Exposure of workers to Volatile Organic Compounds during explosives manufacturing

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GLOSSARY OF ABBREVIATIONS

ACGIH	:	American Conference for Governmental Industrial Hygienists.
ALT	:	Alanine Aminotransferase
AP	:	Alkaline Phoshatase
AST	:	Asperatate Aminotransferase
BDL	:	Below Detectable Limits
CBC	:	Complete Blood Count
GGT/GGTP	:	y-Glutamyl-Transpeptadase
MCV	:	Mean Corpuscle Volume
mg/m^3	:	Milligrams per cubic metre
(m/sec)	:	Meters per second, air velocity
NIOSH	:	National Institute for Occupational Safety and Health
OHSACT	:	Occupational Health and Safety Act, Act No 85 of 1993
OEL	:	Occupational Exposure Limites
OESSM	:	Occupational Exposure Sampling Strategy Manual
PPE	:	Personal Protective Equipment
ppm	:	Parts Per Million
SGOT	:	Serum Glutamic Oxalaocetic Transaminase
SGPT	:	Serum Glutamic Pyruvic Transaminase
TLV	:	Threshold Limit Value
TWA	:	Time Weighted Average
VOC's	:	Volatile Organic Compounds

SUMMARY

This study was conducted at a manufacturer of heavy calibre ammunition as well as electro-detonators for the mines and different defense forces. Thirteen most exposed subjects were chosen (according to the Occupational Exposure Sampling Strategy Manual (OESSM)) in four different departments where these subjects were exposed to volatile organic compounds (VOC's) in the normal line of duty.

VOC's are organic (carbon containing compounds) chemicals that have a high vapour pressure and easily form vapour at normal temperature and pressure. VOC's include solvents such as benzene, alcohols, ethanol, toluene, acetone and more of these solvents can be used as paint additives. As can be seen in the literature review VOC's can cause adverse effects if the worker is exposed to levels above the occupational exposure limits and the control measures are not in place. Because of the severity of exposure to VOC's and the relation between occupational hygiene and occupational health (biological monitoring), the study was conducted to determine worker exposure. Estimating occupational exposure is difficult. This is due to a variety of factors, which include the following: worker exposures vary within the same job position, exposures vary during one workday, multiple routes of absorption, personal protective equipment (PPE) may be used inconsistently, and solvents are commonly used as mixtures. For environmental exposure, similar challenges exist. Thus it is important to take biological monitoring into consideration as a way of determining exposure.

During the study three entities were considered to determine exposure. Firstly activated charcoal tubes were used to determine personal exposure. Secondly, the amount of VOC's that was in the worker's body was determined via biological monitoring. Lastly, control measures were considered and the air flow was measured to determine whether the controls were effective (extractor systems and protective equipment).

After the evaluation process the results indicated that although there was exposure to solvents, the workers health was not at risk. It can thus be seen that if the workers are exposed to VOC's and the control measures are in place it will not lead to the adverse health effects that can be caused by exposure to VOC's. During the study there were a few subjects that were exposed to VOC concentrations above the action levels and the effect of this could also be seen in the biological monitoring results. Where defects were noticed, different control measures according to the hierarchy of control, further biological monitoring was suggested.

OPSOMMING:

Blootstelling van werkers aan vlugtige organiese oplosmiddels tydens die vervaardiging van plofstof

'n Studie van met die blootstelling van werkers aan vlugtige organiese oplomiddels is uitgevoer by 'n ammunisie vervaardiger vir weermag instansies en die mynbou industrie. Dertien mees blootgestelde proefpersone uit vier verskillende departemente is gekies volgens OESSM om aan die studie deel te neem.

Vlugtige organiese oplosmiddles is chemikalieë wat 'n hoë damp druk het en maklik dampe vorm by normale temperatuur en druk. Hierdie substanse sluit in oplosmiddels soos benseen, alkohole, etanol, tolueen, asetoon en baie ander. Hierdie oplosmiddels word dikwels gebruik in verwe. Soos in die literatuur studie gesien, kan oplosmiddels verskillende gesondheid effekte veroorsaak by werkers wat aan vlakke bo die blootstellings drempels blootgestel word.

Hierdie studie is uitgevoer omdat blootstelling aan vlugtige organiese oplosmiddels nadelig vir die gesondheid mag wees asook om die verhouding tussen beroepshigiëne en beroepsgesondheid te bestudeer. Tydens die studie was drie parameters in ag geneem om blootstelling te bepaal. Eerstens is geaktiveerde koolstof buise gebruik om persoonlike blootstelling te bepaal. Tweedens is die hoeveelheid oplosmiddels wat die persone se liggaam binnedring bepaal deur biologiese monitering uit te voer soos lewerfunksies, volbloedtellings en ander relevante bloedtoetse. Laastens is die ingenieurs beheermaatreëls (uitsuig sisteme) getoets om te bepaal hoe effektief dit is.

Na die evaluering toon die resultate dat daar wel afwykings is en dat daar 'n korrelasie bestaan tussen beroepshigiëne en beroepsgesondheid monitering. Waar die beheermaatreëls nie volkome effektief is nie is daar 'n toename in blootstelling waargeneem.

Waar afwykings tydens die studie waargeneem is, is verskillende beheermaatreëls volgens die hiëragie van beheer aanbeveel asook verdere biologiese monitering waar nodig.

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HYPOTHESES

Exposure of workers to volatile organic compounds in a explosive plant are lower than the occupational exposure limits and this will be confirmed by the biological monitoring results.

PURPOSE OF THE SURVEY

The purpose of this survey was to identify whether any occupational health and hygiene hazards involving Volatile Organic Compounds exists, to evaluate the degree of the hazard and the existing controls and their effectiveness, to make recommendations if necessary and to determine whether further evaluation and investigation are necessary.

Chapter 1: Introduction

Volatile organic compounds (VOC's) are organic (carbon containing compounds) chemicals that have a high vapour pressure and easily form vapour at normal temperature and pressure. They are also known as solvents. VOC's can be classified by their physical, chemical and toxicological properties. This help group VOC's into families with shared or distinguishing features. VOC's are commonly used for cleaning, degreasing, thinning and extraction (Williams & Brautbar, 2002).

According to Baker (1988), solvents may be classified as aqueous or organic. Since most of the substances that solvents are used to dissolve in industry are organic, most industrial solvents are organic chemicals. Hundreds of individual chemicals are used to make over 30 000 industrial solvents/ VOC's (La Dou, 1997).

VOC's or solvents include trichloroethylene, benzene, toluene, styrene, acetone, ethyl benzene, mixed xylenes, methyl ethyl ketone, alcohols, phenols, methacrylates, acrolein, polyclic aromatic hydrocarbons and pesticides. VOC's are by-products of fossil fuel combustion and come from many sources including industrial and combustion processes and petrol stations. These types of organic solvents can be found in certain paint additives, aerosol sprays, can propellants, fuels, petroleum distillates, dry cleaning products and many other industrial and consumer products ranging from office supplies to building materials, and they all contribute to VOC emissions. According to La Dou (1997), VOC's such as benzene trichloroethylene, toluene, styrene, acetone and mixed xylenes can cause cancer, kidney, liver and brain damage as well as damage to the nervous, reproductive and immune systems. The symptoms of VOC intoxication are as follows: irritation of the eyes, nose and throat, nausea, dizziness, skin rashes, irritation of the lungs, wheezing, coughing, memory problems, anxiety, fatigue, severe allergic reactions, and may cause cancer and difficulty in breathing.

Due to all of the above mentioned effects it is important that both biological and environmental monitoring are done to determine exposure to these chemicals. It is also important that control measures are in place to reduce possible exposure to solvents. If control measures are in place, where occupational exposure does occur it is extremely important that they are evaluated regularly as indicated in the Occupational Health and Safety Act and Regulations (85 /1993) to determine their effectiveness.

Biological monitoring of certain solvents is compulsory as specified in Regulation 9 Table 3 of the Occupational Health and Safety Act and Regulations (85 /1993). Biological monitoring can provide accurate exposure values for some solvents. This is particularly true for those substances whose pulmonary absorption is affected to a large degree by physical work and those with significant dermal exposure and absorption.

Measurement of VOC's in the air plays an important role in determining the air borne concentration of the substances in the breathing zone of the occupationally exposed person's breathing zone. The existing controls (in this case extraction hoods and fume cupboards) must be evaluated to determine whether control measures are effective. Extract hoods, slots, enclosures and fume cupboards are intended to capture air pollutants to prevent them from being released into the general work atmosphere. Therefore some means of checking the airflow patterns and air velocity in and around the inlets are useful so that the full extent of the zone of influence of the device can be ascertained.

In order to study the exposure to VOC's, this study was conducted at a manufacturer of heavy calibre artillery as well as electro detonators for mines and different armics. Thirteen workers were chosen in four different departments namely:

1. FO1: Where electro detonators are manufactured.
2. Paint shop: Where the empty shells are cleaned, phosphated, screen printed and spray-painted.
3. Blue building: Where plants and equipment are manufactured and painted.
4. A03: Melting and filling of empty shells with explosives as well as the stenciling of the shells for packaging and numbering.

Chapter 2: Literature Review

2.1 Chemical properties and the use of Volatile Organic Compounds (VOC's)

The term "organic solvents" refers to a group of volatile compounds or mixtures that are relatively stable chemically and that exist in a liquid state at temperatures of approximately 0° to 250°C (Parrish, 1983). Common organic solvents are classified as aliphatic hydrocarbons, cyclic hydrocarbons, aromatic hydrocarbons, halogenated hydrocarbons, ketones, amines, esters, alcohols, aldehydes and ethers. Many common solvents often exist as mixtures or blends of chemical compounds (e.g., Stoddard solvent and thinners) (WHO, 1985; Parrish, 1983).

Organic solvents share a common structure (at least 1 carbon and 1 hydrogen molecule), low molecular weight, lipophilicity and volatility. They may be grouped further into aliphatic compounds that exist in chain form, such as n-hexane, and aromatic compounds that exist in a 6-carbon ring form, such as benzene or xylene. Aliphatics and aromatics may contain a substituted halogen element and may be referred to as halogenated hydrocarbons, such as perchlorethylene (PCE), trichloroethylene (TCE), and carbon tetrachloride. Alcohols, ketones, glycols, esters, ethers, aldehydes and pyridines exist as substitutions for a hydrogen group. Organic solvents are used routinely in commercial industries. They are used in paints, adhesives, glues, coatings and degreasing/cleaning agents, and in the production of dyes, polymers, plastics, textiles, printing inks, agricultural products and pharmaceuticals. Organic solvents are useful because they can dissolve oils, fats, resins, rubber and plastics (Rutchik, 2006).

According to Rutchik (2006), organic solvents arose in the latter half of the 19th century from the coal tar industry. Their application grew to be wide and diverse in both developed and developing countries. The introduction of chlorinated solvents in the 1920s led to reports of toxicity. Although the number of solvents is in the thousands, only a few have been tested for neurotoxicity.

Table 2.1: Organic Solvents and their common industrial uses (Rutchik 2006)

Compound	Industrial Uses
Acetone	Cleaning solvent
Benzene	Fuel, detergents, paint removers, manufacture of other solvents
n-hexane	Glues and vegetable extraction, components of naphtha, lacquers, metal cleaning compounds
Methane	Industrial settings
Methyl-*n*-butyl ketone	Many industrial uses
Methylene chloride	Solvent, refrigerant, propellant

Compound	Industrial Uses
(dichloromethane)	
Toluene	Paint, fuel oil, cleaning agents, lacquers, paints and paint thinners
Trichloroethylene	Cleaning agent, paint component, decaffeination, rubber solvents, varnish
Xylene	Fixative for pathological specimens, paint, lacquers, varnishes, inks, dyes, adhesives, cements

Results from studies of indoor irritation symptoms are consistent with the hypothesis that ozone (O_3) in combination with unsaturated hydrocarbons contributes to nasal resistance and eye irritation (Höppe *et al.*, 1995; Stephens *et al.*, 1961). It is well known that reactions between oxidants (e.g. ozone) and unsaturated VOC's occur to form aldehydes and carboxylic acids, in addition to hydroperoxides and radicals (Weschler & Shields, 1997). Experimental evidence has shown that strong airway irritants can be formed in ozone/turpentine mixtures (Wolkoff *et al.*, 1999).

Organic solvents are used for extracting, dissolving, or suspending materials such as fats, waxes and resins that are not soluble in water. The removal of the solvent from a solution permits the recovery of the solute intact with its original properties.

2.2 Potential for occupational exposure to organic solvents

Organic solvents are commonly used in industries because of their dissolving, degreasing and other chemical uses. Most occupational exposures are to solvent mixtures (Stacey & Winder, 2004). Occupations in which these agents are used include printers, paint manufacturing or painting, microelectronics workers, degreasers, dry cleaners, carpet layers, coating workers, gluers, dye workers, carpenters, anesthesia personnel, petrol filling workers, laboratory workers, inkers, textile workers, and those who work with polymers, pharmaceuticals, synthetic fabrics, agriculture products, refining, or in airplane refitting. Because solvents are found in a wide range of products and processes, many workers are at the risk of exposure. According to Parker (1989), 43% of all organic solvents in Europe in 1980 were used in paints and other surface coatings, 10% for metal cleaning, 8.1% for dry cleaning, and 20% for other uses. Parker (1989) also states that aliphatic and aromatic hydrocarbons accounted for approximately half of all the solvents used in Western Europe.

An evaluation of organic solvents used in Denmark found that 93 different solvents were used in industry, most commonly ethanol, gasoline, toluene, isopropanol and acetone (Seedorff & Olsen, 1990). Seedorff and Olsen (1990) also found that the highest exposure in Denmark occurs in the printing and chemical

industries. According to Kalliokoski (1986), some authors distinguish between two types of work activities and processes (painting and degreasing), occupational exposure to solvents and application work involves the creation of an open surface from which solvents evaporate. It is usually associated with intermitted high-level exposure. Solvent exposure in these settings can be highly variable and is related to both the work setting and ventilation (Baker & Smith, 1984). Much variability of exposure can exist between individuals, even those performing the same tasks (Droz *et al.*, 1989). There is the potential of multiple routes of entry (Seedorf *et al.*, 1990) and personal protective equipment may be used.

There are several methods for determining occupational exposure to solvents, including environmental monitoring, biological monitoring and semi-quantitative retrospective exposure estimation.

Estimating occupational exposure is difficult and, therefore, it is important to take biological monitoring into consideration as a way of determining exposure.

2.3 The intake of Volatile Organic Compounds (VOC) in the body and toxicological effects

The harmful effects of organic solvents follow inhalation of vapour, eye and skin contact with liquid or vapour, or ingestion, which are described below. Inhalation is usually the most significant route of entry by which organic solvents enter the human body at work. Some organic solvents may be absorbed through the skin without any noticeable effect on the skin. Others may cause serious damage to the skin itself. Ingestion is of relatively minor significance in occupational exposure. Toxic atmospheric contaminants may have local effects if they harm only the part of the body they come in contact with, or systemic effects causing changes to the function of other organs (Stacey & Winder, 2004).

2.4 Health effects

Many occupational diseases caused by chemicals result from breathing air that contains harmful substances. Exposure to hazardous material may be acute or chronic. Acute exposure generally refers to single dose, high concentration exposure over short periods, while chronic exposure involves repeated or continuous exposure over long periods. These exposures may have acute immediate effects or chronic, long-term effects. The extent of any health effects are dependent on the duration and frequency of exposure and the concentration of the substance. Some examples of health effects are listed below.

- **Respiratory tract:** The vapour of many organic solvents is irritating to the lining of the respiratory tract, affecting the nose, throat and lungs. Asthma-like reactions have been reported with some organic solvents (National Occupational Health and Safety Commission, 1990).
- **Skin:** Skin contact often causes drying, cracking, reddening and blistering of the affected area. These signs of inflammation of the skin are called dermatitis and enhance solvent absorption and encourage secondary infection. Dermatitis may be irritant or allergic in nature. Solvent-induced

dermatitis may persist for a long time after exposure. Absorption of solvents through the skin may produce systemic health effects (National Occupational Health and Safety Commission, 1990).

- **Eyes:** Direct contact with organic solvent vapour or liquid may cause eye irritation. This is usually reversible and permanent eye damage is rare (National Occupational Health and Safety Commission, 1990).
- **Liver:** Many organic solvents are potentially toxic to the liver, either alone or in combination with other solvents. For example, liver damage is associated with exposure to carbon tetrachloride, other chlorinated hydrocarbons and ethanol. Consumption of alcoholic drinks may enhance the effects of many solvents (National Occupational Health and Safety Commission, 1990).
- **Kidney:** Both short and long term exposure to certain organic solvents has been found to be harmful to the kidneys. Carbon tetrachloride, trichloroethane and petroleum distillates, for example, gasoline, jet fuel and turpentine, are among the most toxic (National Occupational Health and Safety Commission, 1990).
- **Cardiovascular system:** Chlorinated hydrocarbon solvents, such as methylene chloride and trichloroethane, may cause harmful effects to the heart. Abnormal heart rhythms have been reported arising from trichloroethylene exposure. Chronic exposure to carbon disulphide is considered to be a contributory factor in coronary heart disease (National Occupational Health and Safety Commission, 1990).
- **Nervous system:** Exposure to organic solvents can result in a variety of serious effects in both the central nervous system (CNS - brain and spinal cord) and the peripheral nervous system (PNS - nerves supplying the rest of the body). The acute effects of organic solvent exposure range from an alcohol-like intoxication to narcosis (stupor or insensibility), which may lead to unconsciousness and eventually death from respiratory failure. Intermediate symptoms include drowsiness, headache, dizziness, dyspepsia (gastric discomfort) and nausea. Long term gross exposure to both n-hexane and methyl n-butyl ketone is associated with degeneration of nerve cells in the PNS, resulting in symptoms such as restless legs, muscle cramps, pains and weakness in limbs and loss of sensation in the limbs. Chronic CNS effects resulting from long term repeated exposures to organic solvents include fatigue, mood disturbance and difficulty in concentrating, memory loss, personality changes and loss of motivation. This damage may become virtually permanent (National Occupational Health and Safety Commission, 1990).

2.5 Toxicity of VOC's

Workers in industries that utilize VOC's may have occupational exposure, while other individuals may have environmental exposure if they live in proximity to industrial installations and/or come into contact with contaminated water, soil, air or food. Exposure is often to mixtures of solvents. Some of these may occur deliberately when an individual recreationally inhales paints, glues and other products (Stacy & Winder, 2004).

Short-term, high-level exposure, frequently stated in case reports have been noted to result in acute reversible and irreversible health effects that involve the central and peripheral nervous systems. Intermediate and long-term low-level exposure has been reported in population studies to lead to reversible and non-reversible, sub-clinical and clinical abnormalities in the central and peripheral nervous systems. In some cases, these exposures have been estimated to be below levels designated in regulations as acceptable for workers. Neurophysiological, neuropsychological and neuro-imaging diagnostic tools have been utilized to evaluate individuals and groups exposed to organic solvents (Rutchik, 2006).

The central nervous system is primarily affected and symptoms can include diminished cognition, memory loss, reaction time, and hand-eye and foot-eye coordination, balance and gait disturbances. Exposure can also lead to mood disorders with depression, irritability and fatigue being common symptoms. Peripheral neurotoxicity usually results in paresthesias, tremors and diminished fine and gross motor movements. VOC's have been implicated in kidney damage. They have been associated with immunological problems, including increased cancer rates and immunotoxicity. The typical presentation of low-dose formaldehyde exposure includes upper respiratory irritations (rhinitis, sinusitis and pharyngitis), lower respiratory symptoms of wheezing, and persistent flu-like symptoms (Rutchik, 2006).

Phenol causes local burns and may be absorbed both through the lungs and dermally. Although phenol causes severe local burns, systemic systems may also occur. These include headache, vertigo, salivation, nausea, vomiting and diarrhea. In severe intoxication, urinary albumin excretion may also be increased. Red cells and casts are found in the urine. The potentially disastrous consequences of transdermal absorption should not be under estimated. Patients may present with hypothermia, which is followed by convulsions. The urine may be dark and oliguria may develop. Phenol is metabolized to hydroquinone, which when excreted in the urine may be oxidized to coloured substances, causing the urine to change to green or brown (carboluria). Prolonged exposure has been reported to result in proteinuria (La Dou, 1997).

The symptoms of acute VOC intoxication are as follows: irritation of the eyes, nose and throat, nausea, dizziness, skin rashes, irritation of the lungs, wheezing, coughing, anxiety, fatigue, severe allergic reactions and difficulty in breathing (La Dou, 1997).

2.6 Kidney toxicity

Acute Effects

Acute tubule necrosis (ATN) is a potentially life-threatening renal disorder charaeterized by azotemia and oliguria. It is one cause of renal failure. Short-term, high-level exposure to selected solvents is universally accepted as a cause of ATN (Schier & Conger, 1980). Solvents that have been described as causing ATN include the halogenated hydrocarbons, petroleum distillates, ethylene glycol, ethylene glycol ethers, diethylene glycol, dioxane and toluene (Phillips *et al.*, 1988), ATN has been reported to follow both intentional inhalation exposure (volatile substance abuse) and occupational inhalation exposure. The mechanism of solvent-including tubule damage is poorly understood (Lauwerys *et al.*, 1985). Solvent induced ATN is not associated with glomerular disease. When it occurs, ATN shortly follows solvent exposure, so the association with exposure is usually easy to establish. Some authors have concluded that the risk of ATN associated with solvent exposure is low because few reports of solvent induced acute renal failure are available despite the widespread use of solvents (Barrientos *et al.*, 1977). Although in the past ATN was universally fatal, recovery is common now that renal dialysis is readily available. The initial tubes regenerate in approximately three weeks. While complete recovery is possible, renal insufficiency may persist . The most well established solvent risk factors for kidney damage are glycol ether and some chlorinated solvents (Winder & Stacy, 2004).

Chronic Effects

Glomerulonehritis

Three types of chronic effects might occur namely glomerulonephritis, tubule and glomerular dysfunction and finally renal effects.

Glomerulonephritis according to Glassock & Benner (1987) is a disorder characterized by, either individually or in combination, hematuria, proteinura, reduced glomerular filtration rate and hypertension. It is caused by alterations in the structure and functional integrity of the glomerular capillary circulation. Glomerlonephritis is the most commonly cited renal disease following long-term exposure to solvents (Phillips *et al.*, 1988). Several comprehensive literature reviews according to Churchill *et al.*, (1983), Nelson *et al.*, (1990) and (Phillips *et al.*, 1988) were done relating solvent exposure to glomerulonephritis. All include a discussion of the many case reports of individual parties or series of patients with glomerular disease who have a history of exposure to solvents. Agreement exists that the result of these case series, while indicating the need for additional research, are not conclusive.

According to Harrington *et al.* (1989), one of the most carefully preformed case controlled studies of the relationship between glomerular disease and exposure to organic solvents was done via a biopsy, using appropriate control groups, and ensuring that interviewers were blinded to disease status of the subject.

Of these studies a significant association between solvent exposure to glomerular disease was observed by Ravinskov *et al.*, (1979) and Bell *et al.*, (1985). Statistical power was sufficient to detect substantial increase in risk only in the two negative studies.

Tubule and Glomerular Dysfunction

According to Gerr and Letz (1992), several cross-sectional studies of urinary excretion of proteins and cells in subjects occupationally exposed to organic solvents have been performed. These studies were done to detect renal tubule and glomerular dysfunction at an early or sub-clinical stage. Outcomes of interest have included not only conventional clinical measures of renal function such as proteinuria, albuminuria, and the presence of cells in urine but also novel measures of renal function, such as excretion of low-molecular weight enzymes and proteins, including N-Acetylglucosaminidase, retinol binding protein and β2-microglobin. The results of such studies have been mixed, showing both mild tubule dysfunction (Franchini *et al.*,1983) and glomerular effects (Hotz *et al.*, (1989) and Asloergren (1986). Studies in which no effect was found on a variety of measures of renal function have also been reported (Krusell *et al.*, 1985; Vyskocil *et* al., 1989).

Some inconsistency exists regarding the effect of solvents on measures of renal function among working populations exposed to solvents. Evidence according to Gerr and Lentz (1992), suggests that mild tubule effects of unknown clinical significance are detectable in solvent exposed workers.

Renal Effects

Gerr and Lentz (1992) also indicated that solvents are widely recognized as one of the causes of ATN and hence acute renal failure. In addition the results of several case-control studies of glomerulonephritis indicate that solvent exposure is associated with that disorder as well. The results of cross-sectional studies indicate that mild tubule and glomerular effects can be observed among solvent-exposed groups.

Biological effects of solvents on the kidneys

i. Diffraction diagnosis

Renal tubular dysfunction, including acidosis, can be a primary disease that first manifests itself in early adulthood or may occur secondary to a variety of metabolic and hyperglobulinemic states and exposure to toxic agents, including antibiotics and heavy metals. (La Dou, 1997).

ii. Laboratory findings

According to La Dou (1997), renal tubular dysfunction from solvents may be manifested by polyria, glycosuria, proteinuria, acidosis and electrolyte disorders. Hypokalemia, hypophosphatemia, hypercholremia and hypocarbonatemia have been seen as manifestations of renal tubular acidosis in

toluene abusers. Acute renal failure from halogenated solvents is similar to that of other causes. Routine monitoring of renal function is not generally recommended for workers exposed to solvents. However, the measurement of unary excretion of low molecular weight enzymes such as N-acetyl-B-glucosaminidase, B-glucuronidase and muramidase appears to offer promise as a monitor for evidence of early tubular dysfunction (La Dou, 1997).

