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Lead Exposure and Physiological Enzymes among Industrial versus Non-Industrial Workers in Al-Ain, United Arab Emirates.

Fatima Rashid Mattar Said Al-Neamy

LEAD EXPOSURE AND PHYSIOLOGICAL ENZYMES AMONG INDUSTRIAL VERSUS NON-INDUSTRIAL WORKERS IN AL-AIN, UNITED ARAB EMIRATES

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By

Fatima Rashid Mattar Said Al-Neamy

Faculty of Science
United Arab Emirates University

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SUPERVISORS

Dr. Abdulbari Bener, Ph.D.,
Senior Supervisor
Associate Professor
Department of Community Medicine
Faculty of Medicine & Health sciences
United Arab Emirates University

Dr. Ahmed Mirza Almehdi, Ph.D.,
Supervisor
Assistant Professor of Biochemistry
Department of Chemistry
Faculty of Science
United Arab Emirates University

The Thesis of **Fatima Rashed Mattar Al-Neaimi** for the Degree of Master of Science in Environmental is approved.

8 May 2000

Examining Committee Member, Dr. Abdulbari Bener

8 May 2000

Examining Committee Member, Dr. A. G. Nicol

8 May 2000

Examining Committee Member, Dr. Ahmad Ayoob Alqadal

Dean of the Faculty of Science, Prof. A. S. Al-Sharhan

United Arab Emirates University
2000/99

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ABSTRACT

Lead, which is considered to be as a toxic metal without any useful phy biochemical function in human body was widely used since the evolution of the industrialized era as a chemical part of much machinery and commodities. Lead containing gasoline, lead paints, lead-soldered cans, lead-glazed earthenware and tobacco are examples of sources of lead. Therefore, lead continues to be a significant public health concern globally.

Nowadays it is particularly impossible to find a person with blood lead level lower than 0.48µmol/L (100 µg/ dL) especially in city areas. Changes in neurobehavioral, cardiovascular, hematological, and renal functions are found to be associated with lead even low blood lead levels. At the molecular levels, lead tends to cause damages to multitude of enzymes and essential cellular structures.

The aim of the present study is to determine the Blood Lead Levels (BLLs) among expatriate industrial (exposed) and non-industrial (unexposed) workers and investigate predictor factors that are influencing BLLs in UAE population. Additionally, the present study aiming to determine the effect of lead exposure on the plasma levels of amino acids and serum liver enzymes, cardiac enzymes and renal functions protein in industrial workers in Al-Ain, UAE. Finally, to study acute and chronic respiratory symptoms occurred exposure among industrial.

Although this study is not intended for generalization of the lead population problem to the whole UAE population, but without any doubt this study would be a representative sample of concurrent measures of lead exposures in the specific community. The first part of the study was screening of blood lead levels among industrial versus non-industrial individuals taking in consideration age, ethnic group, and smokers and non-smokers criteria. The second part of the study is focussing on the correlation between blood lead levels and levels of amino acids, liver function enzymes, cardiovascular enzymes and renal function proteins.

This study was based on matched exposed and unexposed study of subject selected from Al-Ain City. This study included 100 exposed and 100 unexposed, matched for age, sex and nationality. The field survey was conducted during the period from 1st of February to July, 1999.

The relative importance of some factors on the likelihood of industrial workers having lead exposure was assessed using statistical methods and models.

This matched case-control study was conducted in three parts, namely:

a) An epidemiological study of eating habits, life style, reported symptoms and lead level in industrial and non industrial workers.

b) Plasma amino acid profiles among industrial and non-industrial workers.

c) Laboratory analysis of serum liver enzymes, cardiac enzymes and renal function proteins for cases and controls.

The Socio-demographic characteristics of industrial and non-industrial workers were similar among the population surveyed. Most of the industrial workers were not certain about lead exposures. Both groups, industrials and non-industrials workers were demographically similar with regards to age groups, nationality and marital status. The majority of industrial workers (38%) were illiterate, which was the expected result. The majority of non-industrial workers (48%) were educated with secondary or high school educational certificates. Most industrial workers were not aware with the amount of lead they might have been exposed.

