$^3$. The analysis also included the

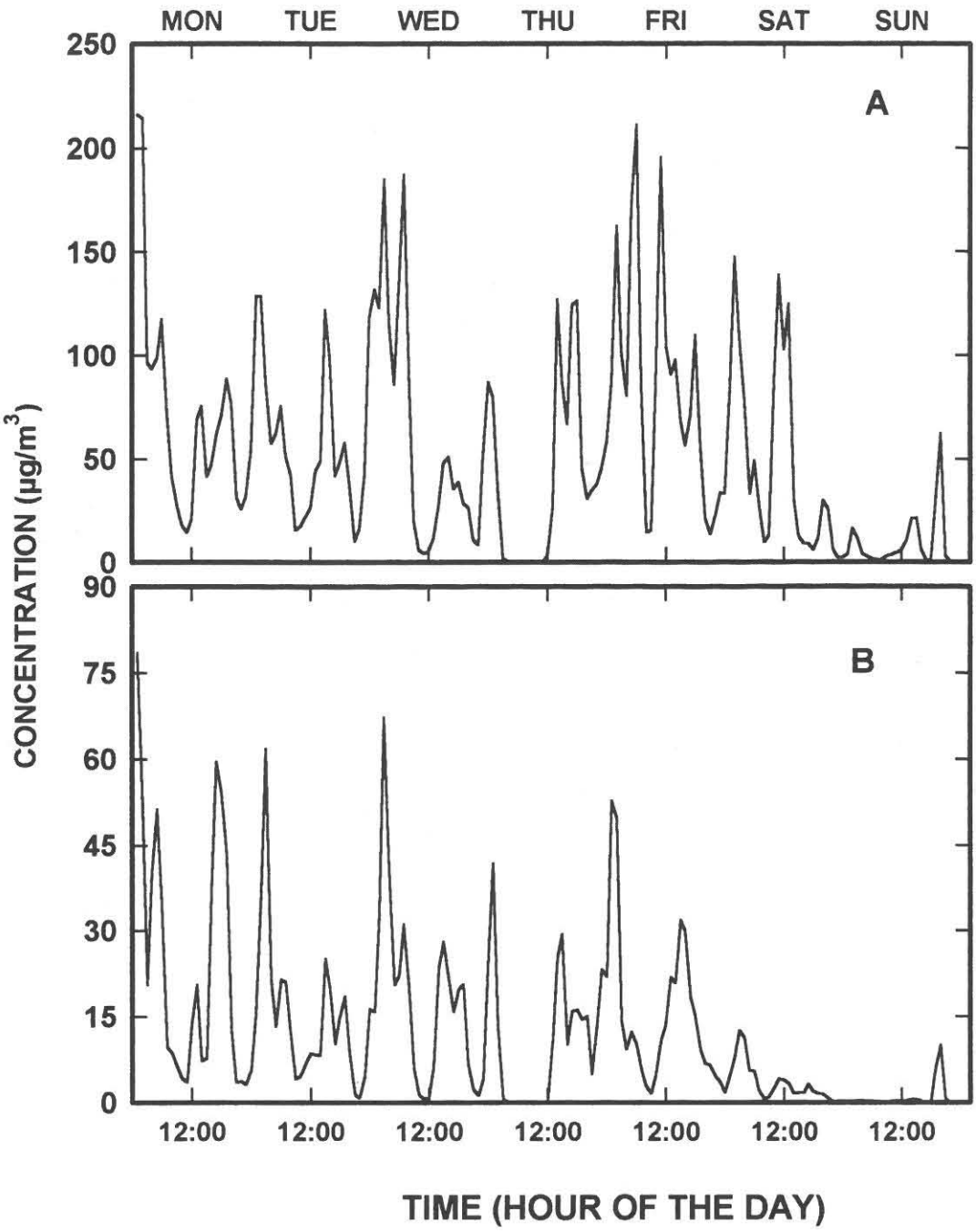


**FIGURE 4:** A: Respirable dust Fe-concentrations (µg/m$^3$) measured in foundry air.
B: Respirable dust Mn-concentrations (µg/m$^3$) measured in foundry air.

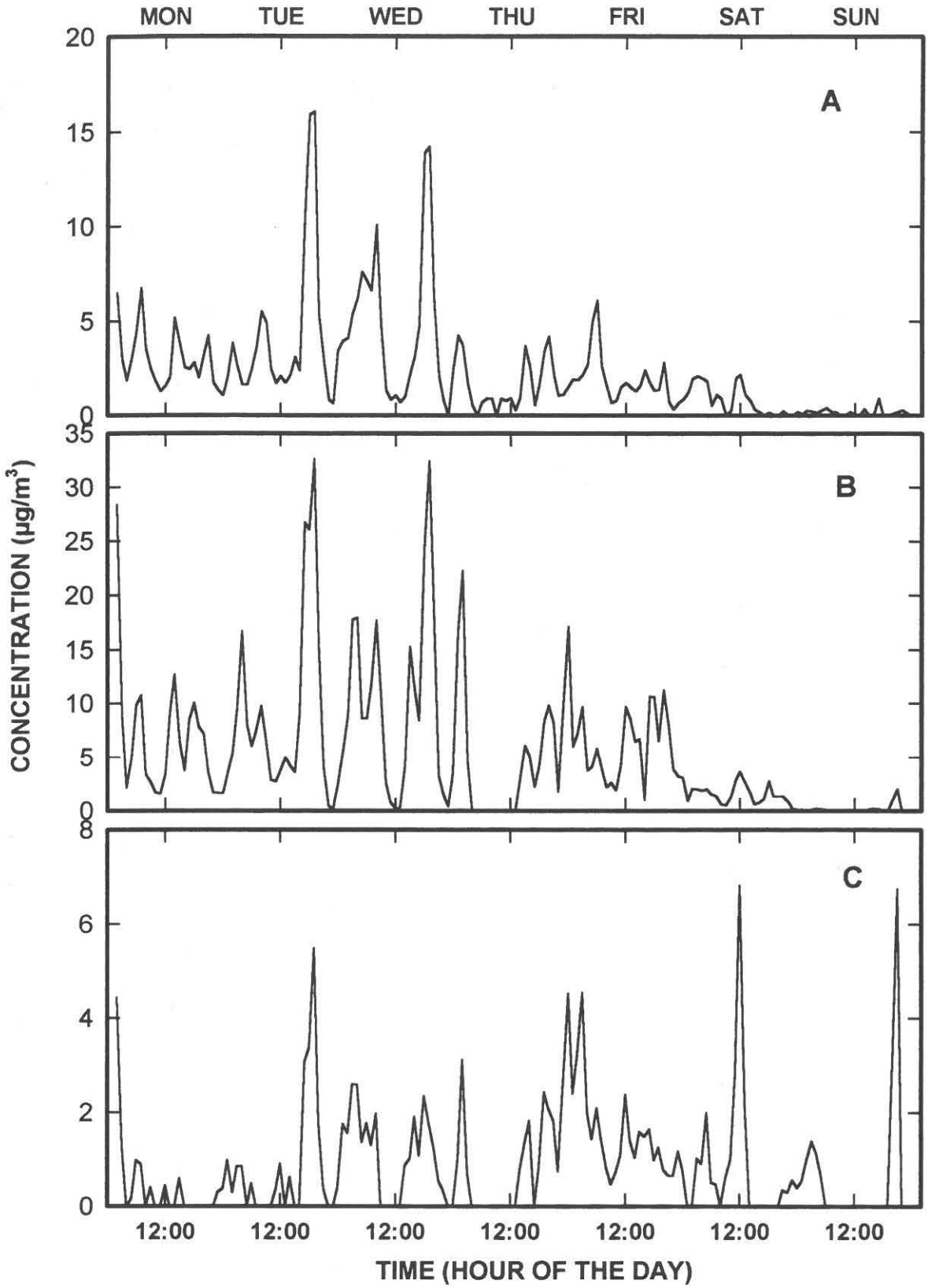


**FIGURE 5:** A: Respirable dust Cl-concentrations (µg/m$^3$) measured in foundry air.
B: Respirable dust Zn-concentrations (µg/m$^3$) measured in foundry air.
C: Respirable dust Pb-concentrations (µg/m$^3$) measured in foundry air.

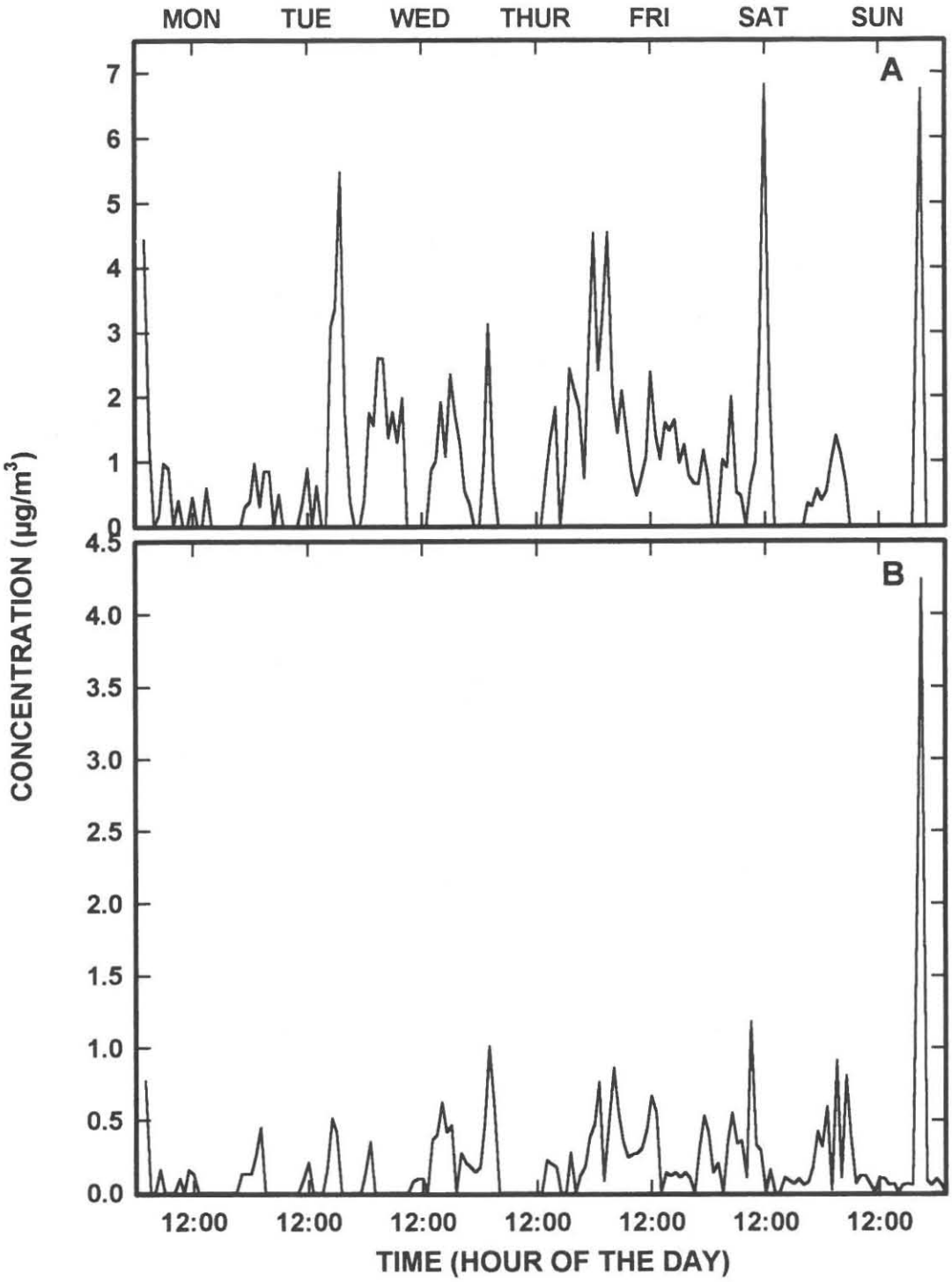


**FIGURE 6:** **A: Respirable dust Pb-concentrations (µg/m$^3$) measured in foundry air.**
**B: Respirable dust Br-concentrations (µg/m$^3$) measured in foundry air.**

percentages of $\alpha$-quartz and tridymite which were 14 % and 0.4 % respectively. The maximum concentrations for these two forms of silica were 137.0 and 3.9 µg/m$^3$.

**Potassium:** Potassium concentrations ranged from 0 to 7.5 µg/m$^3$ with a TWA concentration of 1.8 $\pm$1.6 µg/m$^3$. K-concentrations are shown in Figure 7D.

**Calcium:** Calcium concentrations are shown in Figure 7E with a TWA concentration of 5.2 $\pm$5.4 µg/m$^3$. The maximum value obtained during sampling was 28.4 µg/m$^3$.

**Titanium:** Titanium concentrations with a TWA concentration of 0.9 $\pm$1.0 µg/m$^3$ are shown in Figure 7F. It ranged from 0 to 4.9 µg/m$^3$. The maximum $TiO_2$ concentration was 8.3 µg/m$^3$.

**Vanadium:** The measured V-concentrations were below the 7.0 ng/m$^3$ detection limit (Annegarn *et al.*, 1988) of the instrument and considered as negligibly low.

**Nickel:** The Ni-concentrations with a maximum concentration of 0.5 µg/m$^3$ are shown in Figure 8A. $NiO_2$ concentrations ranged from 0 to 0.7 µg/m$^3$.

**Sulphur:** Sulphur concentrations ranged from 0 to 15.5 µg/m$^3$ with a TWA concentration of 3.3 $\pm$2.1 µg/m$^3$. The S-exposure is depicted in Figure 8B.

**Source apportionment:** A source apportionment was done on the results from the foundrys' measurement during the sampling period and are depicted in Figure 9.

### 3.3.2 Welding shop

The results obtained after analyses of the streaker samples in the welding shop are depicted in Figures 10 to 17. For the purpose of exposure characterisation, a daily logsheet of the activities inside the welding shop was compiled. The workers have the following daily routine (Table 8):

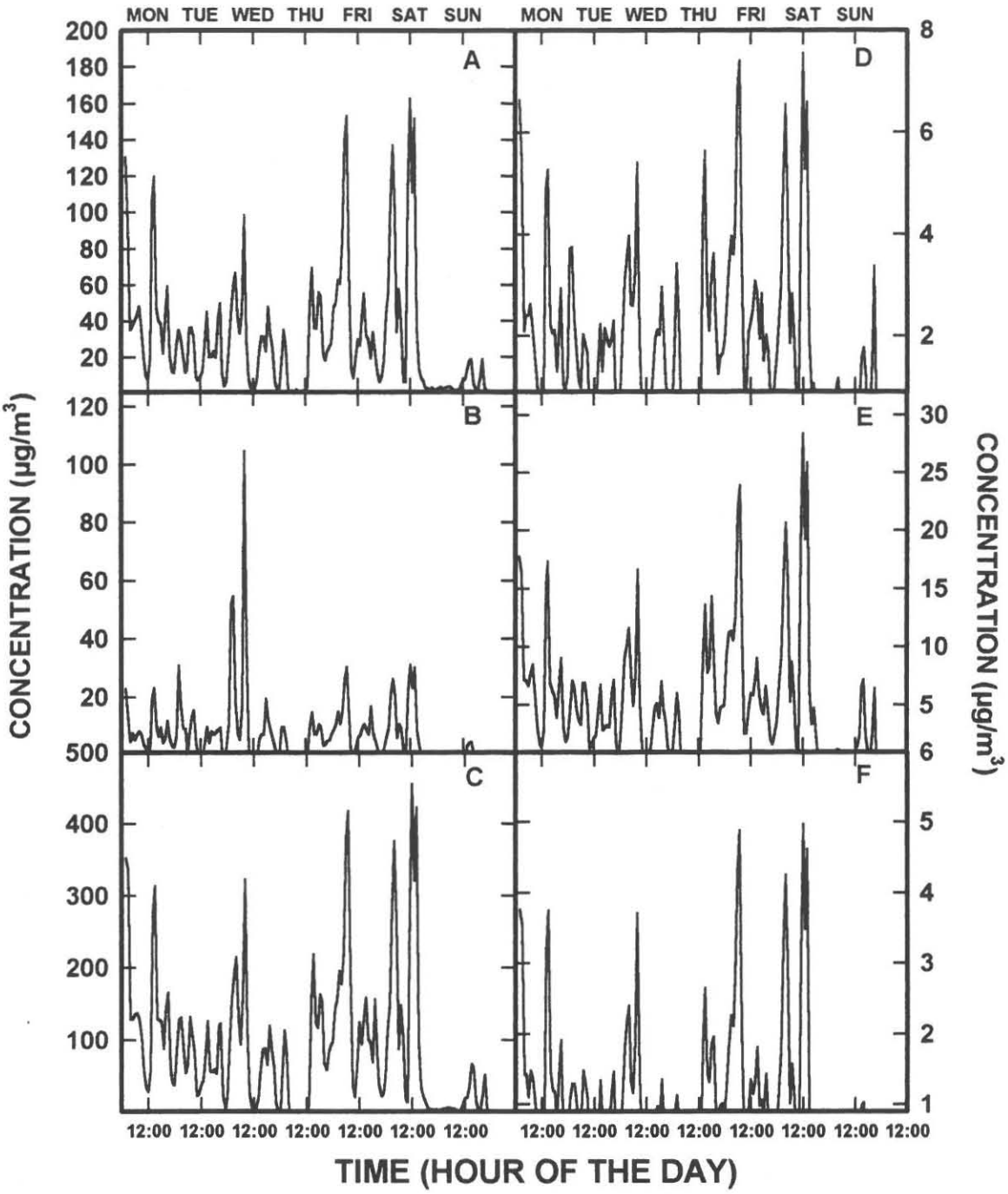


**FIGURE 7:**

**A: Respirable dust Al-concentrations (µg/m$^3$) measured in foundry air.**
**B: Respirable dust Mg-concentrations (µg/m$^3$) measured in foundry air.**
**C: Respirable dust Si-concentrations (µg/m$^3$) measured in foundry air.**
**D: Respirable dust K-concentrations (µg/m$^3$) measured in foundry air.**
**E: Respirable dust Ca-concentrations (µg/m$^3$) measured in foundry air.**
**F: Respirable dust Ti-concentrations (µg/m$^3$) measured in foundry air.**

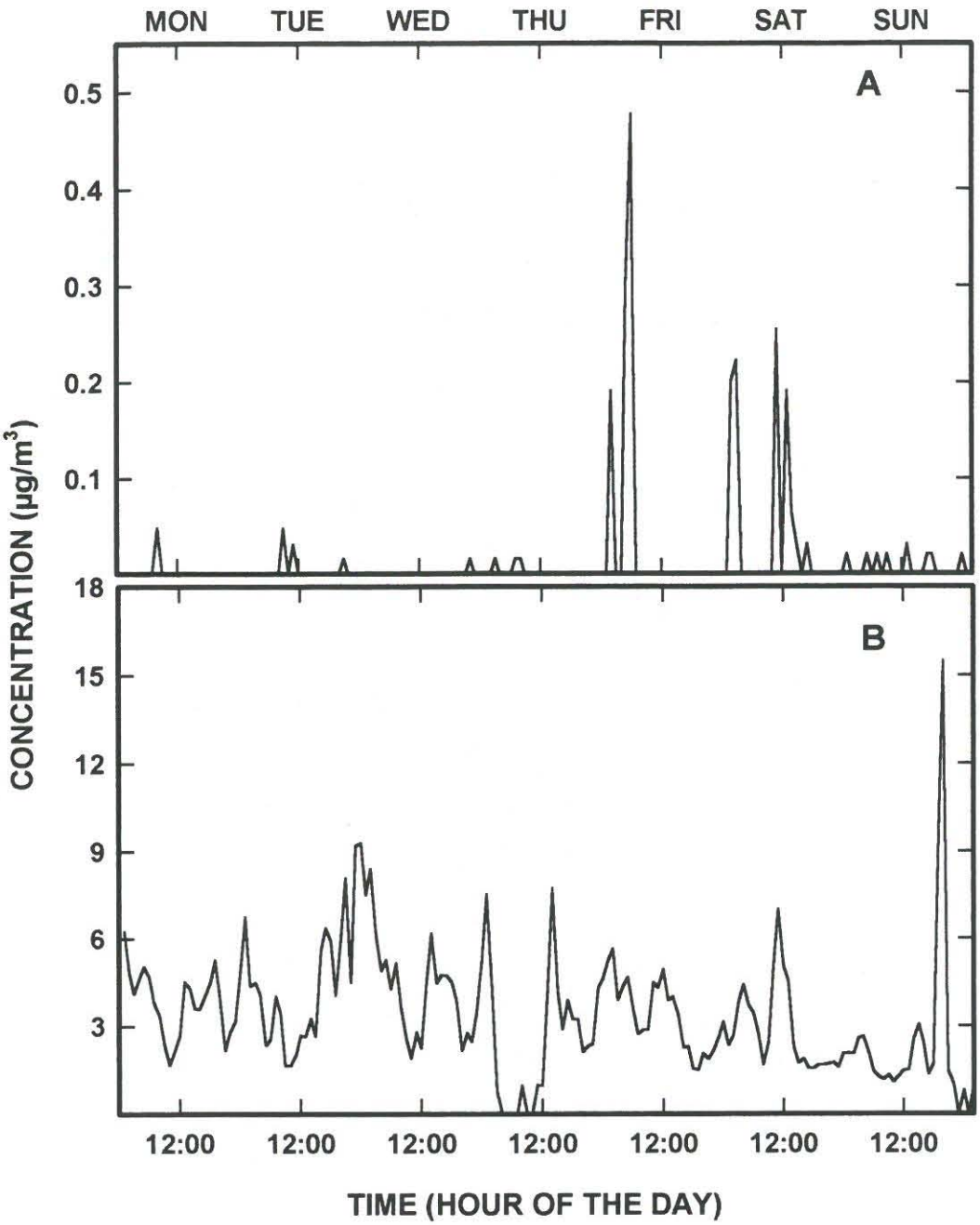


**FIGURE 8:** **A: Respirable dust Ni-concentrations (µg/m$^3$) measured in foundry air.**
**B: Respirable dust S-concentrations (µg/m$^3$) measured in foundry air.**

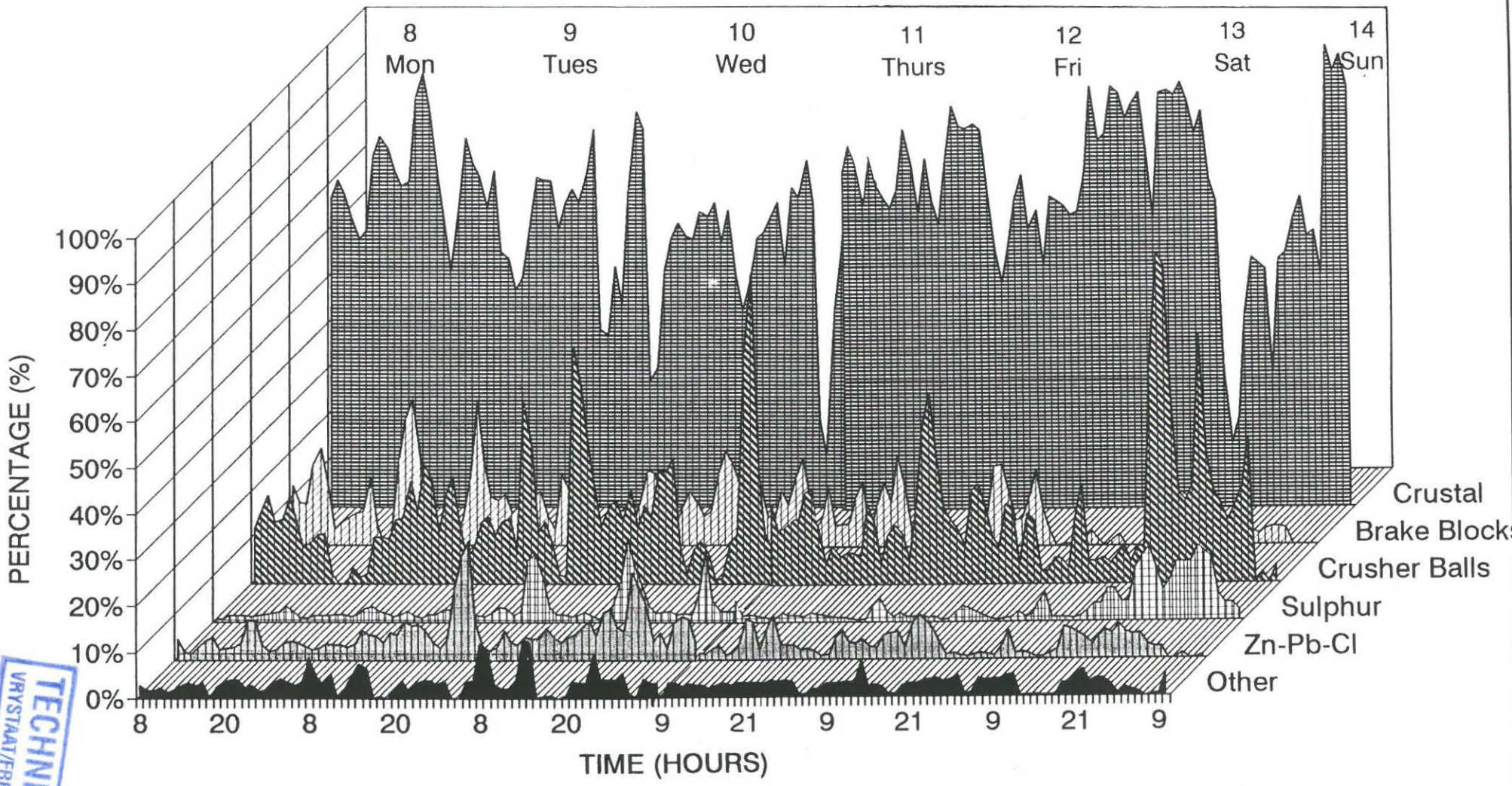


**FIGURE 9:** **Concentration of the different sources in the air of the foundry expressed as a percentage of the total exposure during the sampling period**

**TABLE 8:** Daily routine of welders

| Time | Activity |
|---|---|
| 06:50 | Safety talk |
| 07:00 | Start working in welding shop |
| 08:50 | Tea time. Process stops |
| 09:00 | Resume duties |
| 11:55 | Lunch break. Process stops |
| 12:30 | Resume duties |
| 14:50 | Tea time. Process stops |
| 15:00 | Resume duties |
| 16:20 | Shut off systems if work is finished |
| | Workers leave for home |

To obtain the chemical composition of the materials that are used in the welding shop, data were collected from the manufacturers of the different products. Table 9 gives a summary of the composition of the different welding products used.