2.7 Liver toxicity

Halogenated Hydrocarbons

Carbon tetrachloride, terachlorethane and chloroform are well-known hepatotoxins, acutely causing hepatic necrosis and steatosis (Zimmerman & Ishalk., 1987). In addition to this, hepatic cirrhosis has been observed following long-term exposure to carbon tetrachloride (Xiao & Levin, 2000). Use of these substances has diminished over the past decades, in part because of their recognized hepatotoxicity and the availability of less toxic substitutes. (Gerr & Letz, 1992). The evidence on human exposure to other halogenated hydrocarbon solvents such as trichloroethylene and 1,1,1-trichoroethane suggests that they are substantially less hepatotoxic than carbon tetrachloride and chloroform (Kramer *et al.,* 1978). A relative paucity of data from carefully performed epidemiologic studies of exposed workers necessitates guarded conclusion. In addition case reports of diffuse liver disease including hepatic necrosis and steatosis in workers exposed to 1,1,1-trichoroethane (Hodgson *et al.,* 1989) and hepatic necrosis with fibrosis in solvent abuses heavily exposed to trichloroethylene (Baerg & Kimberg, 1970) suggests that these chlorinated hydrocarbon solvents have potential.

Nonhaloginated hydrocarbons

According to Gerr and Lentz (1992), few or no hepatotoxic effects have been observed in well-performed cross-sectional epidemiological studies of subjects exposed to nonhalogenated solvents, including both aliphatics (kerosene, n-hexane and others) and aromatics (xylene, toluene, styrene and others). These studies have utilized conventional noninvasive laboratory methods, such as measurement of serum hepatocellular enzymes, including aspartane aminotransferase (AST) and alanine aminotransferase (ALT) to identify the potential hepatotoxic effects. Lundberg and Hakansson (1989) studied serum hepatic enzyme activity in 47 paint industry workers exposed to a mixture of solvents, of which xylene and toluene were most common. No significant differences were found between the solvent-exposed workers and the unexposed age-matched referents. Pedlesen and Rasmussen (1982) compared 122 subjects with suspected "solvent poisoning' to 64 solvent exposed subjects without poisoning as well as the unexposed referents. The exposed subjects had been exposed to many solvents, the most common being turpentine, toluene and xylene. Some use of chlorinated solvents was reported. No differences were found in serum ALT, the only measure of liver function performed in this group. Ørback *et al.*, (1985) performed a

comprehensive study of 50 male workers exposed to solvent in the paint industry and a comparison group matched for age and education level. The paint industry subjects had been exposed to non-halogenated aliphatic and aromatic hydrocarbon solvents. The mean serum AST level was significantly decreased and mean serum lactic acid dehydrogenase (LDH) was significantly increased in the exposed group. Mean serum ALT, alkaline phosphatase (AP) and y-glutamyl-transpeptadase (GGTP) were not significantly different. A similar study performed demonstrated no differences in serum AST or ALT between solvent exposed painters and age–matched comparison subjects. Elofsson *et al.*, (1980) compared AP, ALT, AST and serum bilirubin levels of 80 spraypainters exposed to a mixture of solvents and of an unexposed comparison group. The most common toxicant was toluene, but exposure to xylene, trichloroethylene, and white spirits also occurred frequently. Serum AP was found to be significantly elevated in the solvent exposed subjects. No significant differences were found for serum AST, ALT or bilirubin. Outbreaks of liver disease in the occupational setting, such as a well–documented episode of liver disease due to dimethylformamide at a coated-fabric factory and the case report of two workers with fulminant hepatic failure following exposure to 2-nitropropane, indicate that selected non-halogenated hydrocarbon substances are capable of inducing acute and chronic liver disease in exposed populations. These outbreaks have underscored the need to identify solvents that can induce hepatic disorders before they are made available for widespread use.

Diagnostic tests for liver dysfunction

Biochemical tests for liver dysfunction

Serum enzyme activity: The tests most commonly used to detect liver disease are serum glutamic-oxalaocetic transaminase (SGOT), also known as asperatate aminotransferase (AST) and serum glutamic–pyruvic transaminase (SGPT), also known as alanine aminotransferase (ALT). Transaminase release is due to release of enzyme protein from liver cells as a result of cell injury. Elevations of serum aminotransferase levels may occur with minor cell injury, making such determinations useful in the early detection and monitoring of liver disease of drug or chemical origin. However, transaminase levels may be elevated in viral, alcoholic or ischemic hepatitis as well as extra hepatic obstruction, limiting the specificity of these tests. In addition, significant elevations of transaminase levels have been noted in a few normal healthy subjects due to diets high in sucrose, and false positives have been reported in patients receiving erythomucin and aminosalisicylic acid and during diabetic ketoacidosis (La Dou 1997). Conversely, significant liver damage may be present in individuals with normal levels of transaminase. A serum AST/ALT ratio of less than 1, especially when the transaminase levels are below 300IU/L, may be suggestive of the diagnosis of occupational liver disease (La Dou, 1997).

Chronic alcohol use can include a wide variety of metabolic changes in a variety of organ systems. Alcohol can also cause direct injury to certain organ systems, such as the liver and bone marrow, which

can be reflected in the peripheral blood. A number of studies according to La Dou (1997) have shown that the most useful blood studies are the CBC, especially the mean corpuscle volume (MCV) and certain liver enzyme studies, particularly the gamma-gutyl transpeptidase. A number of studies have shown an elevated MCV, particularly when combined with an elevated GGT and can identify over 90 % of alcohol abusing-patients. Chronic alcohol use exerts a number of effects on red blood cells, which can cause the appearance of abnormally large numbers of macrocytic cells to appear in the peripheral blood (La Dou, 1997). The first of these effects of alcohol solvents action on the red blood cells are to increase in size. Likewise, hypersplenism and hepatomegaly induced by alcohol abuse tend to cause accelerated red cell destruction, which is compensated for by the release of immature red cells from the bone marrow. These immature red cells tend to be lager than mature cells. Finally nutritional deficiencies occur, particularly a deficiency in vitamin B_{12} and pyridoxine (La Dou, 1997). The GGT is also commonly elevated with any significant alcohol usage. This test alone, while sensitive is not very specific. It can be elevated in starvation, obesity and disease of the bile ducts or gall bladder as well as alcohol usage. The GGT, in itself, cannot be used as a diagnostic maker for alcohol dependence, nor can the MCV or any other single laboratory test. It is important that the physician remembers that careful history and physical examination is important in making a diagnosis (Rees, 1993).

2.8 Dermal toxicity

Organic solvents have the ability to dissolve grease and fat from the skin when cutaneously exposed. It can also deplete the intact skin of lipids that are physiologically necessary for its integrity. The above mentioned can result in contact dermatitis characterized by dryness, scaling and fissuring of the skin, especially the hands (Andersen & Burrows, 1995). This occurs either because the work requires handling materials wet with solvents as in the case of manual cleaning and degreasing or where solvents are used to wash the hands to remove glues, plastics or other materials from the skin. These effects are reversible upon cessation of skin contact and are prevented by avoiding direct skin contact with solvents. According to La Dou (1997), up to 20% of cases of occupational dermatitis are caused by solvents and the typical appearance ranges from an acute irritant dermatitis like erytheme and edema to a chronic dry, cracked eczema.

2.9 Reproductive toxicity

Numerous articles in the press have raised alarming questions about a world wide decline in sperm counts in humans and animals. Various chemicals and other physical agents have been blamed for the damage to the male reproductive system. According to Chia *et al.* (1994), there are more than 104 000 chemical and physical agents listed as environmentally hazardous by the National Institute of Occupational Safety (NIOSH) and the Health Registry of Toxic Effects of Chemical substances. The effects of these on reproduction have not been investigated in more than 95% of them. In spite of this, NIOSH has ranked infertility among the 10 leading national occupational illnesses and injuries in that year. The fact that little

is known about most of these toxic agents highlights the importance of understanding and perhaps preventing these potential reproductive injuries in the workplace.

Organic solvents are widely used in the chemical industries and evidence concerning the reproductive effects of these agents is poor and generally no definitive conclusions can be drawn. According to Karakaya e*t al.* (1997), solvents have been found to lower testosterone and luteinizing hormone (LH). They have also been associated with infertility and a decreased sperm count according to Sallmen e*t al.* (1998). According Oudiz and Zenick (1986) and Zenick *et al.* (1984) special attention should be given to the glycol ethers a class of organic solvents widely used in paints, printing inks and paint thinners. Several investigators have studied the effects of these solvents in animals. These studies according to Zenich *et al.* (1984), indicated that the most sensitive target cell appears to be the primary spermotocyte during its division in the early and late pachytene stages, but spermatogenia may also be affected in higher doses. Welch *et al.* (1986) studied 73 shipyard painters exposed to glycol ether and compared these workers to cohort of 40 non exposed men. They found a higher percentage of oligospermia and azoospermia in the exposed group, but the strength of the association was equivocal. No hormonal differences were observed between the two groups.

Materials of ethylene glycol ether variety, particularly 2-methoxyethanol represent an important group of organic solvents found in paints, dyes, thinners as well as many others. Studies in laboratory animals have demonstrated damage by 2 methoxyethanol (me) and 2 ethoxyethanol (ee) to male fertility and physical and aesthetic structure of offspring. Cherry *et al.* (2001) found a significant association between intensity of exposure to solvents and clinical findings of less than 12 x 10^6 motile sperm (odds ratios ors) were 2.07 (95% confidence interval (95% CI) 1.24 to 3.44) for moderate exposure and 3.83 (95% CI) 1.24 to 3.44) for moderate exposure to solvents (Krurinczuk & Clarke, 2001).

Only a few epidemiologic studies are available in which reproductive physiology or outcome is assessed in solvent-exposed workers. This limited evidence that is available does suggest that adverse reproductive effects can occur in both male and female solvent-exposed workers according to the Washington DC: US Government Printing Office. Occupational exposure to both carbon disulfide and benzene has been associated with menstrual abnormalities (Hunt, 1979) and exposure to ethylene oxide has been associated with spontaneous abortion (Hemminiki, 1995).

Benzene has been proved to cause testicular atrophy, oligospermia and teratospermia in mice (Ward *et al.*, 1985).

Many animal studies are available for exposure to solvents and additional research is needed to clarify reproductive effects caused by solvents in humans (Gerr & Letz, 1992).

According to Stacey and Winder (2004), evidence is also available from animal studies to indicate that 2 –etoxyethanol, monomeric methacrylates and methyl ketone cause teratogenic effects. Dicloroethylene and xylene cause foetotoxicity.

2.10 Carcinogen

The organic solvents benzene and the chloromethyl ethers, bis-chloromethyl ether (BCME), ethanol (alcohol drinking), mineral oils (untreated), vinyl chloride monomer and technical grade chloromethyl ether (CMME), are human carcinogens well-established by the International Agency for Research on Cancer (IARC) as group 1 carcinogens (Winder & Stacey, 2004). Out of all of these Group 1 carcinogens, benzene is the most important as it causes leukemia and other hematopoietic disorders in humans (Gerr & Letz, 1992). According to Winder and Stacey (2004), the use of bezene is virtually discontinued, Thiugh benzene may appear as an impurity in small concentrations in some thinners, and petrol can contain up to 5% benzene. BCME is also classified as a group 1 carcinogen and is known to cause small cell carcinoma of the lung. It is used as an analytical agent and solvent during the manufacture of polymers, ion-exchange resins and waterproof coatings. Epidemiological studies demonstrating excess small cell lung cancer in working populations exposed to BCME have come from the United States, the Federal Republic of Germany and Japan (Commission of European Communities). The categorization of ethanol as a Class 1 carcinogen is based principally on the production of cancer in the liver in chronic alcohol abusers. This raises issues of non-threshold carcinogens, acceptable exposures and mechanisms of carcinogenesis (Winder & Stacey, 2004).

2.11 Toxicity to the central nervous system

Neurotoxicity induced by the exposure to organic solvents has emerged as one of the most important issues in occupational health (Baker, 1988). Substantial concern stems from the essential life function performed by the nervous system as well as the fact that damage to it may be irreversible (Gerr & Letz, 1992). Although much work has been done, Substantial uncertainty still exists, particularly with regard to the effects on the central nervous system of long-term, low-level exposure to solvents.

Solvents can cause depressant intoxication following acute exposure, which appears to be related to physical or chemical interactions with membranes or neurotransmitters. Long-term heavy exposure to solvents may also cause persistent, potentially irreversible impairment in cognitive function and affect, which may be associated with structural changes in neural tissue (NIOSH 1987).

According to Gerr and Lentz (1992), solvents may exert their primary effect on the central nervous system (CNS), the peripheral nervous system (PNS), or both, and CNS effects are typically investigated with behavioural tests or electrophysiologic evaluations. It is also said that the information about the effect of occupational solvent exposure on the PNS has come from studies that utilize clinical evaluation,

electrophysiologic examination and histophathologic examination of biopsy specimens. Gerr and Lentz (1992) also indicated that the effects of occupational solvent exposure on the PNS are more clearly defined and easier to identify than those of the CNS, owing to the relative simplicity of both the structure and the function of the PNS.

The acute neurotoxic effects of organic solvent exposure in workers and laboratory animals are narcosis, anaesthesia, central nervous system (CNS) depression, respiratory arrest, unconsciousness and death (Gerr & Letz, 1992).

Acute experimental exposures of human volunteers to one or several organic solvents have impaired psychomotor function as measured by reaction time, manual dexterity, coordination or body balance (NIOSH, 1987).

Chronic animal studies with a limited number of organic solvents support the evidence for peripheral neuropathy and mild toxic encephalopathy in solvent-exposed workers (NOISH, 1987).

Epidemiologic studies of various groups of solvent-exposed workers have demonstrated statistically significant chronic changes in peripheral nerve function (sensory and motor nerve conduction velocities and electromyographic abnormalities) that persisted for months to years following cessation of exposure (Grasso *et al.*, 1988). Epidemiologic studies have also shown statistically significant increases in neurobehavioural effects in workers chronically exposed to organic solvents. These effects include disorders characterized by reversible subjective symptoms (fatigability, irritability and memory impairment), sustained changes in personality or mood (emotional instability and diminished impulse control and motivation), and impaired intellectual function (decreased concentration ability, memory and learning ability). Among organic solvent abusers, the most severe disorders reported are characterized by irreversible deterioration in intellect and memory (dementia) accompanied by structural CNS damage. According to La Dou (1997), drug or alcohol abuse may result in a clinical state identical to chronic solvent toxicity, distinguished only by history and other evidence of exposure. Diffuse organic brain disease, particularly Alzheimer's disease or less commonly, Creutzfeld Jacob disease must also be considered.

On the basis of the identified adverse health effects of solvent exposure, the National Institute for Occupational Safety and Health (NIOSH, 1987) recommends that employers use engineering controls, personal protective equipment and clothing, and worker education programmes to reduce exposure to organic solvents, at least to the concentrations specified in existing Occupational Safety and Health Administration (OSHA) permissible exposure limits (PEL's), or to NIOSH recommended exposure limits (REL's) or the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit values (TLVs) if they provide a greater degree of protection.

2.12 Biological monitoring

General

Biological monitoring according to Lauwerys *et al.* (1985), is the evaluation of the internal exposure of the organism to a chemical agent by a biological method. According to Heinrich-Ramm *et al.* (2000), biological monitoring is the measurement of a chemical, its metabolite, or a non adverse biochemical effect in a biological specimen for the purpose of assessing exposure. This means measuring the substance itself or its metabolites in various biological media like blood, urine, exhaled air, hair, dispossess tissue, etc. (Lauwerys *et al.*, 1985).

Biological monitoring provides a measure of the route of absorption (eg. inhalation, skin contact or ingestion). Total exposure rather than only workplace exposure is measured. The biological levels may not correlate well with environmental measurements. This variability occurs for several reasons: (1) actual work practices vary among employees doing identical work eg. one worker may have more skin contact or may inhale more of a chemical than another worker; (2) a high respiratory rate can increase pulmonary absorption of solvents by a factor of 3-4; (3) the rate of metabolism and excretion will vary between individuals even when hepatic or renal function is normal; and (4) lipid soluble chemicals may accumulate to a grater extent in a person with excess adipose tissue (La Dou, 1997).

Workplace exposure to different chemicals can be estimated in an individual by measuring the chemical in the blood, urine or exhaled air. Depending on the pharmacokinetics of the target substance, the body fluid sampled and the time of sampling, the measured level will reflect the duration of the exposure ranging from acute recent exposure or accumulated life time exposure (La Dou, 1997).

The advantages of biological monitoring are that it accounts for all routes of absorption, non occupational exposure is also assessed, and individual differences in the rate of uptake due either to use of personal protection or differential uptake secondary to work load or other factors are accounted for (Lauwerys *et al.*, 1985). Biological monitoring also has its disadvantages such as the need to obtain biologic media from the workers, limited understanding of the association between biological exposure measures and the workers' health and the limited number of solvents for which biological measures are available.

Biological monitoring of solvent exposure

According to La Dou *et al.* (1997), biological monitoring can provide a more accurate measure of exposure than environmental monitoring for some solvents. This is particularly true for substances whose pulmonary absorption is affected to a large degree by physical work and those with significant dermal exposure and absorption. Because excretion kinetics vary among compounds, kinetics must be considered in planning biological monitoring in which elimination of these compounds is measured as an estimate of solvent uptake (Baker *et al.*, 1985).

Solvents also have the properties that tend to make biological monitoring less practical: (1) they tend to be rapidly absorbed and excreted, so that biological levels change rapidly over time and (2) exposure over very short intervals is often a more important determinant of adverse heath effects than 8 hour or longer exposures. However, biological monitoring has been investigated for a number of solvents (Heinrich-Ramm *et al.*, 2000). According to La Dou (1997), the ACGIH has recommended biological exposure indices (BEIs) for the following solvents: n-Hexane, benzene, toluene, xylenes, ethyl benzene, styrene, phenols, methyl ethyl ketone, perchloroethylene, trichorothylene, dimethylformamide and carbon disulfide. Laboratories offer whole- blood or plasma analysis of solvents. For solvents with relatively slow excretion, such as perchloroethylene and methyl chroroform, analysis of blood is a reasonable alternative to analysis of solvents. However, for those with relatively fast excretion values the timing of the samples is critical and the results are, therefore, difficult to interpret. Most solvents distribute into several compartments of the body, so that the decline in blood levels exhibits several consecutive half times, with the first being very short in the order of 2-10 minutes. A blood sample taken immediately after an exposure will reflect primary peak exposure at that time, a sample taken 15-30 minutes after termination of exposure will reflect exposure over the preceding few hours, while a sample taken 16-20 hours after exposure will (prior to the next shift) reflect mean exposure over the preceding day. The distribution of exposure over an 8-hour shift will also affect the validity of the biological sample (La Dou, 1997).

2.13 Occupational exposure measurement sampling strategies

When a determination is made that indicates the possibility of any significant employee exposure to airborne concentrations of a toxic substance, the employee is obligated to take measurements of the exposure to substances. Several considerations are involved in formulating an employee exposure monitoring programme. The following questions are the most frequently asked when deciding on the methods used:

- Which employees are to be sampled?
- Where should the sampling device be located in relation to the employee sampled?
- How many samples should be taken?
- How long should the sampling intervals be?
- What time of the day should the sampling of the employee be done?

The proposed health regulations OHSACT require that once a positive determination is made that indicates the possibility of any employee exposure, the employee is required to take an exposure measurement of the employee believed to have the greatest exposure. The concept is known as the Maximum risk employee. This is used to reduce the sampling burden on the employer reasonably.

When sampling the employee presumed to be the most/highest exposed, the following criteria must be used to select the employee closest to the source of the hazardous material being generated. The distance from the source also plays a very important role and it is the only factor in determining risk potential. With the distance, employee mobility is another consideration. Careful observation is required to obtain an accurate picture of the workers movement within his work environment so that valid time exposure can be estimated. Air movement patterns within the workroom should be analysed to determine accurately the risk potential of employees. Differences in work habits of individuals can also affect levels of exposure.

2.14 Environmental monitoring of VOC's

Environmental monitoring involves measuring the concentrations of solvent vapour in ambient air that are available for respiratory uptake by workers. Both direct reading and indirect sampling methods are available as measuring techniques. According to Gerr and Letz (1992), direct reading equipment includes indicator tubes, portable gas chromatographs and portable infrared analyzers, among others. This method is not as accurate as the indirect method.

The indirect active method for sampling of solvents is the most commonly used and is more accurate then the direct method.

During the indirect method, sorbent tubes with a media like silica gel or activated carbon are used which is connected to a pump for the collecting of the sample. The solvent is absorbed onto this material. The tube contains two layers of active charcoal or silica gel (grain size 0.4 to 0.8 mm) separated by a foam plug. The first layer is known as the absorption layer (100 mg) and the second the backup layer (50mg). The contaminant is supposed to be absorbed in the first layer, but in certain instances it can pass into the backup layer once the capacity of the first layer is exhausted. Thus breakthrough must be prevented by regulating the sampling period. After sampling the tube is sealed and sent to a laboratory where it is analysed.

This method of assessing exposure is most useful when the composition of the airborne solvent contaminants is well known. Gerr and Letz (1992) also indicated that methods of sampling include grab samples, collection of time weighted average samples, use of solid absorbents and diffusion badges. Environmental measures of exposure can also be obtained from fixed locations at the work site and are called "area or static samples." Alternatively, samples can also be taken in the breathing zone of the worker where the worker must wear a portable device that samples air near the nose and mouth. Breathing zone samples are considered more representative of individual exposure than area samples.

The uptake of solvents according to Gerr and Letz (1992), depends on several factors in addition to the concentration of solvents in air, including both dermal uptake and work load.

The disadvantage of using environmental monitoring as the sole index of exposure is that the contribution of these other factors to solvent uptake is not measured and, therefore, the actual dose may be poorly estimated (Gerr & Letz, 1992).

2.15 Evaluation of extraction ventilation systems

Ventilation is normally used as control measure to reduce exposure to air born particles. There are three types of ventilation, natural, general and local exhaust ventilation (LEV). Where air is required to be moved some distance, ducting is used which may be of considerable length and may contain bends, changes of section and branch pieces and other fittings. Coupled with the capacity to draw in fresh air or to recyle it, a ventilation system irrespective of whether it is a LEV or a general ventilation system may contain filters, heaters, coolers, humidifiers or a combination of these. To prevent atmospheric pollution from the discharge of dirty air, dust collectors and various air cleaners may be used (Plog & Quinlan, 2002).

Performance of ventilation systems requires to be checked from time to time to ensure satisfactory operation. This involves measuring air volume flow rates, velocities, pressures inside the ducts and the tracing of air flow patterns around ventilation terminals such as extraction hoods, slots, enclosures and fume cupboards. The routine checking of ventilation systems is recommended for all places where it has been considered necessary to maintain comfort or a healthy working environment. Air volume flow rates are quoted in units of cubic metres per second (m^3/s) or air changes per hour.

A local exhaust ventilation system relies on a totally different principle to general ventilation. Instead of allowing the contaminant to become airborne and then dispersing it, the idea of LEV is to capture the contaminant close to the source. It is then removed (sucked out of) from the workplace.

The key components of an LEV system are an extraction hood, where the contaminant is drawn in, an extraction fan to power the system, duct work to connect the fan to the hood, an air cleaner to clean emissions before being discharged to the outside of the workplace by means of a discharge point or stack (Plog & Quinlan, 2002)..

The capture velocity is important in measuring the effectiveness of a LEV. Capture velocity can be defined as the air velocity necessary at the point of contaminant generation to capture the contaminant and draw it into the hood. The capture velocity needed to capture a certain practical depends on the size of the particle. The larger the practical the greater the suction power and thus the greater the capture velocity. This is reflected in the table where typical capture velocities are given.

Table 2.2: Recommended capture velocity (Plog & Quinlan, 2002)

Source Conditions	Typical Situations	Capture Velocity m/s
Release into still air with no velocity	Degreasing tanks, dying paint	0.25-0.5
Released at a low velocity or into a moving air steam	Container filling, sieving, planting, pickling, debugging	0.5-1.0
Release at a moderate velocity or into turbulent air	Paint spraying, crushing, barrel filling	1.0-2.5
Release at high velocity or into very turbulent air	Grinding, fettling, blasting	2.5-10.0

For air to move in any ventilation system the following basic laws of air flow apply (Plog & Quinlan, 2002).

- For air to move between 2 points there must be a pressure difference between the points.
- Air will always move from a high to a low pressure
- The quality of air that will flow will depend upon the pressure difference.
- The higher the difference the greater the volume of air that will flow.
- Resistance to air flow will decrease the pressure difference and therewith the volume of air.

In order to measure air flow (velocity of flow multiplied by area flow) there are different methods and equipment available such as the following.