Few industrial workers reported exposure to lead. One type of exposure derives from the working in jobs related to lead as 40% of industrial workers reported being involved with smelling spraying of toxic gases and chemicals directly, 13% of the subjects reported working in radiation areas or magnetic fields, and 14% of them reported dealing with battery recycling.

The exposure to lead among industrial and non-industrial workers was investigated. The majority of industrial workers reported not using masks as the main method of protection during their work. The majority of industrial workers reported different clinical symptoms. Reported symptoms among industrial workers were higher than non-industrial workers had. It was observed that, most of the symptoms occurred among industrial and non-industrial were significant differences as follows Nausea/vomiting [OR = 4.235, 95% CI (1.35-13.25)], (p = 0.008), Red/irritated eye/blurred vision [OR=1.61, 95% CI(0.731-3.547)],(p = 0.235)], Increased anxiety [OR = 2.68, 95% CI (1.16-6.20)], (p = 0.017), Dizziness [OR = 1.926, 95% CI (0.808-4.592)], (p = 0.134), Headache [OR = 1.176, 95% CI (0.617-2.243)], (p = 0.622), Muscular symptoms [OR = 3.146, 95% CI (1.46-6.75)], (p = 0.002), Memory loss [OR = 11.0, 95% CI (1.38-87.64)], (p = 0.005), Fatigue [OR= 3.439, 95% CI (1.303-9.074)], (p = 0.009), Insomnia [OR = 2.16, 95% CI (0.834-5.612)], (p = 0.106), Chest pain [OR = 1.0, 95% CI (0.41-2.42)], (p = 1.000). Kidneys [OR = 3.20, 95% CI (1.20-8.51)], (p = 0.008), Gastrointestinal [OR = 2.55, 95% CI (1.17-5.55)], (p = 0.016), Anemia [OR = 3.43, 95% CI (1.30-9.07)], (p = 0.009), Difficulty in breathing [OR = 2.72, 95% CI (1.07-6.88)], (p = 0.030), Cardiovascular [OR = 3.61, 95% CI (1.27-10.30)], (p = 0.011), Mania [OR = 6.62, 95% CI (1.87-23.39)], (p = 0.0016), Abdominal pain [OR = 1.18, 95% CI (0.53-2.63)], (p = 0.684), Myalgia and Anorexia [OR = 1.26, 95% CI (0.32-4.84)], (p = 0.733).

Further more we have investigated plasma amino acid profiles among industrial workers (exposed) and non-industrial workers (unexposed). Most plasma amino acid tests showed higher values among industrial than non-industrial workers did. There were statistically very highly significant differences between industrial non-industrial with the respect of some amino acids levels as can be seen as follows: Taurine (112.02 ± 38.70 µmol/l, Mean ± S.D), N= 100 in industrial workersand (55.15 ± 24.97 µmol/l, Mean ± S.D) in non-industrial workers, N= 100 , (p = 0.0001), Serine (159.33 ± 34.68 µmol/l, Mean ± S.D) in industrial workers and (108.09 ± 26.42 µmol/l, Mean ± S.D) in non-industrial workers, (p = 0.003), Glutamic acid (236.89. ± 149.91 µmol/l, Mean ± S.D) in industrial workers and (82.26 ± 57.09 µmol/l) in non-industrial workers, (p = 0.000), Glycine , (236.89 ± 149.91 µmol/l, Mean ± S.D) in industrial workers and Mean ± S.D (244.25 ± 72.88