**TABLE 9:** Chemical composition ( %) of welding products used

| Welding Products | C | Si | Mn | P | S | Cr | Ni | Fe | Cu | Ti | Mo |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 3CR12 Plate | 0.03 | 1.00 | 1.50 | 0.04 | 0.03 | 14.00 | 1.50 | 81.0 | | 0.60 | |
| Stainless Steel Welding Wire | 0.01 | 0.38 | 1.91 | 0.02 | 0.03 | 23.40 | 13.90 | 60.0 | | | 0.07 |
| Corten Steel Plate | 0.12 | 0.75 | 1.30 | 0.15 | 0.05 | 1.25 | 5.65 | 90.0 | 0.55 | | |
| Corten Welding Wire | 0.03 | 0.65 | 2.50 | 0.02 | 0.02 | 25.00 | 14.0 | 57.0 | 0.05 | | |

A few observations were made during the sampling process. Due to the extremely cold condition during winter months, workers burn coal (cooks) and wood in large drums to provide heating in the welding shop. This gives rise to smoke inside the building. Diesel smoke is also present in the workplace as a result of diesel forklifts being used.

In the one corner of the building, workers degrease train parts. The parts are dipped into tanks with a caustic potash (KOH) solution. Heating of the tanks produce steam and irritating fumes.

**Total inorganic fumes:** The exposure is shown in Figure 10. The total inorganic fume concentration ranged from 0 to 781.1 µg/m$^3$ with a TWA concentration of 106.5 ±164.6 µg/m$^3$ during the measurement period. A 95 % confidence limit of 36.6 µg/m$^3$ and a 99 % confidence limit of 48.6 µg/m$^3$ were calculated from the results. The exposure showed a tendency of higher concentrations during mornings.

**Chromium:** The Cr concentrations measured showed a TWA concentration of 10.7 ±17.1 µg/m$^3$ with a maximum concentration of 86.2 µg/m$^3$. The Cr exposure is shown in Figure 11A. Although peaks appear to be higher during normal working hours, no specific pattern of exposure is shown. $CrO_3$ exposure exhibited a maximum concentration of 165.7 µg/m$^3$.

**Copper:** The Cu concentrations measured are shown in Figure 11B. This exposure ranged from 0 to 10.9 µg/m$^3$ with a TWA concentration of 1.1 ±2.0 µg/m$^3$. The maximum CuO concentration was 13.6 µg/m$^3$.

**Iron:** Iron concentrations showed the highest exposure of all measured elements with a maximum concentration of 476.1 µg/m$^3$ and a TWA concentration of 61.6 ±97.9 µg/m$^3$ and are depicted in Figure 12A. The TWA $Fe_2O_3$ concentration was 140.1 µg/m$^3$ with a maximum concentration of 680.9 µg/m$^3$.

**Manganese:** Manganese concentrations are shown in Figure 12B. A TWA concentration of 10.2 ±7.1 µg/m$^3$ with a maximum concentration of 79.5 µg/m$^3$ were obtained. The $MnO_2$ concentration reached a maximum of 125.8 µg/m$^3$.

**Chlorine:** The Cl concentrations measured are shown in Figure 13A. The maximum Cl-concentration was 14.3 µg/m$^3$ with a TWA concentration of 2.3 ±2.8 µg/m$^3$.

**Zinc:** Zinc concentrations fluctuated between 0 and 24.6 µg/m$^3$ with a TWA concentration of 2.9 ±4.8 µg/m$^3$. The highest ZnO concentration was 30.6 µg/m$^3$. Measured Zn-concentrations are shown in Figure 13B.

**Lead:** The Pb-concentrations are shown in Figure 13C. The Pb-concentration ranged between 0 and 9.6 µg/m$^3$ with a TWA concentration of 0.5 ±1.5 µg/m$^3$. PbO concentrations ranged from 0 to 10.4 µg/m$^3$.

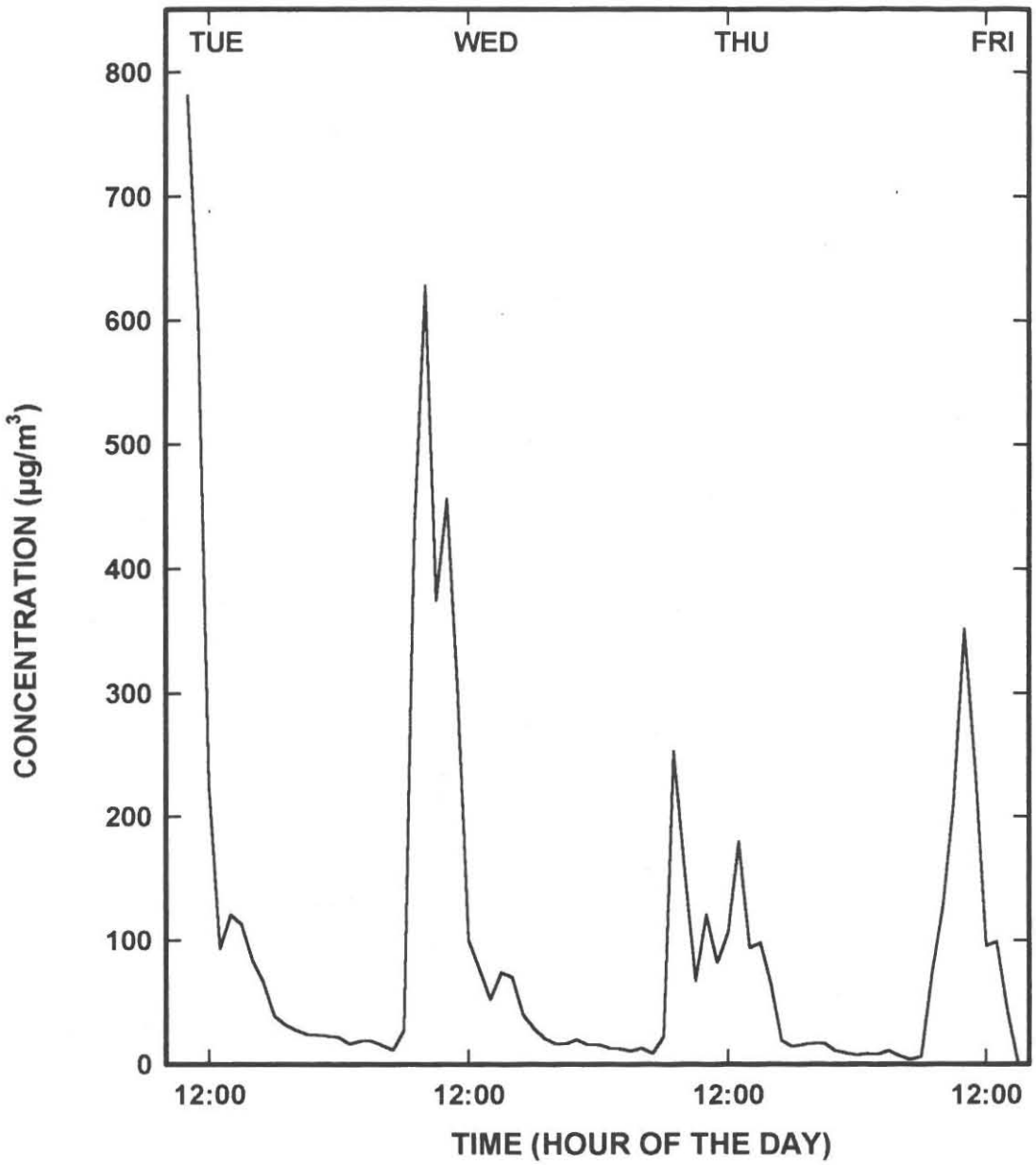


**FIGURE 10: Total respirable inorganic welding fume concentrations (µg/m$^3$).**

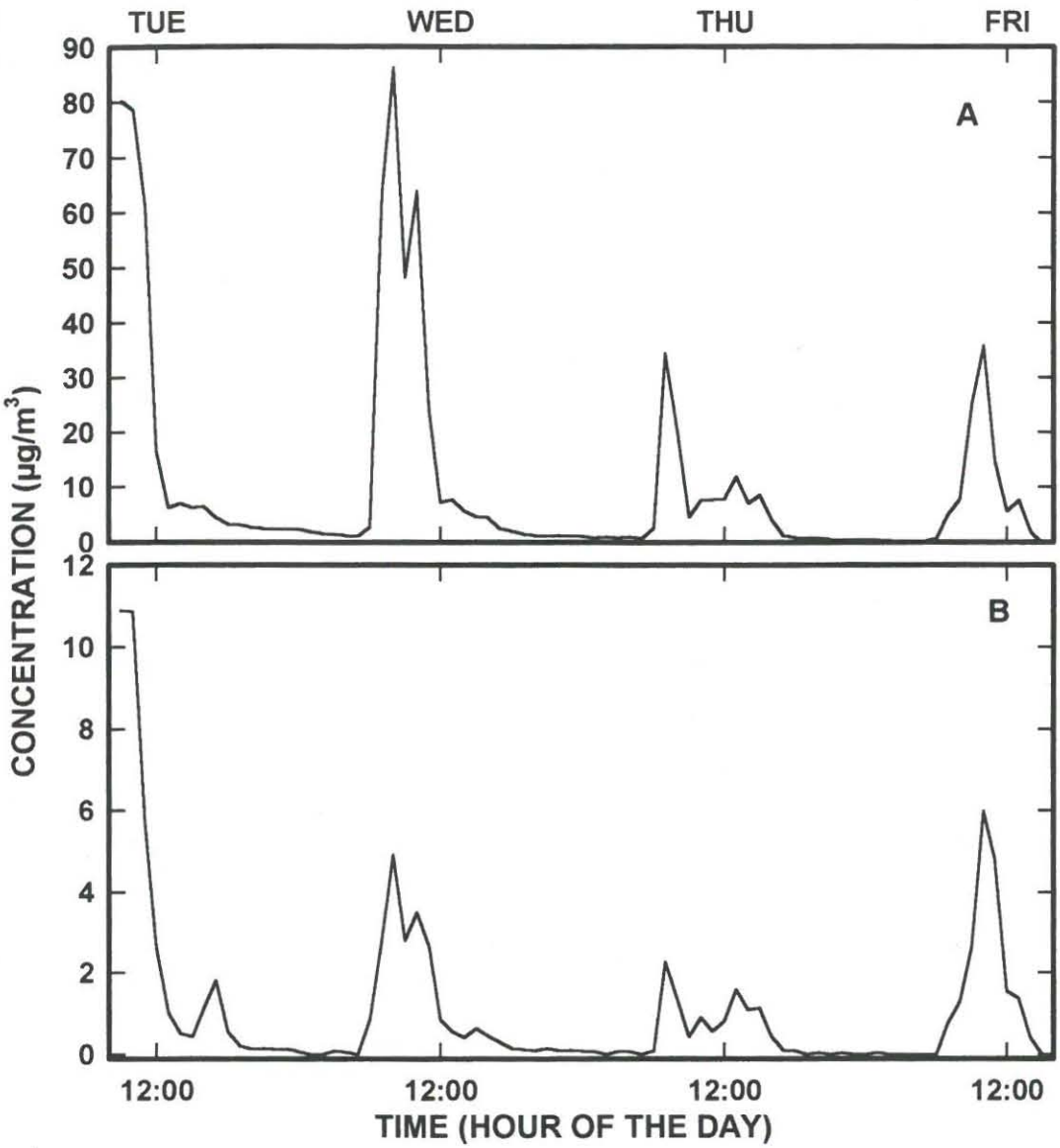


FIGURE 11: A: Respirable dust Cr-concentrations (µg/m$^3$) measured in welding shop air.
B: Respirable dust Cu-concentrations (µg/m$^3$) measured in welding shop air.

**Bromine:** Bromine concentrations are shown in Figure 14B in comparison with the measured Pb concentrations. The maximum concentration of Br was 1.7 $\mu g/m^3$ with a TWA concentration of 0.1 $\pm$0.3 $\mu g/m^3$.

**Aluminium:** Aluminium concentrations are shown in Figure 15A. The maximum value was 7.3 $\mu g/m^3$ with a TWA concentration of 1.2 $\pm$1.5 $\mu g/m^3$. $Al_2O_3$ concentrations showed a maximum value of 13.8 $\mu g/m^3$.

**Magnesium:** A maximum Mg concentration of 7.1 $\mu g/m^3$ with a TWA concentration of 0.2 $\pm$1.0 $\mu g/m^3$ were obtained. Mg-concentrations are shown in Figure 15B.

**Silicon:** Silicon concentrations are shown in Figure 15C. The silicon concentration ranged between 0 and 42.4 $\mu g/m^3$ with a TWA concentration of 6.0 $\pm$8.4 $\mu g/m^3$. No $\alpha$-quartz or tridymite was found in the welding fume samples. The value for these two substances can thus be regarded as zero.

**Potassium:** Potassium concentrations are shown in Figure 15D. Potassium exposure ranged from 0 to 18.1 $\mu g/m^3$ with a TWA concentration of 3.3 $\pm$4.6 $\mu g/m^3$.

**Calcium:** Calcium exposure exhibited a maximum concentration of 7.5 $\mu g/m^3$ and a TWA concentration of 1.4 $\pm$1.9 $\mu g/m^3$. Ca-concentrations are shown in Figure 15E.

**Titanium:** Titanium concentrations are shown in Figure 15F. Ti-concentrations were one of the lowest exposures with a maximum concentration of 1.3 $\mu g/m^3$ and a TWA concentration of 0.2 $\pm$0.4 $\mu g/m^3$. $TiO_2$ showed a maximum concentration of 2.2 $\mu g/m^3$.

**Vanadium:** Vanadium concentrations were below the 7.0 $ng/m^3$ detection limit (Annegarn *et al.*, 1988) of the instrument and therefore considered as negligibly low.

**Nickel:** The Ni-concentrations with a maximum concentration of 27.1 $\mu g/m^3$ are shown in Figure 16A. The corresponding maximum concentration for $NiO_2$ was 41.8 $\mu g/m^3$.

**Sulphur:** Sulphur concentrations ranged from 0 to 8.6 $\mu g/m^3$ with a TWA concentration of 2.7 $\pm$1.7 $\mu g/m^3$. The S-exposure is depicted in Figure 16B.

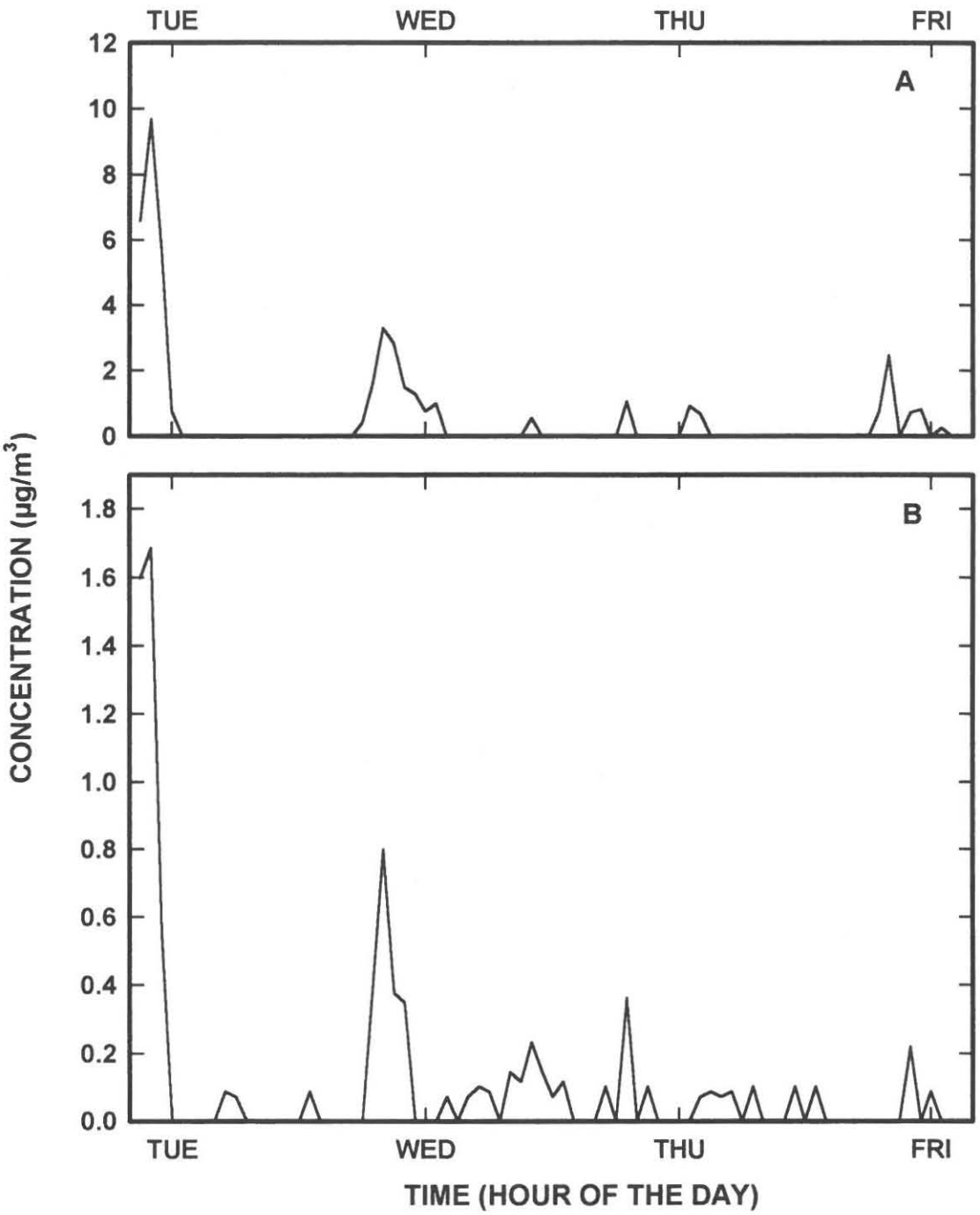


**FIGURE 14:** **A: Respirable dust Pb-concentrations (µg/m$^3$) measured in welding shop air.**
**B: Respirable dust Br-concentrations (µg/m$^3$) measured in welding shop air.**

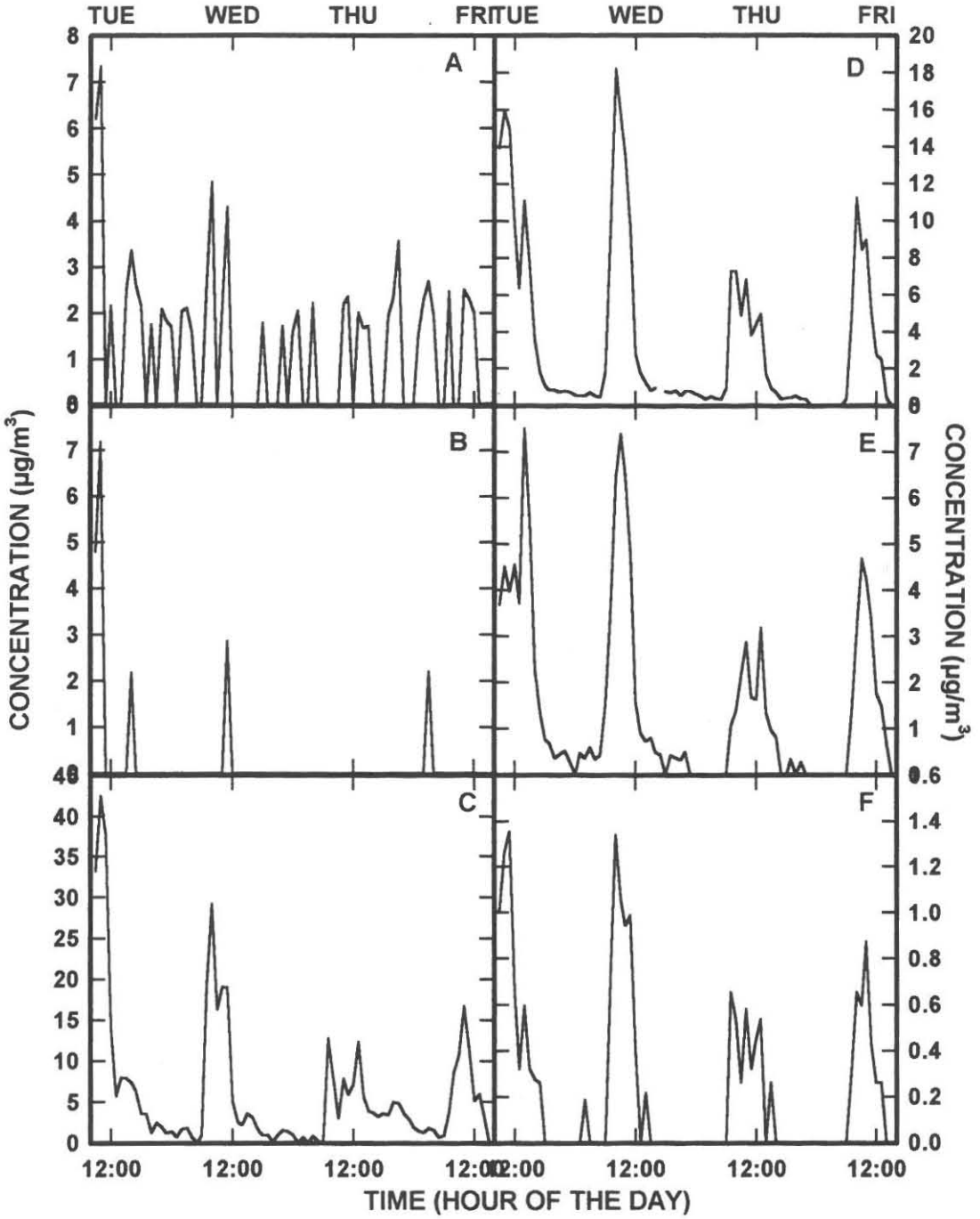


FIGURE 15: A: Respirable dust Al-concentrations (µg/m$^3$) measured in welding shop air.
B: Respirable dust Mg-concentrations (µg/m$^3$) measured in welding shop air.
C: Respirable dust Si-concentrations (µg/m$^3$) measured in welding shop air.
D: Respirable dust K-concentrations (µg/m$^3$) measured in welding shop air.
E: Respirable dust Ca-concentrations (µg/m$^3$) measured in welding shop air.
F: Respirable dust Ti-concentrations (µg/m$^3$) measured in welding shop air.