- Barometric pressure instruments
- Smoke tube kit
- Pressure measuring instruments
    - Velocity pressure devices
    - Air velocity measuring instruments
- Anemometers
    - Thermal or hot wire anemometers
    - Vane Anemometers
    - Calibration devices.

In practice one can determine the quantity of air moving by determining velocity. If the system is closed, however, it is impossible to do this and the pressure gauges are used.

The instrument used during this survey to determine air flow of the extraction systems was a hot wire anemometer. This device relies upon the cooling power of the air to cool a sensitive head. Essentially the

heated head is a hot wire, thermocouple to thermister bead through which an electric current is passing to maintain it at a constant temperature. As air blows over it cooling takes place depending upon the air velocity. The current which is required to keep the temperature constant is registered on a meter which has been previously calibrated in units of air velocity. This instrument requires careful handling and regular calibration against known air speeds. Also they may not be used in flammable atmospheres. They should be zero calibrated before use. Some of these instruments have a cowl over the sensing head to direct air over it which means that they must be carefully placed in the air stream with no yaw (Plog & Quinlan, 2002).

Chapter 3: Methods

3.1 General

This survey was conducted with SABS approved apparatus and instrumentation in accordance with requirements as set out in SABS Codes of Practice for Technical methods, NIOSH method (2549) and OHS Act 85 of 1993 as a set standard for evaluation purposes. The 13 most exposed workers were chosen according to OESSM.

Because of the cost implications for biological and occupational hygiene sample analyses only thirteen occupationally exposed workers were chosen in four different departments namely:

1. FO1:	Where electro detonators are manufactured.
2. Paint shop:	Where the shells are cleaned, phosphated, screen printed and spray-painted
3. Blue building:	Where plants and equipment are manufactured and painted
4. A03:	Melting and filling of empty shells with explosives as well as the stenciling of the shells for packaging and numbering.

3.2 Biological monitoring

Methodology

The biological level of a chemical is determined by its rate of absorption, elimination and metabolism. All the biological monitoring samples were taken by a Occupational Health Nurse (OHN) under the supervision of the medical practitioner. The following samples were taken depending on the solvent to which the worker was exposed.

- Full blood count
- Differential white cell count
- Benzene and phenol exposure
- Liver function
- Xileen exposure
- Tumor makers
- Testosterone.

3.3 Time of collection

The biological samples were taken directly after the end of a work shift. Prior to taking the samples the OHN consulted with the laboratory about the medium/tubes in which the blood samples must be taken.

Before the urine samples were taken it was ensured that the workers washed their hands. The samples were sent to a laboratory directly after they were taken.

The following tests were done:

- Full blood count
- Differential white cell count
- Benzene and phenol exposure / concentration
- Liver function
- Xileen exposure / concentration
- Tumor makers (BHCG)
- Testosterone level

3.4 Selecting a laboratory

Blood analysis was done by Drs. Du Buisson, Bruinette & Kramer, an accredited laboratory. This laboratory was chosen because it conforms to the necessary quality standards and requirements.

3.5 Extraction fans

Instrumentation

- A hot wire anemometer with a temperature probe – Extech, Serial No. L719698 calibrated by the SABS Air and Gas Flow Metrology Department was used.
- Tape measure and marker pen.

Methods Used

At each fume hood cupboard a series of air velocity measurements were taken across the open area, in order to obtain face/capture velocities. Where cupboards were fitted with moveable sashes, three conditions were evaluated, namely with sash 100%; 50% open and 25% open respectively. It is assumed that the 25% open condition is standard practice. Where possible the capture velocities at duct extraction openings were measured.

3.6 Air quality – organic vapour

Instrumentation / Methods Used

NIOSH method 2549 were used to determine the amount of VOC's in the workers breathing zone.

Pre-selected approved SKC charcoal tubes connected to Gilian low-flow personal pumps were used to sample volatile organic compounds in the employees breathing zones.

Sorbent Tubes

Content:

- The sorbent tubes contain 2 layers of active charcoal (grain size 0.4 to 0.8 mm) separated by a foam plug
- The absorption layer contains 100mg active charcoal/ silica gel
- The backup layer contains 50 mg active charcoal
- On the inlet side of the tube is a glass fiber fixing element in front of the absorption layer with a foam fixing element at the end of the back-up layer (tube outlet) and polythene sealing caps.

Preparation of the tubes and pump:

- The tips of the active charcoal tube are opened
- The tube is then connected to a suitable calibrated pump
- The backup layer (shorter) of material faces the pump
- The sorbent tube should be kept in a vertical position.

Air sampling volume:

- The flow rate of the pumps was set at 0.01 and it is designed to measure average concentration of flow concentrations over a measured time interval of 8 hours or less
- The organic contaminant is adsorbed by diffusion on the active charcoal and in the ideal situation is completely adsorbed in the first layer
- The amount of contaminant adsorbed is determined by exposure time and contaminant concentration present in the sampled environment.
- After the sample has been collected the tube is sealed with the polyethylene caps
- The tubes are dispatched to the laboratory for analyses.

Laboratory analysis of tubes:

- The sorbet tube is analyzed in the laboratory whereby the substance collected on the active charcoal is desorbed. In parallel a blank test is carried out with an unused tube i.e. the used and unused tubes are treated by the same laboratory method. Gas chromatography analysis is performed on sample tubes.
- An action index that serves as a precautionary measure was used to evaluate the results and the action index should not exceed the number one.

3.7 Statistical analysis of results

The Statistics Department of North West University made use of Statistica to do the statistical analysis of the results obtained.

Chapter 4: Results

4.1 Air quality – organic vapour

Air quality index is an index that is used as an action level. It is lower than the exposure limit and serves as a warning that some kind of action should be taken before the hazard reach the actual exposure limit. The action index can be determined by dividing the measured air concentration by the occupational exposure limit. The total must then be multiplied by two. If the value obtained is more the 1 then the action index is exceeded. If the action indexes of mixed exposure are added and the values obtained are equal to or more than one, the action index is exceeded.

Table 4.1: Volatile organic vapours measured at Dunfilm

AREA	SUBSTANCE MONITORED	Concentration [mg/m³]	OEL (S.A) mg/m³	ACTION INDEX	COMMENTS
SAMPLE G – A. P – POLISH ROOM					
	Acetone	BDL	1780	-	Within standard
	Ethanol	1053	1900	1.10*	Within standard but exceeding action index of "1"
	n-Hexane	0.16	70	0.004	Within standard
	n-Heptane	0.23	1600	0.0002	Within standard
	Methyl Ethyl Ketone	BDL	590	-	Within standard
	Ethyl Acetate	0.12	1400	0.0001	Within standard
	Toluene	0.10	188	0.001	Within standard
	Xylene	0.27	435	0.001	Within standard
	Ethyl Benzene	0.09	435	0.0004	Within standard
	n-Butyl Acetate	BDL	710	-	Within standard
	1,2,3-Trimethyl Benzene	BDL	123	-	Within standard
	1,2,4-Trimethyl Benzene	BDL	123	-	Within standard
	1,3,5-Trimethyl Benzene	BDL	123	-	Within standard
AQI				1.1067*	**AQI>1**
SAMPLE H – L. B – POLISH ROOM					
	Acetone	2.25	1780	0.002	Within standard
	Ethanol	BDL	1900	-	Within standard
	n-Hexane	0.03	70	0.0008	Within standard
	n-Heptane	BDL	1600	-	Within standard
	Methyl Ethyl Ketone	BDL	590	-	Within standard
	Ethyl Acetate	BDL	1400	-	Within standard
	Toluene	BDL	188	-	Within standard
	Xylene	BDL	435	-	Within standard

AREA	SUBSTANCE MONITORED	Concentration [mg/m³]	OEL (S.A) mg/m³	ACTION INDEX	COMMENTS
	n-Butyl Acetate	BDL	710	-	Within standard
	1,2,3-Trimethyl Benzene	BDL	123	-	Within standard
	1,2,4-Trimethyl Benzene	BDL	123	-	Within standard
	1,3,5-Trimethyl Benzene	BDL	123	-	Within standard
AQI				**0.0028**	**AQI = <1**
SAMPLE I – M.S. L – POLISH ROOM					
	Acetone	0.74	1780	0.0008	Within standard
	Ethanol	3.91	1900	0.004	Within standard
	n-Hexane	0.10	70	0.002	Within standard
	n-Heptane	0.09	1600	0.0001	Within standard
	Methyl Ethyl Ketone	2.50	590	0.008	Within standard
	Ethyl Acetate	4.12	1400	0.005	Within standard
	Toluene	BDL	188	-	Within standard
	Xylene	BDL	435	-	Within standard
	Ethyl Benzene	0.18	435	0.0008	Within standard
	n-Butyl Acetate	0.64	710	0.0018	Within standard
	1,2,3-Trimethyl Benzene	BDL	123	-	Within standard
	1,2,4-Trimethyl Benzene	2.99	123	0.048	Within standard
	1,3,5-Trimethyl Benzene	12.90	123	0.20	Within standard
AQI				**0.2705**	**AQI = <1**

Table 4.2: Volatile organic vapours measured at FO1A (Electro detonators).

AREA	SUBSTANCE MONITORED	Concentration [mg/m³]	OEL (S.A)	ACTION INDEX	COMMENTS
SAMPLE K – E. MO					
	Acetone	1480	1780	1.66*	Within standard but exceeding action index of "1"
SAMPLE L – C. MA					
	Acetone	1298	1780	1.45*	Within standard but exceeding action index of "1"
SAMPLE M – S.J. KE					
	Acetone	52.83	1780	0.05	Within standard.

Table 4.3: Volatile organic vapours measured at Paint Shop BWO7

AREA	SUBSTANCE MONITORED	Concentration TWA [mg/m³]	OEL (S.A)	ACTION INDEX	COMMENTS
SAMPLE E - W. R - SPRAY BOOTH 1					
	Acetone	BDL	1780	-	Within standard
	Methyl Ethyl Ketone	BDL	590	-	Within standard
	Ethyl Acetate	0.04	1400	0.00002	Within standard
	2-Butoxyethanol	BDL	120	-	Within standard
	Toluene	0.08	188	0.0004	Within standard
	Xylene	0.02	435	0.00004	Within standard
AQI				0.00046	1>0.00046
SAMPLE F - F. P - SPRAY BOOTH 6					
	Acetone	BDL	1780	-	Within standard
	Ethanol	BDL	1900	-	Within standard
	n-Hexane	BDL	70	-	Within standard
	n-Heptane	BDL	1600	-	Within standard
	Methyl Ethyl Ketone	0.12	590	0.0002	Within standard
	Ethyl Acetate	0.30	1400	0.0002	Within standard
	Toluene	0.70	188	0.003	Within standard
	Xylene	1.79	435	0.004	Within standard
AQI				0.0074	1>0.0074
	Ethyl Benzene	0.59	435	0.0013	Within standard
	n-Butyl Acetate	0.62	710	0.0008	Within standard
	1,2,3-Trimethyl Benzene	0.02	123	0.0001	Within standard
	1,2,4-Trimethyl Benzene	0.05	123	0.0004	Within standard
	1,3,5-Trimethyl Benzene	0.11	123	0.0008	Within standard
AQI				0.0034	1>0.0034

Table 4.4: Volatile organic vapours measured at Paint shop BW02 air craft & bomb painting

AREA	SUBSTANCE MONITORED	Concentration TWA [mg/m³]	OEL (S.A)	ACTION INDEX	COMMENTS
Sample A – Ca - Paint Line					
	Acetone	BDL	1780	-	Within standard
	Methyl Ethyl Ketone	0.01	590	0.00001	Within standard
	Ethyl Acetate	0.04	1400	0.00002	Within standard
	2-Butoxyethanol	BDL	120	-	Within standard
	Toluene	0.67	188	0.0035	Within standard
	Xylene	0.81	435	0.0018	Within standard
	Ethyl Benzene	0.20	435	0.0004	Within standard

AREA	SUBSTANCE MONITORED	Concentration TWA [mg/m³]	OEL (S.A)	ACTION INDEX	COMMENTS
	n-Butyl Acetate	0.10	710	0.0001	Within standard
	1,2,3-Trimethyl Benzene	0.20	123	0.0016	Within standard
	1,2,4-Trimethyl Benzene	0.08	123	0.0006	Within standard
	1,3,5-Trimethyl Benzene	0.02	123	0.0001	Within standard
AQI				0.008	1>0.008
Sample B – H. M – Paint Line					
	Acetone	BDL	1780	-	Within standard
	Methyl Ethyl Ketone	0.17	590	0.0002	Within standard
	Ethyl Acetate	0.27	1400	0.00019	Within standard
	2-Butoxyethanol	0.09	120	0.0007	Within standard
	Toluene	0.50	188	0.0026	Within standard
	Xylene	0.55	435	0.0012	Within standard
	Ethyl Benzene	0.18	435	0.0004	Within standard
	n-Butyl Acetate	0.03	710	0.00004	Within standard
	1,2,3-Trimethyl Benzene	0.08	123	0.0006	Within standard
	1,2,4-Trimethyl Benzene	0.04	123	0.0003	Within standard
	1,3,5-Trimethyl Benzene	0.08	123	0.0006	Within standard
AQI				0.0068	1>0.0068
Sample C – Mar – Paint Line					
	Acetone	0.92	1780	0.0005	Within standard
	Methyl Ethyl Ketone	BDL	590	-	Within standard
	Ethyl Acetate	BDL	1400	-	Within standard
	2-Butoxyethanol	0.01	120	0.00008	Within standard
	Toluene	1.29	188	0.0068	Within standard
	Xylene	0.87	435	0.002	Within standard
	Ethyl Benzene	0.21	435	0.0004	Within standard
	n-Butyl Acetate	0.22	710	0.0003	Within standard
	1,2,3-Trimethyl Benzene	0.15	123	0.0012	Within standard
	1,2,4-Trimethyl Benzene	0.07	123	0.0005	Within standard
	1,3,5-Trimethyl Benzene	0.19	123	0.0015	Within standard
AQI				0.0132	1>0.0132
Sample D1 – P. N - Darkroom					
	Acetone	2.97	1780	0.0016	Within standard
	Methyl Ethyl Ketone	BDL	590	-	Within standard
	Ethyl Acetate	BDL	1400	-	Within standard
	2-Butoxyethanol	BDL	120	-	Within standard
	Toluene	0.46	188	0.0002	Within standard
	Xylene	0.24	435	0.0005	Within standard
	Ethyl Benzene	0.06	435	0.00013	Within standard

AREA	SUBSTANCE MONITORED	Concentration TWA [mg/m³]	OEL (S.A)	ACTION INDEX	COMMENTS
	n-Butyl Acetate	0.05	710	0.00007	Within standard
	1,2,3-Trimethyl Benzene	0.06	123	0.0004	Within standard
	1,2,4-Trimethyl Benzene	0.02	123	0.0001	Within standard
	1,3,5-Trimethyl Benzene	0.01	123	0.00008	Within standard
AQI				0.003	I>0.003
Sample D2 – P. N – Ink Room					
	Acetone	1.82	1780	0.0010	Within standard
	Methyl Ethyl Ketone	BDL	590	-	Within standard
	Ethyl Acetate	BDL	1400	-	Within standard
	2-Butoxyethanol	0.19	120	0.0015	Within standard
	Toluene	BDL	188	-	Within standard
	Xylene	BDL	435	-	Within standard
AQI				0.0025	I>0.0025

Table 4.5: Volatile organic vapours measured at Blue Building

AREA	SUBSTANCE MONITORED	Concentration TWA [mg/m³]	OEL (S.A)	ACTION INDEX	COMMENTS
SAMPLE N – S. Ra – BLUE BUILDING					
	Acetone	1.40	1780	0.00078	Within standard
	Benzene	0.02	16	0.00125	Within standard
	Toluene	1.23	188	0.0065	Within standard
	Xylene	0.92	435	0.0021	Within standard
	Ethyl Benzene	0.21	435	0.0004	Within standard
	2-Butoxyethanol	0.08	120	0.00067	Within standard
	1,2,3-Trimethyl Benzene	0.22	123	0.00178	Within standard
	1,2,4-Trimethyl Benzene	0.64	123	0.0052	Within standard
	1,3,5-Trimethyl Benzene	0.13	123	0.00010	Within standard
AQI				0.01968	AQI<1

* Above Occupational Exposure Limit (RL) - Time Weighted Average (TWA - Calculated for 8 hour day)

4.2 Air quality – extractor fans

Actual values for the various parameters evaluated in each area at the specific fume hood cupboard are reflected in the following tables:

Table 4.6: Extraction ventilation velocities measured at Dunfilm polishing room: laboratory fume hood cupboard

Area	Average capture velocity ((m/sec))	Required Capture Velocity ((m/sec))	Average face velocity measurements sash fully open (100%) ((m/sec))	Face velocity measurements sash 50% open ((m/sec))	Average face velocity measurements sash 50% open ((m/sec))	Minimum face velocity Capture required ((m/sec))	Comments
Vapour Cabinet at Dunfilm Room 11 (Polishing Room)	-	-	-	0.8 0.7 0.8 0.7 0.7	0.74	0.5 Face Velocity	Within standard.

* Below minimum required.

Table 4.7: Extraction ventilation velocities measured at FO1A : fume hood cupboards

Area	Capture velocity measurements ((m/sec))	Required Capture Velocity ((m/sec))	Average face velocity measurements sash fully open (100%) ((m/sec))	Comments
A1 – TOOLING CLEANING	0.2 0.3 0.3 0.2	0.5 – 1.0	0.25*	Below standard.
A8.1 SPRAY VARNISH D.E.T. INTERMEDIATE. Cleaning of equipment.	4.0 3.5 1.9 1.5 2.3 2.0	0.5 – 1.0	2.5	Within standard

* Below minimum required.

Table 4.8: Extraction ventilation velocities measured at FO1: laboratory fume hood cupboards

Area	Average capture velocity ((m/sec))	Required Capture Velocity (m/sec)	Average face velocity measurements sash fully open (100%) (m/sec)	Minimum capture velocity Capture required (m/sec)	Comments
A5A - Canopy	2.5	0.2 0.3	0.25*	0.5 – 1.0	Below standard.
A5B - Canopy	4.3	0.4 0.2 0.4	0.33*	0.2 – 1.0	Below standard

* Below minimum required

Table 4.9: Extraction ventilation velocity measured at BWO2 aircraft & bomb painting: fume hood

Location	Area	Measuring Points (m/sec)	Average velocity (m/sec)	Min. Required (m/sec)	Comments
BWO2	Varnishing Machine	3.9 1.8 1.1 1.2	2	0.5 – 1.0	Within minimum requirement.

Table 4.10: Extraction ventilation velocity measured at BWO2 aircraft & bomb painting: fume hoods

Location	Area	Measuring Points (m/sec)	Average velocity (m/sec)	Min. Required (m/sec)	Comments
BWO2	Aircraft and Bomb Painting cubicle 1.	1.6 1.1 1.8 1.7 2.2 0.3 0.3 0.4 0.4	0.97	0.5 – 1.0	Within minimum requirement.

Location	Area	Measuring Points (m/sec)	Average velocity (m/sec)	Min. Required (m/sec)	Comments
BWO2	Aircraft and Bomb Painting Paint cubicle 2 Paper spray on low surface	0.7 0.8 0.5 0.4 0.3 0.4	0.52	0.5 – 1.0	Within minimum requirement
BWO2	Aircraft and Bomb Painting Paint cubicle 3 Bomb fin	1.6 1.4 1.4 0.3 0.3 0.4	0.9	0.5 – 1.0	Within minimum requirement.
BWO2	Paint shop 30cm above silkscreen At brim of canopy (underside) At extraction duct outlet in canopy	0.1 0.6 0.7 5	1.6	0.5 – 1.0	Within minimum requirement.

* Below minimum required.

Table 4.11: Extraction velocity measurements BWO7: fume hoods

Location	Area	Measuring Points (m/sec)	Average velocity (m/sec)	Min. Required (m/sec)	Comments
BWO7	Blackburn & Varnish Machine	0.4 0.5 0.8 0.5 0.7	0.58	0.5 – 1.0	Within minimum requirement.
BW 07	Top of Spray booth 8	1.5 1.2 1.1 1.1 0.9 1.0 1.0 0.9	1.08	0.5 – 1.0	Within minimum requirement
BWO7	At Spray booth 9	0.7 0.8 0.5	0.58	0.5 – 1.0	Within minimum

Location	Area	Measuring Points (m/sec)	Average velocity (m/sec)	Min. Required (m/sec)	Comments
		0.4 0.5			requirement.
BWO7	At Spray booth 10	0.3 0.7 0.8 0.9 0.5 0.5	0.62	0.5 – 1.0	Within minimum requirement.
BWO7	At Spray booth 7 Rear	0.1 0.3 0.4 0.7 0.8 0.9	0.53	0.5 – 1.0	Within minimum requirement
BWO7	At Spray booth 1 At workers breathing zone	0.6 0.8 1.0 0.6 0.9 0.8	0.78	0.5 – 1.0	Within minimum requirement
BWO7	At Spray booth 2	0.5 0.8 0.8 0.5	0.65	0.5 – 1.0	Within minimum requirement
BWO7	At Spray booth 3 Filter blocked When measurements were taken	0.1 0.2 0.7	0.34*	0.5 - 10	Below minimum requirement
BWO7	At Spray booth 4	1.4 1.0 1.2 1.2 1.2 0.8 1	1.11	0.5 - 10	Within minimum requirement
BWO7	At Spray booth 5	0.3 0.5			

Location	Area	Measuring Points (m/sec)	Average velocity (m/sec)	Min. Required (m/sec)	Comments
		0.4			
		0.5	0.43*	0.5 - 10	Below minimum requirement
		0.3			
		0.5			
		0.1			
		0.5			
	Rear at duct	0.8			
BWO7	At Spray booth 6	0.5			
		0.7			
		0.5			
		0.9			
		0.7	0.67	0.5 - 10	Within minimum
		0.1			requirement
	At Rear	1.1			
		0.9			

* Below minimum required

Table 4.12: Extraction ventilation velocities at A03k Touch-Up Station

Area	Average capture velocity (m/sec)	Average face velocity measurements	Minimum capture velocity Capture required (m/sec)	Comments
Extract hood middle when paint spraying – Touch- up Station At face of hood				
	3.0	2.85	0.5 – 1.0	
	2.9		Face Velocity	Within standard
	2.7			
Below hood brim	2.8			
			0.5 – 1.0	
	0.9	0.75	Face Velocity	Within standard
	0.5			
	0.6			
	1.0			

• Below Minimum Standard

4.3 Blood analysis

Table 4.13: Blood analysis done after exposure at Dunfilm Polishing Room

Location	Person	Type of test	Results	Specifications units	Comments
Dunfilm	**M.SL**	**Full blood count**			With in Specifications
		-Hemoglobin	14.7	12.0-16.0 G/dL	
		-Red blood count	4.93	4.00-5.00 X 10^{12}/L	
		-Hematokrit	43.0	36-46 %	
		-GKV	87.0	80-100 Fl	
		-GKH	30.0	27-32 pg	
		-GKHK	34.3	32.0-35.0 g/Dl	
		-RDW	11.8	11.7-13.6%	
		White cell count differential			
		-White cell count	5.5	4.0-10.0 X 10^9/L	
		-Neurofils %	61.0	%	
		-Neurofils abs	3.36	1.90-7.40 X 10^9/L	
		-Lymphocytes %	30.0	%	
		-Lymphocytes abs	1.65	1.0-4.5 X 10^9/L	
		-Monocytes %	9.0	%	
		-Monocytes abs	0,50	0.2- 1.0 X 10^9/L	
		-Plate counts	220	140-450 X 10^9/L	
		Benzene and phenol exposure			
		-Urine phenol		0-250 mg/g	
		-Phenol creatine		0-250 mg/g	