µmol/l) in non-industrial workers, (p = 0.0001), Histidine Mean ± S.D (107.50 ± 29.61 µmol/l) in industrial and Mean ± S.D (95.90 ± 18.53 µmol/l) in non-industrial workers, Ornithine Mean ± S.D (178.94 ± 59.16 µmol/l) in industrial workers and (104.84 ± 30.64 µmol/l) in non-industrial workers (p = 0.001), Lysine Mean ± S.D (218.71 ± 51.73 µmol/l) in industrial workers and (176.77 ± 36.71 µmol/l) in non-industrial workers (p = 0.006), Leucine Mean ± S.D (162.41.99 ± 38.73 µmol/l) in industrial workers and in non-industrials workers (135.57 ± 26.94 µmol/l), (p = 0.002), Valine Mean ± S.D (264.15 ± 65.88 µmol/l) in industrial workers and (234.05 ± 58.02 µmol/l) in non-industrial workers (p = 0.62), Threonine Mean ± S.D (161.67 ± 42.04 µmol/l) in industrial workers, (p=0.055) and Mean ± S.D (130.34 ± 34.55 µmol/l) in non-industrial workders, (p = .055), Alanine Mean ± S.D (526.59 ± 151.35 µmol/l) among the industrial workersand in non-industrial workers Mean ± S.D (417.64 ± 92.08µmol/l), (p=0.0001), Glutamine Mean ± S.D (622.86 ± 139.85 µmol/l) in industrial workersand (583.89 ± 146.50 µmol/l) in non-industrial workers, (p = 0.346) and Proline Mean ± S.D (339.31 ± 137.29 µmol/l) in industrial workers and (261.15 ± 82.464 µmol/l) in non-industrial workers (p = 0.006).

Finally, the liver function tests were estimated among industrial (exposed) and non-industrial (unexposed) workers. Only Lactate Dehydrogenase and Alkaline Phosphates were higher in industrial workers than non-industrial workers. And there were statistically significant differences between industrial and non-industrial workers, for Lactate Dehydrogenase in industrial workers (211.27 ± 57.27u/l, Mean ± S.D); versus in non-industrial workers (194.77 ± 48.69 u/l, Mean ± S.D), (P=0.029) and Alkaline Phosphates (84.27 ± 24.64 u/l, Mean ± S.D) for u/l) for industrial and (76.18 ± 20.48 u/l, Mean ± S.D) in non industrial workers, (p=0.012).

Also kidney proteins such as Blood Urea Nitrogen and Creatinine were estimated among industrial and non-industrial workers without any statically significant differences.

The results of this study determined the exact Blood Lead Levels among industrial and non-industrial workers, in UAE population. Also lead and associated environmental risk

factors will hold great promise for future planning, prevention and programming in Al-Ain City for the Ministry of Health, Universities, Federal Environmental Agency, WHO, Municipality and other concerned academic researcher institutions.

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CHAPTER I

INTRODUCTION

1.1 INTRODUCTION

1.1.1 Historical Aspects of Lead:

Lead and its compounds are potentially toxic (Ho, et al, 1998). It does not seem to have any known physiological function. It is widely distributed in the environment as a result of man's activities. The effect of lead on the health have been recognized for many years and there are some cases of lead poisoning in the wold (Jenkin, 1980). The diagnosis of excessive lead absorption was based on a blood lead level of 50 μg/dL or more and the absence of sings and symptoms of lead poisoning.

Evidence showed that lead is one of the first metals that man used at 4000 BC, and the Egyptian are among the first to use lead, and Phoenicians mined lead ores in Spain around 2000 BC. The Romans were important lead consumers, using metallic lead for piping, cooking utensils, and lead compounds for pottery glazes (Carlzenz, 1988).

The western region of Saudi Arabia contains numerous small veins and lodes of polymetallic copper-zinc-lead-silver sulfides that have been oxidized intensively in some localities (Al-Saleh, 1992). Many of these prospects contain vestiges of very ancient workings of unknown age. The Western region of Saudi Arabia is endowed with abundant lead-silver deposits that have been mined since ancient times. The polymetallic nature of these minerals would make them highly prized as the basic material for making leaded bronzes –and brasses particularly during the early Bronze Age. It also provides a

means of fingerprinting the artifacts fashioned at the western Saudi Arabian ore locations (Nriagu, 1983).