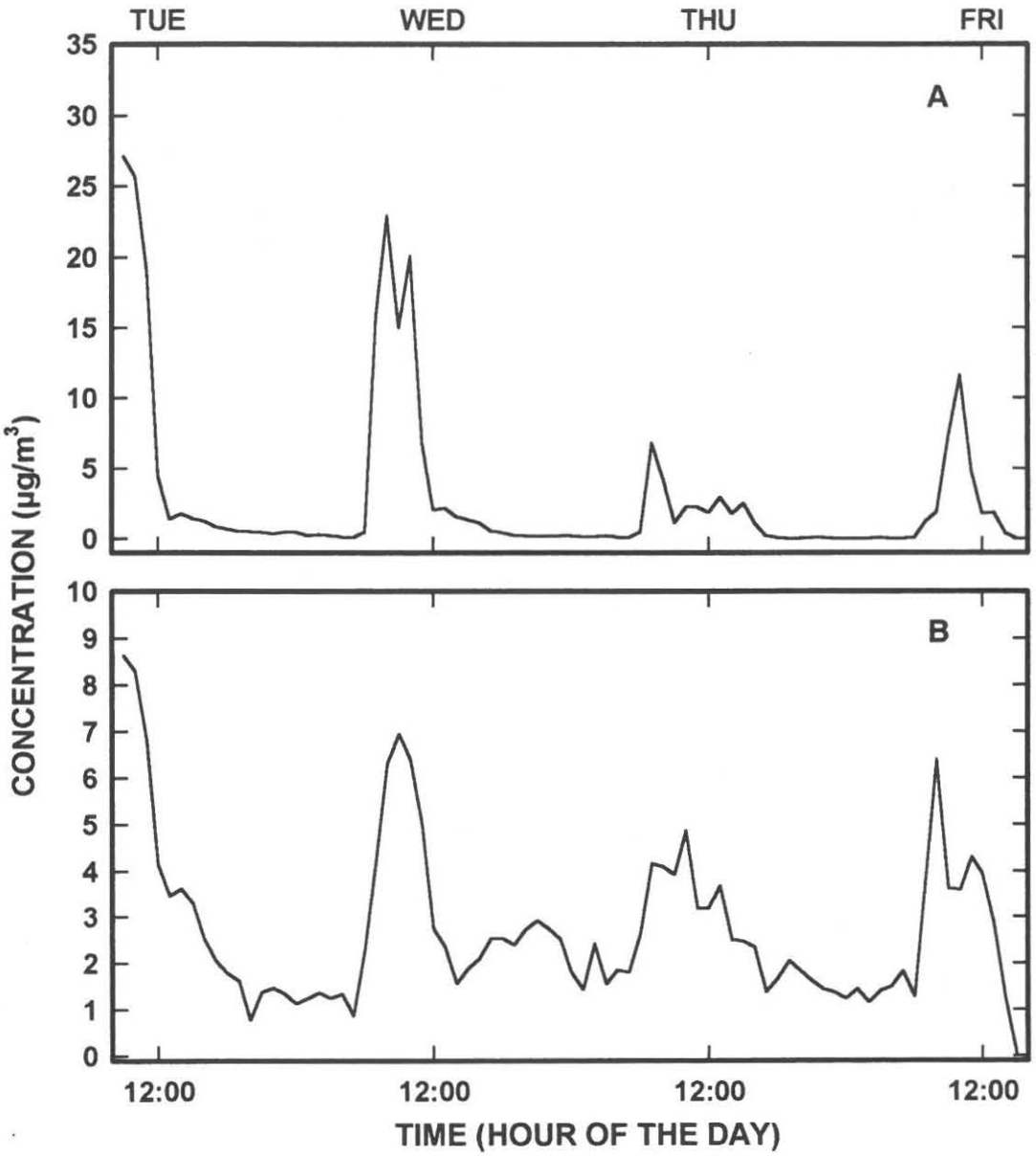


**FIGURE 16:** **A: Respirable dust Ni-concentrations (µg/m$^3$) measured in welding shop air.**
**B: Respirable dust S-concentrations (µg/m$^3$) measured in welding shop air.**

**Source apportionment:**

A source apportionment was done on the results from the welding shops' measurement and depicted in Figure 17.

## 3.4 Weather data

**TABLE 10:** Results of weather conditions during study period

| DATE | TEMPERATURE (° C) | | WIND SPEED m/s | CLOUD CONDITION (overcast) | | |
|---|---|---|---|---|---|---|
| | MIN | MAX | | 08:00 | 14:00 | 20:00 |
| 2 May | 8.0 | 22.0 | 0.52 | Slight | Moderate | Moderate |
| 3 May | 5.8 | 21.3 | 0.71 | | Moderate | |
| 4 May | 6.2 | 22.0 | 0.35 | Slight | Moderate | Moderate |
| 5 May | 8.1 | 20.5 | 0.40 | | Heavy | Slight |
| 8 May | 3.0 | 17.3 | 0.36 | | Heavy | Slight |
| 9 May | 2.6 | 19.7 | 0.65 | Slight | Slight | |
| 10 May | 3.9 | 20.5 | 0.57 | Slight | Slight | |
| 11 May | 3.8 | 22.2 | 0.81 | Slight | Moderate | |
| 12 May | 3.9 | 22.9 | 1.57 | | | |
| 13 May | 2.8 | 21.6 | 0.85 | Slight | Slight | |
| 14 May | 2.9 | 21.5 | 1.76 | | | |

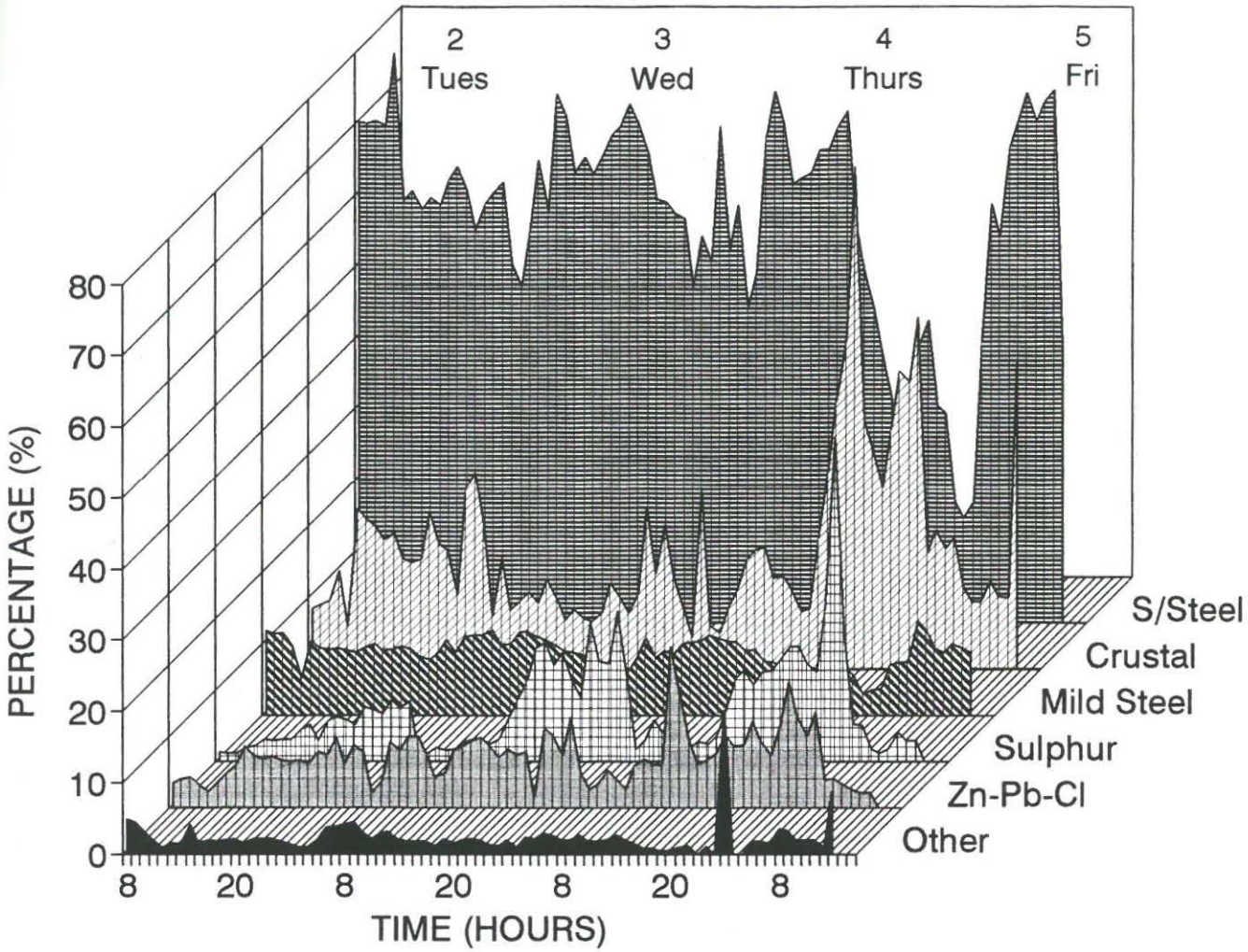


**FIGURE 17: Concentration of the different sources in the air of the welding shop expressed as a percentage of the total exposure during the sampling period**

# 4. Discussion

Recognition of occupational illness has largely depended on the alert clinician, suspecting an association between work and a patients' health situation. Isolated cases of rare disease or clusters of common illness associated with a specific occupation sometimes have been the first clue that work exposure may be causally related to a specific disease. Percivall Pott initiated the concept of occupational cancer when he observed in eighteenth century England that work as a chimney sweep was associated with death at an early age from scrotal cancer (Letz, 1991; Schoeman and Schröder, 1994).

Occupational disease, by definition, is preventable. For the individual, an accurate diagnosis should result in the elimination of further overexposure, thereby reducing the risk of irreversible health effects. Early diagnosis could ensure that the medical treatment will be successful. Identification of occupational disease, in a patient, would imply modifications in environmental working conditions or practices, thus keeping exposures within acceptable limits.

The first step in the process of hazard evaluation is to identify potentially dangerous materials (Letz, 1991). The lack of information on exposure of metal workers to airborne substances, initiated this study. A toxicological profile of each ingredient as compared to basic toxicological information on many commonly used industrial substances is proposed.

Many environmental substances are absorbed via the respiratory tract, making the identification of respiratory disorders prominent in this occupationally related study. The association of respiratory disease with workplace exposures may be complicated by the effects of smoking, non-specific allergic reactions or viral infections. The pathological response, however is only rarely specific or pathognomonic of occupational exposure. Moreover, many disorders of the respiratory tract may be affected by occupational factors. Thus, the diagnosis of a work-related respiratory illness depends on careful integration of clinical, epidemiological and industrial hygiene data such as aerosol characterisation.

A number of chemical agents have been reported to cause acute renal failure after high-dose exposure. Relatively few chemical exposures, however, have been systematically evaluated for more subtle renal effects and only a handful have been positively associated with chronic renal disease (Letz, 1991). The situation is

puzzling, given the number of known or suspect nephrotoxins in widespread industrial use, and given the many theoretical reasons why the kidney should be highly susceptible to the presence of environmental toxins in the body. Biological monitoring of urine was done since this parameter is directly related to adverse health effects (Lauwerys and Hoet, 1993b). It therefore represents a better estimate of the risk than ambient monitoring, because it takes into consideration absorption by routes in addition to the lungs such as the skin or the gastrointestinal tract.

This study provides information on the identification of hazardous substances to which workers are chronically exposed. Furthermore, it provides information on biological parameters for comparison with known limits and finally, it contributes to the estimation of the workers health risk by the integration of clinical, epidemiological and industrial hygiene data.

## 4.1 Urine concentrations

### 4.1.1 Zinc

Zinc in blood and urine has been used as a biological indicator for the assessment of occupational exposure (Cirla *et al.*, 1978; D'Andrea *et al.*, 1981; Bruzzone *et al.*, 1983; Trevisan, Buzzo and Gori, 1983). Although the levels observed are generally significantly higher compared to the controls, no correlation between these values and the levels of exposure or toxic effect has yet been established, and no biological threshold is proposed by the literature (Lauwerys and Hoet, 1993b). Fortunately, Zn has a reputation for being one of the less toxic metals (Harris, 1991).

Reference values of 2.8 - 13.0 µmol/ℓ with a toxic concentration of > 18.4 µmol/ℓ were proposed for urine-Zn in adults (Baselt, 1980; Jacob, 1981). The Zn exposure of both localities is depicted in Figure 18A. No significant differences exist at the 0.05 level after a student *t*-test was carried out. The test resulted in $P = 3.997 \times 10^{-1}$ and $t = 10.61 \times 10^{-1}$. However, the small sample size may have an influence on the significance of the *t*-test values.

**Foundry:** A mean value of 24.0 ± 11.0 µmol/ℓ was calculated for the urine analyses of foundry workers. Since the proposed toxic concentration of Zn is > 18.4 µmol/ℓ (Baselt, 1980), the workers show overexposure to Zn as a result of either nutrition, water or occupational exposure. The results of ambient air concentrations of ZnO ruled out the possibility of overexposure from inhalation.

This high urine-Zn levels can thus only be attributed to food and water intake. Studies on the nutritional status of the workers should confirm this deduction.

**Welding shop:** Urine analyses resulted in a mean value of 15.5 ±3.0 µmol/ℓ after exposure. Although these results are almost within the toxic concentration limit of Baselt (1980), the welders are considered to be overexposed. Once again, ambient air concentration showed that overexposure due to inhalation of ZnO fumes should not be considered, although lower ZnO concentration in the welding shop compared to the concentration in the foundry, resulted in lower urine-Zn concentrations of welders' urine than in foundry workers' urine. However, the statistical significance showed no significant differences between the mean values of the two exposed groups. The welders are from the same social status as the foundry workers and therefore, have the same diet. The high urine-Zn levels can therefore be regarded as a result of food and water intake with a high Zn content.



### 4.1.2 Cadmium

The significance of Cd in urine was summarised by Lauwerys, Buchet and Roels (1976) and Lauwerys *et al.* (1979). At low level exposures (i.e. general environment), when the total amount of Cd absorbed has not yet saturated all the available Cd-binding sites in the body, the Cd concentration in urine reflects mainly the Cd level in the body and hence in the kidney. When integrated exposure has been so high as to cause a saturation of the binding sites, Cd in urine may then be related partly to the body burden and partly to the recent exposure. The relative importance of each factor depends on the intensity of exposure.

When renal damage develops a considerable increase of urinary excretion occurs. Urinary excretion is no more than 18 nmol/24h (unless renal damage has occurred), although there is a small increase with age. Combined biliary and urinary excretion is normally less than 10 % of the retained Cd (Taylor, 1994).

The World Health Organisation (WHO, 1981), OSHA (1990) and ACGIH (1995 - 1996) has recommended that the urinary concentration of Cd should not exceed 10 µg/g creatinine and that control measures should be applied if a value of 5 µg/g creatinine is exceeded. A reference range of 0.09 - 5.16 µmol/ℓ was proposed by Iyengar and Woittiez (1988) and it is believed that urine Cd levels > 10 µg/ℓ are indicative of renal tubular damage.



The values for Cd are depicted in Figure 18B. The student *t*-test showed no

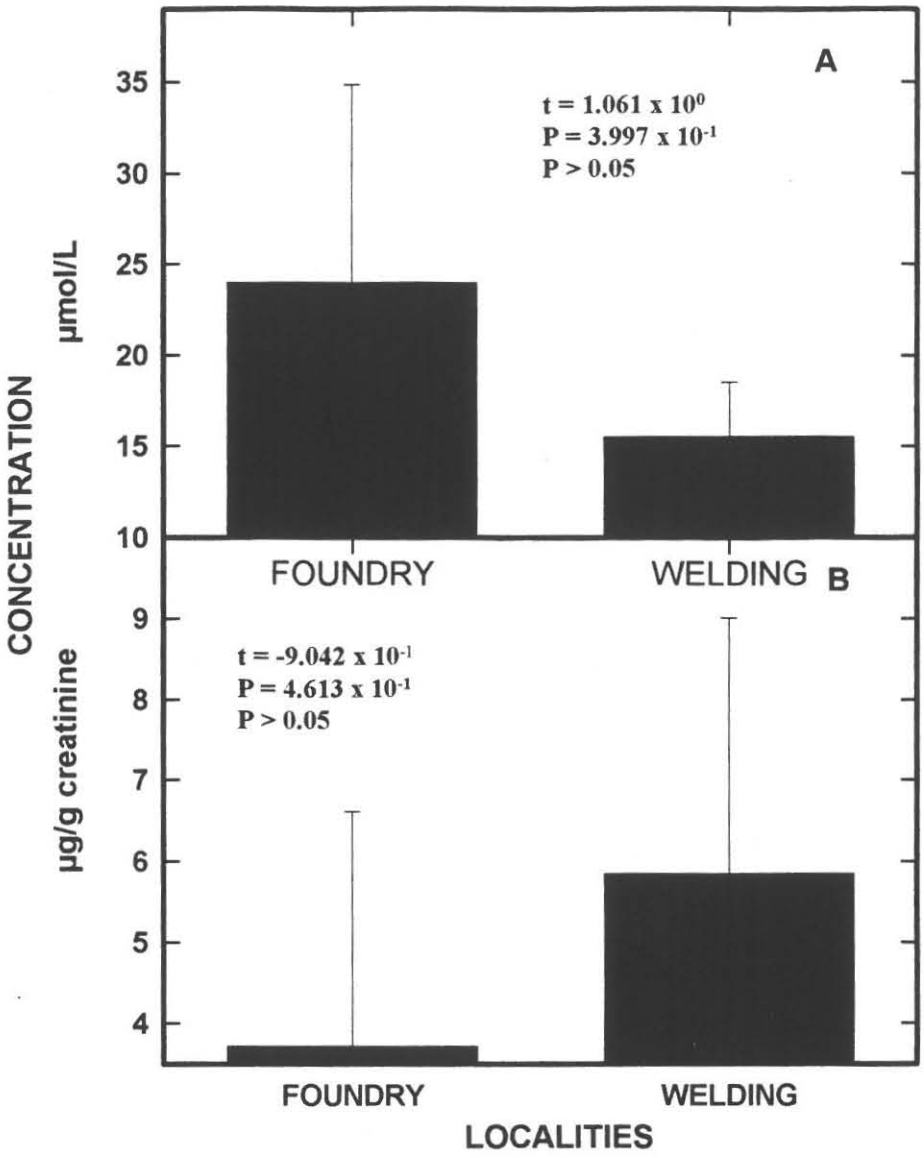


**FIGURE 18:** **A: Mean urine-Zn concentrations (µmol/L) of workers.**
**B: Mean urine-Cd concentration (µg/g creatinine) of workers.**

significant differences between the mean values of the two localities with a P-value of $4.613 \times 10^{-1}$.

**Foundry:** The analyses of workers urine showed a mean value of 2.9 ±2.0 µg/g creatinine. Although the mean value of this exposure does not exceed the biological exposure indices, the 95 % (7.25 µg/g creatinine) and 99 % (16.52 µg/g creatinine) confidence limits predict high exposure levels. Workers exposed to Cd might show signs of pulmonary emphysema (NIOSH, 1976a; Stokinger, 1981a), anaemia, back pain, difficulty in walking (Friberg, Nordberg and Piscator, 1977), suppression of testicular function and damage to the olfactory organs (NIOSH, 1976a; Stokinger, 1981a).

Ambient air monitoring showed small concentrations of Cd and therefore the higher urine-Cd levels cannot be attributed to occupational inhalation exposure. However, it is possible that the urine-Cd levels are indicative of low chronic exposure to Cd fumes in the workplace. It was noted that none of the workers wore dust masks and the exposure at workstations was higher than the ambient measurements at the sampling point in this study. This assumption was verified by means of personal sampling of workers at their workstations. The Cd content of their food and water should also be determined with further research.

**Welding shop:** A mean value of 5.8 ±3.1 µg/g creatinine, which exceeded all recommended biological limits, was obtained with urine analyses. The calculated confidence limits (95 % = 7.9 µg/g creatinine; 99 % = 18.0 µg/g creatinine) also confirmed a high presence of this metal in the workers' urine. Compared to the non-occupational exposure range of 1.0 -2.0 µg/g creatinine (Baselt, 1980), the levels found in the urine of the welders are very high. During chronic exposure the values can reach 10 µg/g creatinine accompanied by renal damage. It is envisaged that some of the above-mentioned symptoms may be present in the workers. Control of exposure should be implemented and biological monitoring should take place afterwards to decrease levels to below a safe limit.

Due to the lower sensitivity of PIXE for Cd, low ambient air concentrations were measured inside the welding shop, but once again none of the workers wore dust masks and personal monitoring should be conducted to determine whether the high urine-Cd levels is a product of occupational exposure. Although the steel and welding consumable manufacturers do not specify any Cd content in their products (Table 9), it was envisaged that Cd is present in a low percentage of the products' composition. Once again, the diet of the welders should be investigated for a

possible source of the high urine-Cd levels. It is proposed that renal function tests be conducted on the workers to detect early renal damage.

The toxic effects of Cd are enhanced by the presence of a low body calcium, copper and Zn content (Taylor, 1994). Workers should therefore be encouraged to consume food which restores such elements in the body.

### 4.1.3 Cobalt

In workers exposed to Co, the urinary Co concentration increases proportionally more than that in blood. In practice, the concentration of Co in urine is mainly influenced by recent exposure, but in view of the biological half-life of Co excretion, under stable exposure conditions, its urinary concentration increases during the workweek.

Observations made on workers exposed to hard metal dust, Pellet *et al.* (1984) concluded that the difference between end- and beginning of shift urinary Co concentrations reflects the day exposure and the concentration in the Friday urine is an indicator of the cumulative exposure during the week.

Posma and Dijstelberger (1985) have estimated that a mean level of 10 µg/g creatinine in the morning urine sample corresponds to a TWA of 0.1 mg/m$^3$. In 1985, Christensen and Mikkelsen suggested that exposure should be kept at a level such that urine concentration of Co at the end of the workday generally do not exceed 0.4 µg/mmol creatinine or 3.5 µg/g creatinine which falls within their control values (0.004 to 1.21 µg/mmol creatinine). According to Scansetti *et al.* (1985), a TWA exposure of 0.1 mg/m$^3$ would lead to a mean Co concentration of about 60 µg/$\ell$ urine at the end of the last workshift of the week.

Similar results were reported by Ichikawa, Kusaka and Goto (1985) who observed that for a TWA exposure of 0.1 mg/m$^3$ the Co concentration in urine collected toward the end of a workshift on Wednesday or Thursday ranged from 59 to 78 µg/$\ell$ (95 % confidence limits) and a TWA of 50 µg/m$^3$ leads to a mean concentration of 34 µg/$\ell$. Lauwerys and Hoet (1993b) found an average Co concentration of 46 µg/g creatinine in urine collected on a Friday and workers exposed to a TWA of 50 µg/m$^3$. The ACGIH (1995 - 1996) recommended a biological exposure indice of 15 µg/$\ell$ sampled at the end of a workshift.

A reference value of 17 - 34 nmol/ℓ was proposed by Iyengar and Woittiez (1988). Non-occupationally exposed reference values ranged from 0 to 2.1 µg/g creatinine while values for occupationally exposed adults ranged from 2.0 to 50 µg/g creatinine (Baselt, 1980).

The Co-concentrations are shown in Figure 19A. No significant differences between the mean values of the two localities existed at the 0.05 level ($P = 9.093 \times 10^{-1}$), taking the small sample size into consideration.

**Foundry:** The mean exposure of 3.7 ± 1.5 µg/g creatinine is well within the above findings and recommendations of the authoritative agencies. With reference to the above mentioned findings, the results correlate with both the biological as well as the ambient air sampling. Due to the insensitivity of PIXE for Co and the low air Co concentrations found, the Co exposure can be regarded as very low. The very low Co concentrations found in the urine complements this assumption.

**Welding shop:** In the case of urine analyses of welders, the mean concentration of 3.6 ± 0.7 µg/g creatinine was below the recommended limits. Although the obtained values are higher than 2.0 µg/g creatinine, indicating industrial exposure, it is lower than the limit of 50 µg/g creatinine and the same interpretation as in the case of the foundry is applicable. No harmful exposure to this element would thus be expected.

### 4.1.4 Nickel

The excretion of Ni is predominantly via the urine (Tossavainen *et al.*, 1980; Sunderman *et al.*, 1989). Sparingly soluble Ni compounds (e.g., stainless steel welding fumes) are poorly absorbed by the lung. In this case, significant amounts of Ni may be deposited in the respiratory tract without rapid and important change in plasma or urinary Ni concentration (Akesson and Skerfving, 1985; Kalliomaki *et al.*, 1981). Nevertheless, Ni is slowly released from the lungs and serum and urine levels may, to a certain extent, reflect lung burden (Grandjean, Andersen and Nielsen, 1988). This may explain the elevated concentration of Ni in plasma and urine of nickel refinery workers, three to four years after cessation of exposure (Boysen *et al.*, 1984). When workers are exposed simultaneously to insoluble and readily soluble Ni compounds, the concentration of Ni in plasma and urine will mainly reflect the exposure to the soluble compounds.

A urine-Ni reference range of 0.1 - 8.0 µg/ℓ was proposed for non-occupationally exposed persons and for nickel refinery workers, a range of 8 -800 µg/ℓ, while the range for nickel carbonyl poisoning is 100 - 2500 µg/ℓ. A Biological Exposure Index (BEI) was set between 10.0 and 50 µg/ℓ with an action level of > 150 µg/ℓ for urine-Ni (Baselt, 1987).

Figure 19C shows the urine Ni-concentrations for the two localities. No significant differences between the mean values of the two localities existed at the 0.05 level with a P-value of $2.75 \times 10^{-1}$.

**Foundry:** Nickel concentrations in foundry workers urine resulted in a mean value of $3.2 \pm 1.7$ µg/ℓ. The results confirmed low occupational exposure with no hazard. Furthermore, the results obtained correlate with the low percentage Ni involved in the manufacturing of the crusher balls.

**Welding shop:** The mean value for urine-Ni from the welders was $12.4 \pm 9.2$ µg/ℓ. The higher occurrence of $NiO_2$ fumes in the welding shop, as a result of the Ni content of the welding consumables, contributed to a higher urine-Ni concentration in welders than in foundry workers. Although the Urine-Ni concentrations are within the limits of the BEI, the 99 % confidence limit exceeds the upper limit of the range.

It is suggested that personal sampling of air intake, as well as urine should be conducted regularly on a yearly basis to monitor the chronic exposure to $NiO_2$ in the welding shop. This recommendation is in accordance with the maximum $NiO_2$ of 42.0 µg/m$^3$ found which approached the new threshold limit value of 0.05 mg/m$^3$, set by the ACGIH (1995 - 1996). Overexposure is envisaged during winter months when the doors and windows are closed in order to prevent wind blowing through the factory and the further cooling down of equipment and people.