Table 4.14: Blood analysis done after exposure at F01A

Location	Worker	Type of test	Results	Specifications units	Comments
FOI	E. Mo	**Full blood count**			
		-Hemoglobin	11.5	12.0-16.0 G/dL	Low
		-Red blood count	4.51	4.00-5.00 X 10^{12}/L	
		-Hematokrit	35.0	36-46 %	Low
		-GKV	99.8	80-100 Fl	
		-GKH	32.9	27-32 pg	High
		-GKHK	32.9	32.0-35.0 g/Dl	
		-RDW	13.2	11.7-13.6%	
		White cell count differential			
		-White cell count	9.5	4.0-10.0 X 10^9/L	
		-Neurofils %	53.6	%	
		-Neurofils abs	5.2	1.90-7.40 X 10^9/L	
		-Lymphocytes %	35.6	%	
		-Lymphocytes abs	3.40	1.0-4.5 X 10^9/L	
		-Monocytes %	8.8	%	
		-Monocytes abs	0.8	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	1.5	%	
		-Eosinofiles abs	0,1	0.0-0.5 X 10^9/L	
		-Basophiles %	0.5	%	
		-Basophiles abs	0	0.0-0.10 X 10^9/L	
		-Plate counts	273	140-450 X 10^9/L	
		Biochemistry			
		-s-Gamma GT	43	5-40 U/L 37°C	High
		-s-ALT (SGPT)	9	10-32 U/L 37°C	Low
		-s-AST (SGOT)	14	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	8.5	0-250 mg/g	
		-Phenol creatine	17.5	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- Mytile hypo creatine	<0.02	0-2.5 g/g	
F01	M. Sey	**Full blood count**			
		-Hemoglobin	12.10	12.0-16.0 G/dL	
		-Red blood count	3.95	4.00-5.00 X 10^{12}/L	Low
		-Hematokrit	36.20	36-46 %	
		-GKV	91.70	80-100 Fl	
		-GKH	30.60	27-32 pg	
		-GKHK	33.40	32.0-35.0 g/Dl	
		-RDW	13.10	11.7-13.6%	
		White cell count differential			
		-White cell count	4.20	4.0-10.0 X 10^9/L	
		-Neurofils %	34.60	%	
		-Neurofils abs	1.50	1.90-7.40 X 10^9/L	Low
		-Lymphocytes %	45.2	%	
		-Lymphocytes abs	1.90	1.0-4.5 X 10^9/L	
		-Monocytes %	11.60	%	
		-Monocytes abs	0.50	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	7.80	%	
		-Eosinofiles abs	0,30	0.0-0.5 X 10^9/L	
		-Basophiles %	0.80	%	
		-Basophiles abs	0	0.0-0.10 X 10^9/L	
		-Plate counts	281	140-450 X 10^9/L	

Location	Worker	Type of test	Results	Specifications units	Comments
		Biochemistry			
		-s-Gamma GT	22	5-40 U/L 37°C	
		-s-ALT (SGPT)	18	10-32 U/L 37°C	
		-s-AST (SGOT)	17	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	6.20	0-250 mg/g	
		-Phenol creatine	5.40	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	
FOI	C. Ma	**Full blood count**			
		-Hemoglobin	13.30	12.0-16.0 G/dL	
		-Red blood count	4.68	4.00-5.00 X 10^{12}/L	
		-Hematokrit	39.90	36-46 %	
		-GKV	85.20	80-100 Fl	
		-GKH	28.40	27-32 pg	
		-GKHK	33.30	32.0-35.0 g/Dl	
		-RDW	12.50	11.7-13.6%	
		White cell count differential			
		-White cell count	7.70	4.0-10.0 X 10^9/L	
		-Neurofils %	60.40	%	
		-Neurofils abs	4.60	1.90-7.40 X 10^9/L	
		-Lymphocytes %	31.20	%	
		-Lymphocytes abs	2.40	1.0-4.5 X 10^9/L	
		-Monocytes %	7.20	%	
		-Monocytes abs	0.60	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	1.00	%	
		-Eosinofiles abs	0.10	0.0-0.5 X 10^9/L	
		-Basophiles %	0.20	%	
		-Basophiles abs	0.00	0.0-0.10 X 10^9/L	
		-Plate counts	333	140-450 X 109/L	
		Biochemistry			
		-s-Gamma GT	12	5-40 U/L 37°C	
		-s-ALT (SGPT)	11	10-32 U/L 37°C	
		-s-AST (SGOT)	20	10-32 U/L 37°C	
		Benzene and phenol exposure			
		-Urine phenol	6.10	0-250 mg/g	
		-Phenol creatine	4.00	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	
FOI	SJ Ke	**Full blood count**			
		-Hemoglobin	11.8	12.0-16.0 G/dL	Low
		-Red blood count	3.74	4.00-5.00 X 10^{12}/L	Low
		-Hematokrit	36.00	36-46 %	
		-GKV	96.80	80-100 Fl	
		-GKH	31.60	27-32 pg	
		-GKHK	32.70	32.0-35.0 g/Dl	
		-RDW	11.40	11.7-13.6%	Low
		White cell count differential			
		-White cell count	6.50	4.0-10.0 X 10^9/L	
		-Neurofils %	50.80	%	
		-Neurofils abs	3.20	1.90-7.40 X 10^9/L	
		-Lymphocytes %	40.90	%	

Location	Worker	Type of test	Results	Specifications units	Comments
		-Lymphocytes abs	2.70	1.0-4.5 X 10^9/L	
		-Monocytes %	5.70	%	
		-Monocytes abs	0.40	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	2.40	%	
		-Eosinofiles abs	0,20	0.0-0.5 X 10^9/L	
		-Basophiles %	0.20	%	
		-Basophiles abs	0	0.0-0.10 X 10^9/L	
		-Plate counts	304	140-450 X 10^9/L	
		Biochemistry			
		-s-Gamma GT	11	5-40 U/L 37°C	
		-s-ALT (SGPT)	11	10-32 U/L 37°C	
		-s-AST (SGOT)	16	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	5 20	0-250 mg/g	
		-Phenol creatine	5.40	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	

Table 4.15: Blood analysis done after exposure at Bwo2

Location	Worker	Type of test	Results	Specifications units	Comments
Paint shop	**LW Rab**	**Full blood count**			
		-Hemoglobin	16.00	12.0-16.0 G/dL	
		-Red blood count	5.34	4.00-5.00 X 10^{12}/L	High
		-Hematokrit	48.10	36-46 %	High
		-GKV	90.10	80-100 Fl	
		-GKH	29.90	27-32 pg	
		-GKHK	33.20	32.0-35.0 g/Dl	
		-RDW	12.60	11.7-13.6%	
		White cell count differential			
		-White cell count	6.30	4.0-10.0 X 10^9/L	
		-Neurofils %	52.30	%	
		-Neurofils abs	3.40	1.90-7.40 X 10^9/L	
		-Lymphocytes %	39.90	%	
		-Lymphocytes abs	2.50	1.0-4.5 X 10^9/L	
		-Monocytes %	7.10	%	
		-Monocytes abs	0.40	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	0.50	%	
		-Eosinofiles abs	0.00	0.0-0.5 X 10^9/L	
		-Basophiles %	0.2	%	
		-Basophiles abs	0	0.0-0.10 X 10^9/L	
		-Plate counts	294	140-450 X 10^9/L	
		Biochemistry			
		-s-Gamma GT	24.00	5-40 U/L 37°C	
		-s-ALT (SGPT)	21.00	10-32 U/L 37°C	
		-s-AST (SGOT)	25.00	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	5.00	0-250 mg/g	
		-Phenol creatine	2.10	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	

Location	Worker	Type of test	Results	Specifications units	Comments
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	
Paintshop	H. M	**Full blood count**			
		-Hemoglobin	13.7	12.0-16.0 G/dL	
		-Red blood count	5.17	4.00-5.00 X 10^{12}/L	High
		-Hematokrit	42.50	36-46 %	
		-GKV	82.20	80-100 Fl	
		-GKH	26.40	27-32 pg	Low
		-GKHK	32.20	32.0-35.0 g/Dl	
		-RDW	12.7	11.7-13.6%	
		White cell count differential			
		-White cell count	8.00	4.0-10.0 X 10^9/L	
		-Neurofils %	39.50	%	
		-Neurofils abs	3.20	1.90-7.40 X 10^9/L	
		-Lymphocytes %	46.60	%	
		-Lymphocytes abs	3.70	1.0-4.5 X 10^9/L	
		-Monocytes %	8.00	%	
		-Monocytes abs	0.60	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	5.70	%	
		-Eosinofiles abs	0.50	0.0-0.5 X 10^9/L	
		-Basophiles %	0.20	%	
		-Basophiles abs	0.00	0.0-0.10 X 10^9/L	
		-Plate counts	211	140-450 X 10^9/L	
		Biochemistry			
		-s-Gamma GT	6.00	5-40 U/L 37°C	
		-s-ALT (SGPT)	9.00	10-32 U/L 37°C	Low
		-s-AST (SGOT)	16.00	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	3.80	0-250 mg/g	
		-Phenol creatine	2.50	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	
Paint Shop	J .Ca	**Full blood count**			Within Specifications
		-Hemoglobin	14.80	12.0-16.0 G/dL	
		-Red blood count	4.84	4.00-5.00 X 10^{12}/L	
		-Hematokrit	43.70	36-46 %	
		-GKV	90.20	80-100 Fl	
		-GKH	30.70	27-32 pg	
		-GKHK	34.00	32.0-35.0 g/Dl	
		-RDW	12.30	11.7-13.6%	
		White cell count differential			
		-White cell count	4.80	4.0-10.0 X 10^9/L	
		-Neurofils %	31.60	%	
		-Neurofils abs	1.50	1.90-7.40 X 10^9/L	
		-Lymphocytes %	55.80	%	
		-Lymphocytes abs	2.70	1.0-4.5 X 10^9/L	
		-Monocytes %	10.80	%	
		-Monocytes abs	0.50	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	1.40	%	
		-Eosinofiles abs	0.10	0.0-0.5 X 10^9/L	
		-Basophiles %	0.40	%	
		-Basophiles abs	0.00	0.0-0.10 X 10^9/L	
		-Plate counts	230	140-450 X 10^9/L	
		Biochemistry			

Location	Worker	Type of test	Results	Specifications units	Comments
		-s-Gamma GT	18	5-40 U/L 37°C	
		-s-ALT (SGPT)	13	10-32 U/L 37°C	
		-s-AST (SGOT)	24	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	5	0-250 mg/g	
		-Phenol creatine	17.5	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	
Paint Shop	**Mar**	**Full blood count**			
		-Hemeglobin	11.5	12.0-16.0 G/dL	Low
		-Red blood count	4.51	4.00-5.00 X 10^{12}/L	
		-Hematokrit	35.0	36-46 %	Low
		-GKV	99.8	80-100 Fl	
		-GKH	32.9	27-32 pg	Low
		-GKHK	32.9	32.0-35.0 g/Dl	
		-RDW	13.2	11.7-13.6%	
		White cell count differential			
		-White cell count	9.5	4.0-10.0 X 10^9/L	
		-Neurofils %	53.6	%	
		-Neurofils abs	5.2	1.90-7.40 X 10^9/L	
		-Lymphocytes %	35.6	%	
		-Lymphocytes abs	3.40	1.0-4.5 X 10^9/L	
		-Monocytes %	8.8	%	
		-Monocytes abs	0.8	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	1.5	%	
		-Eosinofiles abs	0,1	0.0-0.5 X 10^9/L	
		-Basophiles %	0.5	%	
		-Basophiles abs	0	0.0-0.10 X 10^9/L	
		-Plate counts	273	140-450 X 10^9/L	
		Biochemistry			
		-s-Gamma GT	43	5-40 U/L 37°C	High
		-s-ALT (SGPT)	9	10-32 U/L 37°C	Low
		-s-AST (SGOT)	14	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	8.5	0-250 mg/g	
		-Phenol creatine	17.5	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	

Table 4.16: Blood analysis done after exposure at BWO2 Screen Printing

Location	Worker	Type of test	Results	Specifications units	Comments
Paint shop	**C Har**	**Hemoglobin**			
		-Red cell count	4.46	4.60-6.00 X 10^{12}/L	Low
		White cell count differential			
		-White cell count	8.30	4.0-10.0 X 10^9/L	
		-Platelet count	290.00	140-450 X 10^9/L	
		Biochemistry			

Location	Worker	Type of test	Results	Specifications units	Comments
		-s-Gamma GT	69.00	5-50 U/L 37°C	High
		-s-ALT (SGPT)	50.00	10-45 U/L 37°C	High
		-s-AST (SGOT)	23.00	10-40 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	27.70	0-250 mg/g	
		-Phenol creatine	22.90	0-250 mg/g	
		Xilene Exposure			
		-Metile hypoacid	37.31	0-2.5 g/g creatine	
		-Mitiel hupuur creatine	0.03	0-2.5 g/g	
		-U-hipuursuur	0.2 g/L	0-2.5 g/g creatine	
		-Hippuursuur: Kreat	0.2 g/g	0-2.5 g/g creatine	
		Tumor markers			
		BHCG	0.00	0.00-5.00 mIU/mL	
		Testosterone			
		-SHBG	16.9	7.30-43.00 nmol/L	
		- Total	9.00	4.0-18.7 nmol/L	
		- Free calculated	243.00	162-452.0 pmol/L	
Paint shop	P. N	**Hemoglobin**			
		-Red cell count	4.39	4.60-6.00 X 10^{12}/L	Low
		White cell count differential			
		-White cell count	5.6	4.0-10.0 X 10^{9}/L	
		-Platelet count	322	140-450 X 10^{9}/L	
		Biochemistry			
		-s-Gamma GT	21.00	5-50 U/L 37°C	
		-s-ALT (SGPT)	20.00	10-45 U/L 37°C	
		-s-AST (SGOT)	23.00	10-40 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	18.40	0-250 mg/g	
		-Phenol creatine	9.10	0-250 mg/g	
		Xilene Exposure			
		-Metile hypoacid	20.65	0-2.5 g/g creatine	
		-Mitiel hupuur creatine	0.01	0-2.5 g/g	
		-U-hipuursuur	0.30	mg/g	
		-Hippuursuur: Kreat			
		Tumor markers			
		BHCG	0.00		
		Testosterone			
		-SHBG	53.90	0.00-5.00 mIU/mL	High
		- Total	20.00		High
		- Free calculated	309.60	7.30-43.00 nmol/L	
				4.0-18.7 nmol/L	
				162-452.0 pmol/L	

Table 4.17: Blood analysis done after exposure at Bwo7

Location	Worker	Type of test	Results	Specifications units	Comments
Paintshop	F.P	**Full blood count**			Within
		-Hemoglobin	15.30	12.0-16.0 G/dL	Specifications
		-Red blood count	4.93	4.00-5.00 X 10^{12}/L	
		-Hematokrit	45.60	36-46 %	
		-GKV	92.50	80-100 Fl	
		-GKH	31.00	27-32 pg	
		-GKHK	33.50	32.0-35.0 g/Dl	
		-RDW	12.70	11.7-13.6%	
		White cell count differential			
		-White cell count	5.70	4.0-10.0 X 10^9/L	
		-Neurofils %	47.50	%	
		-Neurofils abs	2.70	1.90-7.40 X 10^9/L	
		-Lymphocytes %	42.8	%	
		-Lymphocytes abs	2.40	1.0-4.5 X 10^9/L	
		-Monocytes %	6.70	%	
		-Monocytes abs	0.40	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	2.90	%	
		-Eosinofiles abs	0.20	0.0-0.5 X 10^9/L	
		-Basophiles %	0 10	%	
		-Basophiles abs	0.00	0.0-0.10 X 10^9/L	
		-Plate counts	312	140-450 X 109/L	
		Biochemistry			
		-s-Gamma GT	26.00	5-40 U/L 37°C	
		-s-ALT (SGPT)	17.00	10-32 U/L 37°C	
		-s-AST (SGOT)	27.00	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Unne phenol	1.9	0-250 mg/g	
		-Phenol creatine	2.1	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	

Table 4.18: Blood analysis done after exposure at A03K

Location	Worker	Type of test	Results	Specifications units	Comments
A03	SD.T	**Full blood count**			
		-Hemoglobin	12.70	12.0-16.0 G/dL	
		-Red blood count	4.35	4.00-5.00 X 10^{12}/L	
		-Hematokrit	38.80	36-46 %	
		-GKV	89.10	80-100 Fl	
		-GKH	29.30	27-32 pg	
		-GKHK	32.80	32.0-35.0 g/Dl	
		-RDW	12.20	11.7-13.6%	
		White cell count differential			
		-White cell count	6.50	4.0-10.0 X 10^9/L	
		-Neurofils %	34.8	%	
		-Neurofils abs	2.30	1.90-7.40 X 10^9/L	
		-Lymphocytes %	54.0	%	
		-Lymphocytes abs	3.50	1.0-4.5 X 10^9/L	

Location	Worker	Type of test	Results	Specifications units	Comments
		-Monocytes %	7.60	%	
		-Monocytes abs	0.50	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	3.10	%	
		-Eosinofiles abs	0.20	0.0-0 5 X 10^9/L	
		-Basophiles %	0.50	%	
		-Basophiles abs	0	0.0-0.10 X 109/L	
		-Plate counts	344	140-450 X 109/L	
		Biochemistry			
		-s-Gamma GT	11	5-40 U/L 37°C	
		-s-ALT (SGPT)	9	10-32 U/L 37°C	Low
		-s-AST (SGOT)	19	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	5.90	0-250 mg/g	
		-Phenol creatine	6.40	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	
A03	**S. Ram**	**Full blood count**			
		-Hemoglobin	15.10	12.0-16.0 G/dL	
		-Red blood count	5.17	4.00-5.00 X 10^{12}/L	High
		-Hematokrit	45.40	36-46 %	
		-GKV	87.80	80-100 fL	
		-GKH	29.30	27-32 pg	
		-GKHK	33.40	32.0-35.0 g/Dl	
		-RDW	12.10	11.7-13.6%	
		White cell count differential			
		-White cell count	4.80	4.0-10.0 X 10^9/L	
		-Neurofils %	50.70	%	
		-Neurofils abs	2.40	1.90-7.40 X 10^9/L	
		-Lymphocytes %	39.80	%	
		-Lymphocytes abs	1.90	1.0-4.5 X 10^9/L	
		-Monocytes %	8.30	%	
		-Monocytes abs	0.40	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	1.1	%	
		-Eosinofiles abs	0.10	0.0-0 5 X 10^9/L	
		-Basophiles %	0.10	%	
		-Basophiles abs	0.00	0.0-0.10 X 10^9/L	
		-Plate counts	296	140-450 X 10^9/L	
		Biochemistry			
		-s-Gamma GT	29.00	5-40 U/L 37°C	
		-s-ALT (SGPT)	12.00	10-32 U/L 37°C	
		-s-AST (SGOT)	21.00	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	2.60	0-250 mg/g	
		-Phenol creatine	4.00	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15.00	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	
A03	**TW. P**	**Full blood count**			
	Acetone Spray Paint	-Hemoglobin	15.60	12.0-16.0 G/dL	
		-Red blood count	5.36	4.00-5.00 X 10^{12}/L	High
		-Hematokrit	46.70	36-46 %	High
		-GKV	87.00	80-100 Fl	
		-GKH	29.10	27-32 pg	

Location	Worker	Type of test	Results	Specifications units	Comments
		-GKHK	33.50	32.0-35.0 g/Dl	
		-RDW	12.00	11.7-13.6%	
		White cell count differential			
		-White cell count	7.90	4.0-10.0 X 10^9/L	
		-Neurofils %	38.20	%	
		-Neurofils abs	3.00	1.90-7.40 X 10^9/L	
		-Lymphocytes %	46.70	%	
		-Lymphocytes abs	3.80	1.0-4.5 X 10^9/L	
		-Monocytes %	8.00	%	
		-Monocytes abs	0.60	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	6.70	%	
		-Eosinofiles abs	0.50	0.0-0.5 X 10^9/L	
		-Basophiles %	0.40	%	
		-Basophiles abs	0.00	0.0-0.10 X 10^9/L	
		-Plate counts	238	140-450 X 10^9/L	
		Biochemistry			
		-s-Gamma GT	29.00	5-40 U/L 37°C	
		-s-ALT (SGPT)	27.00	10-32 U/L 37°C	
		-s-AST (SGOT)	21.00	10-32 UL 37°C	
		Benzene and phenol exposure			
		-Urine phenol	7.80	0-250 mg/g	
		-Phenol creatine	6.30	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		- -Mitiel hupuur creatine	<0.02	0-2.5 g/g	

Table 4.19: Blood analysis done after exposure at Blue building

Location	Worker	Type of test	Results	Specifications units	Comments
Blue building	**N. P**	**Full blood count**			
		-Hemoglobin	11.5	12.0-16.0 G/dL	
		-Red blood count	4.51	4.00-5.00 X 10^{12}/L	
		-Hematokrit	35.0	36-46 %	
		-GKV	99.8	80-100 Fl	
		-GKH	32.9	27-32 pg	
		-GKHK	32.9	32.0-35.0 g/Dl	
		-RDW	13.2	11.7-13.6%	
		White cell count differential			
		-White cell count	9.5	4.0-10.0 X 10^9/L	
		-Neurofils %	53.6	%	
		-Neurofils abs	5.2	1.90-7.40 X 10^9/L	
		-Lymphocytes %	35.6	%	
		-Lymphocytes abs	3.40	1.0-4.5 X 10^9/L	
		-Monocytes %	8.8	%	
		-Monocytes abs	0.8	0.2- 1.0 X 10^9/L	
		-Eosinofiles %	1.5	%	
		-Eosinofiles abs	0,1	0.0-0.5 X 10^9/L	
		-Basophiles %	0.5	%	
		-Basophiles abs	0	0.0-0.10 X 10^9/L	
		-Plate counts	273	140-450 X 10^9/L	
		Biochemistry			
		-s-Gamma GT	43	5-40 U/L 37°C	High
		-s-ALT (SGPT)	9	10-32 U/L 37°C	High
		-s-AST (SGOT)	14	10-32 U/L 37°C	
		Benzene and phenol exposure			
		-Urine phenol	8.5	0-250 mg/g	
		-Phenol creatine	17.5	0-250 mg/g	
		Xilene Exposure			
		- Metile hypoacid	<15	0-2.5 g/g creatine	
		-Mitiel hupuur creatine	<0.02	0-2.5 g/g	