1.1.2 Environmental Exposure to Lead

The introduction of lead in the developing countries has been associated with the growth of industry and the expanding use of motor vehicle in transport. Medical and health professionals, scientists, ecologist and legislators all have been interested in man's exposure to lead. The scientific and lay literature is extensive and mounting evidence of its exposure contributes to making it a major public issue (Al-Saleh, 1992). Exposure to lead is considered a major health problem for the people in the industrial and oil-producing countries. Major poisoning among public are fairly well characterized, but the health effects of routine, smaller exposure are uncertain. In UAE studies, although limited by small sample size and inadequate comparison groups, revealed lead in blood in children, the pathophsiological significance of which remain unclear(Al khayyat and Almehdi,1999)

In the United Arab Emirates, and in Gulf Countries, concern of lead exposure is in its early stage. The literature is sparse and the issue has not yet been paid full attention. Historical evidence indicates that lead was used in the earliest periods for commercial (Parry 1970, Hamarneh 1973) and military purpose (Cook 1974) and there was foreign trade in leaded products with India, Egypt, Syria and Iraq. Traditional cosmetics such as kohl and henna, which have significant amounts of lead, are being used widely in the United Arab Emirates. As in other developing countries, use of lead has multiplied greatly in everyday life in the United Arab Emirates, particularly over the last 20 years. Pollution

of air, soil, water are undoubtedly accompany, industrial developments is one of the problems that accompanies economic progress.

The discovery of oil just before the middle of the century has changed many aspects of life in the UAE. Road construction programs have increased parallel to other national developments, and with an increase in reaching serious public health problems (Bener et al.1994). These processes have been accompanied by rapid urbanization, an increasing number of factories, an expansion of modern houses, extensive network of motorways and free availability of cars and other motor transport (Bener et al., 1997, 1999).

As a result, nowadays its particularly impossible to find a person with blood level lower than 0.48 µmol/l (100 µg/dL) specially in city areas (Schutz et al.1987). Changes in neurobehavioral, cardiovascular, hematological and renal functions are found to be associated with lead- even at low blood lead levels. At the molecular levels, lead tends to cause damage to multitude of enzymes and essential cellular structures. Researches showed that blood lead levels are directly influenced by recent exposure to inorganic lead (O`Flahetry 1986 and Hodgkins et al.1991).

Evidence suggests that the response is more pronounced in newly exposed subjects because internal lead stores exert a dampening influence on the transmission of exposure variability to changes in blood lead levels (McGrail et. al. 1995). In contrast, limited evidence on a small of subjects suggests that exposure to organic lead dose not exert the same effect on blood lead level.

1.2. LEAD

1.2.1. Occurrence Of Lead

Lead occurs in the nature as a compound of two isotopes, Pb^{206} and Pb^{208}. It is distributed ubiquitously in the earth crust. The igneous rocks of the earth's crust contain about 12 ppm of lead down to a depth of 30km, while the soil film covering the outermost surface of the continents contains an average of 16ppm. It is found that uncontaminated natural water contains less than 0.1ppm of lead, while seawater contains from 0.08 to 8ppm. In the industrial areas where lead is mined, refined, smelted, or where coal is burned, the lead content in soil may reach several thousand parts pemillion (Carlzenz, 1988).

Nature deposits of lead have been found in Canada, the United States, Mexico, Peru, Ireland, Poland, Sweden, Spain, the Soviet Union, West Germany, Yugoslavia, South-West Africa, China, Japan, North Korea, and Australia. Lead is mainly occurs as sulphide (PbS), in association with other metallic sulphate. It is also occurs as cerussite (carbonate), anglesite (sulfate), pryomorphite (chlorophosphate), crocoisite (chromate), wulfenite (molybdate), matlockite (chloride) and vanadinite (vanadate) (Harrington et al, 1992).

Due to the ubiquitous distribution of lead, no lead free medium exists or has ever existed. So man is and always has been exposed to lead from air or food or drinking water. This exposure varies from place to place, and it most probably was lower before the Industrial Revolution than it is today (Carlzenz, 1988).