### 4.1.5 Manganese

In industry, workers absorb manganese mainly through the lungs (Freeland-Graves and Lin, 1991). The normal concentration of Mn in urine is usually less than 3 µg/ℓ (Buchet, Lauwerys and Roels, 1976; Watanabe *et al.*, 1978). Minoïa *et al.* (1990) suggest a mean reference value of 1.02 µg/ℓ (n = 777; range: 0.12 - 1.9 µg/ℓ) and Järvisalo *et al.* (1992) that of 0.3 µg/ℓ.

Although the excretion in urine is low (about 1 % of the absorbed dose), the determination of manganese in urine has been proposed for estimating recent exposure. Tanaka and Lieben (1969) found that, on a group basis, the urinary Mn concentration of exposed workers shows a rough correlation to the average air concentration. Similar results were obtained by Roels *et al.* (1987). Smyth *et al.* (1973), on the other hand, only found a slight correlation between the airborne concentration of Mn and its concentration in urine of exposed workers.

On a group basis, Mn in urine seems partly influenced by recent exposure, and it was suggested that its periodic determination in a group of workers may detect changes in environmental pollution and / or time trend in the risk of overexposure (Lauwerys and Hoet, 1993b). The study of Järvisalo *et al.* (1992) on manual arc welders of mild steel also indicate that the measurement of Mn in urine or blood may be used for monitoring Mn exposure at the group level only. No biological threshold level is currently proposed, but it is recommended to measure Mn in urine for confirmation of Mn absorption (Lauwerys and Hoet, 1993b).

Iyengar and Woittiez (1988) proposed a urine-Mn concentration range of 9.1 - 178 nmol/$\ell$ (0.5 - 9.8 µg/$\ell$) with a toxic concentration of > 182 nmol/$\ell$ (>10 µg/$\ell$) for adults. The value for non-occupationally exposed adults is < 3.0 µg/$\ell$ (Baselt, 1980).

Urine-Mn concentrations are shown in Figure 19B. No significant difference between the means of the two localities were found. The *t*-test showed a P-value of $2.403 \times 10^{-1}$ and a *t*-value of $-16.51 \times 10^{-1}$.

**Foundry:** A mean Mn-concentration of $1.2 \pm 0.2$ µg/$\ell$ was found in the urine. This value is well within the ranges proposed by the authors (Buchet *et al.*, 1976; Watanabe, *et al.*, 1978; Iyengar and Woittiez, 1988). The 95 and 99 % confidence limits of 0.6 and 1.4 µg/$\ell$ also predict values within the safe ranges. It is therefore recommended that minor attention be given to Mn exposure of foundry workers. The exposure is in accordance with the approximate amount of 1 % Mn used in the alloys during foundry operations.

**Welding shop:** The urine of the welders showed a mean concentration of $1.7 \pm 0.4$ µg/$\ell$. Once again, both the 95 and 99 % confidence limits are within the safe limits although it can be assumed that small amounts of Mn are absorbed by the workers. Although the absorption varies greatly according to Lauwerys and Hoet (1993b), less than 10 % of the total Mn intake is absorbed. Thus, it is

recommended that attention be given to Mn exposure of welders, especially when air concentrations of Mn reach the proposed threshold limit values (ACGIH, 1995 - 1996).

### 4.1.6 Copper

In the occupational setting, absorption of dust and fumes containing copper compounds probably occurs via the respiratory tract. There is, however no data available on absorption rates of copper after inhalation (Lauwerys and Hoet, 1993b). Excretion occurs mainly via the bile, with only a small fraction being eliminated in the urine.

The mean urinary levels reported by Versieck (1985) range from 15 to 36 µg/24h. This is in accordance with the estimate of Triebig and Schaller (1984) which reported a mean urinary daily excretion between 20 µg and 50 µg/ℓ. A study by Minoïa *et al.* (1990) recorded a mean value of 23 µg/ℓ (n = 507; range 4.2 - 50 µg/ℓ) for healthy non-occupationally exposed workers.

According to Lauwerys and Hoet (1993b), data concerning the relationship between occupational exposure, internal dose and effect are at present too scarce to suggest reliable biological limit values. Two urine-Cu ranges for adults are proposed in the literature; 0.03 - 1.26 µmol/ℓ (2 - 80 µg/ℓ) by Iyengar and Woittiez (1988) and 0.047 - 0.55 µmol/24h (3 - 35 µg/24h) by Schramel, Lill and Hasse (1985).

Figure 19D depicts the Cu-concentrations in urine of the two localities. The student *t*-test showed no significant differences between the mean values of the two localities ($P = 4.671 \times 10^{-1}$).

**Foundry:** The mean concentration of Cu in foundry workers' urine was $2.9 \pm 2.0$ µmol/ℓ, which is lower than the reported values of Versieck (1985) and Triebig and Schaller (1984). However, when comparing the results to the values 0.03 - 1.26 µmol/ℓ (2 - 80 µg/ℓ), recommended by Iyengar and Woittiez (1988), the workers might show signs and symptoms of Wilson's disease, as a consequence of an inability to excrete the metal (Taylor, 1994).

According to Taylor (1994), urinary excretion is considerably increased (> 1.5 µmol/24h) during Wilson's disease. It is therefore suggested that urine sampling together with measurement of caeruloplasmin in serum be carried out as

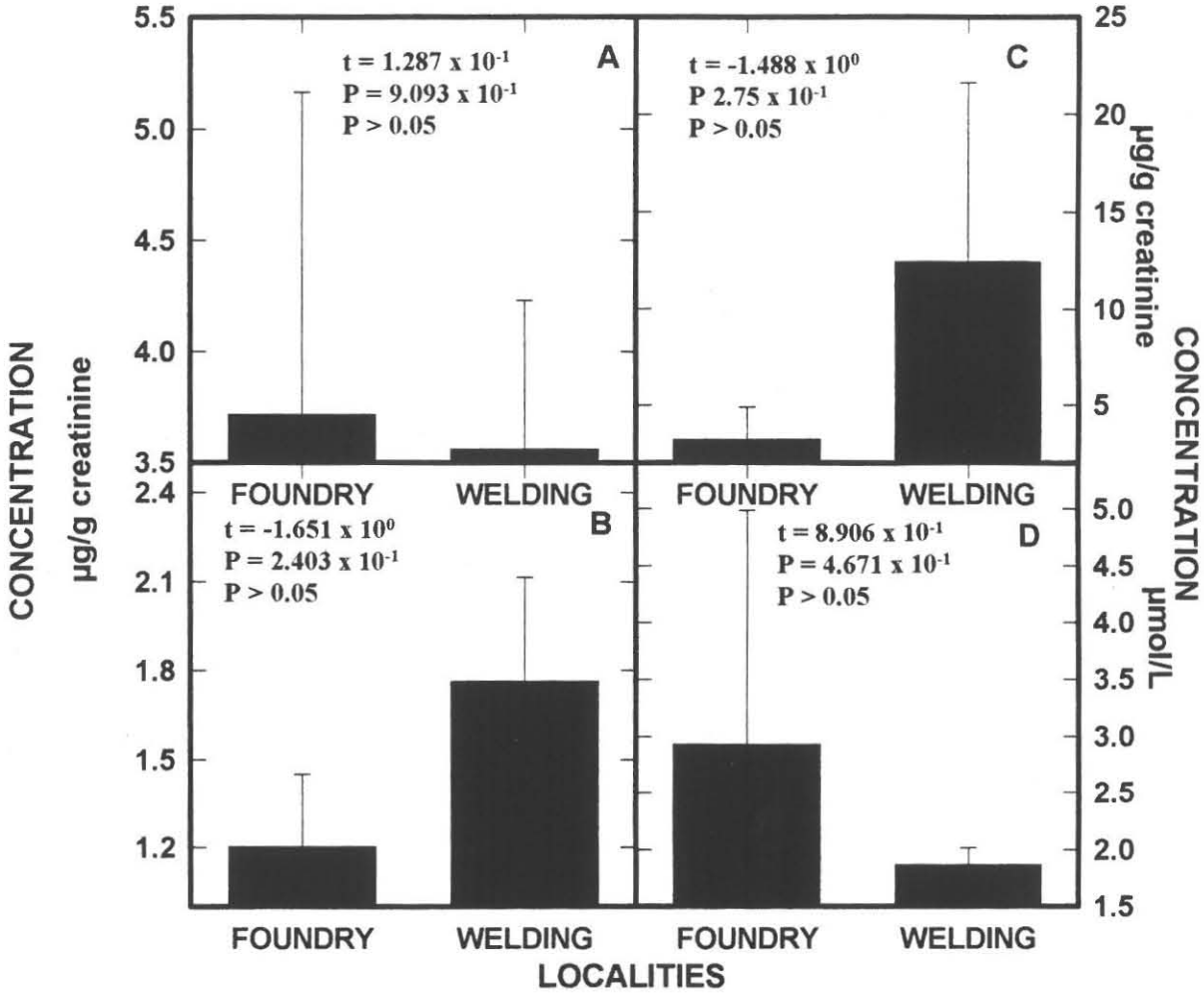


**FIGURE 19:** **A: Mean urine-Co concentrations (µg/g creatinine) in workers.**

**B: Mean urine-Mn concentrations (µg/g creatinine) in workers.**

**C: Mean urine-Ni concentrations (µg/g creatinine) in workers.**

**D: Mean urine-Cu concentrations (µmol/L) in workers.**

biochemical investigations for the diagnosis of Wilson's disease in the foundry workers. If these results are ambiguous a liver biopsy specimen can be collected for copper measurement which will show greatly increased Cu concentrations if positive (Taylor, 1994).

If the copper overload is due to inability to excrete it, the possibility of renal failure as mentioned may be linked to the high urine-Cd levels. This in turn can then be linked to the high urine-Zn levels.

The urine-Cu level cannot be as a result of inhalation exposure due to the low ambient Cu-concentrations measured during the study period. If such levels are exposure related, it is the result of historical exposures. However, the intake of Cu through food and water cannot be disregarded in the evaluation of this condition. Studies on food composition should give more facts for consideration of toxic exposures.

**Welding shop:** The welders had a lower urine-Cu concentration with a mean value of 1.8 $\pm$0.1 µmol/$\ell$. This concentration can also be regarded as a safe level, depending on the reference values being used. If the 0.03 - 1.26 µmol/$\ell$ by Iyengar and Woittiez (1988) are used, workers ought to be showing symptoms of Wilson's disease. The same diagnosis will be applicable as in the case of the foundry workers.

Ambient Cu-concentrations were in accordance with the process, not containing very much copper in the steel or the welding wires. The maximum Cu content in welding consumables, used in this welding shop, is approximately 0.1 % which contribution could result in minimal Cu exposure.

### 4.1.7 Chromium

Industrial workers may be exposed to trivalent and hexavalent chromium compounds whose metabolic handling and toxicity are strikingly different.

**Hexavalent Cr compounds:** The toxicity of Cr is mainly attributed to hexavalent compounds which can be absorbed by the lungs, gastrointestinal tract and even by the intact skin to a certain extent (Baranowska-Dutkiewicz, 1981). It seems that the carcinogenic risk for respiratory tract is principally related to the more insoluble hexavalent Cr compounds. Once absorbed, Cr is mainly excreted via urine and the excretion occurs rapidly (Franchini *et al.*, 1975). With the exception of the

lungs (in which Cr concentration increases with age), tissue levels of Cr decline with age (WHO, 1988a).

According to Lauwerys and Hoet (1993b), the determination of Cr in urine seem to be the most practical biological monitoring method for assessing exposure to hexavalent Cr compounds. Studies carried out by several authors (Berode and Guillemin, 1977; Gylseth, Gundersen and Langard, 1977; Sjögren, Hedstrom and Ulfvarson, 1983; Tola *et al.*, 1977) suggest the following relation: a TWA exposure of 0.025 or 0.05 mg/m$^3$ hexavalent Cr is associated with an average urinary concentration at the end of the exposure period of 15 or 30 µg/g creatinine, respectively. This relation is only valid on a group basis as in this study. Following exposure to 0.025 mg/m$^3$ hexavalent Cr, the lower 95 % confidence limit value is approximately 5 µg/g creatinine. This estimate is in good agreement with the relationship observed by Mutti *et al.* (1985).

The ACGIH (1995 - 1996) has adopted as BEI for Cr VI (water-soluble fume) a 10 µg total Cr/g creatinine as increase during shift and 30 µg/g creatinine measured at the end of a shift (end of workweek) which is equivalent to a TWA exposure of 0.05 mg/m$^3$. An urine-Cr reference range of 1.9 - 38.4 nmol/ℓ (0.1 - 2.0 µg/ℓ) was proposed (Iyengar and Woittiez, 1988; Veillon, Patterson and Bryden, 1982).

**Trivalent Cr compounds:** In the trivalent state, Cr is poorly absorbed. Lim, Sagent III and Kusubov (1983) found some accumulation in the liver, spleen, soft tissues and bones.

Although, these compounds are poorly absorbed, increased Cr urinary concentrations have been observed in workers exposed to trivalent Cr, indicating that Cr III is absorbed at least to some extent (Kiilunen *et al.*, 1983; Aitio *et al.*, 1984; Saner , Yuzbasiyan and Cigdem, 1984).

Figure 20A shows the Cr-concentrations in the urine of workers from both localities. No significant differences between the means of the two localities existed at the 0.05 level with a P-value of $9.093 \times 10^{-1}$. Once again, the small sample size may have an influence on the significance.

**Foundry:** The Cr-concentration of the foundry workers was low with a mean value of 1.9 $\pm$ 0.6 µg/g creatinine. This low urine-Cr concentration is in accordance with the findings of several authors mentioned (Berode and Guillemin, 1977; Gylseth *et*

*al.*, 1977; Sjögren *et al.*, 1983; Tola *et al.*, 1977). Furthermore it corresponds with the limits of the ACGIH (1995 - 1996) and is possibly the result of the low TWA-concentration of 0.1 $\pm$0.1 µg/m$^3$ in the foundry air. It can be concluded that the workers of the foundry are not at risk as a result of Cr VI exposure.

**Welding shop:** The mean concentration found for urine-Cr in welders was 9.3 $\pm$9.7 µg/g creatinine which nearly exceeds the ACGIH limit of 10 µg/g creatinine. The 95 and 99 % confidence limits emphasized these high concentrations with limits of 24.1 and 54.9 µg/g creatinine, respectively. This level of exposure was anticipated due to the welding of stainless steel when using 3CR12 plates with a Cr content of 12 %. The Cr content of the welding consumables is 23.4 %.

In subjects chronically exposed to hexavalent Cr, a mean urinary chromium increase (pre-exposure value subtracted from the end of shift value) of 12.2 µg/g creatinine or a mean total concentration of 30 µg/g creatinine is equivalent to an air concentration of 50 µg/m$^3$ Cr VI from welding fumes (Mutti *et al.*, 1985). Lindberg and Vesterberg (1983) also found a correlation between air and urinary Cr concentrations and determined that a postshift Cr level of $\leq$ 100 nmol/$\ell$ (5.2 µg/$\ell$) would reflect a TWA exposure of $\leq$ 2 µg/m$^3$ Cr hexavalent from chromic acid. According to the results of Angerer *et al.* (1987), urinary Cr in the order of 40 µg/$\ell$ would correspond to an exposure of 100 µg/m$^3$ Cr trioxide in stainless steel welders. It can therefore be assumed that the high urine-Cr levels in the welders are a direct result of the TWA concentration of approximately 40 - 50 µg/m$^3$ measured at some time intervals. The maximum concentration of 86.2 µg/m$^3$ ($CrO_3$[ ] = 165.8 µg/m$^3$) confirms this interpretation.

Special attention should be given to the Cr exposure of the welders in this study and symptoms such as lesions of the mucosa and submucosa of the respiratory tract may be found (Carson, Ellis III and McCann, 1991). Signs and symptoms from chronic inhalation exposure include allergic contact dermatitis, skin ulcers, nasal membrane inflammation and ulceration, nasal septum perforation, perforated eardrums, rhinitis, nosebleed, liver damage, pulmonary congestion and oedema, epigastric pain, tooth erosion and discoloration, and nephritis. The possibility of renal damage, as mentioned previously for Cd and Cu, may be also be aggravated by Cr exposure during welding operations.

### 4.1.8 Aluminium

In subjects with normal renal function, urinary excretion of aluminium is a more sensitive indicator of aluminium exposure than its concentration in serum. Hence, in the case of occupational exposure, the concentration of aluminium in urine may be increased, whereas plasma or serum levels hardly differ from those found in control subjects (Mussi *et al.,* 1984; Valentin, Preusser and Schaller, 1976; Alessio *et al.,* 1983; Savory and Wills, 1988). Occupational exposure to fumes seem to produce higher urinary levels of aluminium than exposure to dust (Mussi *et al.*, 1984).

The urinary excretion of aluminium amongst welders was shown to be related to the number of years of exposure to aluminium (Sjögren *et al.,* 1988). In practice, workers chronically exposed to aluminium, the concentration in urine collected 1 or 2 d after the end of exposure, is probably a good indicator of the amount accumulated in the body. The relationship between external exposure and urinary concentration may vary depending on the bio-availability of various aluminium compounds, however this aspect has not been extensively investigated.

Limited available data (Sjögren *et al.,* 1985) suggests that for some aluminium compounds to which workers may be exposed (e.g. aluminium welding fumes, etc.), a mean urinary concentration of 200 µg/g creatinine at the end of the shift corresponds to a TWA exposure of approximately 5 mg/m$^3$. However, this only applies to workers exposed for less than a few weeks. The concentration of aluminium in urine collected at the end of the shift is likely to reflect recent exposure. Lauwerys and Hoet (1993b) proposed a tentative biological limit of 150 µg/g creatinine. A urine-Al reference range of 0.19 - 1.11 µmol/$\ell$ was proposed by Schramel *et al.* (1985). Furthermore, Lauwerys and Hoet (1993b) suggested that a concentration of < 50 µg/g creatinine be representative of non-occupational exposure.

Figure 20B shows the Al-concentrations in the urine of workers from both localities. No significant differences existed between the means of the two localities at the 0.05 level with a P-value of $6.313 \times 10^{-1}$.

**Foundry:** The mean concentration of 12.0 $\pm$7.1 µg/g creatinine is well below the proposed limit of 150 µg/g creatinine (Lauwerys and Hoet, 1993b). This result is in accordance with results of Sjögren *et al.* (1985). Although the manufacturing process of crusher balls contains Al (Table 7), it is only 0.03 % of the total alloy

and should not contribute to a high $Al_2O_3$ concentration in air as found during the study. Occupational exposure to Al in the foundry did not show any concern for negative effects on the health of the workers.

**Welding shop:** Urine analyses of welders revealed a mean concentration of 8.6 ± 3.5 µg/g creatinine after exposure to welding fumes. Although Mussi *et al.* (1984) states that fume exposure results in a higher urinary level than dust exposure, the opposite was found during this measurement. However, it must be emphasised that the welding process did not contain materials with an Al content. The lower urine-Al value could thus be ascribed to the absence of Al in the process and be seen as Al exposure from other sources such as food, water, soil, etc.

### 4.1.9 Iron

Chronic inhalation of Fe fumes leads to mottling of the lungs, a siderosis that is considered a benign pneumoconiosis, nonfibrotic and not favourable to tubercle bacilli (Carson, Ellis III and McCann, 1991). Excess Fe, which is not absorbed, appears in the urine. Normal losses are through sloughing of intestinal cells and bleeding. According to Hammond and Beliles (1980) excessively absorbed Fe is excreted in the urine.

No BEI for Fe or its oxides could be found in the literature. The results obtained thus provide new information and cannot be compared to any standard. This aspect should present strong challenges to clinicians and nutritionists for further research. A urine-Fe reference range of 0.04 - 1.3 µmol/ℓ (2 - 70 µg/ℓ) was proposed by Schramel *et al.* (1985). They also suggested a daily excretion range of 0.05 - 1.8 µmol/24h (3 - 98 µg/24h) for non-occupationally exposed adults. Although it was mentioned that Fe excretion is higher in menstruating females, such information is not applicable during this interpretation because no females were used during this study.

Iron concentrations are shown in Figure 20C. The student *t*-test did not show any significant differences between the means of the two localities. The P-value was $2.318 \times 10^{-1}$.

**Foundry:** A mean concentration of 84.3 ± 50.6 µg/ℓ was found. The 95 % confidence limit was 125.0 µg/ℓ compared to the 99 % confidence limit of 285.0 µg/ℓ. The mean concentration exceeds the upper limit of the range specified by Schramel *et al.* (1985) which indicates occupational exposure to Fe in

the foundry. Snyder *et al.* (1975) stated that Fe in urban air would contribute ~27 µg/24h to the total Fe intake. Therefore it can be assumed that the iron foundry workers have a higher Fe intake by means of inhalation. The materials used in the casting process contains more than 94 % Fe and the Fe-concentration in the foundry air was the second highest concentration of all elements present.

Although the mean urine-Fe concentration exceeds the 70 µg/ℓ upper limit, it is within the 98 µg/24h limit specified by the same authors (Schramel *et al.*, 1985). However, the 95 % and 99 % confidence limits predict high levels and further research should be conducted to enlighten this phenomenon. It should also be mentioned that the traditional black workers use iron pots to cook food and Walker and Arvidsson (1950) suggested that this practice could contribute to a higher Fe intake and accompanying excretion of excess Fe in urine (Hammond and Beliles, 1980).

Shils and Young (1988) state that the Fe intake of adult black males may exceed 100 mg/24h. According to them, African siderosis reaches its greatest severity between the ages of 40 and 60 years and is more severe in males because their alcoholic consumption tend to be greater. However, Bothwell (1994) concluded that iron-loaded individuals were heterozygous for a Fe-loading gene which only becomes manifest when there is an excess of bio-available Fe in the diet. If the findings of Walker and Arvidsson (1950) and Gordeuk *et al.* (1992) are confirmed, it is difficult to overload the body with Fe even when the dietary intake is many times greater than normal, unless there is some genetic predisposition.

**Welding shop:** The urine samples showed a mean concentration of 52.7 µg/ℓ ± 19.0 µg/ℓ with a 95 % confidence limit of 47.4 µg/ℓ. The 99 % confidence limit was calculated at 108.0 µg/ℓ. The mean urine-Fe concentration was within both the range, as well as the upper limit of daily Fe excretion.

Iron concentrations were the highest of all elements present in the welding shop air samples due to Fe being the main constituent of steel and welding consumables (Table 9). The upper limits of the confidence limits (Table 3) once again, stressed the need for regular sampling and further research to monitor the chronically exposed workers. It is also envisaged that $Fe_2O_3$ concentrations will be higher at the source of origin and therefore personal monitoring inside welding helmets should be conducted for occupational hygiene purposes. Such results should present relevant data for the protection of welder's health.

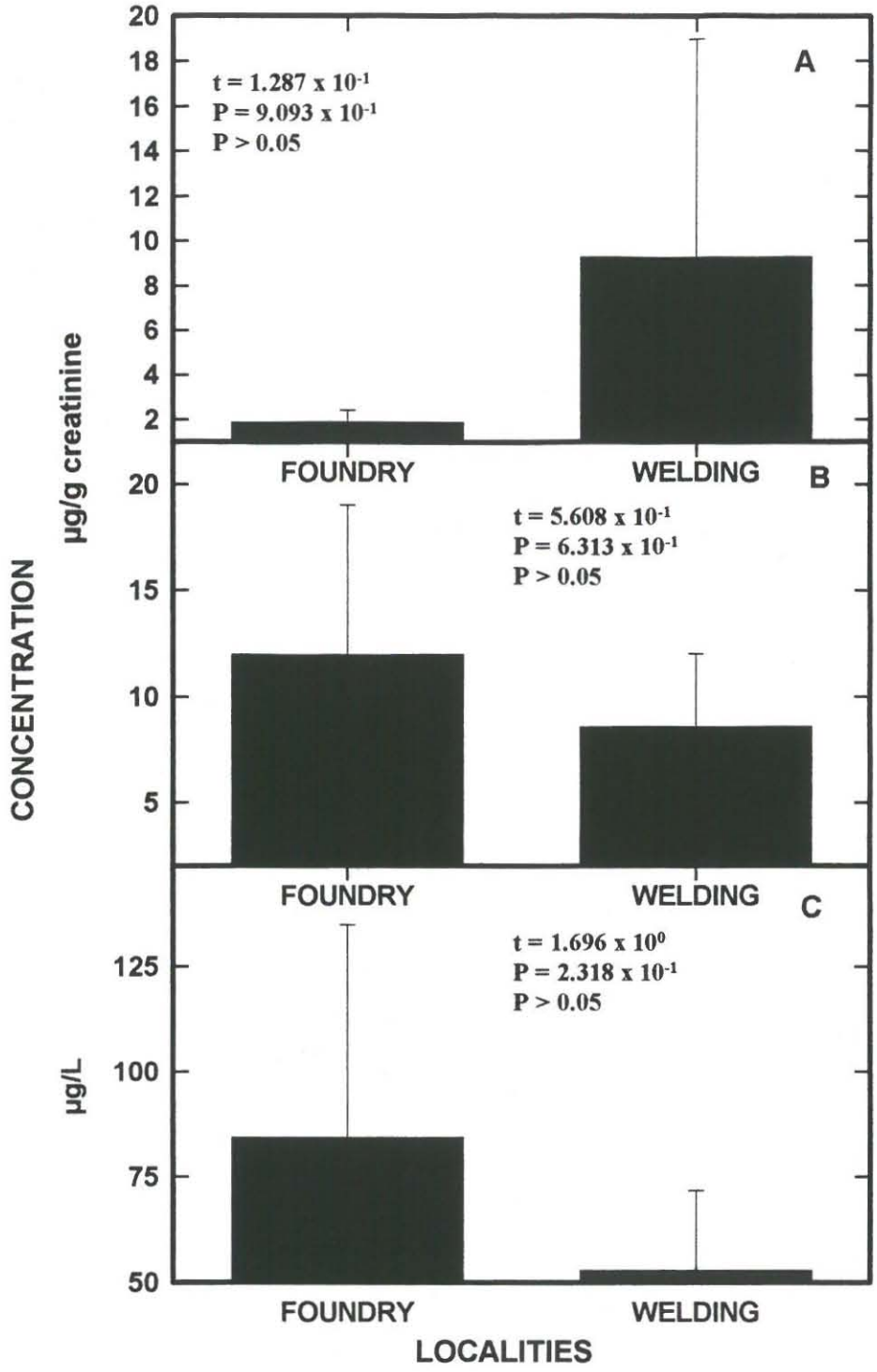


FIGURE 20: A: Mean urine-Cr concentrations (µg/g creatinine) in workers.
B: Mean urine-Al concentrations (µg/g creatinine) in workers.
C: Mean urine-Fe concentrations (µg/g creatinine) in workers.