4.4 Graphs

Normal spread of all measured chemical exposure and ventilation systems

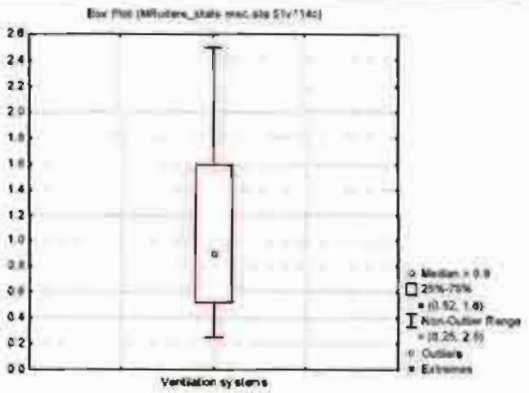

Figure 4.1: Ventilation system

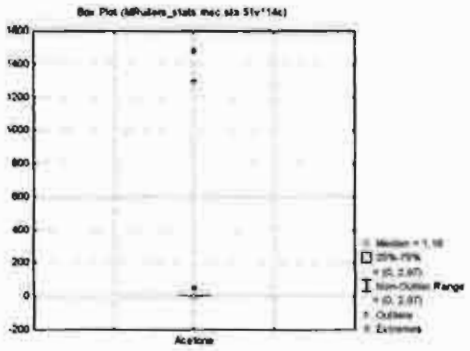

Figure 4.2: Acetone normal spread

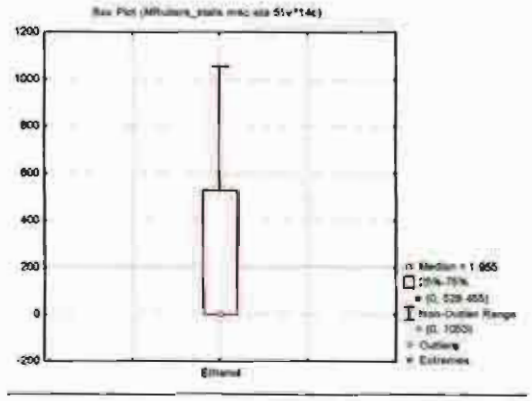

Figure 4.3: Ethanol normal spread

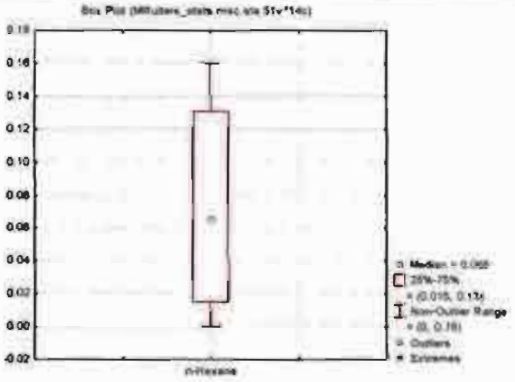

Figure 4.4: n-Hexane normal spread

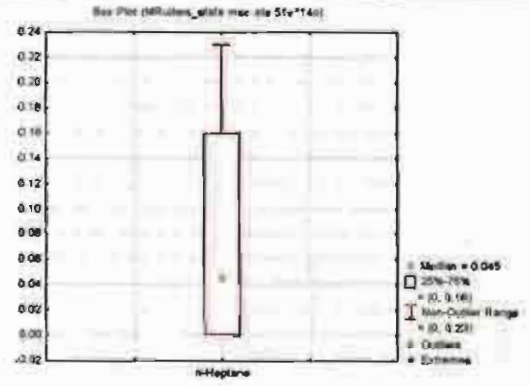

Figure 4.5: n-Heptane normal spread

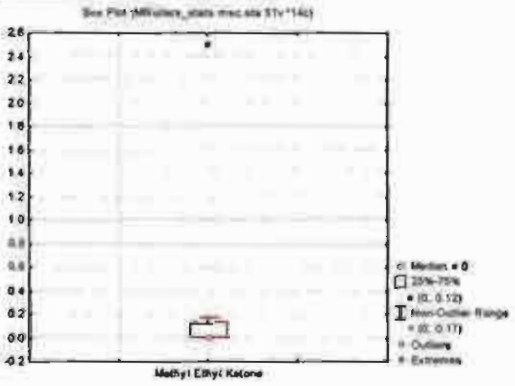

Figure 4.6: Methyl Ethyl Ketone normal spread

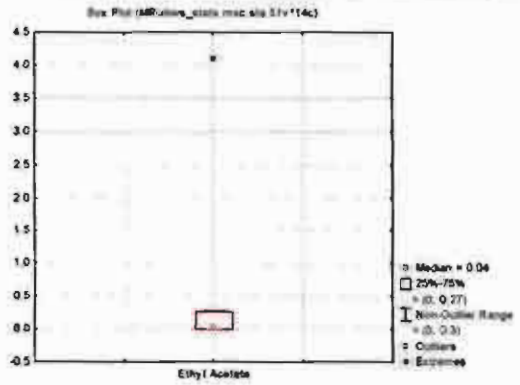

Figure 4.7.Ethyl Acetate normal spread

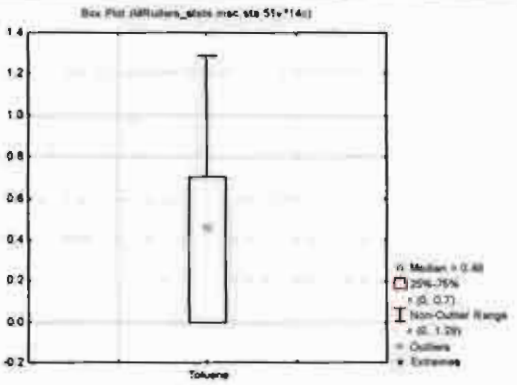

Figure 4.8. Toluene normal spread

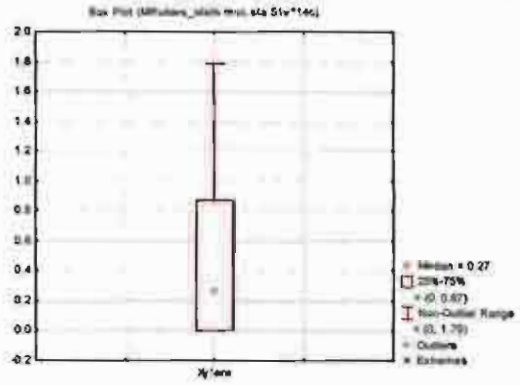

Figure 4.9.Xylene normal spread

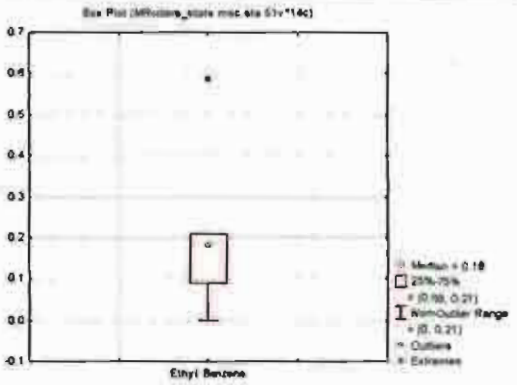

Figure 4.10.Ethyl Benzene normal spread

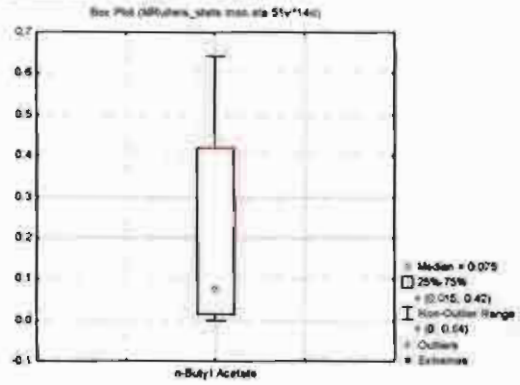

Figure 4.11. n-Butyl Acetate normal spread

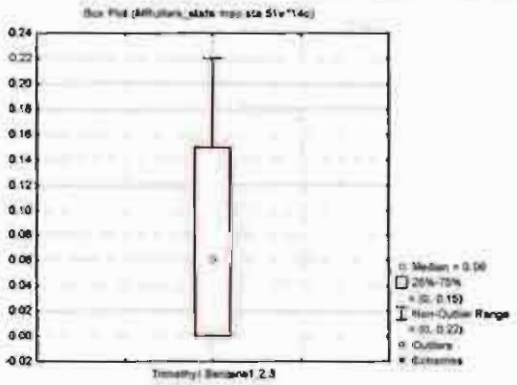

Figure 4.12.Trimethyl Benzene 1,2,3 normal spread

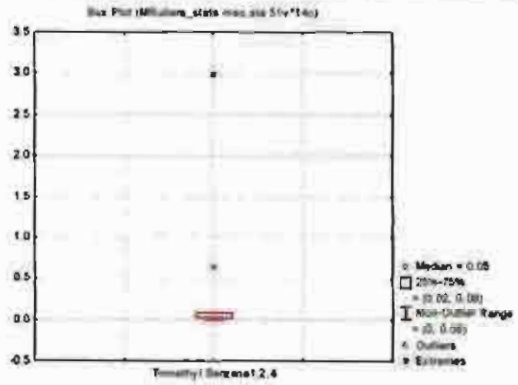

Figure 4.13. Trimethyl Benzene 1,2,4 normal spread

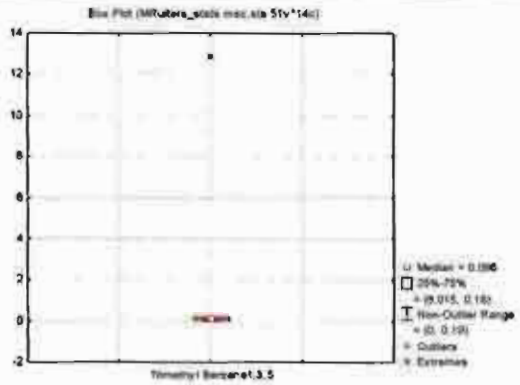

Figure 4.14. Trimethyl Benzene 1,3,5 normal spread

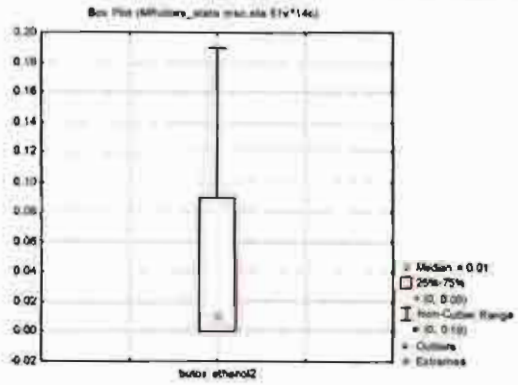

Figure 4.15. Butox ethanol normal spread

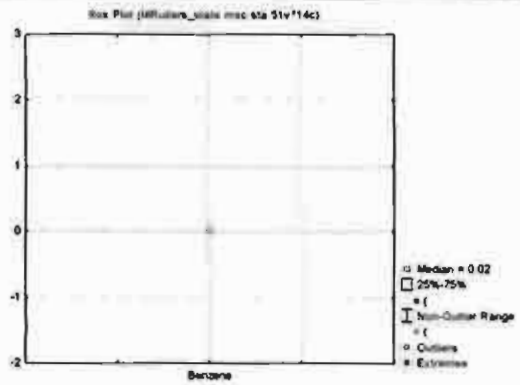

Figure 4.16. Benzene normal spread

Normal spread of all blood analysis done

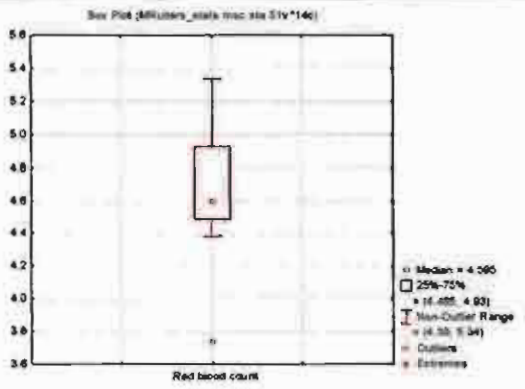

Figure 4.17.Red blood count normal spread

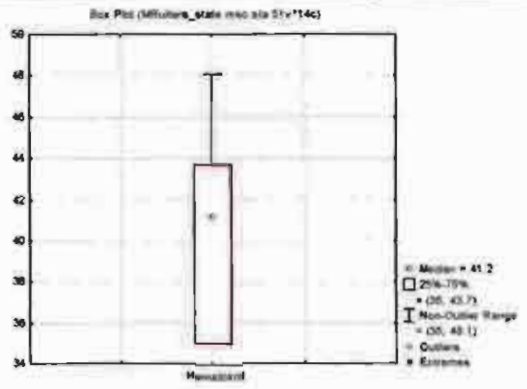

Figure 4.18. Hematokrit normal spread

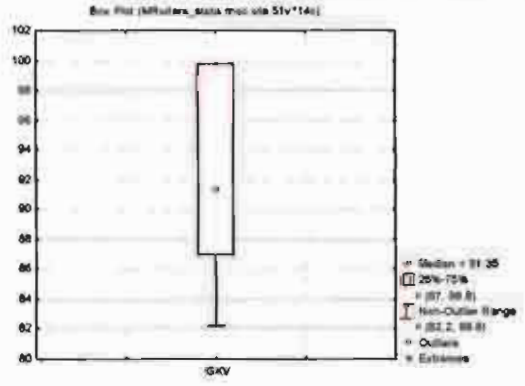

Figure 4.19.GKV normal spread

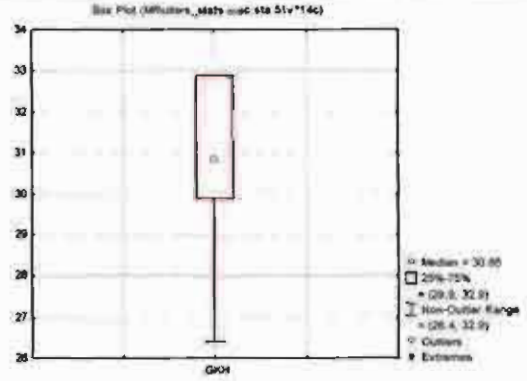

Figure 4.20. GKH normal spread

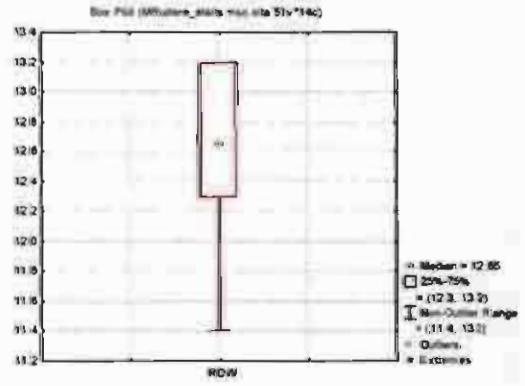

Figure 4.21. RDW normal spread

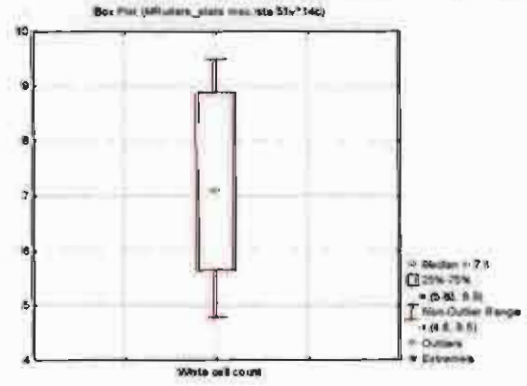

Figure 4.22. White cell count normal spread

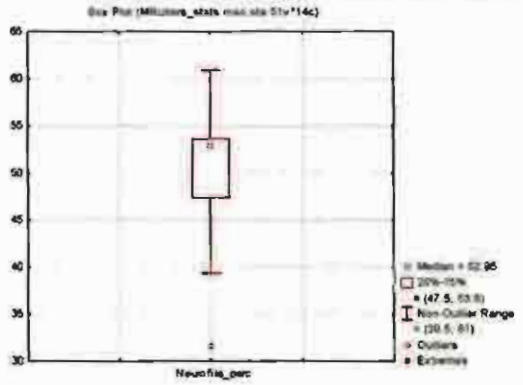

Figure 4.23. Neurofils perc normal spread

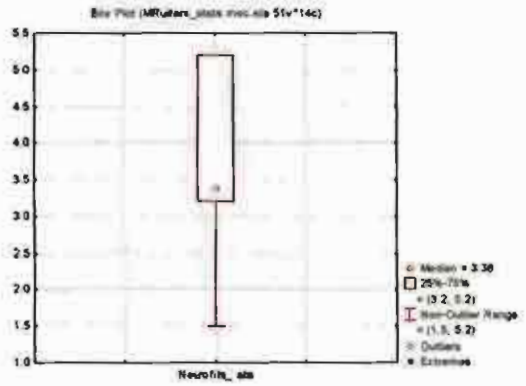

Figure 4.24. Neurofils abs normal spread

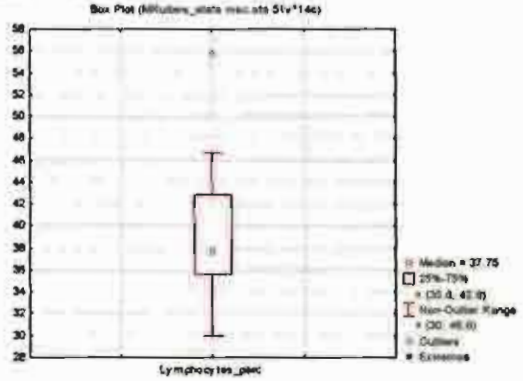

Figure 4.25. Lymphocytes perc normal spread

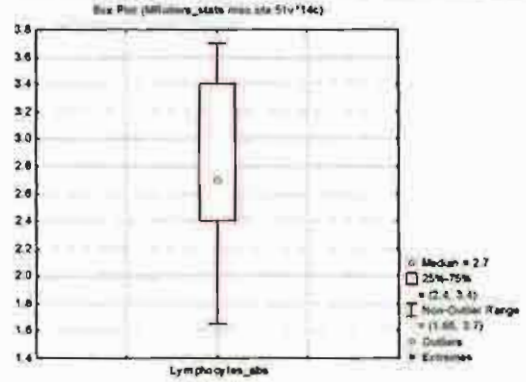

Figure 4.26. Lymphocytes abs normal spread

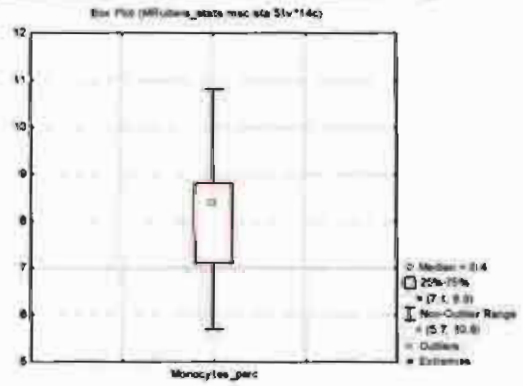

Figure 4.27. Monocytes perc normal spread

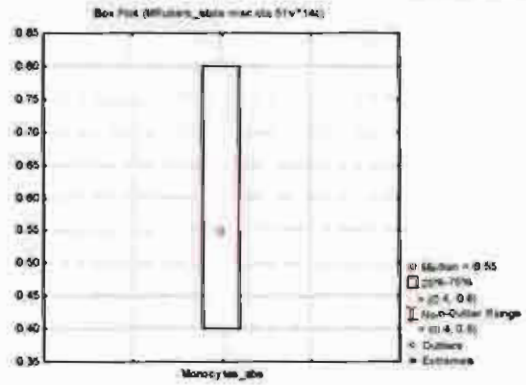

Figure 4.28. Monocytes abs normal spread

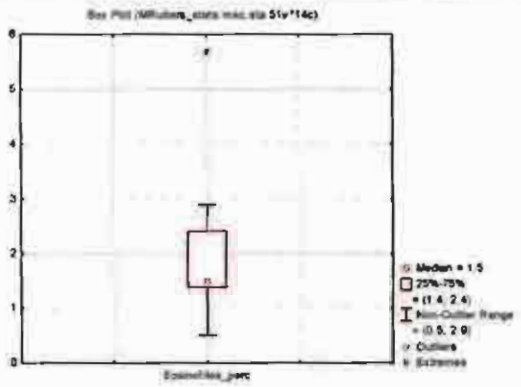

Figure 4.29. Eosinofiles perc normal spread

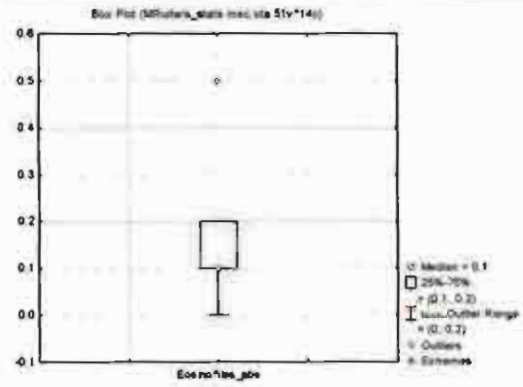

Figure 4.30. Eosinofiles abs normal spread

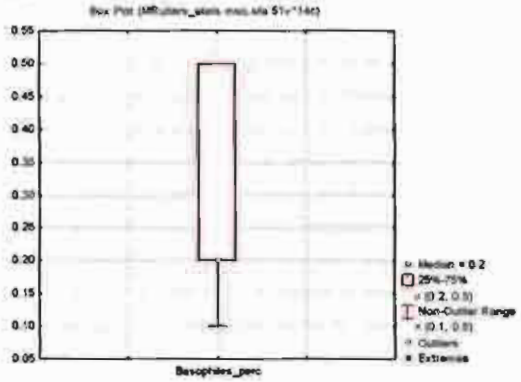

Figure 4.31. Basophiles perc normal spread

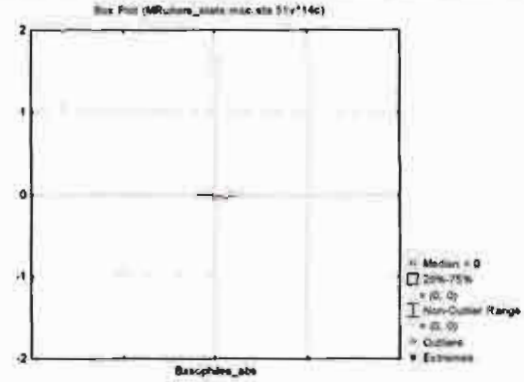

Figure 4.32. Basophiles abs normal spread

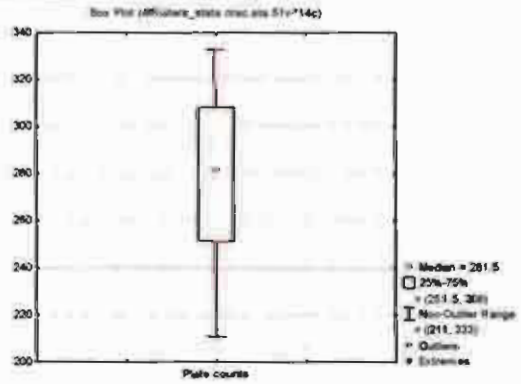

Figure 4.33. Plate counts normal spread

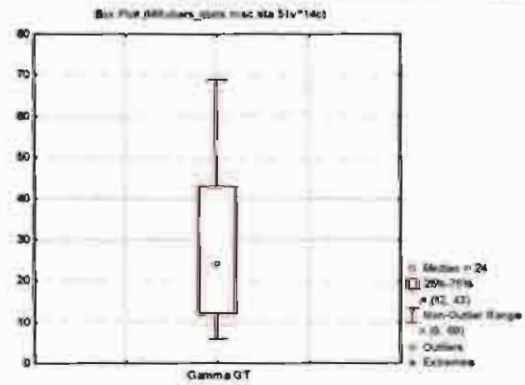

Figure 4.34. Gamma GT normal spread

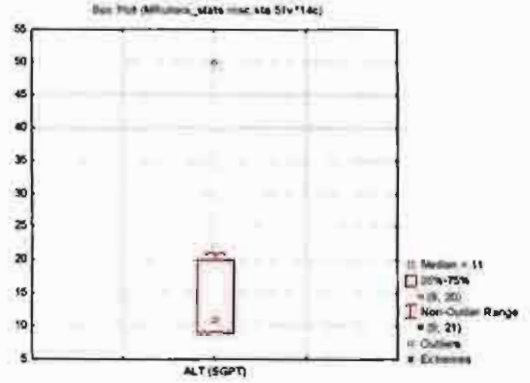

Figure 4.35. ALT (SGPT) normal spread

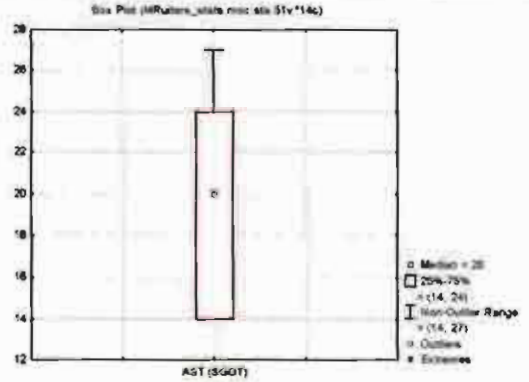

Figure 4.36. ALT (SGOT) normal spread

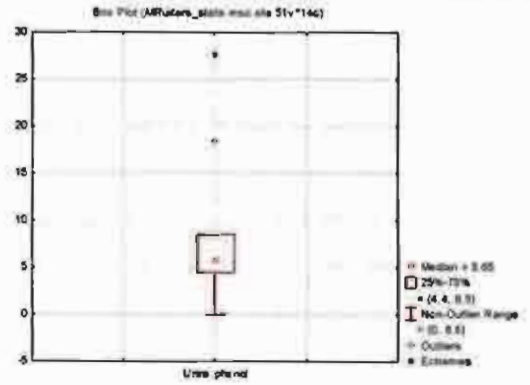

Figure 4.37. Urine Phenol normal spread

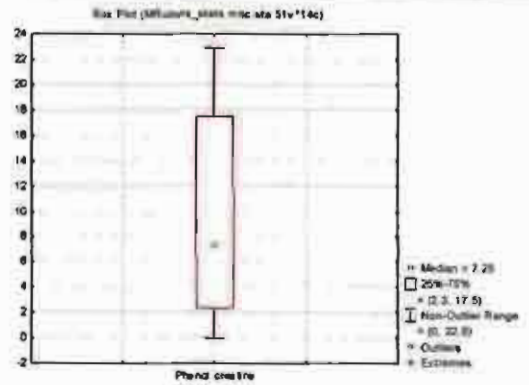

Figure 4.38. Phenol creatine normal spread

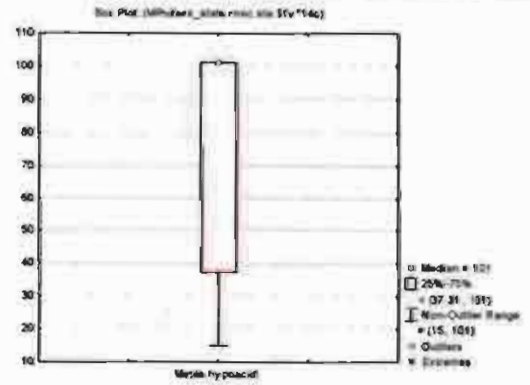

Figure 4.39. Metile hypoacid normal spread

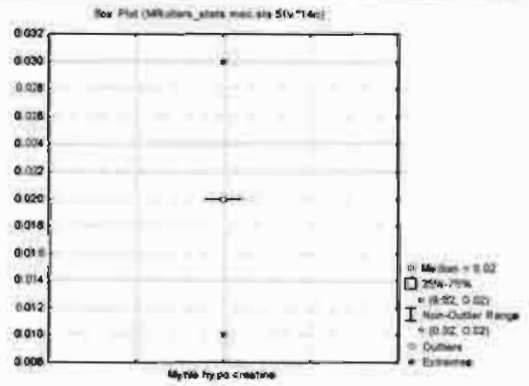

Figure 4.40. Mytile hypocreatine normal spread

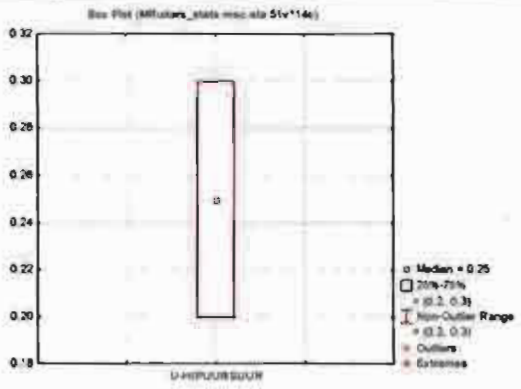

Figure 4.41. U –Hipuric acid normal spread

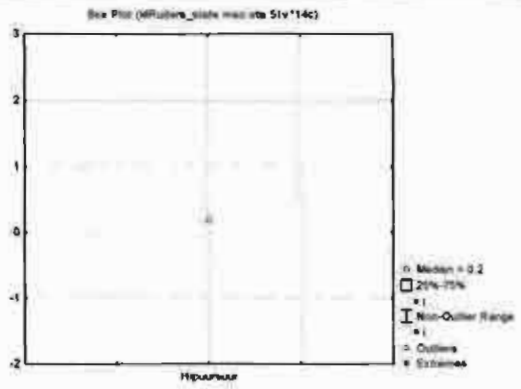

Figure 4.42. Hipuric acid normal spread

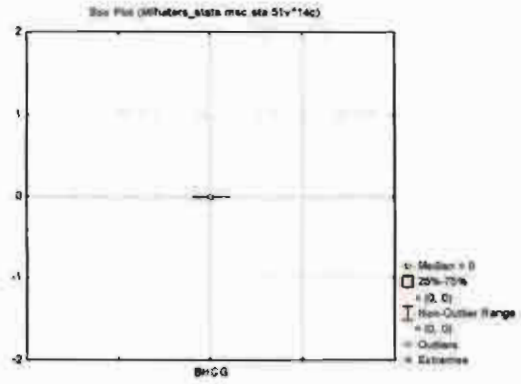

Figure 4.43. BHCG normal spread

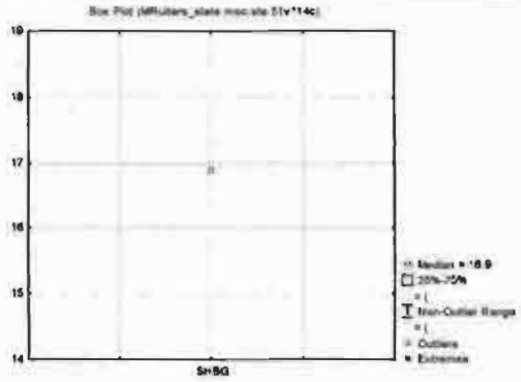

Figure 4.44. SHBG normal spread

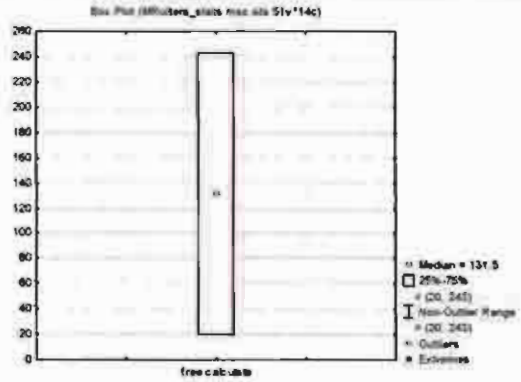

Figure 4.45. Free caltate normal spread

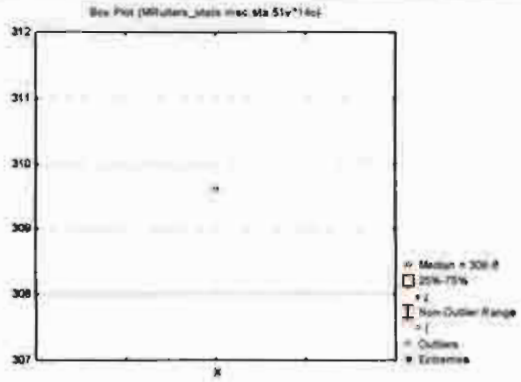

Figure 4.46. X normal spread

4.5 Results with deviations in the different areas

The following Figures contains results deviating from normal specifications and exposure limits and are revered to as abnormal.