1.2.2. Chemical Properties of Lead

Atomic weight of lead is 207.21and its density is 11.34. It is a bluish or silver gray, soft, heavy metal with an atomic number of 82. It melts at 327.4°C and its boiling point at atmospheric pressure is 1620°C. Evaporation increases with the rising of temperature, lead is poorly soluble in both cold and hot water and in diluted acids, but it dissolves in nitric acid, acetic acid and hot concentrated sulfuric acid. The valence states of lead are +2 and +4. Within the common lead compounds, the acetate and the nitrate are easily soluble in cold water (44.3 and 37.7 gm\100ml respectively). Moderately soluble compounds include the chloride (1.0gm/ 100 ml). The water solubility of other compounds is low (0.012-0.001 gm/100 ml; e.g., the carbonate, the sulfate, and the sulfide) or they are practically insoluble (the basic chromate, the molybdate, the red lead or (Pb_3O_4) and the silicate). Lead also forms organic compounds, the most important are tetraethyl lead (TEL) and tetramethyl lead (TML). They are practically insoluble in water but dissolve readily in organic solvents, fats, and lipids.

1.2.3. Sources of Lead

Without doubt that man has always absorbed some lead from his environment, although this contribution is usually small in comparison with that derived from lead result from his own activities. The increase of production and industrial use of lead may also give rise to much dust and fumes within factories and their surrounding environment. All sources of lead exposure such as water, food, air and paint are well known and different measures

including regulation have been done much to reduce the risk of lead poising (Jenkin, 1980).

1.2.3.1. Water

A recent survey done by consumers Union showed that many communities had lead in water over the Environmental Protection Agency standard (Needleman, 1994). The most important sources of the contamination of the tap water with the lead is lead piping and lead-lined tanks (Carlzenz, 1988). It has been found that the amount of lead in a sample of tap water is primarily determined by the chemical characteristics of the water and length of lead piping through which it flow. Although it is known as soft, acid waters dissolve lead, the national survey reported that it could be dissolved by some harder water. The concentration of lead is found in places where the supply of drinking water is drawn from the down pipe. This concentration is influenced by the length of the pipe, time that the water remain in the pipes and also influenced by how far and how quickly the tap is turned appears to be greater with the increase of temperature in the summer. It is noticed that the concentration of lead increase within hours if the water remains in the pipes but flushing may achieve a substantial reduction in the lead content of water intended for consumption. In general, the people exposed to lead for two reasons firstly, substantial proportion of people run some water to waste or for other purposes before drawing it for consumption and the lead content is generally lower after flushing and secondly, Survey have shown that there is marked variation in the concentration of lead in tap water within any household (Jenkin, 1980).

1.2.3.2. Food

Usually, solder that is used in the manufacture of cans is a main source of contamination of food by lead during processing. The maximum concentration of lead permitted in prepared food specially intended for babies or young children is 200µg/kg. This mean that the manufacturers have to use pure tin in solder for canning infant foods (Jenkin, 1980). Lead can be introduced to the food from ceramic tableware, some of the decorations on chinaware, and leaded crystal. So it is always advised not to feed the baby from a crystal nursing bottle (Needlman, 1994).

1.2.3.3. Air

Lead found in the air as a result of the combustion of lead containing petrol, from certain industrial processes and from lesser extent from the combustion of coal and weathering of paints. The chemical and physical form of the lead varies according to the source. Often most of the lead is emitted from cars and industrial processes after the waste gases have been cleaned are fine enough to remain in airborne for long periods and is within the respirable size range. These small particles are widely dispersed and lead resulting from man's activities has been detected at the polar region. Coarse particles are generally deposited close to their source and may contribute to the lead content of dust. The short period concentration of lead found in the atmosphere close to sources is likely to vary considerably as strength and factors affecting dispersion change (Jenkin, 1980).

1.2.3.4. Paints

For a long time lead compounds have been used in the manufacture of domestic paints. Most of the leaded paints are used in the industry of shipbuilding and construction. Since it has been considered as a serious health hazard, especially for children; it has been estimated that leaded paints now make up less than 3% of total sales of paints. Although the hazard from lead in new paints has been clearly reduced, but we are concern that the risk of lead is still from lead paint used out-of-doors in places accessible to children or from domestic use of lead paint intended for industry. There should be labels warning in these places and the second step is the complete elimination of the use of these paints (Jenkin, 1980).