### 4.1.10 Lead

Lead is a cumulative toxin that is absorbed by the lungs and the gastrointestinal tract (WHO, 1978). The urine reflects the amount of Pb recently absorbed. The concentration of Pb in urine is usually lower than 50 µg/g creatinine. A concentration in blood of 50 µgPb/100 ml corresponds to a concentration in urine of approximately 150 µgPb/g creatinine. Pb in urine will probably average 50 µg/g creatinine for a mean blood Pb level of 40 µg/100 ml.

The ACGIH (1994 - 1995) has proposed 150 µg/g creatinine as a Biological Exposure Index. The latter value was removed from the BEI list (ACGIH, 1995 - 1996), probably in view of the poor association between Pb in urine and in blood and the great risk of external contamination during sampling. The Lead Regulations (South Africa, 1991), enforce an urine-Pb concentration of < 120 µg/$\ell$ for male workers exposed to Pb. Schramel *et al.* (1985) recommended an acceptable reference concentration of < 0.58 µmol/$\ell$ (120 µg/$\ell$) for Pb in urine of adults with industrial exposure.

Figure 21A shows the Pb-concentrations in urine of both localities. No significant differences between the results of the two localities existed at the 0.05 level ($P = 2.663 \times 10^{-1}$).

**Foundry:** A mean Pb concentration of 24.2 ± 8.8 µg/$\ell$ was calculated after analyses of the urine sampled at the foundry. This concentration was low and did not indicate any significant exposure, as was the findings with the aerosol sampling in the workplace. Pb is not used in the process and this urine-Pb concentration can be regarded as exposure from sources other than the foundry.

**Welding shop:** The mean urine-Pb concentration of 31.8 ± 9.5 µg/$\ell$ was within the specified limits for Pb in urine. The exposure should therefore, not pose any health risk to welders. No Pb is used in the process and the exposure can be regarded as safe, taking into consideration that the upper limit of the 99 % confidence limit is within the biological safety standard of 120 µg/$\ell$.

### 4.1.11 Silicon

The presence of Si in urine was investigated in the light of the high $SiO_2$ concentrations workers are exposed to. The possibility of Si uptake and urinary excretion was investigated. Since no BEI could be found for this substance, it

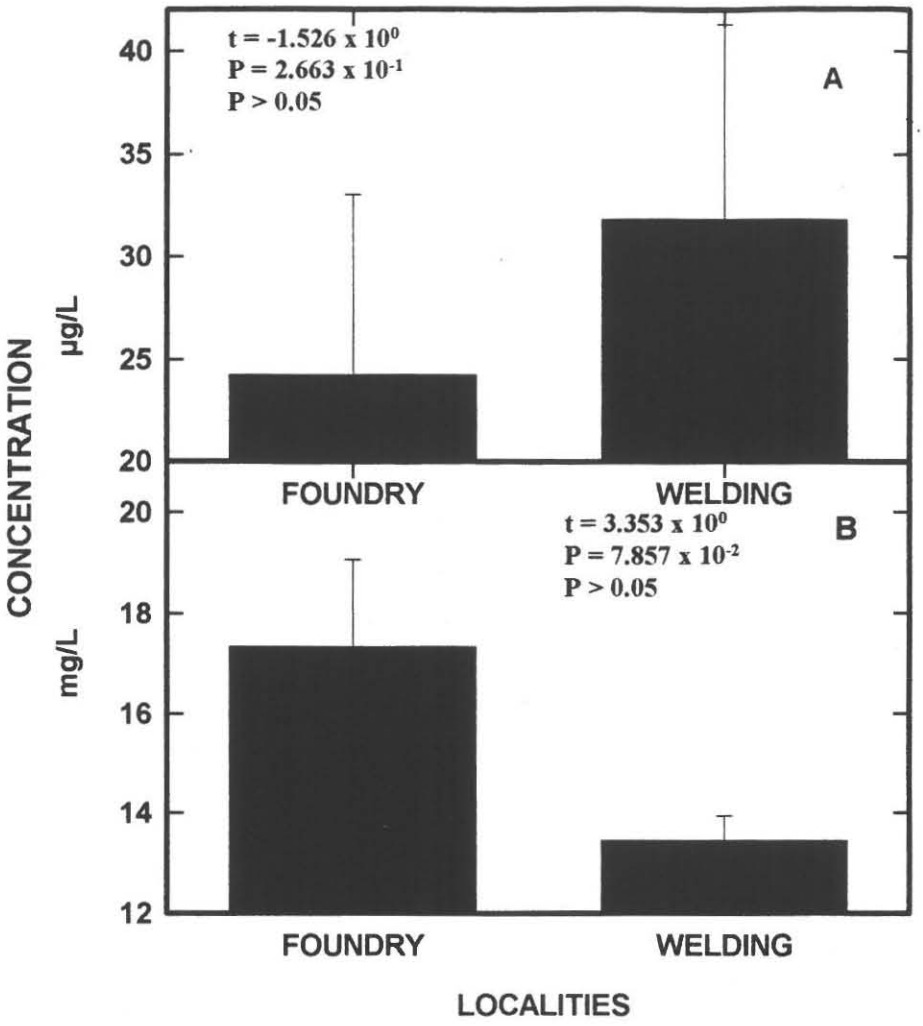


**FIGURE 21:** **A: Mean urine-Pb concentrations (µg/L) in workers.**
**B: Mean urine-Si concentrations (mg/L) in workers.**

presents challenges to clinicians for further research. The results of this analysis can be used for further research on this specific aspect. A reference range of 3.5 - 18.5 mg/$\ell$ was proposed for adults (Gitelman and Alderman, 1990).

The Si-concentrations in the urine of workers are depicted in Figure 21B. No significant differences between the mean values of the two localities were found after a student *t*-test was carried out. It showed a P-value of $7.857 \times 10^{-2}$ and a *t*-value of $33.53 \times 10^{-1}$.

**Foundry:** The results showed a mean Si concentration of $17.3 \pm 1.7$ mg/$\ell$ in the sampled urine which exceeds the above upper limit of the reference range for adults. Si-concentrations were the highest of all elements measured in the foundry air and it is envisaged that some form of absorption with excretion in the urine takes place and therefore exceeding of the normal reference range for urine-Si.

**Welding:** A mean concentration of $13.4 \pm 0.5$ mg/$\ell$ was found in the urine of the welders. This concentration was within the reference range of Gitelman and Alderman (1990) as expected. The Si-concentrations measured in the air of the welding shop are very low and can be regarded as a result of dust exposure due to normal air (wind) movement. The 99 % confidence limit supports the unproved theory of Si uptake through inhalation and excretion in the urine because the workers are exposed to normal low ambient concentrations of Si and all values, including the 99 % confidence limits are within the range.

### 4.1.12 Vanadium

In industry, V is mainly absorbed by the pulmonary route. It has been estimated that $\pm 25$ % of soluble compounds may be absorbed (WHO, 1988b). Urinary V excretion is probably a more suitable indicator of exposure than blood V (Maroni *et al.*, 1983, 1984, 1987; Stonard, Sullivan and Duffield, 1984; Alessio, Maroni and Dell'Orto, 1988; Schaller and Triebig, 1987; Buchet *et al.*, 1985; White *et al.*, 1987; Kawai *et al.*, 1989). In highly exposed workers, urinary V levels may increase up to 30 times over a workshift. However, the majority of studies have obtained poor correlation between V concentrations in air and the amounts excreted in urine (WHO, 1988b).

A tentative biological threshold limit value of 50 µg/g creatinine has been proposed for urinary V (ACGIH, 1995 - 1996), but investigations on occupationally exposed persons are needed to test the validity of the proposal (Lauwerys, Buchet and

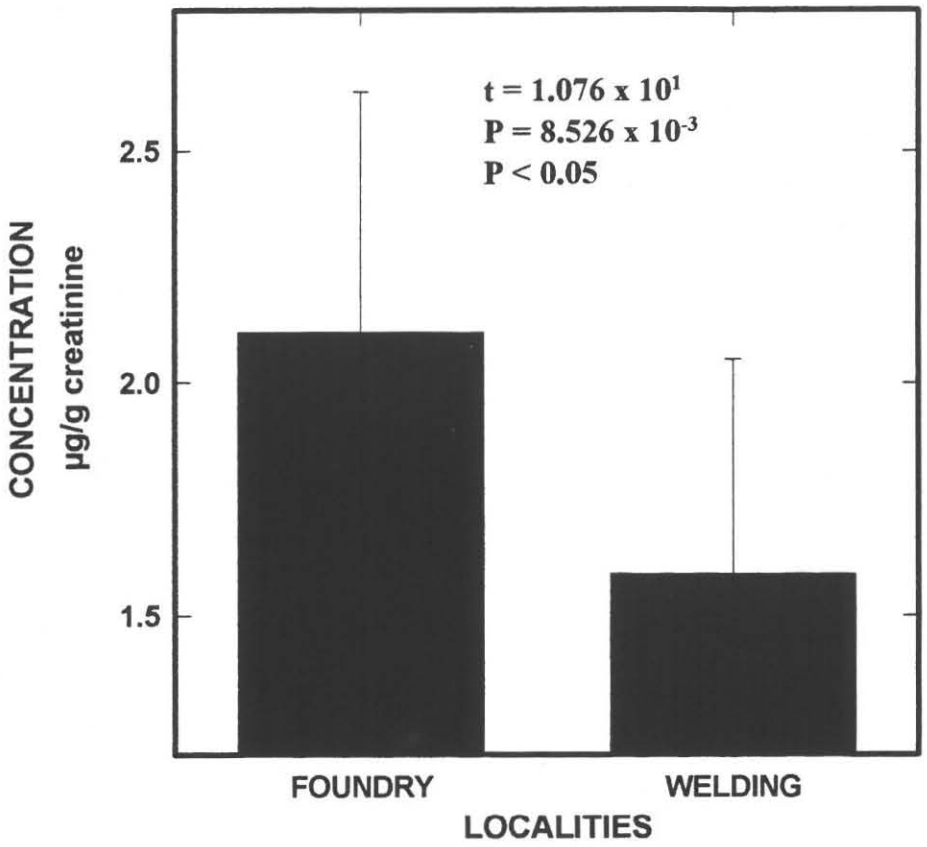


**FIGURE 22: Mean V-urine concentrations (µg/g creatinine) in workers.**

Roels, 1980). A toxic concentration of > 40 µg/ℓ (> 784 nmol/ℓ) for urine-V was set by Tietz (1990) derived from the literature of Crews and Hopkins (1981) and Nechay *et al.*, 1986). Another reference range of < 8 µg/24h was also set by the same author.

Vanadium concentrations are shown in Figure 22. The means of the samples showed significant differences after a student *t*-test was carried out. It showed a P-value of $8.526 \times 10^{-3}$ and a *t*-value of $1.076 \times 10^{1}$. The materials used in the foundry process contain V (Table 7) in known quantities compared to the welding materials which only contain V in trace quantities. The statistical significant higher exposure of the foundry workers is a result of exposure to V fumes in their workplace compared to the welders which are only exposed to trace quantities of V. It must however be mentioned, that the small sample size may have an influence on the statistical significance.

**Foundry:** The very low mean concentration of 2.1 ±0.5 µg/g creatinine in foundry workers is below the proposed BEI and it can be assumed that the workers are only exposed to very small quantities of V. The ambient sampling could not detect V in significant quantities, therefore the V exposure can be regarded as negligible.

**Welding shop:** As expected, a very low mean concentration of V was found in the urine samples of the welders. The exposure to V in the welding shop is so low that no significant concentrations could be detected in the air samples. Thus, exposure to V in the welding environment should not pose any health risk to the welders. However, it is well known that $V_2O_5$ is a by-product of welding fumes (NIOSH, 1988; Sferlazza and Beckett, 1991) and therefore personal sampling inside welding helmets is recommended to verify the low concentrations obtained. Neither the welding wires nor the steel contains V and might be the reason that no significant V concentrations were detected.

## 4.2 Spirometry

The measurement of pulmonary function attempts to detect and quantitate abnormal lung function. This detection of abnormal lung function helps to assess the severity and progression of a disease process as well as response to therapy (Garay, 1992).

### 4.2.1 Foundry

It is well known that foundry workers are at high risk for silicosis due to the use of silica in mould making and exposure during fettling (Balaan and Banks, 1992). The exposure to various metals and $SiO_2$ caused concern with regard to the development of silicosis. Spirometry was used to detect any signs of abnormal lung function.

Significant differences ($P < 0.05$) existed for FVC and $FEV_1$ between the subjects of the experimental and control group as a whole. P-values of $3.8 \times 10^{-2}$ for FVC and $4.9 \times 10^{-2}$ for $FEV_1$ were calculated and are indicative of a significant decrease in lung volumes. A statistical significant difference was also found between the smokers of the experimental and the control group with a P-values of $2.11 \times 10^{-2}$ for FVC and $9.92 \times 10^{-3}$ for $FEV_1$. However, no significant differences were found for the same parameters between the non-smokers of the experimental and the control group. P-values were $6.4 \times 10^{-1}$ for FVC and $8.9 \times 10^{-1}$ for $FEV_1$. The results and statistical analyses show that the lung volumes of the exposed foundry workers are normal, although it is noted that more rapid expiration took place in the exposed group than in the control group.

A restrictive process may be suggested by reduced VC and normal $FEV_1$/FVC ratio on spirometry (Garay, 1992). The faster expiration of the foundry workers could thus be ascribed to a slight restrictive condition which is typical image of silicosis. The $FEV_1$/FVC ratio shows slight restriction due to the faster expiration of the smaller airways which are not obstructed in a relative shorter time. All the values (smokers and non-smokers) fit well with the starting of a clinical restrictive image for silicosis. The values obtained for the smaller airways confirm this statement.

The significant decrease measured for FVC an $FVC_1$ in smokers compared to the same parameters of the smokers in the control group confirms smoking as an aggravating factor in the development of lung disorders in foundry workers. The effect of pollution in the workplace together with the smoking of tobacco, is responsible for the development of respiratory disorders in the experimental population.

### 4.2.2 Welding shop

No significant differences ($P < 0.05$) existed for FVC and $FEV_1$ between the subjects of the experimental and control group as a whole. P-values of $2.6 \times 10^{-1}$

for FVC and $3.9 \times 10^{-1}$ for $FEV_1$ were calculated. No statistical significant differences were found between the smokers of the experimental and the control group with P-values of $8.3 \times 10^{-1}$ for FVC and $6.7 \times 10^{-1}$ for $FEV_1$. No significant differences were also found for the same parameters between the non-smokers of the experimental and the corresponding control group. P-values were $1.4 \times 10^{-1}$ for FVC and $7.6 \times 10^{-2}$ for $FEV_1$.

Studies of pulmonary function suggest that some welders may experience reductions in air flow, VC and diffusing capacity, the latter two apparently more likely among welders who smoke (Stern *et al.,* 1986; Kilburn and Warshaw, 1989 Cotes *et al.,* 1989). This is in accordance with the obtained results of this study. The degree to which these changes progress after they appear is in some doubt (Mur *et al.*, 1989), but an effect of welding at least on flow in small airways appears to be independent of smoking or asbestos exposure (Kilburn and Warshaw, 1989).

Considering the known respiratory effect of smoking, the measured respiratory parameters were analysed comparatively for exposed and non-exposed smokers. Both groups did not show significantly different values. Similar results were obtained when the lung functions were measured for exposed non-smokers and compared to those of non-smoking controls. This indicates that smoking had no increased effect on the exposure. The results are in accordance with the results of Kalliomäki *et al.* (1982) which showed no statistically significant effect of smoking on the measured parameters. Pollution in the workplace can thus be regarded as the main agent responsible for the first signs of a clinical obstructive image. However, the results might have been due to a selection among welders, because the subjects who were particularly susceptible to a noxious influence of welding fumes tended to quit welding (Sjögren, Ulfarson and Tech, 1985).

The results and statistical analyses showed that the lung volumes of the exposed welders tend to show the first clinical signs of an obstructive image. This is reflected in the $FEV_1$/FVC % which is lower in the exposed group than in the control group. It must be emphasised that, when it is possible, serial testing is often useful in establishing a diagnosis of restrictive or obstructive disease in patients whose values are borderline or slightly elevated.

## 4.3 Aerosol concentrations

### 4.3.1 Total inorganic elements

In general, dust is referred to as aerosols consisting of solid particles made airborne by the mechanical disintegration of bulk solid material. Airborne particles tend to form heterogeneous systems of poor stability consisting of various sizes and shapes. The geometric diameter of dust particles can vary between 0.001 µm and 100 µm (Schoeman and Schröder, 1994). Usually, the term "total dust" is used to describe all particles with a size of < 50 µm sampled. For the purposes of this study, the term "total dust" will refer to the alveolar fraction containing particles with an aerodynamic diameter of less than 7 µm ($PM_{10}$) and includes the total complement of all inorganic elements sampled.

An excessive and / or prolonged exposure to airborne dust can cause a variety of occupational diseases, depending on the physical, chemical or toxicological characteristics of the inhaled substance. The biological response to the various inhaled particles may be non-injurious, slight or serious. Thus soluble particles may evoke only a temporary response with no permanent or severe damage to tissue, while insoluble particles may irritate the tissue to a greater or lesser extent depending on their interaction with the tissue.

Apart from pneumonitis caused by metallic dusts and fumes, silicosis or other form of pneumoconiosis, individuals often suffer from an obstructive (blocked-up) type of lung impairment due to total dust exposure (Schröder and Schoeman, 1994). Affected persons may complain about moderate to severe coughing fits and salivation, or breathlessness on exertion, and they may have a measurable alteration of their lung function.

The Threshold Limit Value (TLV) for total respirable particles not otherwise classified (PNOC) is 3 $mg/m^3$ (ACGIH, 1995 - 1996; South Africa, 1995). This value is particulate matter containing no asbestos and < 1 % crystalline silica.

**Foundry:** The total respirable inorganic dust concentration never exceeded the TLV of 3.0 $mg/m^3$ (Figure 2) at the point of measurement. However, if the calculated exposure ratio of 1:8.25 (streaker sampler to individual workplace) is taken into account, all concentrations > 370 $\mu g/m^3$ would be indicative of overexposure. Concentrations changed rapidly and no specific pattern was observed. The low concentrations on Thursday, 11 May, were due to a breakdown in the foundry process during which no work was carried out. The results showed

~ 310.0 µg/m$^3$ during the day and can be regarded as the average exposure concentration of the foundry workers. The peak concentrations may be as a result of the continuous cleaning and sweeping operation in the vicinity of the sampler. Workers will also be exposed to such concentrations when cleaning is carried out near them.

It was also observed that the concentrations were lower when clouds were absent. The concentrations during the last 12 hours of Saturday and the whole of Sunday, can be regarded as normal ambient crustal aerosol concentrations due to the effect of wind and weather conditions.

It is envisaged that some workers, working in the tunnels and the fettling process are exposed to higher concentrations than those mentioned. Chronic exposure to the measured concentrations could contribute to obstructive airway disease, but the fact that the dust contains > 1 % $\alpha$-quartz, makes restrictive airway disease a more reliable diagnosis. The early signs of restriction were already present during spirometry tests. It was also observed that many workers showed signs of salivation during spirometry, which confirms the diagnosis of chronic restrictive airway disease.

**Welding shop:** The TLV for total respirable inorganic dust was never exceeded during the sampling period. Although concentrations varied from day to day, a more specific pattern of exposure was observed. Workers light fires in drums from about 06:00 to warm up the welding shop. During such periods, the place was filled with smoke from the burning of wood and coal.. Such smoke was the main cause of the high morning concentrations measured during which doors and windows were closed to prevent chilly wind blowing through the factory.

Depending on the increase of the outside ambient temperature, doors and windows were opened for fresh air at noon. Figure 10 shows a sudden drop in concentration after such operation because natural ventilation cleans the welding shop very rapidly. The afternoon exposure (from 12:00 to 17:00) would represent a normal exposure to welding fumes when the temperature was acceptable for the workers. The concentrations measured from 18:00 to 05:00 represents soil concentrations and suspended dust particles during which time the doors and windows were locked. Thursday concentrations, which were lower than the rest may be as a result of overcast weather conditions during the day.

The welders may be subjected to higher concentrations of welding fumes than the concentrations measured. Workers are welding inside confined spaces such as truck bodies and tanks. An exposure ratio of 1:7.2 (streaker sampler to work station) was calculated for workers welding inside truck bodies, resulting in concentrations from 0.7 up to 4.6 mg/m$^3$ and exceeding the TLV. Such an overexposure, confirms the clinical signs of obstructive airway disease as shown with the spirometry (Table 5).

### 4.3.2 Chromium

The water-soluble hexavalent Cr compounds, such as chromic acid mist and certain chromate dusts are severe irritants of the nasopharynx, larynx, lungs and skin. Exposure to certain hexavalent Cr compounds, mainly water-insoluble, appears to be related to an increased risk of lung cancer.

Furthermore, hexavalent Cr compounds have been implicated to be responsible for such effects as ulcerated nasal mucosa, perforated nasal septa, rhinitis, nosebleed, perforated eardrums, pulmonary oedema, asthma, kidney damage, erosion and discoloration of the teeth, primary irritant dermatitis, sensitisation dermatitis and skin ulceration (Enterline, 1974).

The ACGIH (1995 - 1996) and the Regulations for Hazardous Chemical Substances, 1995 (South Africa, 1995) recommended a TWA [ ] of 0.5 mg/m$^3$ for occupational exposure to Cr metal, Cr(II) and Cr(III) compounds. A TLV of 0.05 mg/m$^3$ is applied for Cr(VI) compounds by the above mentioned organisations. The TLV's for Cr metal and di- and trivalent Cr compounds are based on the prevention of pulmonary disease and other toxic effects such as dermatitis. The TLV for Cr(VI) compounds is set to protect against irritation of the respiratory tract and possible renal and hepatic damage. The same TLV for certain water-insoluble Cr compounds (e.g. $CrO_3$) is based on respiratory cancer in workers (ACGIH, 1986).

**Foundry:** The Cr-concentrations measured in the foundry air were very low and in accordance with the amount of Cr used in the process. The TLV's were not exceeded and thus no adverse health effects due to this Cr exposure were suspected. An aerosol inhalation exposure risk (AIER) of 9.5% was calculated for the workers exposed to the foundry air. This low risk also confirms that Cr is not used in significant quantities during the smelting or casting process. The exposure pattern may also indicate the presence of Cr in soil of which Cr is a basic element.

**Welding shop:** The measured Cr-concentrations in the air of the welding shop were high, most likely as a result of the materials used containing high percentages of Cr. The Cr(VI) TLV of 50 $\mu g/m^3$ was exceeded every day during peak concentrations with the highest concentration of 165.7 $\mu g/m^3$ during Wednesday. All Cr (total) concentrations below 25 $\mu g/m^3$ can be regarded as safe exposures for workers, taking into consideration the Cr(VI) content of the total exposure.

It can be concluded that the workers were overexposed for 4 to 5 hours per day during which the windows and doors were closed. Once the natural ventilation cleans the air, exposures return to safe limits. Night-time concentrations decreased gradually after 17:00 until operations restarted the next morning at 06:00. Such overexposures are not envisaged during summer time when the doors and windows are opened with the start of the shift and no fires are made.

The aerosol inhalation exposure risk of 47.6% emphasised the overexposure, taking into consideration that it was calculated for ambient air in the welding shop. When the same risk is calculated for personal exposure at the different work stations, it amounts to 54.6%. The risk at the work station can be controlled by the wearing of respirators, however, the risk in the welding shop should be eliminated by engineering controls such as a mechanical ventilation extraction system with a natural supply of fresh air.

The results of the overexposure are in accordance with the urine-Cr levels found in the exposed subjects. Such chronic overexposure may also contributed to other medical conditions such as irritation, pulmonary congestion and possible renal damage. The spirometry results (Table 5) support the findings of such exposure.