Deviations from normal at Dunfilm

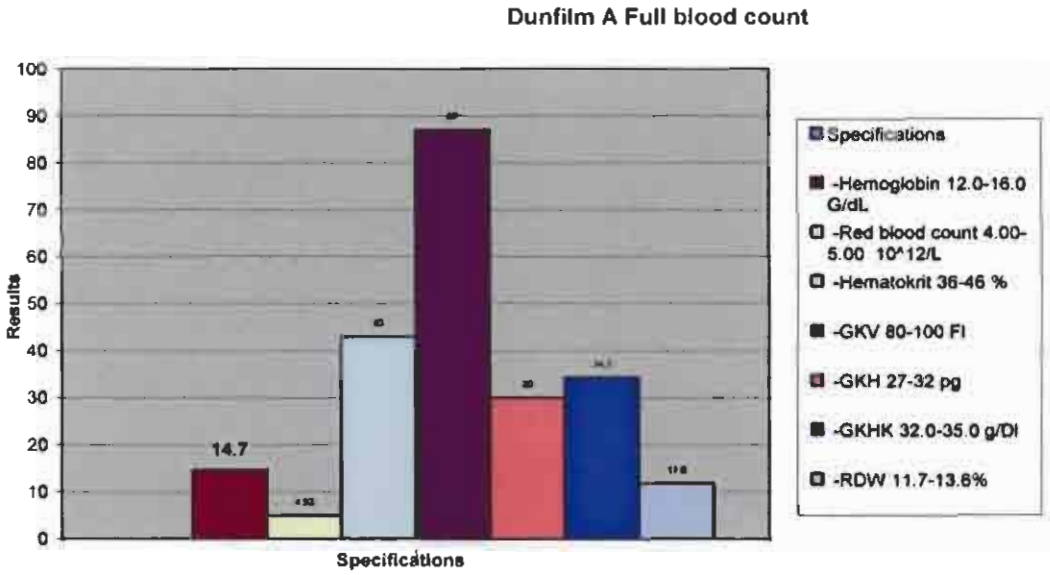

Figure 4.47. Dunfilm Full Blood Count results with deviations from normal specifications

Deviations from normal at FO1

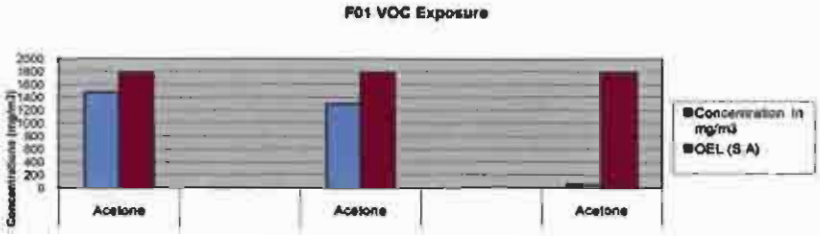

Figure 4.48.Abnormal acetone exposure in building FO1

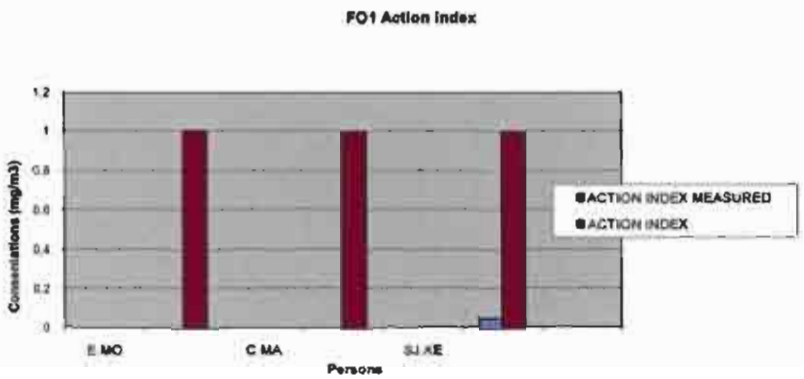

Figure 4.49. Abnormal acetone exposure with regard to the action index in building FO1

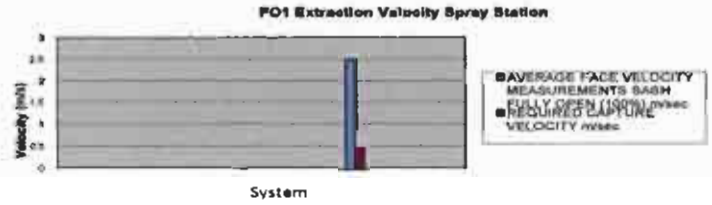

Figure 4.50. Abnormal Extraction Velocity results at building FO1 Painting Station

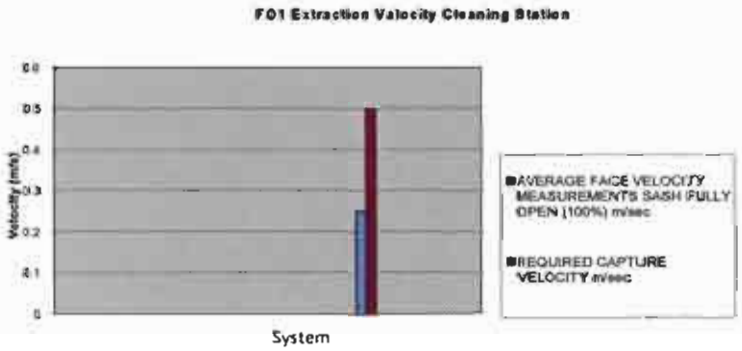

Figure 4.51. Abnormal Extraction Velocity results at building FO1 Cleaning Station

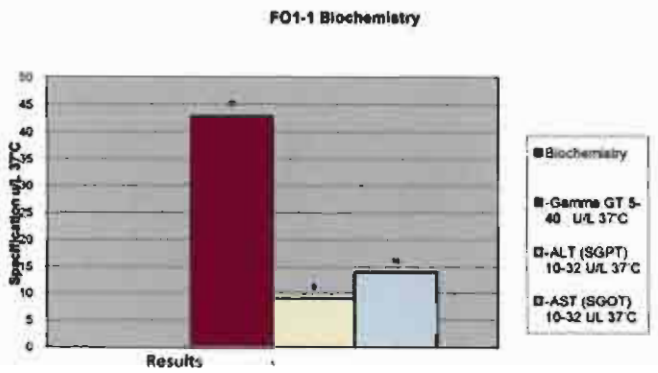

Figure 4.52. Abnormal Biochemistry results measured at building FO1

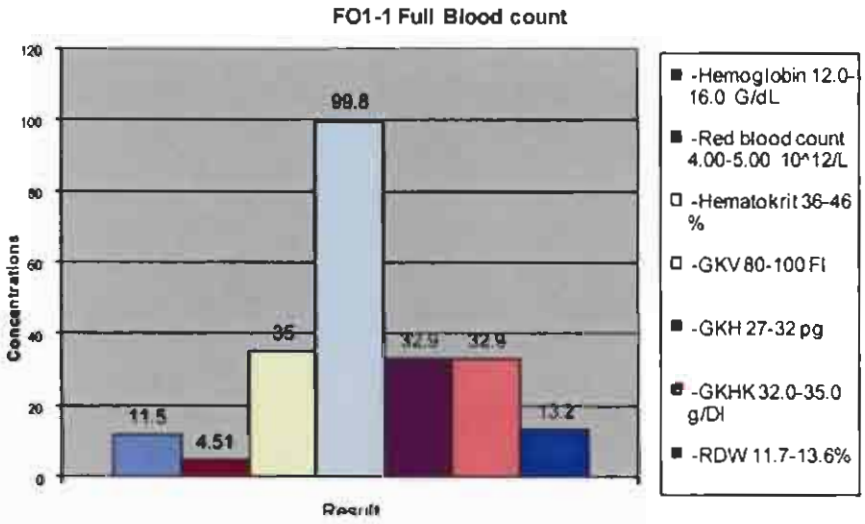

Figure 4.53. Abnormal Full Blood Count results measured at building FO1

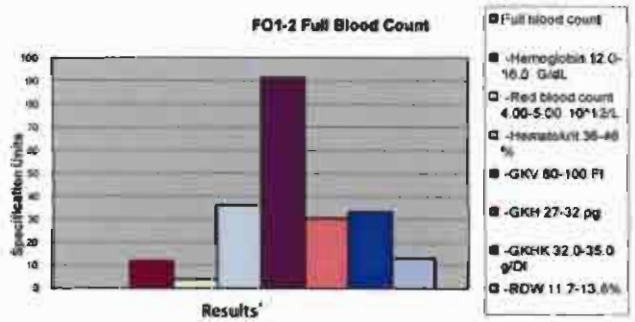

Figure 4.54. Abnormal Full Blood Count results measured at building FO1

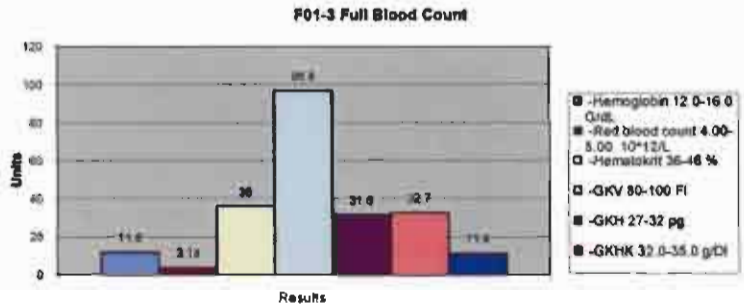

Figure 4.55. Abnormal Full Blood Count results measured at building FO1

Deviations from normal at Paint Shop BWO2

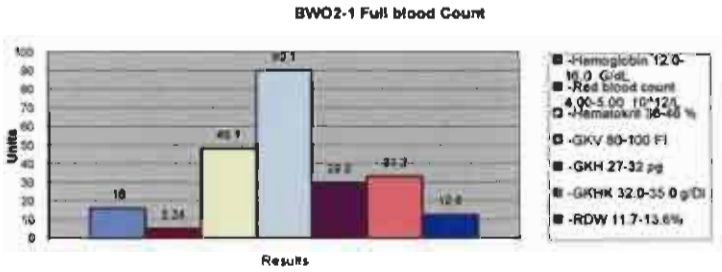

Figure 4.56. Abnormal Full Blood Count results measured at Paint shop BW02

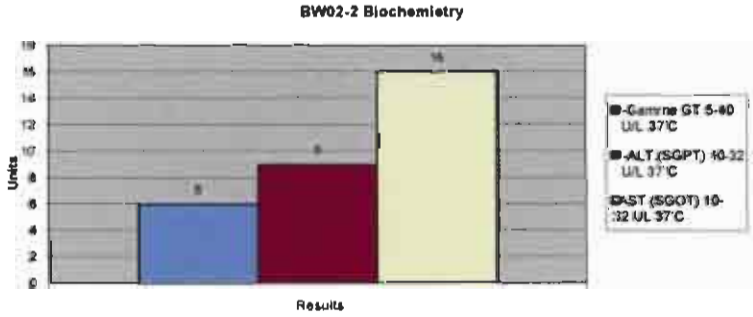

Figure 4.57. Abnormal biochemistry results measured at Paint shop BWO2-2

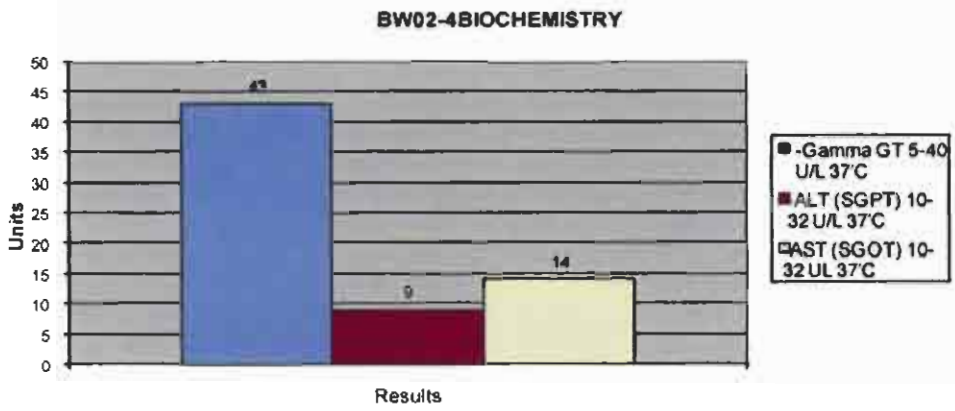

Figure 4.58. Abnormal biochemistry results measured at Paint shop BW02-4

Deviations from normal at Paint Shop Silk Screen and BWO7

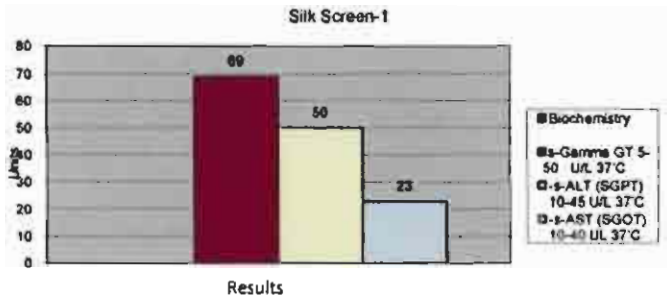

Figure 4.59. Abnormal Full Blood Count results measured at Paint shop Silk Screen-1

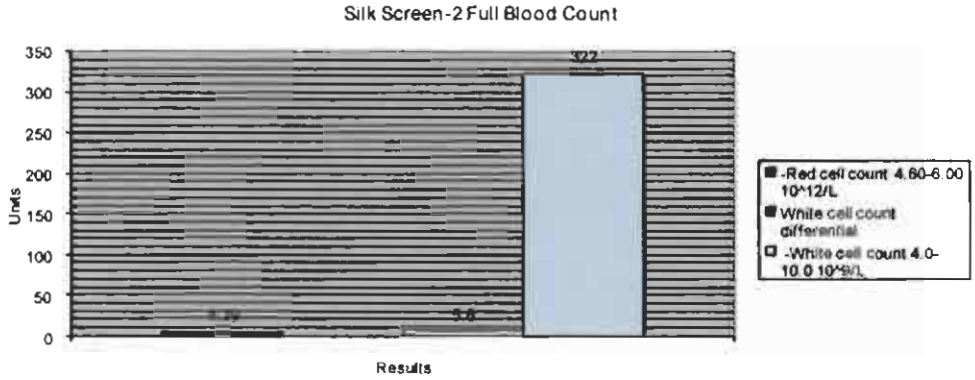

Figure 4.60. Abnormal Full Blood Count results measured at Paint shop Silk-Screen-2

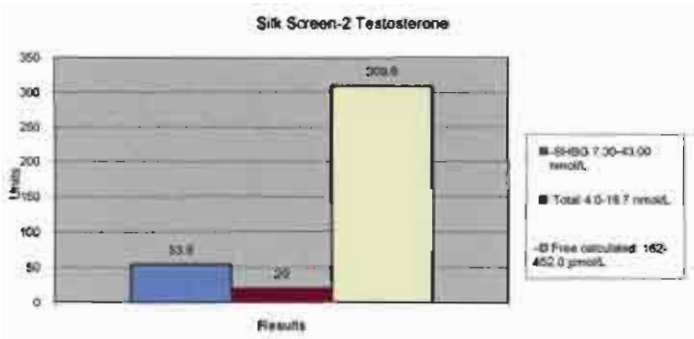

Figure 4.61. Abnormal Testosterone results measured at Paint shop Silk Screen-2

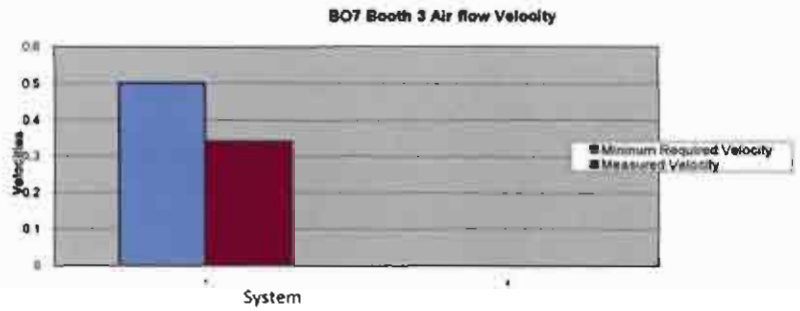

Figure 4.62. Abnormal Air Flow Velocity results measured at Paint shop Bwo7 Booth 3

Deviations from normal at building AO3

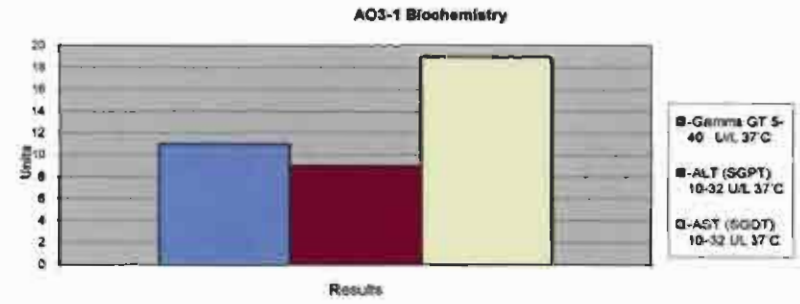

Figure 4.63. Abnormal Biochemistry results measured at building AO3-1

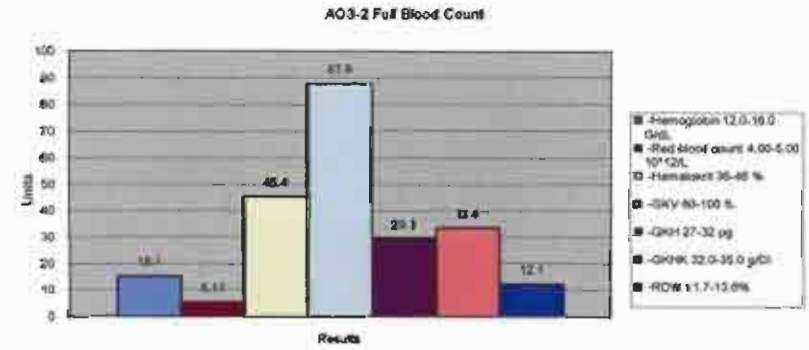

Figure 4.64.Abnormal Full Blood Count results measured at building AO3-2

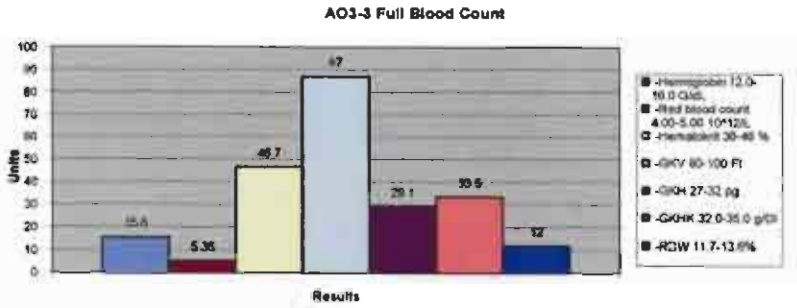

Figure 4.65.Abnormal Full Blood Count results measured at building AO3-1

Deviations from normal at Blue Building

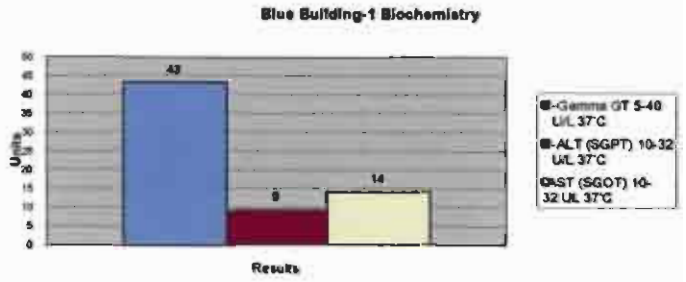

Figure 4.66. Abnormal Biochemistry results measured at Blue Building

4.6 Summary of results

The results obtained, as summarized in the table below are indicative of the conditions that prevailed during the sampling period, and could change with changes in processes and materials used as well as during possible failure of control systems, change in climatic conditions etc.

Table 4.20: Summary of all results

Area	Blood analysis results	VOC monitoring results	Air flow velocity results
Paint Shop			
BWO7	All within standard	All within standard	Within standard
BW02	Some not within standard	Within standard	Some not within standard
Screen Print	Not within standard	Within standard	Within standard
Dunfilm	All within standard	Some not within standard	Within standard
FO1	Not within standard	Some not within standard	Some not within standard
AO3	Some not within standard	All within standard	All within standard
Blue Building	Some not within standard	All within standard	Not applicable

Chapter 5: Discussion

The results indicate that the general health of the occupationally exposed workers is good, exposure to volatile organic compounds is low and the controls and PPE are in place and in a good condition. However, there are a few outliers and deviations from normal.

Statistical analyses done resulted in the following outliers and extremes:

In Figure 4.6 statistical analysis indicated that workers exposure to Methyl Ethyl Ketone (MEK) are at an extreme of 2.50 mg/m^3. This is far above the non outlier range of 0 to 0.17 mg/m^3. In all the different departments there were ten samples analyzed for exposure to MEK and 60% of the samples indicated that exposures were below the detectible limits (See samples G; H; E; F; D_1 and D_2). The other 40% of exposure ranked from 0.01 to 2.50 mg/m^3 (See sample I; F; A and B). Sample I taken at Dunfilm with a value of 2.50 mg/m^3 was the extreme, although this value is above the non outlier range, it is far below the OEL of 590 mg/m^3. Figure 4.7 indicated that exposure to ethyl acetate ranged from below detectable limits to 4.31 mg/m^3. Sample I taken from an occupationally exposed worker at Dunfilm is the extreme that is far above the non outlier range but lower than the OEL of 1400 mg/m^3.