Leaded paints are considered a serious health problem for the children. The Lead Paint Poising Prevention Act of 1971 banned the use of lead in household paint, but as many as 5% of the American houses have lead in interior paint (Needleman, 1994). It is clear that household paint is the most important threat. Peeling paint is one of the hazards to the poor children in the oldest houses. In the United States of America there are about 2 million houses with peeling paint where children live. These need the most urgent attention.

1.2.4. TOXICITY

1.2.4.1. Definition

Toxicology is the study of the adverse effects of chemical or physical agents on biological systems; it is the science of poisons. A poison is any substance (chemical, physical or biological) that is harmful or destructive to biological (living) system. A poison derived from a biological source is a toxin (Hayes, 1994). The lethal dose of toxicity is (LD 50) expressed as milligram (mg) of toxicity per kilogram (kg) of body weight, the dose that kill 50 percent of the test animals (Hassal, 1990).

There are two types of toxicity namely Acute and Chronic:

a. Acute Toxicity

Acute toxicity related to hazard in connection with acute short-term exposure. This can cause a disruption of biochemical or physical system (Hogstedt, 1992). The acute toxicity tests give (1) a quantitative estimate of acute toxicity (LD 50) for comparison to other substances, (2) identify target organs and other clinical manifestations of acute toxicity, (3) establish the reversibility of the toxic response and (4) give dose-ranging guidance for other studies. Acute inhalation studies are performed simito the acute toxicity studies except the route of exposure is inhalation. Most often, the length of exposure is four hours (Amdur, 1986).

b. Chronic Toxicity

Chronic toxicity is the long-term exposure that results from uptake of the chemical into the body at low doses, but has strong tendencies to bio-accumulated and represent hazard (Barnes, 1976; Hassal, 1990; Hogstedt, 1992). Chronic exposure is usually from six months to two years. Chronic toxicity tests are performed to assess the cumulative toxicity of chemicals, but the study design and evaluation often include a consideration of the carcinogenic potential of chemicals so that a separate lifetime feeding study to address carcinogenicity dose not have to be performed (Amdur, 1986). Long term exposure can causes cancer, teratogenic effect, sterility, spontaneous abortion and cognitive deficits (Rodnitzky et. al. 1975 and Moses, 1989).

1.2.4.2. Toxicity of Lead

The systemic toxicity of lead has been extensively studied, and most of the studies in humans have used blood lead levels (PbB) as biomarker of exposure, which occurs through the inhalation (air pollution) and oral (diet). Lead is not considered to be toxic excepted at extremely high levels. The normal or "safe" level of lead exposure have been reduced 48 times, and nowadays in adults l PbB levels is between 25-40µg/dl which is found to be associated with gastrointestinal pain and impaired hematopoietic, neurological, endocrine, reproductive and immunological functioning; while for children with PbB level between 10-24µg\dl, no pharmacological treatments are currently available and the only intervention is to try to reduce the lead exposure (Hayes, 1994).

Different forms of lead can be absorbed through lung, skin and through the gastrointenstinal tract. Absorption through the lung can't be happen unless there is a penetration of dust or fumes into deep lung structures and if there is a degree of water solubility of deposited material. The organic forms of lead are absorbed more rapidly and efficiently, but these are absorbed to limited extent through the skin. The most complex absorption of lead occurs through gastrointestinal; which depends on the type of lead compound consumed, the physiological state of the individual and the type of food also present. Undissolved lead is absorbed to a minor degree. It is found that the absorption of lead is influenced by nutritional status with respect to calcium balance. When calcium demand is high, lead is more readily absorbed from the gastrointestinal tract because the mechanisms that have been mobilized to increase calcium absorption do not readily distinguish between calcium and lead. Foods, which present on the gastrointestinal, have also effect on the absorption of lead. Fatty foods are found to enhance lead absorption, but why this happening is still unknown or remain unclear. Other materials in the gastrointestinal tract may bind or physically absorb lead to decrease absorption. Only from 5 to 10 % of ingested lead in human is actually absorbed and the rest excreted with the feces (Carlzenz, 1988).