It is recommended that the welding shop be furnished with heating mechanisms to warm the workplace so that open fires can be prohibited. Local extract ventilation can also be provided to extract fumes at the point of origin for the prevention of a contaminant build up in the welding shop's atmosphere. Additionally, although very costly, an artificial ventilation system can be installed which supplies fresh heated air for the dilution of contaminant concentrations.

### 4.3.3 Copper

Copper fumes cause irritation of the upper respiratory tract and metal fume fever, an influenza-like illness. In humans, effects of Cu fumes include irritation of the upper respiratory tract, metallic or sweet taste, and in some instances, discoloration of the skin and hair. Exposure of workers to concentrations of 1 to 3 $mg/m^3$ for short periods resulted in an altered taste response but no nausea; levels from 0.02 to 0.4 $mg/m^3$ produced no complaints (ACGIH, 1986).

Lung damage after chronic exposure to fumes in industry has not been described. The higher incidence of respiratory cancer in copper smelters is due to the presence of arsenic in the ore (Triebig and Schaller, 1984). Transient irritation of the eyes has followed exposure to a fine dust of oxidation products of Cu in an electric arc.

A TLV of 0.2 mg ($Cu/m^3$) TWA fume and 1.0 mg $(Cu)/m^3$ dust and mists were prescribed (ACGIH, 1995 - 1996; South Africa, 1995). The TLV's for Cu are based on the prevention of irritant effects (ACGIH, 1986). Because copper is ubiquitous in the environment, the largest relative source contribution would be expected from food and drinking water (USEPA, 1985).

Data pertinent to evaluation of the inhalation carcinogenicity of Cu and its compounds are lacking (Calabrese and Kenyon, 1991). Copper has been placed in group D, unclassified substances, under the EPA weight-of-evidence classification (USEPA, 1984a; 1987).

Copper is an essential element in humans and other animals with about 2 - 3 mg/24h required for proper nutrition. Furthermore, Cu is a component of several important enzymes (e.g. cytochrome oxidase and erythrocyte subperoxide dismutase) and is also necessary for haemoglobin formation, carbohydrate metabolism, catecholarmine biosynthesis, and cross-linking of collagen , elastin and hair keratin. The concentration of Cu in various body tissues is controlled by well-developed homeostatic mechanisms; this is thought to account for the relatively uncommon occurrence of toxicity attributable to excess Cu (USEPA, 1985; 1987).

In humans, ingestion of high concentrations of copper salts produces gastric irritation resulting in salivation, nausea, vomiting, gastric pain and haemorrhage, and diarrhoea. In animals exposed to copper salts in the diet, effects on the liver,

kidneys, blood, gastro-intestinal tract and brain have been reported, but the concentrations producing these effects are relatively high and of questionable relevance to human exposure situation (only approximately 20 % of inhaled copper is thought to be absorbed in humans). These studies have been thoroughly reviewed by U.S. EPA (United States Environmental Protection Agency) (USEPA, 1985; 1987).

Data concerning the inhalation toxicity of copper and its salts are generally lacking. A 50 % increase in volume density of alveolar type II cells and no changes in lung lysozyme levels were reported in rabbits exposed to 0.6 mg/m$^3$ $CuCl_2$ 6 hours per day, 5 days a week for 4 to 6 weeks (Johansson *et al.,* 1984; Lundborg and Camner, 1984). Guinea pigs exposed to an atmosphere saturated with Bordeaux mixture (an aqueous solution of lime and 1.5 % $CuSO_4$) three times a day (duration not reported) for 6.5 months, developed micronodular lesions and small histiocytic granulomas (Pimental and Marques, 1969). Similar lesions have been found in vineyard workers exposed to Bordeaux mixture, but the interpretation of these findings is complicated by the lack of exposure data and contaminant exposure to other agents (e.g. arsenic) (USEPA, 1987).

Occupational exposure to metallic copper dust at a concentration of approximately 0.1 mg/m$^3$ has been reported to produce nausea and metal fume fever, a reversible condition characterised by influenza-like symptoms (Gleason, 1968). It is stated in the TLV documentation (ACGIH, 1986) that "Extensive industrial experience with copper-welding operations and copper-metal reining in Great Britain, support the view that no adverse effects will result from exposure to fumes at concentrations of up to 0.4 mg (Cu/m$^3$)".

In addition to the effects above, Cu fumes are also reported to cause upper respiratory tract irritation and metallic- or sweet taste. The latter effect was observed in welding operations at concentrations of 1 - 3 mg/m$^3$, but not at levels of 0.02 - 0.4 mg/m$^3$. Dust and mists of copper salts are also capable of producing irritation of the upper respiratory tract through inhalation and itchy eczema on skin contact, as well as eye irritation and ulceration of the nasal septum if sufficiently high concentrations are present (ACGIH, 1986).

**Foundry:** No TLV's were exceeded during the study period. The Cu exposure of foundry workers was very low and in accordance with the composition of the materials used in the process. Copper concentrations showed a similarity to Cr concentration with reference to the occurrence and pattern on the time plots

(Figure 3). The measured concentrations can also be regarded as from soil origin. Small amounts of Cu may be present in the scrap metal which is melted at the foundry.

The excess Cu found in the urine of the workers, cannot be related to inhalation, but rather be a result of an inability to excrete the element taken in by other sources of exposure. No harmful Cu-exposure is expected from normal foundry operations. However, an AIER of 114.1% was calculated. The high risk is mainly due to the Cu concentration found in the urine. It is suggested that further research be carried out on the workers to determine the cause of the high Cu concentrations.

**Welding shop:** Copper concentrations in the welding shop showed similar patterns at the same time intervals. Although the measured concentrations were very low, it is suspected that the welding process is responsible for most of it, due to the similarity with the Cr pattern. However, it never exceeded any TLV for Cu and can be regarded as a safe exposure.

An AIER of 71.7% was calculated for the welders. It can also be stated that the high urine-Cu content cannot be as a result of welding fume exposure, but rather from an inability to excrete Cu already in the body or as a result of the intake of food and water. No harmful exposures for Cu in the welding process are expected. Although the risk is high, the occurrence of Cu in the urine cannot be explained by the aerosol exposure. Further research on food and water intake may reveal more information to explain the phenomenon.

### 4.3.4 Iron

Inhalation of iron oxide fume or dust causes a benign pneumoconiosis (siderosis). Iron oxide alone does not cause fibrosis of the lungs of animals, and it is probable that the same applies to humans (Jones and Warner, 1972). Exposures of 6 to 10 years are usually required before changes recognisable by X-ray occur; the retained dust produces X-ray shadows that may be indistinguishable from fibrotic pneumoconiosis (Sentz and Rakow, 1969; Harding, McLaughlin and Doig, 1958).

Of some 25 welders, exposed mainly to iron oxide for an average of 18.7 (range 3 to 32) years, 8 had reticulonodular shadows on chest X-ray consistent with siderosis, but no reduction in pulmonary function; exposure levels ranged from 0.65 mg/m$^3$ to 47 mg/m$^3$ (Harding *et al.*,1958; Kleinfeld *et al.*, 1969).

In another study, conducted by Stanescu *et al.* (1967), the X-rays of 16 welders with an average exposure of 17.1 (range 7 to 30) years also suggested siderosis; but with normal spirograms. However, the static and functional compliance of the lungs was reduced. Some of the welders were smokers and those with the lowest compliance complained of dyspnea.

Although an increased incidence of lung cancer has been observed among hematite miners exposed to $Fe_2O_3$, presumably owing to concomitant radon gas exposure, there is no evidence that $Fe_2O_3$ alone is carcinogenic to humans or animals (Stokinger, 1984).

The ACGIH (1995 - 1996) and the Hazardous Chemical Substances Regulations (South Africa, 1995) recommended a TLV of 5 mg/m$^3$ for $Fe_2O_3$ dust and fume. The basis for this occupational limit is the prevention of pneumoconiosis.

**Foundry:** The measured concentrations in this study cannot be compared to the TLV because the TLV is for total inhalable dust, while the sample measurements were for total respirable dust only. However, the concentrations measured were high, especially when taking the exposure ratio into account. The calculated maximum concentration was > 2.5 mg/m$^3$ which is more than half the TLV for total dust. The urine-Fe concentrations confirm the high exposure. No specific pattern of exposure could be established, but it is evident that the workers are constantly exposed to high concentration of $Fe_2O_3$.

It is difficult to speculate on the health effects of such exposure. At this time there is no evidence to show that the inhalation of pure Fe or $Fe_2O_3$ particles either in welding or inhalation of red oxide dust from oxygen blown steel making processes produce any disease or disability, though changes may show on X-ray plates (Trevethick, 1980). The latter author suggests that the respirable dust concentration must be kept below 10 mg/m$^3$ and chest X-rays be done every 2 years to eliminate individual susceptibility to dust.

The calculated AIER resulted in 43.5% and it is expected that the lungs of chronically exposed workers may show opacities on X-ray examination. However, there should be no corresponding disablement, which is no fibrous tissue and loss of lung function which can be discerned (Schoeman and Schröder, 1994). The lung condition siderosis, caused by Fe exposure, is sometimes referred to as a benign pneumoconiosis because of the spurious radiological picture.

**Welding:** Welders typically are exposed to a complicated mixture of dust and fume of metallic oxides and are subject to mixed dust pneumoconiosis caused by $Fe_2O_3$ (Jones and Warner, 1972). The materials consist of more than 90 % Fe and thus it was anticipated that Fe-concentrations in the welding shop should be high, as found. Although the $Fe_2O_3$ exposure is high, the spirometry results were in accordance with the results of Stanescu *et al.* (1967).

The concentrations were evident from the welding operations and showed a correlation between the Fe content of the materials and the Fe-concentration in the workroom air. It is recommended that the workers be protected against inhalation of Fe fumes, especially with reference to the high urine-Fe concentrations. Until more information is available on the toxicity of $Fe_2O_3$ exposure and uptake, these results can be regarded as an overexposure of the welders, taking the exposure ratio of 1:7.2 into consideration.

The AIER of 27.5% reflects the need of more information on the toxicity of Fe fumes. Although the mean urine-Fe concentration is approximately half of the BEI, the recommended TLV is set too high to protect workers from siderosis. It would be expected that the AIER will be higher if a more appropriate TLV is available. The ACGIH (1996) states that there are inadequate data on which the agent can be classified in terms of its carcinogenicity in humans and/or animals.

### 4.3.5 Manganese

The major concern of humans exposed to Mn is its effects on the central nervous system following chronic exposure. The neurologic disorder known as chronic manganese poisoning occurs after variable periods of heavy exposure ranging from 6 months to 3 years (USEPA, 1984b; Cook, Fahn and Brait, 1974). The disease begins subtly with headache, irritability and occasionally, psychotic behaviour. The latter, manganese psychosis, occurs more frequently in miners rather than in industrial workers, and consists of transitory psychological disturbances such as hallucinations, compulsive behaviour, and emotional instability (Cook *et al.*, 1974). As Mn exposure continues, symptoms include generalised muscle weakness, speech impairment, in-co-ordination, impotence, tremor, paresthesia and muscle cramps (USEPA, 1984b; Cook *et al.*, 1974; Hine and Pasi, 1975).

A study of 369 workers exposed to 0.3 to 20 mg/m$^3$ suggested that slight neurologic disturbances may occur at exposures < 5 mg/m$^3$ but the disturbances seem to be more prevalent at higher exposures (Saric and Lucic-Palaic, 1977). Factors contributing to inconsistencies in the dose-response relationship include broad exposure ranges, different chemical forms and particle size and a lack of good biologic indicators of exposure (USEPA, 1984b).

Reports of manganism (single cases or clusters of cases) include no longitudinal studies and are therefore not adequate to identify a dose-reponse relationship, but do permit the identification of lowest-observed effect level (LOEL). The full clinical picture of chronic manganese poisoning is reported less frequently at exposure levels below 5 mg/m$^3$. The reports of a few early signs of manganism in workers exposed to 0.3 to 5 mg/m$^3$ suggest 0.3 mg/m$^3$ (300 µg/m$^3$) as the LOEL. The data available for identifying effect levels below 0.3 mg/m$^3$ is ambiguous or inadequate. This is further complicated by the fact that good biological indicators of Mn exposure are not presently available. Furthermore, no data suggesting the rate of absorption of Mn through the lungs in humans or animals are available. Extrapolating from other routes of exposure would thus be difficult (USEPA, 1984b).

Furthermore, an association between Mn exposure and pulmonary effects including pneumonia, chronic bronchitis and airway disability has been observed. Extrapolation from animal studies suggests that it is unlikely that Mn could be the sole etiologic agent responsible for serious pathological changes in the lungs. It was mentioned that susceptibility to infection, due to Mn exposure is increased (USEPA, 1984b).

The ACGIH TLV (ACGIH, 1995 - 1996) for elemental and inorganic compounds was lowered from 5 mg/m$^3$ for dust and compounds and 1 mg/m$^3$ for fumes to 0.2 mg/m$^3$ in 1996. These occupational limits are based on protection against chronic manganism, a disease affecting the central nervous system and characterised by fatigue, weakness in the legs, mask-like face, slow monotonous voice, muscular twitching and parkingsonian gait. It is noted in the TLV documentation that workers exposed to Mn fumes develop signs of manganism at exposure levels below those of 5 mg/m$^3$ (ACGIH, 1986). Despite this decrease in the exposure limit, the Hazardous Chemical Regulations still prescribe the old limits (South Africa, 1995).

Data on the carcinogenicity of Mn to humans are lacking and studies in animals are generally limited to parental routes of administration. Intraperitoneal administration are generally limited to parental routes of administration of manganese sulphate. Using the Strain A mouse lung tumour system produced results suggestive (rather than indicative) of carcinogenic activity (USEPA, 1984b).

Based on two studies available only as abstracts, prenatal exposure to Mn may result in behavioural abnormalities in the adult offspring of mice. Of particular interest (because of the route of exposure) is the study of Massaro *et al.* (1980) in which female mice were exposed to 0 or 48.9 ± 7.5 mg/m$^3$ $MnO_2$ dust continuously on days 0 through 18 of gestation. As adult pups from exposed mothers were deficient in open field, exploratory and rotarod (balance and co-ordination) behavioural performance tests, and normal offspring fostered to exposed mothers also had decreased rotarod performance (indicating adverse effects due to postpartum exposure.)

It is noted (USEPA, 1984b) that Mn deficiency, during gestation in experimental animals, result in a variety of developmental defects. These include reduced co-ordination, bone and growth deficiencies, reproductive difficulties and central nervous system changes, which are consequential of decreased chondroitin sulphate formation and delayed otolith calcification.

Reports of impotence in the majority of patients with chronic manganese poisoning are common; however, no other supporting human data are available (USEPA, 1984b). Existing animal data addressing reproductive failure in males describe long-term dietary exposure to Mn.

Manganese is used primarily as an alloy together with other metals and in the manufacture of steel to impact hardness (ACGIH, 1986). Because Mn is ubiquitous in the environment and exposure is thought to occur mainly via the diet (USEPA, 1984b), 20 % of the contribution to total exposure is allowed from air (Calabrese and Kenyon, 1991).

**Foundry:** The materials used in the foundry process do not contain high amounts of Mn and accordingly a low exposure concentration was found. The measured Mn-concentrations were below the prescribed TLV's of both the ACGIH and South Africa. No overexposure of foundry workers were expected. This assumption was confirmed by the low urine-Mn levels measured during the study.

The exposure showed a certain similarity between Fe and Mn on the time plots (Figure 4). This pattern can be ascribed to the materials containing the affinitive elements in different percentages and also to their oxidation states. It is thus expected that iron foundries would show Mn in air concentrations in the ratio of Fe:Mn depicted in Figure 4, considering the percentages in the raw materials.

As expected, the calculated AIER was 9.1% for the foundry workers. The Mn exposure can be regarded as safe and no special medical examinations are recommended.

**Welding shop:** Manganese concentrations measured did not exceed the prescribed TLV's, but if the 1:7.2 ratio is taken into consideration, the ACGIH TLV of 0.2 mg/m$^3$ was exceeded on Tuesday, Wednesday and Friday for at least four hours a day. Such exposures could be the reason for the urine-Mn levels which nearly exceeded the upper limit of the Mn range. Once again, it stressed the importance of good ventilation, to clear the area from contaminant build-up during winter conditions.

The similarity in exposure pattern between Fe and Mn was also obtained for the measurements in the welding shop. The overcast weather conditions probably resulted in atmospheric conditions, which lowered the exposure on Thursday to a safe level.

The 11.2% AIER for Mn in the welding shop was higher than the risk of the foundry. If the over-exposure to Mn-concentrations continue for long periods, signs and symptoms of Mn-poisoning can be expected from welders at this plant. Good ventilation practice and the heating of work atmospheres should prevent such conditions.

### 4.3.6 Zinc

Inhalation of ZnO fume cause an influenza-like illness termed metal fume fever (NIOSH, 1975a; McCord, 1960). During exposure to ZnO fume, effects are dryness and irritation of the throat, a sweet or metallic taste, substernal tightness and constriction in the chest, as well as a dry cough. Several hours following exposure, the subject develops chills, lassitude, malaise, fatigue, frontal headache, low back pain, muscle cramps, and occasionally blurred vision, nausea and vomiting (Rohrs, 1957). Physical signs include fever, perspiration, dyspnea, rales

through the chest, and tachycardia; in some instances, there has been a reversible reduction in pulmonary vital capacity.

The critical factor in the development of the syndrome is the size of the ultrafine ZnO particles produced when Zn is heated to temperatures approaching its boiling point of 907 °C in an oxidising atmosphere (Brown, 1988). The particles must be small enough (< 1 µm) to reach the alveoli when inhaled. The syndrome is not produced when normal zinc oxide powder is inhaled or taken orally (McCord, 1960). Only freshly formed fume cause the illness, presumably because flocculation occurs in the air with formation of larger particles that are deposited in the upper respiratory tract and do not penetrate deeply into the lungs (AIHA, 1969).

Data on exposure concentrations and duration associated with metal fume fever are insufficient. Early reports found moderate symptoms after 12 minutes exposure at 600 mg/m$^3$. Other investigators found no signs of chronic toxicity with occupational exposures of 3 to 15 mg/m$^3$ for periods up to 35 years. It has been noted that the highly toxic metal Cd is obtained wherever Zn is refined, and the possibility of Cd exposure should be considered when Zn is refined (Elinder, 1986).

A short-term study of guinea pigs exposed to ZnO fume for 3 h/24h for 6 days at the TLV of 5 mg/m$^3$ revealed pulmonary function changes and morphologic evidence of small airway inflammation and oedema. Pulmonary flow resistance increased, compliance decreased and lung volumes and carbon monoxide (CO) diffusing capacity decreased. Some of these changes persisted for the 72 hours duration of post-exposure follow-up (Lam *et al.*, 1985).

The dust of ZnO is considered a nuisance dust that has little adverse effect on the lungs and does not produce significant organic disease when exposures are kept under reasonable control (Elinder, 1986). The ACGIH (1995 - 1996) and Hazardous Chemical Substances regulations prescribe a TLV 5 mg/m$^3$ for Zn fumes and 10 mg/m$^3$ for total Zn dust. The TLV's are based on the prevention of metal fume fever and NIOSH (1975a) also mentioned the prevention of possible chronic respiratory effects.

A relationship between Zn-Pb-Cl seems to exist when air was sampled on a time variation basis. Figure 13 shows a definite similarity between the three different substances. The reason for the affinity between the elements is unknown,

although the Zn and Cl might be as a result of the degreasing of parts in caustic soda inside the welding shop.

**Foundry:** All measured concentrations were very low and below the prescribed TLV's. This is a result of the very low Zn content in the materials. No harmful effects due to Zn exposure are expected. The high urine-Zn concentrations obtained from the workers cannot be regarded as a result of this exposure, although small quantities of Zn may be present in the scrap metal which is melted at the foundry.

The risk due to inhalation of the Zn aerosols is 92.4% for the exposed foundry workers. Although this risk is high, it cannot be the result of aerosol inhalation because the workers are not exposed to Zn fumes. It can be regarded as a rare metabolism disorder as in the case of Cu.

**Welding shop:** The Zn-concentrations in the welding shop were below all prescribed TLV's and indicative of the process which contains Zn only in very small amounts. No harmful health effects are thus expected from this exposure. Urine-Zn concentrations cannot be attributed to the Zn-concentrations measured in the welding shop. The risk of 59.6% can also be the result of a metabolism disorder as in the case of the foundry workers.

### 4.3.7 Lead

Prolonged absorption of Pb or its inorganic compounds results in severe gastrointestinal disturbances and anaemia. With more serious intoxication, there is neuromuscular dysfunction and the most severe Pb exposure may result in encephalopathy (Hathaway *et al.*, 1991).

The onset of the symptoms of Pb poisoning or plumbism often is abrupt. Presenting complaints are often weakness, weight loss, lassitude, insomnia and hypertension (Kehoe, 1972; Klaassen, 1980). Associated with these complaints is a disturbance of the gastrointestinal tract, which includes constipation, anorexia and abdominal discomfort, or actual colic, which may be excruciating (Kehoe, 1972). Physical signs usually are facial pallor, malnutrition, abdominal tenderness and pallor of eye grounds. Lead absorption is cumulative and elimination of the metal from the body is slow requiring a considerably longer time than the period of storage of toxic amounts (Kehoe, 1972; NIOSH, 1972; Klaassen, 1980).

When asymptomatic Pb workers are subjected to a sudden increase in exposure to and absorption of Pb, they often respond with an episode of typical Pb poisoning. Removal of the worker from exposure to abnormal quantities of lead often leads to a seemingly sudden and apparently complete recovery, even when the individual has had a considerable quantity of residual Pb in the body (Kehoe, 1972).

The basis for Pb toxicity is its ability to bind ligating groups in physiologically critical biomolecules, which may then disrupt their function by competing with essential metal ions for binding sites, inhibiting enzyme activity, and altering ion transport.

Although Pb induces toxicity in a number of organ systems, haematological, neurological and renal effects are generally considered the most significant because they are induced at lower levels of exposure. Lead inhibits the heme synthesis pathway at several specific steps and also causes disturbances in globin biosynthesis. The resulting anaemia is characterised as mildly hypochromic and normocytic with reticulocytosis due to shortened survival time of erythrocytes, which are more fragile and have increased osmotic resistance. In addition, Pb alters vitamin D metabolism secondary to heme synthesis effects at blood levels below 30 µg/dℓ.

In adults, central and peripheral nerve dysfunction occurs at blood lead levels as low as 40 - 60 µg/dℓ, and slowed nerve conduction velocities in peripheral nerves at levels as low as 30 - 50 µg/dℓ with no clear threshold for these effects (Calabrese and Kenyon, 1991). In children, there is evidence for decrements in IQ and behavioural (reaction time and psychomotor performance) and electrophysiological (altered EEG patterns and peripheral nerve conduction velocities) effects at blood levels as low as 15 - 30 µg/dℓ and even lower.

Lead also has effects on the cardiovascular system, liver, gastrointestinal tract, and endocrine system, but these systems are generally not as sensitive to the effects of Pb as the haematological and nervous systems. However, some recent evidence suggests significant elevations in blood pressure in animals and humans may occur following chronic low-level Pb exposure (Calabrese and Kenyon, 1991).

Occupational limits of 0.05 mg (Pb)/mg$^3$ (ACGIH, 1995 - 1996), 0.15 mg/m$^3$ (South Africa, 1995), as well as a recommended TLV of < 0.10 mg (Pb)/mg$^3$ (NIOSH, 1985) exist. All of the occupational limits were set in consideration of systemic toxicity, principally effects on the blood, nervous system, and kidney (ACGIH,

1986; NIOSH, 1978; 1985). The lower limit proposed by NIOSH is apparently based on reports of decreased nerve conduction velocities and other effects in workers with blood leads between 0.05 and 0.07 mg/100 g. The national ambient air quality standard in the United States of America is 1.5 $\mu g/m^3$ (USEPA, 1986a; 1986b; Calabrese, 1978).

**Foundry:** The Pb concentrations found in the foundry air were below all prescribed threshold limit values set by authoritative agencies. The materials used did not contain Pb in meaningful quantities and resulted in the low Pb concentrations in both air and urine. No harmful Pb exposure is thus expected. However, the concentrations ranged between 0 and 7.0 $\mu g/m^3$ exceeding the ambient air quality standard of 1.5 $\mu g/m^3$ and with such chronic exposure, it is expected that the Pb concentration in the body will increase.

It is suggested that the peak concentrations found during this study may be as a result of Pb in vehicle gas emissions. This assumption is made because the Br concentrations (Figure 6) showed a similar pattern as Pb, and it is known that Br is a petrol additive. The foundry is surrounded by roads which are used for general transporting purposes.

The AIER is 8.4% which indicates normal environmental exposure. The workers are not exposed to any Pb fumes in the process and no health risk due to Pb exposure is expected.

**Welding shop:** None of the recommended TLV's were exceeded by the measured Pb concentrations which ranged from 0 to 9.6 $\mu g/m^3$. No materials used in the welding process contains Pb in meaningful quantities and the concentrations found in the air, as well as in the urine can thus be regarded as non-occupational exposure concentrations.

A similarity in exposure pattern was observed between Pb-Zn-Cl concentrations (Figure 13). South Africa still use petrol with Pb based additives, hence vehicle emissions can be recognised by the tracer elements Pb and Br (Figure 14), which have approximately similar time variations. These time variations also show a typical pattern of morning and afternoon peaks due to rush hour traffic.