Ethyl benzene values in Figure 4.10 ranged from 0.09 at Dunfilm to 0.59 at the Paint shop. The measure of 0.59 mg/m^3 is regarded as the extreme and is far below the OEL of 435 mg/m^3. All the statistical analyses of the chemicals listed in Figures 4.10, 4.13 and 4.14 for Ethyl Benzene; 1,2,4 Trimethyl Benzene and 1,3,5 Trimethyl Benzene respectively indicate that there are outlier and/or extremes, but all of the outliers and extremes are below the OEL's as was the case for MEK and Ethyl acetate and none of the above mentioned posed a significant occupational health risk.

Statistical analyses done on the biological samples results that were obtained from an accredited laboratory indicated that there were outliers and extremes in the following test results: Figure 4.17: Red Blood Count; Figure 4.25: Lymphocytes %; Figure 4.29: Eosinofiles %; Figure 4.30: Eosinofiles ABS; Figure 4.35: ALT SGTP; and Figure 4.37: Urine Phenol. Red cell blood count results ranged from 3.74 to 5.36 X 10^{12} where 3.74 X 10^{12} were below the outlier range and although 5.36 X 10^{12} is above the specifications of 4.00-5.00 X 10^{12}/L, it was (according to statistica) within the non outlier range of 4.39-5.34 X 10^{12}/L. Two of the RBC results that were below 4 X 10^{12}/L were of women at FO1 building in their child bearing age and two results that were above 5 X 10^{12}/L were of men that were working at the Paint shop and AO3. Women normally have a lower RBS count than men because of their menstrual cycle as seen in the results.

The Lymphocyte, eosinophiles % and ABS statistical results also indicated that there were outliers and extremes but all were within specifications. The ALT SGPT results in Figure 4.35 indicated six levels that were 9 U/L 37°C and one level of 50 U/L 37°C these levels are not within the normal specifications

range of 10-45 U/L. Worker CHA working at the Paint shop screen print had a ALT SGPT level of 50 U/L 37°C that can be seen in Figure 4.35. The urine Phenol results in Figure 4.37 ranged from 0 to 27.70 with 27.70 mg/g phenol for a worker CHA in the Paint shop being the outlier. The outlier value obtained is within specifications of 0-250 mg/g for occupationally exposed people.

At Dunfilm the results indicate that the concentration ethanol for worker A.P Sample G was much higher (1053 mg/m^3) than that of the other two workers (BDL and 3.91 mg/m^3). It did not exceed the OEL of 1900 mg/m^3 but the AQI of 1. This can be due to the fact that work that the three exposed workers are performing differ and worker A.P with the elevated ethanol levels is cleaning fine components which requires more intensive cleaning that the bigger components that the other two workers are cleaning.

Results at F01 (See Tables 4.2, 4.7, 4.8 and 4.14) indicate that there were deviations in all three of the measured variables. At FO1 the workers worked mainly with Acetone. Two of the three workers on which the activated charcoal tubes were placed do exactly the same work at the same workstations and the results were as follows: 1480 mg/m^3 for Sample K and 1280 mg/m^3 for Sample L. This is much higher than that of another worker Sample M 52.83 mg/m^3 doing almost the same work at another workstation. Although all three the air monitoring results were below the OEL of 1750 mg/m^3, there is reason for further investigation because the action index of one is exceeded and they are working under a hood with lower specified extraction velocities. The air velocities at the two hoods in FO1 where the three workers perform their duties were measured. The results indicated that the hood A1 Table 4.7 where two workers work had an average velocity of 0.25 m/s and thus lower than the minimum required average velocity of 0.5 m/s. The results for hood A8.1 (Table 4.7) were above the minimum required velocity. Hood A1 is near a doorway and an office and this causes heavy traffic where operations are being performed. This can have an effect on the capture velocity. The biological monitoring results showed some deviations in all three the subject's results. It is unlikely that this can be due to occupational exposure since the air monitoring results were within specification. The deviation in the biological monitoring results can also be due to personal reasons for instance the one elevated Gamma GT and lowered ALT can be due to alcohol consumption or a hobby that may include exposure to VOC's. The lowered red blood cell counts can also be due to menstruation because all three the subjects are females and of child bearing age.

At the Paint shop which is divided into two areas, VOC's are used daily in different processes and all workers in this area are men (see results in Tables 4.3, 4.4, 4.9, 4.10, 4.9, 4.15, 4.16 and 4.17). At BWO7 where small components are spraypainted, all three the variables that were measured were within specifications. BWO2 where the big components are spraypainted and all the silk screening activities are performed, a few deviations from normal were found. Air monitoring results indicated that the amount of VOC's in the workers' breathing zones were all below the OEL and the action index. The capture velocity results in the spraypainting booths indicated that there were deviations in two of the booths. Booth 3's capture velocity was 0.34 m/s and booth 5's 0.43 m/s. This was due to a blockage of the filters. The biological monitoring was difficult to interpret because some subjects had low and others had high

red and white blood cell counts, testosterone levels and liver functions. This may be due to non-occupational exposure or certain medical conditions.

The same study was also done at AO3. The results obtained from this building can be seen in Tables 4.12 and 4.18. As is the case with some of the other buildings, all the measured variables were within specification accept the biological monitoring results. The abnormal biological monitoring results as mentioned earlier can be due to non-occupational exposure and/or health related problems. This conclusion was drawn because only the red blood count of two workers were elevated but the other results were within the prescribed limits.

At the Blue building (Tables 4.5 and 4.19) the air monitoring results were within specifications. The spraypainting at this area was done in an open area where there is good natural ventilation. Natural ventilation is the most widely-used method of preventing dangerous concentrations of atmospheric contaminants developing in workrooms, factories and plants. The biological monitoring results indicated the subject had high Gamma GT levels and low ALT levels. This can be due to personal or non–occupation exposure because the other variables were within standard.

VOC's can be absorbed through the skin and there is not a method available to measure the amount of substance absorbed through the skin. It is important that the correct PPE be used and that personal hygiene be emphasized since it can affect the biological monitoring results.

Because biological and personal monitoring are expensive, only the most exposed workers were chosen according to OESSM and because the results were not enough, no reasonable statistical correlations could be made between the buildings and the subjects.

Chapter 6: Conclusions and Recommendations

In conclusion, results obtained by this investigation indicated that although not all the engineering controls, personal and biological monitoring results were within normal specifications and statutory limits, the general health of the exposed subjects is good and not compromised due to occupational exposure to VOC's. The hypothesis was proved to be correct.

All the personal monitoring results were within specification but results at FO1 and Dunfilm were above the AQI. It is recommended that the exposure to VOC's in the Dunfilm polish room as well as FO1 cleaning bay should be kept as low as possible to avoid the exposure levels to increasing to above the OEL. Therefore, it is recommended that the hierarchy of control always be followed to reduce solvent exposures to as low as reasonably practicable. It is also important that the use of the correct PPE be checked regularly to avoid possible skin absorption. Worker education programmes should be instituted to inform workers about the hazards of exposure to organic solvents and to provide information on safe handling practices.

The capture velocity of the fume hoods and spraypaint boots were mostly within specifications. At FO1 hood A1 (Table 4.7) where two people were cleaning components under one very small extraction hood, the capture velocity was lower than specifications. It is recommended that a bigger booth be installed with a higher capture velocity at another location away from heavy traffic aisles, doorways and offices. In the interim the door nearest to the work station can be kept open to provide natural ventilation and the maintenance department can reset the propellers/fans to increase the capture velocity before a bigger hood that can accommodate two workers can be installed.

It is recommended that at the Paint shop the blocked filters be removed and cleaned to increase the capture velocity. It is also important that all the extraction hoods and booths be inspected monthly and that a maintenance schedules be drawn up for all the extraction systems of the factory. The phenol and the ALT SGPT results of Worker CARH were high and the working conditions and other methods of VOC exposure should be evaluated to determine why the levels were higher than the co-worker that is doing almost the same work.

The biological monitoring results in all the areas (Tables 4.13 to 4.19) indicate that the results are inconclusive because the biological indexes did not correlate with the personal monitoring and engineering control measure results. This can be due to the fact that VOC's can be absorbed through the skin and there is no method available to measure the amount of solvent absorbed via the skin. The biological monitoring results that were out of specifications can also be due to non occupational exposure outside the workplace, personal medical conditions or alcohol consumption where liver function problems were experienced. It is thus recommended that solvent exposure and medical condition questionnaires be

drawn up to determine non-occupational exposure, alcohol consumption or medical conditions that can influence the biological monitoring results. These questionnaires can be used with the annual biological monitoring of VOC workers and will help the Occupational Medical Practitioner to interpret the biological monitoring results better.

Evaluating a patient for any chemical dependency and/or exposure laboratory studies, including urine testing, should never be used alone but rather to support findings obtained by careful history taking and a thorough physical examination. Workplace and biological monitoring programmes should always be viewed together because both have pitfalls but complement each other.

It is also important to realize that only rarely does an operation release contaminants into the workroom air at a fairly constant rate. The concentration found in a single sample may have been too high or too low due to a number of factors and if the sample had been collected at another time, the results could very well be considerably different. Several dozen samples may be necessary to define accurately a daily time-weighted average exposure for a worker who performs a number of tasks during a shift and this can also be the reason that there is a variation in the results.

General recommendations for air supply distribution and the selection of the hoods' face velocity and work practices for hoods are as follows:

For typical operations at a fume hood, the worker stands at the face of the hood and manipulates the apparatus in the hood. The in-draft at the hood face creates eddy currents around the worker's body which can drag contaminants in the hood back to the body and up to the breathing zone. The higher the face velocity, the greater the eddy currents. For this reason, higher face velocities do not necessarily result in greater protection against exposure.

Room air currents have a large effect on the performance of the hood. Therefore, the design of the room air supply distribution system is as important in securing good hood performance as the face velocity of the hood. Caplan and Knutson (2006) reported that perforated ceiling panels provide a better supply system than grilles or ceiling diffusers, since the system design criteria are simpler and easier to apply, and precise adjustment of the fixture is not required. They also indicated that an increased hood face velocity may be self-defeating because the increased air volume handled through the room makes the low-velocity distribution of supply air more difficult.

The interaction of supply air distribution and hood face velocity makes any blanket specification of hood face velocity inappropriate. Higher hood face velocities will be a waste of energy and may provide no better or even poorer worker protection. The performance test developed by Caplan and Knutson (2006) may be used as a specification. The specified performance should be required of both the hood manufacturer and the designer of the room air supply system.

For more ordinary exposures, a properly designed hood in a properly ventilated room can provide adequate protection. However, certain work practices are necessary in order for the hood to perform capably.

The following work practices are generally required:

- Conduct all operations, which may generate air contaminants at or above the appropriate OEL inside a hood
- Keep all apparatus at least 15cm back from the face of the hood. A line on the bench surface is a good reminder
- Do not put your head in the hood when contaminants are being generated
- Do not use the hood as a waste disposal mechanism except for very small quantities of volatile materials
- Keep the hood sash closed as much as possible
- Minimize foot traffic past the face of the hood
- Keep doors closed
- Provide adequate maintenance for the hood exhaust system and the building supply system. Use static pressure gauges on the hood throat, across any filters in the exhaust system, or other appropriate indicators to insure that exhaust flow is appropriate.

This study emphasized the importance of work methods or conditions, maintenance of engineering control measures, the use of the correct PPE and the importance of occupational health (biological monitoring) and hygiene results to be used together. These factors play an important role in protecting the occupational exposed workers' health and to allow the worker to work in a healthy work environment.

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ADDENDUM: STATISTICAL DATA

Table 1: Statistical Descriptives

Chemical	Valid N	Mean	Confidence - 95.000%	Confidence +95.000%	Median	Minimum	Maximum	Lower Quartile	Upper Quartile	Std.Dev.	Skewness	Kurtosis
Acetone	14	202.9236	-88.05	493.894	1.1600	0.0000	1480.000	0.0000	2.9700	503.9478	2.31536	3.97033
Ethanol	4	264.2275	-572.52	1100.975	1.9550	0.0000	1053.000	0.0000	528.4550	525.8516	1.99993	3.99975
n-Hexane	4	0.0725	-0.04	0.187	0.0650	0.0000	0.160	0.0150	0.1300	0.0718	0.41905	-2.21525
n-Heptane	4	0.0800	-0.09	0.253	0.0450	0.0000	0.230	0.0000	0.1600	0.1086	1.22327	0.58072
Methyl Ethyl Ketone	10	0.2800	-0.28	0.840	0.0000	0.0000	2.500	0.0000	0.1200	0.7824	3.12726	9.83230
Ethyl Acetate	10	0.4890	-0.43	1.405	0.0400	0.0000	4.120	0.0000	0.2700	1.2808	3.11724	9.78581
Toluene	11	0.4573	0.14	0.779	0.4600	0.0000	1.290	0.0000	0.7000	0.4789	0.75285	-0.64527
Xylene	11	0.4973	0.12	0.876	0.2700	0.0000	1.790	0.0000	0.8700	0.5636	1.23686	1.45732
Ethyl Benzene	9	0.1911	0.06	0.320	0.1800	0.0000	0.590	0.0900	0.2100	0.1674	1.82418	4.66334
n-Butyl Acetate	8	0.2075	-0.02	0.433	0.0750	0.0000	0.640	0.0150	0.4200	0.2703	1.18165	-0.44154
Trimethyl Benzene1,2,3	9	0.0811	0.01	0.149	0.0600	0.0000	0.220	0.0000	0.1500	0.0881	0.66944	-1.28780
Trimethyl Benzene1,2,4	9	0.4322	-0.32	1.186	0.0500	0.0000	2.990	0.0200	0.0800	0.9801	2.78342	7.90660
Trimethyl Benzene1,3,5	8	1.6800	-2.11	5.471	0.0950	0.0000	12.900	0.0150	0.1600	4.5340	2.82724	7.99494
butox ethanol2	7	0.0529	-0.01	0.119	0.0100	0.0000	0.190	0.0000	0.0900	0.0720	1.32838	1.23396
Benzene	1	0.0200			0.0200	0.0200	0.020	0.0200	0.0200			
Hemoglobin	10	13.4100	12.16	14.661	13.5000	11.5000	16.000	11.5000	14.8000	1.7483	0.10146	-1.74821
Red blood count	12	4.6675	4.40	4.933	4.5950	3.7400	5.340	4.4850	4.9300	0.4186	-0.54679	1.24564
Hematokrit	10	40.3800	36.88	43.880	41.2000	35.0000	48.100	35.0000	43.7000	4.8930	0.13651	-1.53512
GKV	10	92.3400	87.70	96.981	91.3500	82.2000	99.800	87.0000	99.8000	6.4875	-0.12498	-1.41699
GKH	10	30.6700	29.16	32.184	30.8500	26.4000	32.900	29.9000	32.9000	2.1166	-0.82313	0.38534
GKHK	10	33.1900	32.75	33.634	33.0500	32.2000	34.300	32.9000	33.5000	0.6208	0.44637	0.06404
RDW	10	12.5600	12.13	12.991	12.6500	11.4000	13.200	12.3000	13.2000	0.6022	-0.77944	0.06295
White cell count	12	7.2417	6.15	8.337	7.1000	4.8000	9.500	5.6500	8.9000	1.7244	0.15104	-1.56677
Neurofils_perc	10	50.3900	43.96	56.822	52.9500	31.6000	61.000	47.5000	53.6000	8.9908	-1.07899	1.06622
Neurofils_ abs	10	3.7560	2.86	4.651	3.3800	1.5000	5.200	3.2000	5.2000	1.2505	-0.24691	-0.68642
Lymphocytes_perc	10	39.4000	33.89	44.909	37.7500	30.0000	55.800	35.6000	42.8000	7.7012	0.99751	1.12831
Lymphocytes_abs	10	2.8250	2.37	3.280	2.7000	1.6500	3.700	2.4000	3.4000	0.6356	-0.31029	-0.46573
Monocytes_perc	10	8.0900	7.05	9.132	8.4000	5.7000	10.800	7.1000	8.8000	1.4571	0.16683	0.17935
Monocytes_abs	10	0.5800	0.46	0.701	0.5500	0.4000	0.800	0.4000	0.8000	0.1687	0.38911	-1.57183
Eosinofiles_perc	9	2.0444	0.86	3.229	1.5000	0.5000	5.700	1.4000	2.4000	1.5412	1.92929	4.27998
Eosinofiles_abs	9	0.1556	0.05	0.265	0.1000	0.0000	0.500	0.1000	0.2000	0.1424	2.02703	5.01547

Chemical	Valid N	Mean	Confidence - 95.000%	Confidence +95.000%	Median	Minimum	Maximum	Lower Quartile	Upper Quartile	Std.Dev.	Skewness	Kurtosis
Basophiles_perc	9	0.3111	0.19	0.435	0.2000	0.1000	0.500	0.2000	0.5000	0.1616	0.22948	-2.02664
Basophiles_abs	9	0.0000			0.0000	0.0000	0.000	0.0000	0.0000	0.0000		
Plate counts	12	277.9167	252.63	303.205	281.5000	211.0000	333.000	251.5000	308.0000	39.8005	-0.49871	-0.80188
Gamma GT	11	28.7273	16.07	41.382	24.0000	6.0000	69.000	12.0000	43.0000	18.8366	0.91852	0.51692
ALT (SGPT)	11	16.2727	8.18	24.368	11.0000	9.0000	50.000	9.0000	20.0000	12.0507	2.56018	7.20035
AST (SGOT)	11	19.6364	16.30	22.972	20.0000	14.0000	27.000	14.0000	24.0000	4.9653	0.07936	-1.82501
Urine phenol	12	8.2167	3.35	13.081	5.6500	0.0000	27.700	4.4000	8.5000	7.6559	1.81252	3.38342
Phenol creatine	12	9.8417	4.67	15.018	7.2500	0.0000	22.900	2.3000	17.5000	8.1472	0.29433	-1.74937
Metile hypoacid	11	80.0873	55.77	104.401	101.0000	15.0000	101.000	37.3100	101.0000	36.1908	-1.26555	-0.39360
Mytile hypo creatine	11	0.0200	0.02	0.023	0.0200	0.0100	0.030	0.0200	0.0200	0.0045	0.00000	5.00000
U-HIPUURSUUR	2	0.2500	-0.39	0.885	0.2500	0.2000	0.300	0.2000	0.3000	0.0707		
Hipuursuur	1	0.2000			0.2000	0.2000	0.200	0.2000	0.2000			
BHCG	2	0.0000			0.0000	0.0000	0.000	0.0000	0.0000	0.0000		
SHBG	1	16.9000			16.9000	16.9000	16.900	16.9000	16.9000			
Totaal	2	31.4500	-253.80	316.704	31.4500	9.0000	53.900	9.0000	53.9000	31.7491		
free calculate	2	131.5000	-1285.24	1548.242	131.5000	20.0000	243.000	20.0000	243.0000	157.6848		
X	1	309.6000			309.6000	309.6000	309.600	309.6000	309.6000			
Ventilation systems	9	1.0900	0.49	1.689	0.9000	0.2500	2.500	0.5200	1.6000	0.7792	0.82243	-0.45941

Table 2: Statistical correlations

Chemical	Hemo-globin	Red blood count	Hema-tokrit	GKV	GKH	GKHK	RDW	White cell count	Neurofils		Lympho-cytes		Monocytes		Eosinofiles		Basophiles	
									perc	abs	perc	abs	perc	abs	perc	abs	perc	abs
Acetone	-.3325	-.1075	-.3429	.0592	.0366	-.0956	.2554	.3807	.3759	.4874	-.4000	.0877	-.0298	.3860	-.2846	-.2186	.1645	--
	N=10	N=12	N=10	N=10	N=10	N=10	N=10	N=12	N=10	N=10	N=10	N=10	N=10	N=10	N=9	N=9	N=9	N=9
	p=.348	p=.739	p=.332	p=.871	p=.920	p=.793	p=.476	p=.222	p=.284	p=.153	p=.252	p=.810	p=.935	p=.271	p=.458	p=.572	p=.672	p= ---
Ethanol	-1.0000	--	-1.0000	-1.0000	-1.0000	1.0000	-1.0000	-1.0000	1.0000	1.0000	-1.0000	-1.0000	1.0000	1.0000	--	--	--	--
	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=1	N=1	N=1	N=1
	p= ---	p= ---	p= ---	p= ---		p= ---	p= ---	p= ---	p= ---			p= ---	p= ---		p= ---	p= ---	p= ---	p= ---
n-Hexane	-1.0000	--	-1.0000	-1.0000	-1.0000	1.0000	-1.0000	-1.0000	1.0000	1.0000	-1.0000	-1.0000	1.0000	1.0000	--	--	--	--
	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=1	N=1	N=1	N=1
		p= ---	p= ---	p= ---		p= ---	p= ---	p= ---	p= ---		p= ---			p= ---	p= ---	p= ---	p= ---	p= ---
n-Heptane	-1.0000	--	-1.0000	-1.0000	-1.0000	1.0000	-1.0000	-1.0000	1.0000	1.0000	-1.0000	-1.0000	1.0000	1.0000	--	--	--	--
	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=2	N=1	N=1	N=1	N=1
	p= ---	p= ---	p= ---	p= ---		p= ---	p= ---	p= ---	p= ---		p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---
Methyl Ethyl Ketone	.1178	.1642	.0127	-.3201	-.0850	.5801	-.7848	-.2900	.6059	.0414	-.6343	-.6955	.1689	-.1175	.9421	.9134	-.6492	--
	N=6	N=8	N=6	N=6	N=6	N=6	N=6	N=8	N=6	N=6	N=6	N=6	N=6	N=6	N=5	N=5	N=5	N=5
	p=.824	p=.698	p=.981	p=.536	p=.873	p=.227	p=.064	p=.486	p=.202	p=.938	p=.176	p=.125	p=.749	p=.825	p=.017	p=.030	p=.236	p= ---
Ethyl Acetate	.1328	.1733	.0265	-.3162	-.0783	.5901	-.7880	-.3021	.6090	.0328	-.6347	-.7093	.1565	-.1350	.7957	.7462	-.7883	--
	N=6	N=8	N=6	N=6	N=6	N=6	N=6	N=8	N=6	N=6	N=6	N=6	N=6	N=6	N=5	N=5	N=5	N=5
	p=.802	p=.681	p=.960	p=.542	p=.883	p=.218	p=.063	p=.467	p=.199	p=.951	p=.176	p=.114	p=.767	p=.799	p=.107	p=.148	p=.113	p= ---
Toluene	-.8344	-.3869	-.8252	.7860	.6371	-.4138	.8334	.5113	-.1040	.5470	.0283	.6883	.2106	.7946	-.0990	-.0901	.7592	--
	N=7	N=9	N=7	N=7	N=7	N=7	N=7	N=9	N=7	N=7	N=7	N=7	N=7	N=7	N=6	N=6	N=6	N=6
	p=.020	p=.304	p=.022	p=.036	p=.124	p=.356	p=.020	p=.159	p=.824	p=.204	p=.952	p=.087	p=.650	p=.033	p=.852	p=.865	p=.080	p= ---
Xylene	-.1924	-.0428	-.1909	.4084	.3542	-.1245	.4706	.0522	-.3001	-.0449	.2910	.2493	-.1820	.0905	.1857	.1312	-.1180	--
	N=7	N=9	N=7	N=7	N=7	N=7	N=7	N=9	N=7	N=7	N=7	N=7	N=7	N=7	N=6	N=6	N=6	N=6
	p=.679	p=.913	p=.682	p=.363	p=.436	p=.790	p=.287	p=.894	p=.513	p=.924	p=.527	p=.590	p=.696	p=.847	p=.725	p=.804	p=.824	p= ---
Ethyl Benzene	.4408	.3600	.4506	.1197	.1445	.1140	.1068	-.1189	-.0115	-.2383	.0902	-.2672	-.7051	-.5331	.0256	-.0660	-.6895	--
	N=6	N=7	N=6	N=6	N=6	N=6	N=6	N=7	N=6	N=6	N=6	N=6	N=6	N=6	N=5	N=5	N=5	N=5
	p=.382	p=.428	p=.370	p=.821	p=.785	p=.830	p=.840	p=.800	p=.983	p=.649	p=.865	p=.609	p=.118	p=.276	p=.967	p=.916	p=.198	p= ---