After the absorption of lead, it distributed within the body. Some of the lead is found in the liver, kidneys, and red blood cells. But 90% of lead is stored in the bone due to its chemical similarity to calcium, so lead can be stored for long period without any toxicological consequences; but when the body needed calcium from the bone, some of the lead will be mobilized and result as lead poisoning. It is clear that measurement of blood lead level is an index of exposure to lead, but not an index of the total body burden,

which is largely stored in the bone. The major toxic effects of inorganic lead are on the nervous system, kidneys, and red blood cells from the bone marrow. In the bone marrow lead affects the formation of hemoglobin and red blood cells and one early sign of lead poising is anemia (Devlin, et. al. 1992).

1.2.5. HEALTH EFFECTS

Lead has been recognized as a potential hazard to human health for a long time. The effects of lead appear to be the same regardless of the mechanism by which it enters the body. It is found that lead affects almost every organ and system in the body (Hsing Lio, 1996).

1.2.5.1. Neurological Effects

The effects of inorganic lead on occupationally exposed adult population have been well studied. Some investigators have been associated moderate exposure to lead in infancy and childhood with impairments of both the central nervous system (CNS) and peripheral nervous system (Stokes, et al, 1998).

a. Central Nervous System

Usually lead encephalopathy occurs in both acute and chronic forms. Also there is a syndrome of permanent brain damage occurring in a large group of patients who have survived one or more attacks of acute encephalopathy. In addition, sub-clinical forms of

lead-induced brain injury have been described. However, these states are not yet well defined.

It is found that in acute encephalopathy there is swelling of the brain, some time in combination with petechial hemorrhages. It is believed that vascular injury is the underlying lesion the ceedema. The most extensive neuronal injury usually is found in the cerebral cortex, but changes frequently in the cerebral cortex as well. The prognosis of acute lead encephalopathy is poor, and in fatal cases the patient often dies within a few days after onset the first seizure. The mortality in the children was about two thirds, before the discovery of chelation therapy. The late encephalopathy may be grave and characterized by severe mental retardation, blindness, and epilepsy with cortical atrophy, and sometimes may be it become more subtle and characterized by lack of sensory perception and perseverance resulting in poor learning ability and by behavioral disorders such as aggressiveness, destructive behavior and hostility (Carlzenz, 1988).

In the chronic encephalopathy, extensive tissue destruction with cavity formation, as well as thickening of veins with cellular disorganization of their walls have been described. The later findings suggest that vascular changes be involved in the development on cerebral injury. The entity of chronic encephalopathy is complex, it can be said that the former is characterized by a more insidious onset and has a more protracted course. In its mildest forms, it may by detectable by psychomotor disturbances, impairment of intelligence function, and personality changes (Carlzenz, 1988).

b. Peripheral Nervous System

Peripheral nervous paralysis, or lead palsy, formerly represented a severe and common complication of occupational lead. At these days, this disorder is seen only sporadically and in cases of uncontrolled lead exposure. The paralysis result from this exposure is characterized by selective involvement of motor neurons with little or no sensory abnormalities. The other typical feature is that paralysis usually affects extensor muscle, often unilaterally. Since the most heavily used muscle groups are most sensitive, the typical picture is wrist drop of the right hand.

However, other muscles of the upper limbs, the extraocular eye muscles, and the extensors of the lower limbs may be involved. Optics neurities leading to blindiness , extraocular eye muscles has been reported in children. In this case the clinical picture is characterized by aching and tenderness of muscles and joints, increased fatigability of muscles of the muscles, and fine tremor. Also there may be weakness and lowered tone in the muscles and even atrophy of extensors of the forearm (CarlZenz, 1988).