The low Pb-Zn-Cl concentrations can possibly also be as a result of vehicle emissions from nearby roads and highways (20 km radius). The rapid variation in Zn concentrations up to 24.6 $\mu g/m^3$ indicates a point source in the area which is

believed to be the degreasing of parts in caustic soda and the adjacent electroplating facility. Despite the fact that the source have not been positively identified, a source profile could be established (Kneen *et al.* 1994).

An AIER of 10.8% was calculated for the Pb exposure of the welders. This risk is higher than the foundry workers' risk because their workplace is closer to roads with heavy traffic and associated emissions.

No harmful effects are expected from exposure to the measured substances. With the recent introduction of unleaded petrol in South Africa, it is expected that this exposure will decrease in future.

### 4.3.8 Aluminium

The aluminas are considered to be nuisance dusts and under conditions of human exposure may cause, at most, a minimal pulmonary nodular response. The group of compounds referred to as aluminas is composed of various structural forms of aluminium oxide, trihydroxide and oxyhydroxide. As these aluminas are heated, dehydration occurs, producing a variety of transitional forms. Temperatures between 200°C and 500°C result in low-temperature-range transitional aluminas characterised by increased catalytic activity and larger surface area (Mitchell, *et al.* 1961).

Despite the problems in defining precise exposures (in terms of structure and form), population studies of potentially exposed workers have shown minimal evidence for pulmonary fibrosis or pneumoconiosis. Epidemiological studies of aluminium smelter workers have confirmed either minimal or absent fibronodular disease and no excess mortality associated with pneumoconiosis (Alfrey, LeGendre and Koehny, 1976; Gibbs, 1981; Wisniewski and Sturman, 1989).

Animal experiments with alumina have shown that the type of reaction in lung tissue is dependent upon the form of alumina and its particle size, the species of animal used, and the route of administration. Intratracheal administration of $\tau$-alumina with a 2 µm average size caused only a mild fibrous reaction of loose reticulin (Milham, 1983). However, intratracheal administration of $\tau$-alumina of 0.02 to 0.04 µm size in rats produced reticulin nodules that later developed into areas of dense collagenous fibrosis (Longstreth, Rosenstock and Heyer, 1985). The latter alumina by the same route in mice and guinea pigs, caused development of a reticulin network with occasional collagen, whereas in rabbits

only a slight reticulin network was observed. Intratracheal administration of another form of alumina in rats, corundum of particle size less than 1 µm, caused the development of compact nodules of reticulin (Milham, 1983).

A review of the animal studies concluded that a fibronodular response has resulted only from intratracheal insufflation of catalytically active, low temperature range transitional aluminas, and high-surface aluminas (Mitchell, 1959). In general, alumina is efficiently eliminated from the lung and has a low degree of fibrogenicity.

The ACGIH and the South African Government prescribes a TLV of 5 mg/m$^3$ for respirable aluminium oxides (ACGIH, 1995 - 1996; South Africa, 1995). Furthermore, the ACGIH makes provision for Al welding fumes with a TLV of 5 mg/m$^3$.

**Foundry:** The measured exposure concentrations were well below the recommended TLV's and should not result in negative health effects on the workers. The materials used in the foundry process contain only small amounts of Al (0.3 %). The small amounts of the substance is also reflected in the AIER of 4.3%. This contribution from the materials, together with the normal occurrence of Al in soil, can be regarded as the sources of this exposure.

**Welding shop:** The welding materials do not contain Al and this might be the reason for the low concentrations found in the air. The concentrations were below all recommended TLV's. The concentration can be regarded as a result of the presence of soil of which Al is an element. The AIER of 2.9% reflects the low exposure to the substance and no negative health effects are expected due to inhalation of Al in the air of this locality.

### 4.3.9 Potassium

Potassium hydroxide (KOH) is a severe irritant of the eyes, mucous membranes and skin. The effects of KOH are similar to those of other strong alkali's such as sodium hydroxide (NaOH). Although inhalation of KOH usually is of secondary importance, the effects from the dust or mist will vary from mild irritation to severe pneumonitis, depending on the severity of exposure (NIOSH, 1975b). The greatest industrial hazard is rapid tissue destruction of the eyes or the skin upon contact with either the solid or concentrated solutions.

A short term occupational exposure limits of 2 mg/m$^3$ is recommended by the Hazardous Chemical Substances Regulations (South Africa, 1995). No limits are specified by the other agencies.

**Foundry:** The measured concentrations were below the South African TLV of 2 mg/m$^3$ which is regarded as a safe limit for chronic occupational exposure. It is not envisaged that this exposure would harm the health of exposed workers. Soil can be regarded as the main source of K in this workplace.

**Welding shop:** The K concentrations did not exceed the TLV and can be regarded as a safe occupational exposure to K. Since K does not form part of any materials used in the welding process, it is assumed that the exposure is mainly a result of soil in the air. The higher than normal ambient K concentrations may be as a result of the degreasing process in the welding shop where caustic potash (KOH) is used in degreasing tanks.

### 4.3.10 Magnesium

Magnesium oxide (MgO) fume is a mild irritant of the eyes and the nose. Experimental subjects exposed to fresh MgO fumes developed metal fume fever, an illness similar to influenza. Effects of exposure were fever, cough, oppression in the chest and leukocytosis (Drinker, Thomsom and Finn,1972). There are, however no reports of metal fume fever resulting from industrial exposure to MgO fume (Drinker *et al.*, 1972; AIHA, 1960).

Examination of 95 workers exposed to an unspecified concentration of MgO dust revealed slight irritation of the eyes and the nose; the Mg level in the serum of 60 % of those examined was above the normal upper limit of 3.5 mg/d$\ell$ (Stokinger, 1981b).

The Hazardous Chemical Substances Regulations (South Africa, 1995) recommended a TLV of 5 mg/m$^3$ for respirable MgO.

**Foundry:** The Mg concentrations were below the TLV and can be regarded as a safe exposure. It is assumed that the Mg in the foundry air is the result of soil in the workroom air, although Mg (Ferro silicon magnesium$^{TM}$) is used as a by-product to harden metals and to increase the ductility and strength. It must be stressed that sand is used excessively in the foundry process and although Mg is a

soil element, it is present in higher than normal ambient concentrations, probably due to the addition of Mg in the molten metal.

**Welding shop:** The very low Mg concentrations in this workroom air could be regarded as typical soil exposure from ambient air and should not pose any health risk to workers when inhaled.

### 4.3.11 Calcium

Calcium oxide (CaO) is an irritant of the eyes, mucous membranes and skin. The irritant effects are probably due primarily to its alkalinity, but dehydrating and thermal effects also may be contributing factors (ACGIH, 1986). Strong nasal irritation was observed from exposure to a mixture of dust containing CaO in the range of 25 mg/m$^3$, but levels of 9 to 10 mg/m$^3$ produced no observable irritation (Wands, 1981). Inflammation of the respiratory tract, ulceration and perforation of the nasal septum and pneumonia have been attributed to inhalation of CaO dust. Severe irritation of the upper respiratory tract ordinarily causes persons to avoid serious inhalation exposure (ACGIH, 1986; Wands, 1981).

A TLV of 2 mg/m$^3$ is recommended by ACGIH (1995 - 1996) and South Africa (1995).

**Foundry:** The Ca concentrations were below the TLV and can be regarded as a safe exposure. It is assumed that the Ca in the foundry air is the result of soil being present in the workroom air. It must be stressed that sand is used excessively in the foundry process and although Ca is a soil element, it is present in higher than normal ambient concentrations.

**Welding shop:** The very low Ca concentrations in this workroom air could be regarded as typical soil exposure from ambient air and should not pose any health risk to workers when inhaled.

### 4.3.12 Silicon

Crystalline silica causes silicosis, a form of disabling, progressive and sometimes fatal pulmonary fibrosis, characterised by the presence of typical nodulation in the lungs (NIOSH, 1974a).

The earliest lesions are seen in the region of the respiratory bronchioles. Lymphatics become obliterated by infiltration with dust laden macrophages and granulation tissue. Morphologically, the typical lesion of silicosis is a firm nodule composed of concentrically arranged bundles of collagen; these nodules usually measure between 1 to 10 mm in diameter and appear around blood vessels and beneath the pleura, as well as in mediastinal lymph nodes. There may be conglomeration of nodules as the disease progresses, leading to massive fibrosis (NIOSH, 1974a). The pulmonary pleura usually is thickened because of fibrosis and often is adherent to the parietal pleura, especially over the upper lobes and in the vicinity of underlying conglomerate lesions (Parkes, 1982).

The progression of symptoms may continue after dust exposure ceases. Although there may be a factor of individual susceptibility to a given exposure to silica dust, the risk of onset and the rate of progression of the pulmonary lesion clearly are related to the character of the exposure (dust concentration and duration) (NIOSH, 1974a). The disease tends to occur in years rather than in months. It is generally accepted that silicosis predisposes to active tuberculosis and that the combined disease tends to be more rapidly progressive than uncomplicated silicosis.

A group of 972 granite shed workers were studied to relate exposure levels to incidence of silicosis (Russel *et al.*, 1929). Those with the highest exposure (37 - 60 mppcf) showed development of early silicosis in 40 % of the workers after 2 years and 100 % after 4 years of exposure. The development of silicosis in the remaining workers appeared to be proportional to the dust exposure. At the second highest exposure level (27 - 44 mppcf), early stages of silicosis appeared after 4 years of exposure, and more advanced stages developed by the seventh year. In the group exposed at an average of 20 mppcf, there was little indication of severe effects upon the health of the workers. In the lowest exposed group (3 - 9 mppcf), there was no indication of any adverse health effects of dust exposure to workers.

The International Agency for Research on Cancer (IARC, 1987) has determined that there is insufficient evidence for the carcinogenicity of crystalline silica to experimental animals and limited evidence for the carcinogenicity of crystalline silica to humans.

Occupational limits of 3.0 mg/m$^3$ (South Africa, 1995), 2.0 mg/m$^3$ (ACGIH, 1995 - 1996) for respirable amorphous silica dust (South Africa, 1995), 0.1 mg/m$^3$ for $\alpha$-quartz and 0.05 mg/m$^3$ for tridymite (ACGIH, 1995 - 1996; South Africa,

1995) are used worldwide. The National Institute for Occupational Safety and Health (NIOSH, 1974a) recommended a TLV of 0.05 mg/m$^3$ for respirable crystalline silica. These TLV's are used because some evidence exist for the carcinogenicity of crystalline silica to experimental animals and humans (ACGIH, 1986).

**Foundry:** Maximum Si-concentrations measured at the sampling point nearly exceeded the prescribed threshold limit values. If the exposure ratio of 1:8.25 is taken into consideration, all concentrations higher than 87 µg/m$^3$ exceeds the threshold limit value for $\alpha$-quartz. This means that the workers are overexposed for between 2 - 5 hours per day. This situation is worsen by the presence of highly toxic tridymite. For tridymite, all concentrations higher than 155 µg/m$^3$ can be considered as an overexposure. Figure 7 shows that tridymite overexposure occurred every day of the week for at least an hour, except for Tuesday and the last part of the weekend during which no work was conducted.

An AIER of 91.3% was calculated and it is envisaged that the foundry workers are at risk of silicosis due to chronic short overexposures during their work. These findings confirm the symptoms of a slight restriction in pulmonary airflow as obtained from the spirometry tests. It also confirms the observation of excessive saliva production in some of the workers, regarded as one of the first symptoms of silicosis. This shows that contaminant control should be enforced in order to protect the workers with a long occupational exposure in this foundry.

It is recommended that blood pressure tests, lung function tests and X-ray examinations be carried out on the workers exposed to crystalline silica (NIOSH, 1974a). Radiological examinations are very important because it is the only method of diagnosing silicosis at an early stage.

**Welding shop:** The Si concentrations measured in the welding shop were very low and should not pose any health risk to the exposed welders. A maximum background concentration of 4.5 µg/m$^3$ during night time is seen in Figure 15. The concentrations higher than 4.5 µg/m$^3$ may be as a result of the Si content in the steel and especially the welding rods. No $\alpha$-quartz or tridymite were found in the samples.

The calculated AIER is 39.2%, reflecting low presence of silica in the workroom air. Although no $\alpha$-quartz or tridymite were found in the samples, it is still believed that the workers are exposed to the substance, considering the fact that the materials

do contain Si and it is heated above 860°C. The method of analyses may not be sensitive enough to determine the $\alpha$-quartz content, however, the AIER formula showed evidence of such exposure. Sferlazza and Beckett (1991) state that some fluxes may contain as much as 20% silica which transforms to silicon dioxide during the welding process.

### 4.3.13 Titanium

Titanium dioxide ($TiO_2$) is a mild pulmonary irritant and generally is regarded as a nuisance dust, i.e. a dust that tends to remain in the lung tissue and although potentially able to produce adverse health effects at higher concentrations, is not generally associated with induction of a proliferate response.

In a study concerning 15 workers, who had been exposed to $TiO_2$ dust, three showed radiographic signs in the lungs resembling "slight fibrosis", without disabling injury. The magnitude and the duration of exposure, however, were not specified (AIHA, 1978a; Browning, 1969). In the lungs of three workers involved in processing $TiO_2$ pigments, deposition of the dust in the pulmonary interstitium were associated with cell destruction and slight fibrosis. The findings indicate that $TiO_2$ is only a mild pulmonary irritant (Elo *et al.*, 1972).

Rats repeatedly exposed to concentrations of 10 to 328 mppcf of air for as long as 13 months showed small focal areas of emphysema, which were attributed to large deposits of dust. There was no evidence of any specific lesion being produced by $TiO_2$ (Christie, Mackay and Fischer, 1963). Exposure of rats to 250 mg/m$^3$ $TiO_2$ in a 2 year inhalation bioassay resulted in the development of squamous cell carcinomas in 13 of 74 female rats and in 1 of 177 male rats, as well as an increase in bronchoalveolar adenomas. No excess tumour incidence was observed at 50 mg/m$^3$. Given the extremely high concentration exposures, the unusual histology, the location of the tumours and the absence of metastases, the authors questioned the biological relevance of these tumours to humans (Lee, Trochimowicz and Reinhardt, 1985).

The TLV documentation states that "there has been no evidence of danger to health from inhalation of $TiO_2$ in concentrations that do not exceed 10 mg/m$^{3\alpha}$ (ACGIH, 1986). It is also indicated by a number of investigators to consider $TiO_2$ to be in the category of a nuisance dust. OSHA proposed to adopt the TLV-TWA for total dust as part of its final rule limits (OSHA,1989). The Hazardous Chemical

Regulations (South Africa, 1995) recommended a TLV of 5 mg/m$^3$ for respirable $TiO_2$.

**Foundry:** The materials contain Ti in very small quantities and $TiO_2$ concentrations never exceeded the TLV, even if the exposure ratio of 1:8.25 was applied. However, $TiO_2$ dust is capable of causing irritation of the eyes, skin and respiratory tract at the exposure levels as found (Carson *et al.*, 1991; OSHA, 1989).

Titanium dioxide occurs as a crystalline solid; the major crystalline forms are anatase and rutile. It is usually considered insoluble in water and because of its physical properties, would exist as a dust in ambient air (ACGIH, 1986). The occurrence of $TiO_2$ in the foundry air can thus be ascribed to the high Ti content in the sand used for moulds. This is confirmed by the similarity in exposure pattern of the soil elements during the sampling (Figure 7). The absence of the dust during the weekend once again confirmed that it is only present in meaningful quantities when the foundry is in operation.

**Welding shop:** From the results in Figure 15F, it is evident that Ti is only present during welding operations and it can be assumed that the welding materials contain small amounts of the metal. The $TiO_2$ concentration, however, never exceeded the TLV.

According to Carson *et al.* (1991), $TiO_2$ can produce irritation at ambient air concentration and such effect can thus be expected during welding operations. No other harmful health effects are expected in the welding shop as a result of $TiO_2$ exposure.

## 4.4 Other elements

### 4.4.1 Chlorine

Oceanic activity and industrial processes are the main sources for $Cl^-$ in the solid phase (Bridgman, 1990). Biomass burning from human sources enriches all ionic constituents in aerosols by between a factor of 2 and 3, except $Na^+$ (Andreae *et al.*, 1988). Much of the change occurs in the fine particulate mode in layers, as the haze adapts itself to small variations in atmospheric stability. Average concentrations of $Cl^-$ in aerosols over Amazonia in the mixing layer were $1.2 \pm 0.9$ nmol/m$^3$ in coarse aerosols and not detected in fine aerosols (Talbot *et al.*, 1988).

Data on atmospheric $Cl^-$ levels are limited because most studies measure "gaseous chloride" and do not distinguish between $Cl^-$, HCl or other Cl ion species. Some investigators have also questioned the presence of $Cl^-$ in the atmosphere based on its high reactivity and hence limited solubility. It is thought that atmospheric reactions involving sodium chloride (NaCl) aerosols are the major source of "gaseous chlorides" (WHO, 1982).

No TLV for $Cl^-$ in the solid phase was adopted at any of the regulatory agencies concerning workroom air quality.

**Foundry:** Comparing atmospheric levels to the measured concentrations inside the factory, $Cl^-$ showed high concentrations for most of the time during the week (Figure 5). The concentrations during Saturday afternoon and Sunday can be regarded as normal atmospheric levels. It is envisaged that the exposed workers would experience ocular and respiratory irritation during working operations (NIOSH, 1976b).

The low exposures during Saturday afternoon and Sunday could be salts from nearby roads and industries (Kneen *et al.,* 1994). However, the high Cl concentrations measured may also be from the use of trichloro-ethylene during cleaning operations in the mould making process. Metal fluxing, being the largest operation in this area, is regarded as the major source of Cl in the foundry air (Hathaway *et al.* 1991).

**Welding shop:** Exposures during the day, especially during the mornings, exceeded the TLV. Workers may experience irritation of the lungs and eyes during such overexposures (NIOSH, 1976b). The concentrations were very high during periods when the doors and the windows were closed. The concentration during Thursday with its overcast conditions, is noticeably lower than the other.

Sources such as salts from roads and industries have been documented (Kneen *et al.,* 1994), but metal fluxing, detinning and dezincing iron has also been described as sources of Cl (Hathaway *et al.,* 1991). It is thus assumed that the majority of the latter processes, during the welding operations, are responsible for the high Cl concentrations.

### 4.4.2 Bromine

Van Leeuwen and Sangster (1987) considered that the diet was the major source of Br exposure in humans and that concentrations in ambient air are due to ocean-derived aerosols and volcanic activity, with some contributions from automobile exhaust and the use of Br-containing compounds. They concluded that: "Although the occurrence of bromine-containing compounds in the atmosphere is not of toxicological concern, it poses an indirect threat to human health since Br, like Cl, can reduce $O_3$, and therefore is a risk to the protective layer of the earth." Since the ambient air level goal (AALG) for Br is based on irritant effects, no relative source contribution from air was factored into the AALG calculation. Soil contains a concentration of 1.6 parts per billion (ppb) Br as one of the basic elements in the earth's crust.

No occupational related TLV's are set for Br compounds in the particulate phase.

**Foundry:** The Br concentration were high during several time-intervals and it reached a maximum concentration of 4.23 $\mu g/m^3$ on Sunday. This maximum peak cannot be explained easily, but it is believed that it can be a result of vehicle exhaust fumes and Br containing crustal particles. The crustal particles may show the same composition as volcanic rock after being heated by the molten metal. Automobile exhaust fumes become obvious when comparing the Br concentration with the graph for Pb which are both petrol additives (Figure 6).

No adverse health effects, due to inhalation of Br in the foundry, is expected and it is believed that the occurrence of Br will decrease in future, especially with the introduction of unleaded petrol in South Africa. However, the workers might experience irritation in the respiratory tract during times of exposure to the substance.

**Welding shop:** Bromine concentrations inside the welding shop were high during the sampling period, especially during the morning shifts on Tuesday and Wednesday. The air temperatures, at these times, were very low and the workers made fires for heating in the building. Figure 14 shows that the concentration dropped to normal after the opening of the windows and doors (± 12:00). It is believed that the high concentrations measured are a result of the burning of wood treated with substances containing Br or even the burning of liquids which contain Br as an additive.

No harmful health effects are expected at exposed workers, but adequate ventilation should be provided to prevent workers from burning Br-containing substances during chilly conditions. Chronic exposure to such overexposures could be irritating to the lungs (Henderson and Haggard, 1943).

### 4.4.3 Sulphur

Sulphur (S) is an important pollutant species that occurs in the atmosphere primarily as $SO_2$, $H_2S$ and particulate $SO_4$, though there are small amounts of mercaptans (Radical-SH) and methyl sulphide $(CH_3)_2S$. Natural sulphur comes from $H_2S$ released by $SO_4$ reducing bacteria; anthropogenic sulphur is usually released as $SO_2$ from coal burning, petroleum combustion and smelting processes.

In industrialised countries, particulate sulphur is present in the atmosphere as $SO_4$, mostly from conversion of ($SO_2$) produced from fossil fuel combustion (Kneen *et al.*, 1994). Sulphur is normally present in soils in an oxidised state ($SO_4^{2-}$), with concentrations varying according to the amount of organic matter. Mineral soils contain only 0.01-0.06 % dry weight (Bridgman, 1990).

A large fraction of fine aerosols are $SO_4^{2-}$. In the nucleation stage, S often occurs as part of a liquid droplet of sulphuric acid ($H_2SO_4$), grows rapidly to accumulation size, and eventually forms a stable non-reactive particle containing $SO_4^{2-}$ (Wolff, 1984). Typical concentrations of sulphate aerosols are 1-2 $\mu g/m^3$ in remote background areas, <10 $\mu g/m^3$ in non-urban continental areas and >10 $\mu g/m^3$ in areas under urban and anthropogenic influence.

Measurements in North America have established that concentrations of all major parameters are higher in summer than in winter, with $SO_4$ up to 2.5 times higher in the warm season. However, at ground level in Scandinavia, aerosol concentrations in winter are higher because of the greater frequency of stable atmospheric conditions and more emissions associated with heating (Lannefors, Hansson and Granat, 1983). Changes from night to day affect fine particles the most, as they react rapidly to changes in sunlight, atmospheric stability and airflow. Changes in coarse aerosols from night to day are normally not very great.

No occupational TLV is established for S or $SO_4$. Although individual air quality standards have been promulgated for $SO_x$ and particulate matter, the health effects of these pollutants are not easily separated. Since they are usually produced by a common source, such as the combustion of coal, high $SO_x$ levels are often

associated with high particulate matter levels. There is also some scientific evidence that particulate matter potentiates the toxic response to $SO_x$ (Godish, 1991). Irritation, respiratory disease, coughing and breathing problems can be associated with S and particulate matter exposure. Lyons and Scott (1990) mentioned that an increased frequency of asthma attacks would occur at levels higher than 150 $\mu g/m^3$.

**Foundry:** As expected, the $SO_4$ concentration was higher than normal atmospheric levels because sampling was carried out inside the factory. The $SO_4$ concentrations in the foundry were higher than those measured in the welding shop, probably due to the higher particulate concentrations inside the foundry plant. No adverse health effects are expected although the exposure might result in irritation, coughing, sinusitis and conjunctivitis (Plunkett, 1987).

It is suspected that the $SO_4$ concentrations measured are a result of wood and coal fires used for heating the foundry, as well as from other sources such as molten metal containing sulphur (Table 7) and vehicle exhaust gases. It may also be from the casting of non-ferrous metal (Hathaway *et al.*, 1991).

**Welding shop:** Sulphate concentrations measured in the welding shop were also higher than normal atmospheric $SO_4$ concentrations. The morning concentrations were higher than the concentrations during the rest of the day, except during Thursday when conditions were overcast (Figure 16), probably due to the absence of sunlight which minimise concentrations (Bridgman, 1990). The health consequences of indoor $SO_4$ exposures may only partly lie in the direct effects of particulates and $SO_2$ gas on the human respiratory system. $SO_2$ is a combustion product of burning of materials containing S (Hathaway *et al.*, 1991) and confirms the presence of the substance in this workroom due to the burning of wood and coal to provide heating of the environment during the winter mornings. It is more likely that health effects attributed to $SO_2$ are due to the highly irritant effects of sulphate aerosols such as sulphuric acid, which are produced from $SO_2$.

It is expected that exposed workers could suffer from irritation, especially workers with asthma. Irritation of the eyes, nose and throat, as well as rhinorrhea, choking and coughing could be also be expected (DOL, 1975).

## 4.5 Source apportionment

Although $SO_4$ is the major component of fine aerosols, trace metal ions can be used to establish the relative importance of sources. Trace metal ions exhibit a wide range of concentrations, depending on site, nearby sources and wind direction frequency. Barrie (1988) established four major sources for aerosols in southern Canada. Fifty-eight percent of the aerosols consisted of a soil factor (Ti, Al, Ca, Fe), 15 % of auto exhaust emissions (mainly Pb and Br), 9 % of smelter and power station stack emissions (Cu, $SO_4$) and 6 % from non-ferrous metals and smelters (Ir, As). Using patterns of time variations and elemental ratios, a number of sources or source categories have been identified in the atmospheres of the two localities.