Chemical	Hemo-globin	Red blood count	Hema-tokrit	GKV	GKH	GKHK	RDW	White cell count	Neurofils		Lympho-cytes		Monocytes		Eosinofiles		Basophiles	
									perc	abs	perc	abs	perc	abs	perc	abs	perc	abs
n-Butyl Acetate	.4425	.2508	.3361	.1352	.3260	.5961	-.4108	-.2627	.7141	.0486	-.6358	-.8338	-.4452	-.5105	-.2207	-.3156	-.5056	--
	N=5	N=6	N=5	N=5	N=5	N=5	N=5	N=6	N=5	N=5	N=5	N=5	N=5	N=5	N=4	N=4	N=4	N=4
	p=.456	p=.632	p=.580	p=.828	p=.592	p=.289	p=.492	p=.615	p=.175	p=.938	p=.249	p=.079	p=.452	p=.379	p=.779	p=.684	p=.494	p= ---
Trimethyl Benzene1,2,3	-.6164	-.3905	-.6243	.5564	.4590	-.2540	.5545	.4749	-.3559	.2547	.3252	.6026	.5988	.6453	-.6387	-.5701	.9093	--
	N=6	N=7	N=6	N=6	N=6	N=6	N=6	N=7	N=6	N=6	N=6	N=6	N=6	N=6	N=5	N=5	N=5	N=5
	p=.193	p=.386	p=.185	p=.252	p=.360	p=.627	p=.254	p=.282	p=.489	p=.626	p=.529	p=.205	p=.209	p=.166	p=.246	p=.316	p=.032	p= ---
Trimethyl Benzene1,2,4	.2061	.1998	.1109	-.2335	-.0352	.5950	-.6878	-.2057	.6686	.0557	-.6507	-.7272	.1363	-.1785	-.3856	-.3721	.5338	--
	N=6	N=7	N=6	N=6	N=6	N=6	N=6	N=7	N=6	N=6	N=6	N=6	N=6	N=6	N=5	N=5	N=5	N=5
	p=.695	p=.668	p=.834	p=.656	p=.947	p=.213	p=.131	p=.658	p=.147	p=.917	p=.162	p=.102	p=.797	p=.735	p=.521	p=.537	p=.354	p= ---
Trimethyl Benzene1,3,5	.3153	.2686	.2261	-.3364	-.1272	.6226	-.7656	-.3067	.6121	-.0468	-.5908	-.7738	.1110	-.2860	-.2058	-.2302	.3249	--
	N=6	N=7	N=6	N=6	N=6	N=6	N=6	N=7	N=6	N=6	N=6	N=6	N=6	N=6	N=5	N=5	N=5	N=5
	p=.543	p=.560	p=.667	p=.514	p=.810	p=.187	p=.076	p=.503	p=.197	p=.930	p=.217	p=.071	p=.834	p=.583	p=.740	p=.709	p=.594	p= ---
butox ethanol2	-.4497	-.2278	-.3671	-.1897	-.3392	-.7525	.3805	.4966	.0427	.3210	-.1859	.7985	-.2191	.4467	.7228	.7173	-.0659	--
	N=5	N=7	N=5	N=5	N=5	N=5	N=5	N=7	N=5	N=5	N=5	N=5	N=5	N=5	N=5	N=5	N=5	N=5
	p=.447	p=.623	p=.543	p=.760	p=.577	p=.142	p=.527	p=.257	p=.946	p=.598	p=.765	p=.105	p=.723	p=.451	p=.168	p=.173	p=.916	p= ---
Benzene	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1
	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---

Chemical	Plate counts	Gamma GT	ALT (SGPT)	AST (SGOT)	Urine phenol	Phenol creatine	Metile hypoacid	Mytile hypo creatine	U-HIPURICACID	HIPURICACID	BHCG	SHBG	Totaal	free calculate	X	Ventilation systems
Acetone	.2779	-.0136	-.2638	-.2894	-.0534	.0734	.2900	-.0005	1.0000	--	--	--	1.0000	-1.0000	--	-.5653
	N=12	N=11	N=11	N=11	N=12	N=12	N=11	N=11	N=2	N=1	N=2	N=1	N=2	N=2	N=1	N=9
	p=.382	p=.968	p=.433	p=.388	p=.869	p=.821	p=.387	p=.999		p= ---	p= ---	p= ---		p= ---	p= ---	p=.113
Ethanol	-1.0000	--	--	--	-1.0000	-1.0000	--	--	--	--	--	--	--	--	--	--
	N=2	N=1	N=1	N=1	N=2	N=2	N=1	N=1	N=0	N=0	N=0	N=0	N=0	N=0	N=0	N=1
	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---
n-Hexane	-1.0000	--	--	--	-1.0000	-1.0000	--	--	--	--	--	--	--	--	--	--
	N=2	N=1	N=1	N=1	N=2	N=2	N=1	N=1	N=0	N=0	N=0	N=0	N=0	N=0	N=0	N=1
	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---
n-Heptane	-1.0000	--	--	--	-1.0000	-1.0000	--	--	--	--	--	--	--	--	--	--
	N=2	N=1	N=1	N=1	N=2	N=2	N=1	N=1	N=0	N=0	N=0	N=0	N=0	N=0	N=0	N=1
	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---
Methyl Ethyl Ketone	-.4850	-.5166	-.3727	-.1523	-.4098	-.4590	.4066	0.0000	--	--	--	--	--	--	--	-.1450
	N=8	N=7	N=7	N=7	N=8	N=8	N=7	N=7	N=2	N=1	N=2	N=1	N=2	N=2	N=1	N=6
	p=.223	p=.235	p=.410	p=.744	p=.313	p=.253	p=.365	p=1.00	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p=.784
Ethyl Acetate	-.4755	-.4954	-.3565	.0692	-.4212	-.4695	.4734	0.0000	--	--	--	--	--	--	--	-.3156
	N=8	N=7	N=7	N=7	N=8	N=8	N=7	N=7	N=2	N=1	N=2	N=1	N=2	N=2	N=1	N=6
	p=.234	p=.258	p=.433	p=.883	p=.299	p=.240	p=.283	p=1.00	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p=.542
Toluene	.0590	-.0134	-.7247	-.6659	-.1831	.3797	-.0074	-.2616	1.0000	--	--	--	1.0000	-1.0000	--	-.4893
	N=9	N=8	N=8	N=8	N=9	N=9	N=8	N=8	N=2	N=1	N=2	N=1	N=2	N=2	N=1	N=6
	p=.880	p=.975	p=.042	p=.071	p=.637	p=.314	p=.986	p=.531	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p=.325
Xylene	.1645	-.1919	-.5130	-.0116	-.3862	.0040	.3077	-.1086	1.0000	--	--	--	1.0000	-1.0000	--	-.7001
	N=9	N=8	N=8	N=8	N=9	N=9	N=8	N=8	N=2	N=1	N=2	N=1	N=2	N=2	N=1	N=6
	p=.672	p=.649	p=.194	p=.978	p=.305	p=.992	p=.458	p=.798	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p=.121
Ethyl Benzene	.2943	.1064	.1455	.4605	-.5371	-.2918	.4478	.4946	--	--	--	--	--	--	--	-.6970
	N=7	N=6	N=6	N=6	N=7	N=7	N=6	N=6	N=1	N=0	N=1	N=0	N=1	N=1	N=1	N=5
	p=.522	p=.841	p=.783	p=.358	p=.214	p=.525	p=.373	p=.319	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p= ---	p=.191

Chemical	Plate counts	Gamma GT	ALT (SGPT)	AST (SGOT)	Urine phenol	Phenol creatine	Metlic hypoacid	Mytlic hypo creatine	U-HIPURICACID	HIPURICACID	BHCG	SHBG	Totaal	free calculate	X	Ventilation systems
n-Butyl Acetate	.0872	.4070	.2558	.4871	-.6257	-.5443	.3528	.3528	--	--	--	--	--	--	--	-.3899
	N=6	N=5	N=5	N=5	N=6	N=6	N=5	N=5	N=1	N=0	N=1	N=0	N=1	N=1	N=1	N=5
	p=.870	p=.497	p=.678	p=.405	p=.184	p=.264	p=.560	p=.560	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p=.516
Trimethyl Benzene1,2,3	-.1104	.4533	-.6155	-.5140	.2471	.9240	-.2021	.3748	--	--	--	--	--	--	--	-.2637
	N=7	N=6	N=6	N=6	N=7	N=7	N=6	N=6	N=1	N=0	N=1	N=0	N=1	N=1	N=1	N=5
	p=.814	p=.367	p=.193	p=.297	p=.593	p=.003	p=.701	p=.464	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p=.668
Trimethyl Benzene1,2,4	-.4217	.5897	-.4385	-.4953	-.4638	-.4280	-.6119	.2643	--	--	--	--	--	--	--	-.3695
	N=7	N=6	N=6	N=6	N=7	N=7	N=6	N=6	N=1	N=0	N=1	N=0	N=1	N=1	N=1	N=5
	p=.346	p=.218	p=.384	p=.318	p=.295	p=.338	p=.197	p=.613	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p=.540
Trimethyl Benzene1,3,5	-.4285	.7085	-.6154	-.6073	-.4804	-.5176	.2032	.5729	--	--	--	--	--	--	--	-.5170
	N=7	N=6	N=6	N=6	N=7	N=7	N=6	N=6	N=1	N=0	N=1	N=0	N=1	N=1	N=1	N=5
	p=.337	p=.115	p=.193	p=.201	p=.275	p=.234	p=.699	p=.235	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p=.372
Butox ethanol2	-.0846	.6231	.6590	-.1296	.6026	.4071	-.3800	.7613	-1.0000	--	--	--	-1.0000	1.0000	--	.2498
	N=7	N=7	N=7	N=7	N=7	N=7	N=7	N=7	N=2	N=1	N=2	N=1	N=2	N=2	N=1	N=5
	p=.857	p=.135	p=.107	p=.782	p=.152	p=.365	p=.400	p=.047	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p=.685
Benzene	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=1	N=0	N=0	N=0	N=0	N=0	N=0	N=0	N=0
	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p= —	p= —

Table 3: Statistical Area breakdowns

Area	Acetone					Ethanol					n-Hexane					n-Heptane				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1	0.9967	3	1.1467	0.00000	2.250	352.3033	3	606.8243	0.00	1053.000	0.096667	3	0.065064	0.030000	0.160000	0.106667	3	0.115902	0.00	0.230000
Area 2	943.6100	3	776.7868	52.83000	1480.000		0					0					0			
Area 3	0.0000	2	0.0000	0.00000	0.000	0.0000	1	0.0000	0.00	0.000	0.000000	1	0.000000	0.000000	0.000000	0.000000	1	0.000000	0.00	0.000000
Area 4	0.3067	3	0.5312	0.00000	0.920		0					0					0			
Area 5	2.3950	2	0.8132	1.82000	2.970		0					0					0			
Area 6	1.4000	1	0.0000	1.40000	1.400		0					0					0			
All Grps	202.9236	14	503.9478	0.00000	1480.000	264.2275	4	525.8516	0.00	1053.000	0.072500	4	0.071822	0.000000	0.160000	0.080000	4	0.108628	0.00	0.230000

Area	Methyl Ethyl Ketone					Ethyl					Toluene					Xylene				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1	0.833333	3	1.443376	0.00	2.500000	1.413333	3	2.344810	0.000000	4.120000	0.033333	3	0.057735	0.000000	0.100000	0.090000	3	0.155885	0.000000	0.270000
Area 2		0					0					0					0			
Area 3	0.060000	2	0.084853	0.00	0.120000	0.170000	2	0.183848	0.040000	0.300000	0.390000	2	0.438406	0.080000	0.700000	0.905000	2	1.251579	0.020000	1.790000
Area 4	0.060000	3	0.095394	0.00	0.170000	0.103333	3	0.145717	0.000000	0.270000	0.820000	3	0.415812	0.500000	1.290000	0.743333	3	0.170098	0.550000	0.870000
Area 5	0.000000	2	0.000000	0.00	0.000000	0.000000	2	0.000000	0.000000	0.000000	0.230000	2	0.325269	0.000000	0.460000	0.120000	2	0.169706	0.000000	0.240000
Area 6		0					0				1.230000	1	0.000000	1.230000	1.230000	0.920000	1	0.000000	0.920000	0.920000
All Grps	0.280000	10	0.782404	0.00	2.500000	0.489000	10	1.289785	0.000000	4.120000	0.457273	11	0.478938	0.000000	1.290000	0.497273	11	0.563633	0.000000	1.790000

Area	Ethyl Benzene					n-Butyl Acetate					Trimethyl Benzene1,2,3					Trimethyl Benzene1,2,4				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1	0.090000	3	0.090000	0.000000	0.180000	0.213333	3	0.369504	0.000000	0.640000	0.000000	3	0.000000	0.000000	0.000000	0.996667	3	1.726277	0.000000	2.990000
Area 2		0					0					0					0			
Area 3	0.590000	1	0.000000	0.590000	0.590000	0.620000	1	0.000000	0.620000	0.620000	0.020000	1	0.000000	0.020000	0.020000	0.050000	1	0.000000	0.050000	0.050000
Area 4	0.196667	3	0.015275	0.180000	0.210000	0.116667	3	0.096090	0.030000	0.220000	0.143333	3	0.060277	0.080000	0.200000	0.063333	3	0.020817	0.040000	0.080000
Area 5	0.060000	1	0.000000	0.060000	0.060000	0.050000	1	0.000000	0.050000	0.050000	0.060000	1	0.000000	0.060000	0.060000	0.020000	1	0.000000	0.020000	0.020000
Area 6	0.210000	1	0.000000	0.210000	0.210000		0				0.220000	1	0.000000	0.220000	0.220000	0.640000	1	0.000000	0.640000	0.640000
All Grps	0.191111	9	0.167365	0.000000	0.590000	0.207500	8	0.270278	0.000000	0.640000	0.061111	9	0.088097	0.000000	0.220000	0.432222	9	0.980061	0.000000	2.990000

Area	Trimethyl Benzene1,3,5					Butox ethanol2					Benzene					Hemoglobin				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1	6.450000	2	9.121677	0.000000	12.90000		0					0				14.70000	1	0.000000	14.70000	14.70000
Area 2		0					0					0				12.20000	3	0.964365	11.50000	13.30000
Area 3	0.110000	1	0.000000	0.110000	0.11000	0.000000	1	0.000000	0.000000	0.000000		0				15.65000	2	0.494975	15.30000	16.00000
Area 4	0.096667	3	0.086217	0.020000	0.19000	0.033333	3	0.049329	0.000000	0.090000		0				13.33333	3	1.680278	11.50000	14.80000
Area 5	0.010000	1	0.000000	0.010000	0.01000	0.095000	2	0.134350	0.000000	0.190000		0					0			
Area 6	0.130000	1	0.000000	0.130000	0.13000	0.080000	1	0.000000	0.080000	0.080000	0.020000	1	0.00	0.020000	0.020000	11.50000	1	0.000000	11.50000	11.50000
All Grps	1.680000	8	4.534043	0.000000	12.90000	0.052857	7	0.072045	0.000000	0.190000	0.020000	1	0.00	0.020000	0.020000	13.41000	10	1.748301	11.50000	16.00000

Area	Red blood count					Hematokrit					GKV					GHH				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1	4.930000	1	0.000000	4.930000	4.930000	43.00000	1	0.000000	43.00000	43.00000	87.00000	1	0.000000	87.00000	87.00000	30.00000	1	0.000000	30.00000	30.00000
Area 2	4.310000	3	0.500899	3.740000	4.680000	36.96667	3	2.589080	35.00000	39.90000	93.93333	3	7.710599	85.20000	99.80000	30.96667	3	2.315887	28.40000	32.90000
Area 3	5.135000	2	0.289914	4.930000	5.340000	46.85000	2	1.767767	45.60000	48.10000	91.30000	2	1.697056	90.10000	92.50000	30.45000	2	0.777817	29.90000	31.00000
Area 4	4.840000	3	0.330000	4.510000	5.170000	40.40000	3	4.714870	35.00000	43.70000	90.73333	3	8.812113	82.20000	99.80000	30.00000	3	3.306055	26.40000	32.90000
Area 5	4.425000	2	0.049497	4.390000	4.460000		0					0					0			
Area 6	4.510000	1	0.000000	4.510000	4.510000	35.00000	1	0.000000	35.00000	35.00000	99.80000	1	0.000000	99.80000	99.80000	32.90000	1	0.000000	32.90000	32.90000
All Grps	4.667500	12	0.418572	3.740000	5.340000	40.38000	10	4.893034	35.00000	48.10000	92.34000	10	6.467458	82.20000	99.80000	30.67000	10	2.116627	26.40000	32.90000

Area	GKHK					RDW					White cell count					Neurofils [perc]				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1	34.30000	1	0.000000	34.30000	34.30000	11.80000	1	0.000000	11.80000	11.80000	5.500000	1	0.000000	5.500000	5.500000	61.00000	1	0.00000	61.00000	61.00000
Area 2	32.96667	3	0.305505	32.70000	33.30000	12.36667	3	0.907377	11.40000	13.20000	7.900000	3	1.509967	6.500000	9.500000	54.93333	3	4.93694	50.80000	60.40000
Area 3	33.35000	2	0.212132	33.20000	33.50000	12.65000	2	0.070711	12.60000	12.70000	6.000000	2	0.424264	5.700000	6.300000	49.90000	2	3.39411	47.50000	52.30000
Area 4	33.03333	3	0.907377	32.20000	34.00000	12.73333	3	0.450925	12.30000	13.20000	7.433333	3	2.400694	4.800000	9.500000	41.56667	3	11.14465	31.60000	53.60000
Area 5		0					0				6.950000	2	1.909188	5.600000	8.300000		0			
Area 6	32.90000	1	0.000000	32.90000	32.90000	13.20000	1	0.000000	13.20000	13.20000	9.500000	1	0.000000	9.500000	9.500000	53.60000	1	0.00000	53.60000	53.60000
All Grps	33.19000	10	0.620642	32.20000	34.30000	12.56000	10	0.602218	11.40000	13.20000	7.241667	12	1.724402	4.800000	9.500000	50.39000	10	8.99079	31.60000	61.00000

Area	Neutrofils [abs]					Lymphocytes [perc]					Lymphocytes [abs]					Monocytes [perc]				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1	3.360000	1	0.000000	3.360000	3.360000	30.00000	1	0.00000	30.00000	30.00000	1.650000	1	0.000000	1.650000	1.650000	9.000000	1	0.000000	9.000000	9.00000
Area 2	4.333333	3	1.026320	3.200000	5.200000	35.90000	3	4.85695	31.20000	40.90000	2.833333	3	0.513160	2.400000	3.400000	7.233333	3	1.550269	5.700000	8.80000
Area 3	3.050000	2	0.494975	2.700000	3.400000	41.35000	2	2.05061	39.90000	42.80000	2.450000	2	0.070711	2.400000	2.500000	6.900000	2	0.282843	6.700000	7.10000
Area 4	3.300000	3	1.852026	1.500000	5.200000	46.00000	3	10.11336	35.60000	55.80000	3.266667	3	0.513160	2.700000	3.700000	9.200000	3	1.442221	8.000000	10.80000
Area 5		0					0					0					0			
Area 6	5.200000	1	0.000000	5.200000	5.200000	35.60000	1	0.00000	35.60000	35.60000	3.400000	1	0.000000	3.400000	3.400000	8.800000	1	0.000000	8.800000	8.80000
All Grps	3.756000	10	1.250522	1.500000	5.200000	39.40000	10	7.70123	30.00000	55.80000	2.825000	10	0.635632	1.650000	3.700000	8.090000	10	1.457128	5.700000	10.80000

Area	Monocytes [abs]					Eosinofiles [perc]					Eosinofiles [abs]					Basophiles [perc]				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1	0.500000	1	0.000000	0.500000	0.500000		0					0					0			
Area 2	0.600000	3	0.200000	0.400000	0.800000	1.633333	3	0.709460	1.000000	2.400000	0.133333	3	0.057735	0.100000	0.200000	0.300000	3	0.173205	0.200000	0.500000
Area 3	0.400000	2	0.000000	0.400000	0.400000	1.700000	2	1.697056	0.500000	2.900000	0.100000	2	0.141421	0.000000	0.200000	0.150000	2	0.070711	0.100000	0.200000
Area 4	0.633333	3	0.152753	0.500000	0.800000	2.866667	3	2.454248	1.400000	5.700000	0.233333	3	0.230940	0.100000	0.500000	0.366667	3	0.152753	0.200000	0.500000
Area 5		0					0					0					0			
Area 6	0.800000	1	0.000000	0.800000	0.800000	1.500000	1	0.000000	1.500000	1.500000	0.100000	1	0.000000	0.100000	0.100000	0.500000	1	0.000000	0.500000	0.500000
All Grps	0.580000	10	0.168655	0.400000	0.800000	2.044444	9	1.541194	0.500000	5.700000	0.155556	9	0.142400	0.000000	0.500000	0.311111	9	0.161589	0.100000	0.500000

Area	Basophiles [abs]					Plate counts					Gamma GT					ALT (SGPT)				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1		0				220.0000	1	0.00000	220.0000	220.0000		0					0			
Area 2	0.00	3	0.00	0.00	0.00	303.3333	3	30.00556	273.0000	333.0000	22.00000	3	18.19341	11.00000	43.00000	10.33333	3	1.15470	9.00000	11.00000
Area 3	0.00	2	0.00	0.00	0.00	303.0000	2	12.72792	294.0000	312.0000	25.00000	2	1.41421	24.00000	26.00000	19.00000	2	2.82843	17.00000	21.00000
Area 4	0.00	3	0.00	0.00	0.00	238.0000	3	31.76476	211.0000	273.0000	22.33333	3	18.87679	6.00000	43.00000	10.33333	3	2.30940	9.00000	13.00000
Area 5		0				306.0000	2	22.62742	290.0000	322.0000	45.00000	2	33.94113	21.00000	69.00000	35.00000	2	21.21320	20.00000	50.00000
Area 6	0.00	1	0.00	0.00	0.00	273.0000	1	0.00000	273.0000	273.0000	43.00000	1	0.00000	43.00000	43.00000	9.00000	1	0.00000	9.00000	9.00000
All Grps	0.00	9	0.00	0.00	0.00	277.9167	12	39.80054	211.0000	333.0000	28.72727	11	18.83662	6.00000	69.00000	16.27273	11	12.05065	9.00000	50.00000

Area	AST (SGOT)					Urine phenol					Phenol creatine					Methi hypoacid				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1		0				0.00000	1	0.000000	0.00000	0.00000	0.00000	1	0.000000	0.00000	0.00000		0			
Area 2	16.66667	3	3.055050	14.00000	20.00000	6.60000	3	1.705872	5.20000	8.50000	8.96667	3	7.423162	4.00000	17.50000	101.0000	3	0.00000	101.0000	101.0000
Area 3	26.00000	2	1.414214	25.00000	27.00000	3.45000	2	2.192031	1.90000	5.00000	2.10000	2	0.000000	2.10000	2.10000	101.0000	2	0.00000	101.0000	101.0000
Area 4	18.00000	3	5.291503	14.00000	24.00000	5.76667	3	2.441994	3.80000	8.50000	12.50000	3	8.660254	2.50000	17.50000	101.0000	3	0.00000	101.0000	101.0000
Area 5	23.00000	2	0.000000	23.00000	23.00000	23.05000	2	6.576093	18.40000	27.70000	16.00000	2	9.758074	9.10000	22.90000	28.9800	2	11.78040	20.6500	37.3100
Area 6	14.00000	1	0.000000	14.00000	14.00000	8.50000	1	0.000000	8.50000	8.50000	17.50000	1	0.000000	17.50000	17.50000	15.0000	1	0.00000	15.0000	15.0000
All Grps	19.63636	11	4.955334	14.00000	27.00000	8.21667	12	7.655875	0.00000	27.70000	9.84167	12	8.147220	0.00000	22.90000	80.0873	11	36.19085	15.0000	101.0000

Area	Mytile hypo creatine					U-HIPUURSUUR					Hipuursuur					BHCG				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1		0					0					0					0			
Area 2	0.020000	3	0.000000	0.020000	0.020000		0					0					0			
Area 3	0.020000	2	0.000000	0.020000	0.020000		0					0					0			
Area 4	0.020000	3	0.000000	0.020000	0.020000		0					0					0			
Area 5	0.020000	2	0.014142	0.010000	0.030000	0.250000	2	0.070711	0.200000	0.300000	0.200000	1	0.00	0.200000	0.200000	0.00	2	0.00	0.00	0.00
Area 6	0.020000	1	0.000000	0.020000	0.020000		0					0					0			
All Grps	0.020000	11	0.004472	0.010000	0.030000	0.250000	2	0.070711	0.200000	0.300000	0.200000	1	0.00	0.200000	0.200000	0.00	2	0.00	0.00	0.00

Area	SHBG					Totaal					free calculate					X				
	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum	Means	N	Std.Dev.	Minimum	Maximum
Area 1		0					0					0					0			
Area 2		0					0					0					0			
Area 3		0					0					0					0			
Area 4		0					0					0					0			
Area 5	16.90000	1	0.00	16.90000	16.90000	31.45000	2	31.74909	9.000000	53.90000	131.5000	2	157.6848	20.00000	243.0000	309.6000	1	0.00	309.6000	309.6000
Area 6		0					0					0					0			
All Grps	16.90000	1	0.00	16.90000	16.90000	31.45000	2	31.74909	9.000000	53.90000	131.5000	2	157.6848	20.00000	243.0000	309.6000	1	0.00	309.6000	309.6000

Area	Ventilation systems				
	Means	N	Std.Dev.	Minimum	Maximum
Area 1	0.740000	1	0.000000	0.740000	0.740000
Area 2	1.026667	3	1.276571	0.250000	2.500000
Area 3		0			
Area 4	0.796667	3	0.242143	0.520000	0.970000
Area 5	1.800000	2	0.282843	1.600000	2.000000
Area 6		0			
All Grps	1.090000	9	0.779214	0.250000	2.500000