**Foundry:** Figure 9 shows that 6 source types have been identified:

- Heavy metal products from smelting at the foundry during the manufacturing of crusher balls - P, S, Cr, Ni, Fe and Mn.
- Heavy metal products from smelting at the foundry during the manufacturing of brake blocks - P, S, Cr, Fe, Mn, Al and Ni. This source is identified mainly due to its high Fe content.
- Crustal particles from sources including local soil, sand for the mould making, resuspended road dust and coal fires in the nearby vicinity - Al, Si, K, Ca, Ti, Mn and Fe.
- Sulphur particles from conversion of $SO_2$ gas in the air with a major S composition.
- Traffic-related combustion products due to petrol additives - Pb, Br and possibly Zn.
- Other heavy metals - Cu, Ni and V.

**Welding shop:** Figure 17 shows that 6 source types have been identified:

- Heavy metal products from welding of stainless steels at the welding shop during the manufacturing of train trucks - P, S, Cr, Fe, Ni, V and Mn. This source is mainly identified due to its high Cr and Fe content.
- Heavy metal products from mild steels at the welding shop during manufacturing of train trucks - Mn, Fe, Ni, P and S. This source is mainly composed of Fe and Mn.

- Crustal particles from sources including local soil, ash from coal burning during mornings, resuspended road dust and coal fires in the nearby vicinity - Al, Si, K, Ca, Ti, Mn and Fe.
- Sulphur particles from conversion of $SO_2$ gas in the air with a major S composition.
- Traffic related combustion products due to petrol additives - Pb, Br and possibly Zn.
- Other heavy metals - Cu, Ni, and V.

In Figure 23 a source apportionment was carried out on the exposure of 9 May 1995, which was randomly selected from the sampling period. The figure shows that the concentration of dust caused by the manufacturing of brake blocks and crusher balls, decreased during the night period (00:00 - 08:00) during which no operations took place. The increase in crustal component during this time period may be the result of sand particles from the moulds, dispersed by the movement of air.

The source apportionment in Figure 24, carried out on the exposure of 3 May 1995, shows that the elements for the stainless steel source, increased during the night period when no operations were conducted This increase may be the result of dispersed welding fumes of which the particle size is too small to settle out. When particles in the smaller aerodynamic ranges (0-0.5 μm) are present in air, it can take several hours to settle out (Schoeman and Schröder, 1994). Settlement would mainly take place due to coagulation and the formation of an agglomerate resulting in gravitation.

Figures 25 and 26 shows the source apportionment during times when the plants were closed and none of the normal activities carried out. The apportionment for the foundry shows that the crustal component increased compared to concentrations during normal operations. The depicted concentrations can be regarded as soil exposure, mainly caused by sand used in mould making and dispersed into the workroom by means of air movement. It is obvious that all sources, other than soil, decreased and may be an indication of the normal occurrence of heavy metal elements in soil.

The apportionment in the welding shops' air (Figure 26) shows that particles from the stainless steel welding remain in the air, probably due to its particle size. It is known that welding fume particles are in the aerodynamic range of 0 - 0.5 μm and thus remaining longer in air before settling out. The building is closed during this

time period (00:00 - 07:00) and very little air movement takes place, therefore no natural cleansing of the indoor atmosphere can take place. However, it is envisaged that the indoor atmosphere of the locality should purify to a greater extent over a longer time period such as a weekend.

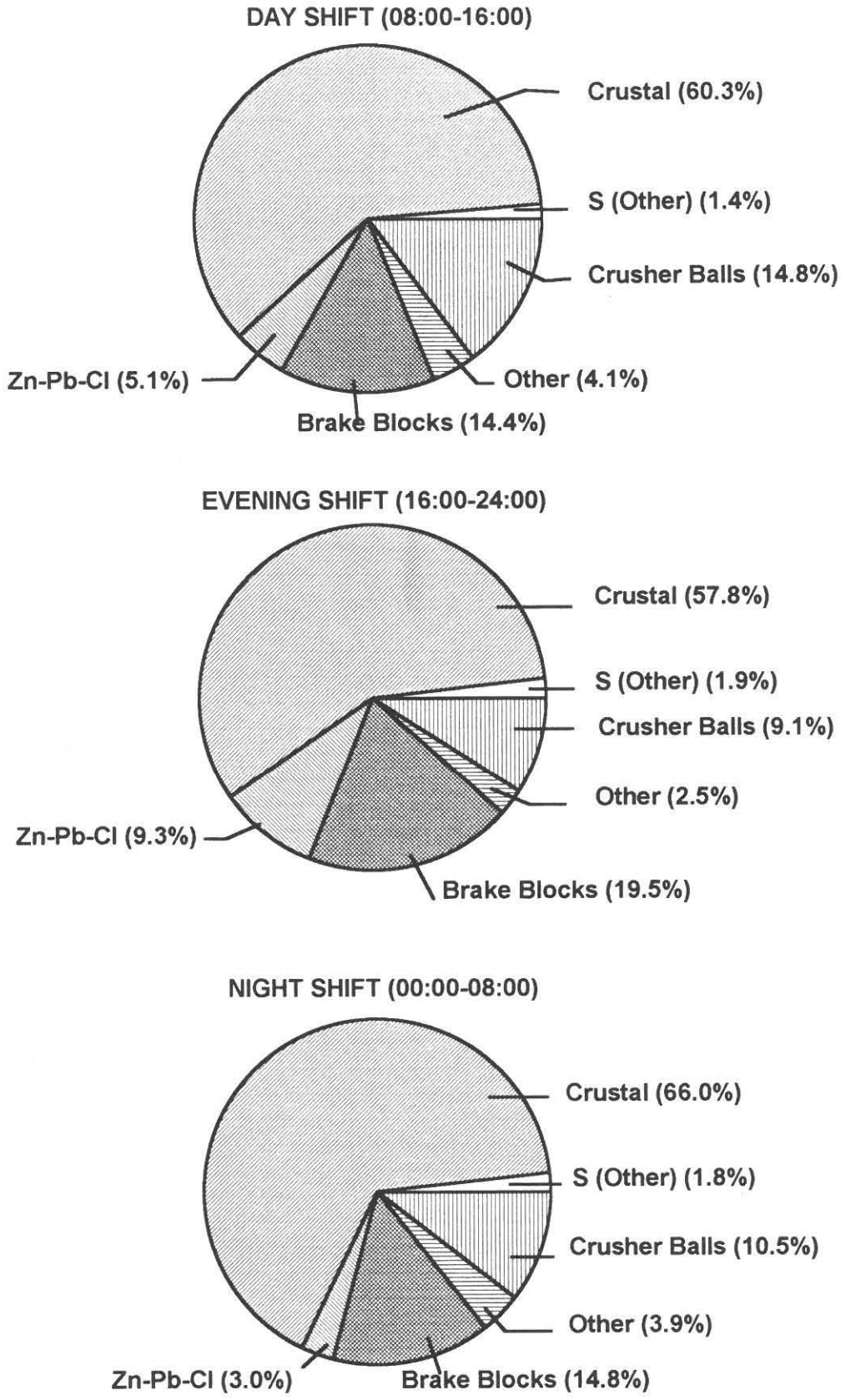


FIGURE 23: Source apportionment for three shifts in foundry on 9 May 1995.

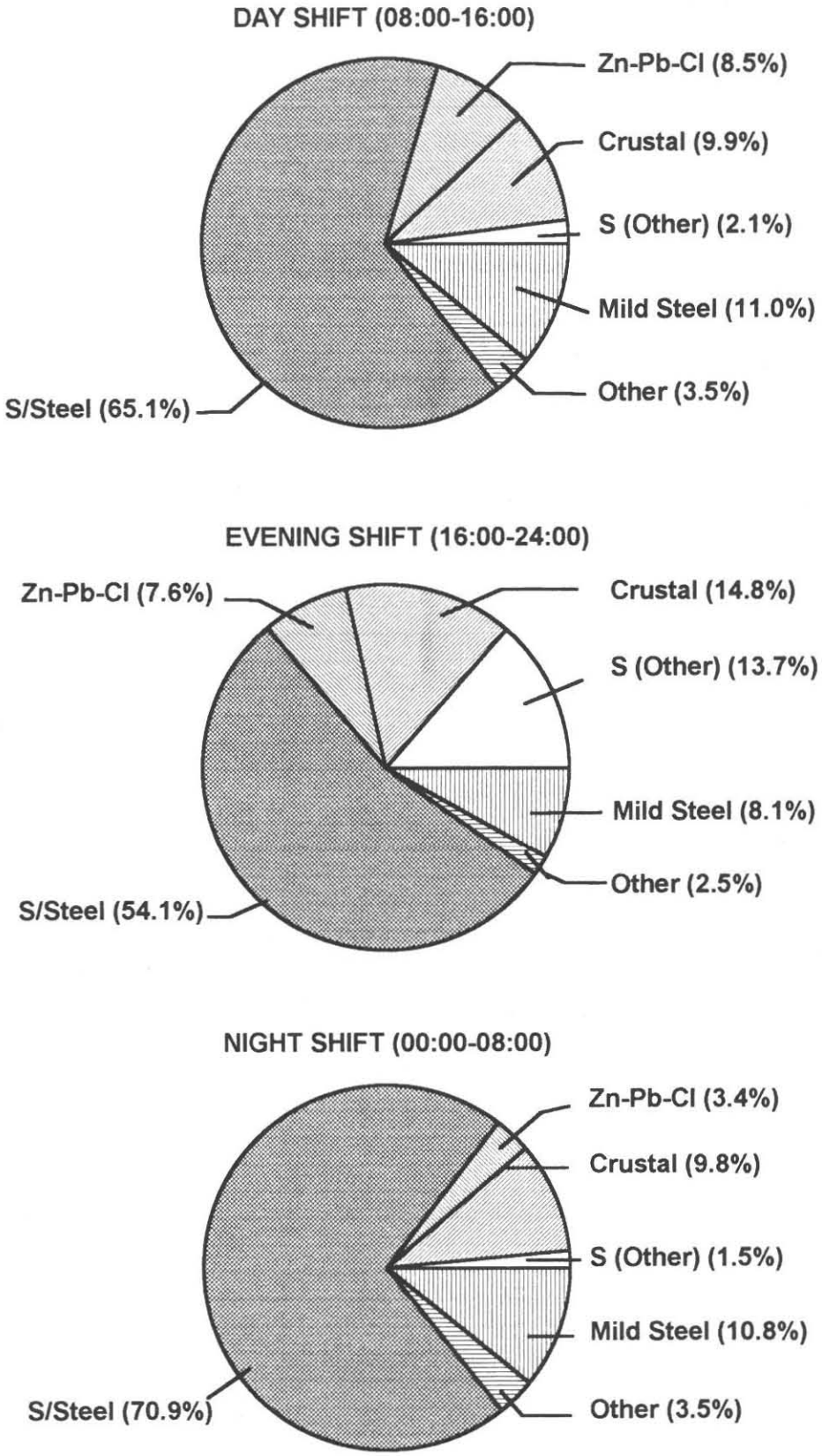


FIGURE 24: Source apportionment for three shifts in welding shop on 3 May 1995.

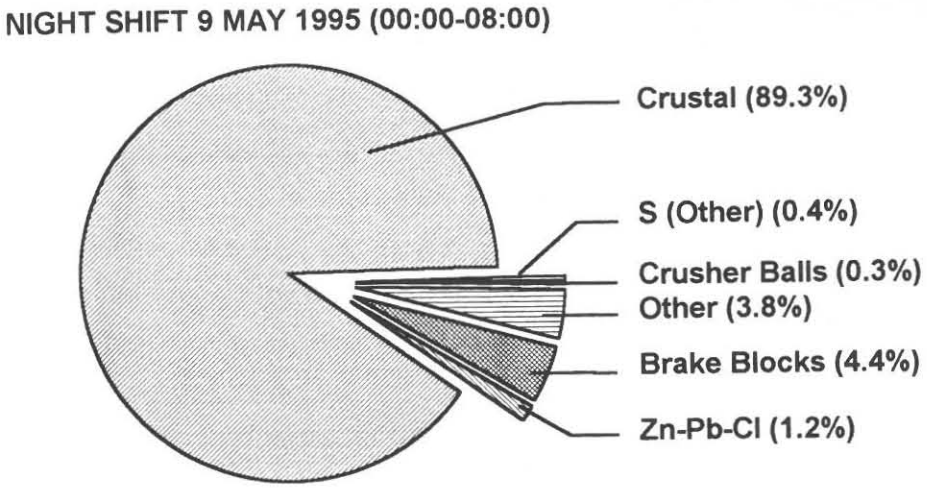


FIGURE 25: Source apportionment of foundry air during no operations.

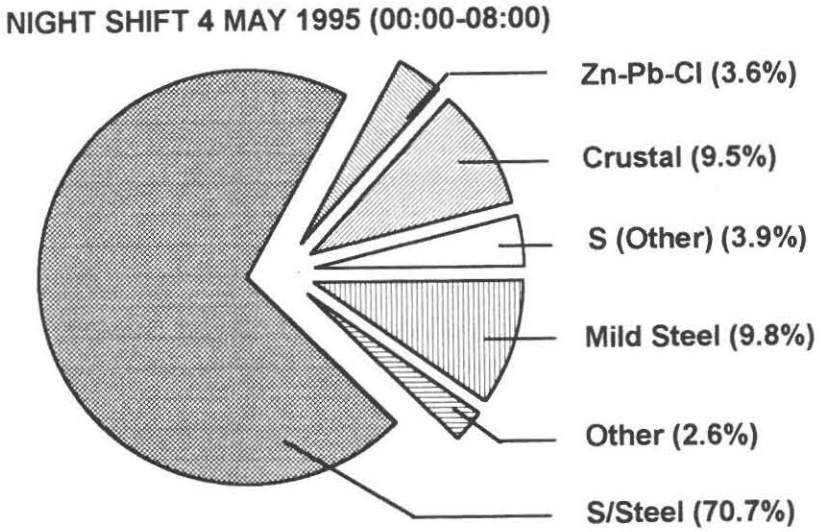


FIGURE 26: Source apportionment of welding shops' air during no operations.

## 5. Conclusion and recommendations

The occupational exposure to dust and fumes of foundry workers and welders at a large engineering plant was characterised to identify possible health hazards. Health hazards were identified for implementing control measures in combatting the harmful exposures and protecting the health of the exposed workers.

The urine of exposed workers was analysed for the urine-metal concentrations of Zn, Cd, Co, Ni, Mn, Cu, Cr, Al, Fe, Pb, Si and V. The air in the respective work areas was analysed for concentrations of Zn, Cd, Co, Ni, Mn, Cu, Cr, Al, Fe, Pb, Si, V, Mg, Ca, Ti, $Cl_2$, Br and S.

Spirometry was conducted for forced vital capacity (FVC), forced expiratory volume in one second ($FEV_1$), peak expiratory flow (PEF), peak inspiratory flow (PIF), average expiratory flow between 25 % and 75 % of FVC ($FEF_{25-75}$), expiratory flow at 25 % of FVC ($V_{max25}$), expiratory flow at 50 % of FVC ($V_{max50}$), expiratory flow at 75 % of FVC ($V_{max75}$) and forced expiration time (100 % FVC) ($FET_{100}$). This was done to provide a physical image of the workers' lungs.

The comparative results for spirometry of foundry workers exposed to dust and fumes from the foundry process showed a statistical significant decrease in $FEV_1$ and FEV in smokers but not in non-smokers. The significant decrease in the smoking population, could be attributed to the effect of smoking. The smoking of tobacco aggravates the susceptibility of lung disorders and workers should be persuaded not to smoke. The dust and fumes created during working operations contributes to the development of restrictive disorders in the foundry population. The total exposed population also showed a significant decrease in $FEV_1$ and FVC which indicates that the pollution in the workplace contributes to the development of restrictive lung disorders in workers working in the foundry.

A comparison of the results of the ventilatory function tests in welders exposed to stainless steel dust and in control workers did not show a statistically significant difference in FVC, $FEV_1$ and $FEV_1$/FVC, in both smokers and non-smokers. This could be attributed to the effect of pollution in the workplace and not to smoking. Although no significant differences were found, the results present a typical clinical image of the first signs of obstructive airway disease. It is concluded that the dust created during the welding of stainless steel is a contributing agent in the development of obstructive respiratory disorders in the welder population.

The urine analysis of foundry workers yielded high concentrations of Cd, Cu, Fe and Si. It is assumed that the Fe and Si concentrations are exposure related because it is present in large quantities in the materials. The measured dust concentrations also show high concentrations for Fe and Si that exceeded the appropriate TLV. The high occurrence of Cd and Cu could not be proved to be a result of occupational exposure because the presence of these metals in airborne dust was not meaningful enough to cause such urine concentrations. It is therefore assumed that the urine-Cu and -Cd concentrations were related to other exposures such as food and water intake.

The analyses of urine from welders yielded high concentrations of Ni, Cr and Fe. Although Ni concentrations in the workroom air were low, the occurrence of all three elements in the urine may be as a result of chronic exposure to welding fumes containing these elements. Fume concentrations measured in the welding shop showed, especially, high concentrations of Cr and Fe. This assumption is in accordance with the composition of the welding materials of which Cr and Fe forms the largest portion.

Urine-Zn analyses showed high concentrations in both foundry workers and welders. Since the occurrence of Zn in the air of both localities was not meaningful, the presence of this metal in urine can also be regarded as the product of food and water intake.

The high occurrence of Fe and Cu in the urine of the workers requires further clinical information. It is also recommended that a study be carried out with a large number of subjects to determine reference values for traditional underprivileged South African workers.

Except for V, no statistical significant differences ($P > 0.05$) were found between the different metal urine concentrations of the workers of the two localities. The statistical insignificance can be explained by the similarity in the exposures from the materials used at the two localities. Vanadium was the only element with known concentrations of the element in the raw materials at the foundry and the exposure resulted in a significant higher concentration in the exposed foundry workers compared to the welders, who are exposed to fumes originating from materials with only trace quantities of V. However, the sample size ($n = 3$) is too small to make a positive conclusion.

Time sequence streaker sampling and PIXE analyses proved to be a reliable method for determining inorganic dust concentrations in foundries and welding

shops. The method provided chemical composition of 1-h increment samples which is not possible with conventional samplers used during occupational hygiene dust monitoring. The use of time sequence sampling proved to be successful for occupational hygiene purposes. Although it is an expensive method of determining inorganic dust concentrations in workplaces, it provides more information than any other method. It also has the advantage of providing composite analyses of a sample which is not possible with normal methods of analysis using cellulose filters. Streaker disks can also be stored for reference purposes and analyses compared to samples which are digested with acid.

Examination of the relationship between elemental variations has allowed identification of several sources and activities contributing to the airborne particles. The similarity of the variations of the soil elements Mg, Ca, Si, Ti and K confirms the assumption that concentrations of elements deriving from the same source behave similarly. Consequently, differences in diurnal variations could be attributed to different or multiple sources.

The aerosol profiles did not show similar diurnal time variation patterns in the foundry on the consecutive days because the processes stopped at irregular intervals when the metal in the pouring pot was finished and the pot returned to the smelter. The pouring activity was interrupted at different times during a shift. At the welding shop, a similar situation was found when welders had to stop the welding activity at irregular intervals, waiting for material or overhead cranes to manoeuvre welded train trucks.

Total dust concentrations in both the foundry and the welding shop exceeded the TLV when concentrations were calculated for individual exposures. The results confirmed the spirometry findings which showed early symptoms of chronic obstructive airway disease. It is recommended that both localities be fitted with local exhaust ventilation systems for removing contaminants from the source of origin. Over and above that, workers must be issued with dust masks and the wearing thereof must be enforced. It is also recommended that spirometry be carried out on a yearly basis on all workers.

The welding fumes contained high concentrations of Cr as a result of the stainless steel welding processes. The results confirmed the findings of the urine analyses and medical conditions such as irritation, pulmonary congestion and possible renal damage can be expected. It is recommended that personal air- and biological monitoring of welders be carried out on a monthly basis.

Iron concentrations measured in both the foundry and welding shops' air can be regarded as an overexposure. The airborne concentrations are a result of the materials containing Fe in large quantities. The workers must be protected against inhalation of Fe-fumes to prevent siderosis. Chest X-rays should be conducted on a yearly basis.

The Mn concentrations in the air of both localities showed high levels, taking into consideration the individual exposures and the new proposed TLV for Mn. It is expected that Mn-poisoning could occur with chronic exposure to current concentrations. Exhaust ventilation which contain the dust is recommended as control measure. Medical examination for neurological changes are essential for early detection of poisoning.

A similarity in exposure pattern was observed between Mn and Fe in the welding shop. This pattern can be ascribed to the materials containing affinitive elements in specific percentages. The similarity might also be due to the fact that both elements have the same oxidation state.

Lead concentrations exceeded the global air quality standard of 1.5 $\mu g/m^3$. Evidence in the streaker time pattern showed that the Pb concentration is a result of motor vehicle exhaust fumes in the surrounding environment, rather than an emission from the processes.

Foundry workers are overexposed to $\alpha$-quartz and tridymite. This finding confirms the early symptoms of a slight restriction in pulmonary airflow obtained during spirometry. It also confirms the excessive saliva production in some workers as a symptom of silicosis. The provision of exhaust ventilation and personal protective equipment should combat the development of silicosis.

Irritation to the eyes and upper respiratory tract may be found at workers of both localities due to overexposure of Cl, Br and $SO_4$. Adequate ventilation in the workplaces should be provided and workers should not be allowed to make fires for heating purposes. Central heating of the workplaces should be provided.

In the foundry air, six sources of atmospheric pollution were identified, namely heavy metal products used in the process, metals containing high Fe amounts, crustal particles from soil and sand from the moulds, sulphur particles from open fires, traffic-related combustion products and other heavy metals containing metals in trace quantities.

The air of the welding shop exhibited six atmospheric pollution sources such as heavy metals from welding stainless steel materials, heavy metal products from mild steel welding, crustal particles from soil, sulphur from open fires, traffic related combustion products and heavy metals in trace quantities.

A new method for rapid evaluation of the inhalation risk to aerosols is proposed. The method is a combination of the ambient aerosol concentrations and metal urine concentrations to which workers are exposed. The method of risk determination, defined in this thesis for the first time, is called aerosol inhalation exposure risk (AIER). The method is designed with the purpose of providing occupational hygienists with a simple and rapid calculation to evaluate the risk of aerosol exposure.

The AIER calculation was applied to the situation using elemental concentrations obtained during this study. The AIER results, were compared to known quantities of toxicants in the materials, air and biological specimens, considering threshold limits specified by world-wide research. The results were representative of the different situations and showed unreliable risks in cases where a lack of scientific evidence on certain BEI's exist. This method of risk determination offers the best alternative for occupational risk determination in developing countries because it estimates the worker's health risk by the integration of clinical, epidemiological and industrial hygiene data. By showing unreliable risks in cases with insufficient clinical and epidemiological available data, the method proved that an occupational health risk cannot be based on industrial hygiene data alone, but on the integration of the three components.

The following substances showed high aerosol inhalation exposure risk (AIER) percentage in the foundry workers' population:
Zn (92.4%), Cu (114.1%), Fe (43.5%) and Si (91.3%).

The substances showing high AIER percentages in the welding shops' population, were as follows:
Zn (59.6%), Cu (71.7%), Cr (47.6%), Fe (27.5%) and Si (39.2%).

The results represent the worst case scenario during winter conditions. It is expected that conditions inside the workrooms will be more healthy during summer time when doors and windows are left open and natural ventilation takes place. The planning and prioritisation for the improvement of indoor air quality in both workplaces can proceed, using the data on the sources of the pollutants. The control measures in the two localities are likely to require different strategies. In

the foundry, reduction of $SiO_2$ concentrations would be the highest priority while in the welding shop, Fe, Cr and $SiO_2$ would be the major concern for providing a healthy work environment.

It is expected that workers of both localities would experience pneumoconiosis and heavy metal related illness after chronic exposure. Engineering control measures such as exhaust ventilation should be applied together with an occupational hygiene and medical surveillance program. Chronic exposure of workers to current conditions would result in occupational related illness costing hundreds of thousands rands in compensation claims.

It can be concluded that workers exposed to conditions as found during this project, will definitely experience health problems after chronic exposure. The $H_0$ is accepted on the fact that workers are chronically exposed to dust concentrations with synergistic effects exceeding the scientific formulated TLV's and resulting in AIE risks greater than 100 %. The spirometry results and metal urine analyses confirm this assumption.

Determination of risk estimates can be useful in the prediction of numbers of specific diseases to obtain an assessment of the magnitude and significance of the occupational health problem related to airborne substances on a national level. The results then obtained, may also serve for international comparisons.

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**APPENDIX I**

**International units and conversion factors for urine analyses**

All over the world, laboratories are gradually converting from conventional units to *Système Internationale d'Unités* (SI Units). The impetus to convert all measurements of substances in body fluids to a molar concentration is based on the fact that substances in the body interact on a molar basis. It would also standardise units internationally.

Reference laboratories in most hospitals as well as most clinicians in certain countries such as the United States of America, have not accepted this change willingly and still use the old conventional reference standards. Due to this practice, some journals still accept both sets of units. These different units were documented in this thesis. The following reference values can be used for converting conventional creatinine standards to SI units (Kaplan, Szabo and Opheim, 1988):

**TABLE 11:** Reference values for creatinine in urine

| Specimen | Reference Range Conventional | SI Conversion Factor* | Reference Range SI Units |
|---|---|---|---|
| **Serum** | 0.6-1.2 mg/dL | 88.4 | 53-106 µmol/L |
| **Urine** | ~1 mg/min | 88.4 | ~88 mmol/L |

* Multiply conventional units by this factor for conversion to SI

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