1.2.5.2. Renal Effects

It is found that the heavy exposure may cause progressive and irreversible renal diseases. The children who ingest lead paint chips and consume the illicit whiskey still exposed to risk. It has been notice that the effect of lead on renal function is accompanied with by hypertension. The renal damage is chronic interstitial fibrosis, tubular degeneration and vascular changes in small arteries and arterioles. A peculiar feature is the abundance of the intranuclear inclusion bodies in renal tubular lining cells. These bodies are composed of lead-protein complex in which lead is bound in non-diffusible form. Goyer and Chiolm

have suggested that the inclusion of body represent a defense mechanism by which the concentration of lead in cytoplasm will be lowered. Furthermore, since those cells finally are excreted in the urine, this phenomenon would provide the kidneys with a means of getting rid of surplus lead without destroying the viability of the tubular lining cells. In several cases, the whole Fanconi triad (hyperaminoaciduria, glucosuria, and hypophosphatemia in combination with hyperphosphaturia) may occur. The functional damage is not necessarily, since even the Fanconi syndrom may be reversible (CarlZenz, 1988).

1.2.5.3. Gastrointestinal Effects

Lead poisoning has a variety of gastrointestinal symptoms. The first symptoms begin to appear at PbB concentrations slightly above 80µg/dL. The symptoms include loss of appetite, digestive disturbances, epigastric discomfort after eating and either constipation or diarrhea. If PbB exceeds 100 µg/dL, the likelihood of more severe symptoms increases. These include occasional to frequent colicky abdominal pain and sever constipation. If lead exposure is not interrupted, and PbB exceeds 150µg/dL, the classic lead colic will develop. Lead colic is characterized by "sharp onset and recurrent spasms in which the patient writhes in pain, retracts his legs spasmodically to his abdomen, groans, clenches his hands, grits his teeth, with beads of sweat on his brow". This dramatic disorder often results in inappropriate laparotomy, sometimes due to the negligence of penetrating the history of the patient and sometimes because the source of exposure is not easily found. If untreated, colic may persist for several days –even for a

week. Intravenous injection of calcium gluconate or other antispasmodic drugs usually results in prompt relief (CarlZenz, 1988).

1.2.5.4. Cardiovascular Effects

It has been found that in the case of acute lead poisoning and if the patient has colic, the blood pressure often is elevated. Alternatively, hypotonia may occur and myocardial damage may also occur. The studies showed that the main findings were tachaycardia, arterial arrhthmia, inverted T waves and or abnormally wide QRS-T angle. All these findings return to normal after treatment. In another study it was reported that there were disturbances in a trioventricular conduction, which return to normal after the treatment, but retain after re-exposure to lead. But until now little information is known about the acute toxic effects of lead on the cardiovascular system.

The chronic effects have been studied to some extent, but with at least superficially divergent result. Dingwall-Fordyce and Lane found a 2.5 fold excess of cerebrovascular deaths in a population of heavily exposed accumulator factory workers. This view is further supported by the fact that there was no effect of moderate lead exposure on the blood pressure in a Swedish study of 364 accumulator factory workers. In view of that and other similar results that have been reported, it appears that work in conditions where exposure is controlled dose not give rise atherosclerosis, hypertension, myocardial infarction, or cerebrovascular disease (Carlzenz, 1988).

1.3. LIVER FUNCTION ENZYMES

Liver is considered as the largest organ in the body, it weighs about 1200-1500g and comprises one-fiftieth of the total adult body weight. It is relatively larger in infancy, comprising one-eighteenth of the birth weight (Sherlock, 1989). The design of the liver enables this complex organ to unique functional demands. It is the place of the metabolism of amino acids, carbohydrates, lipids, and vitamins. It also filters foreign substances and handles the products of human metabolism (Gitnick, 1991).

The major liver function enzymes includes the following Enzymes:

1.3.1. Lactate Dehydrogenase

Lactate dehydrogenase (LDH) is an enzyme that catalyzes the inter-conversion of lactic and pyruvic acids. It is a hydrogen-transfer enzyme and utilizes the coenzyme NAD+ (Bishop, 1984). Myocardial infraction may be associated with the elevations of total LDH. It should be contrasted with creatine kinase (CK) and aspartate aminotransferase (AST).

1.3.2. Aspartate Amino Transaminase

Aspartate amino trasferase (AST) is an enzyme belonging to the class of transferase. It catalyzes the interconversion of amino acids and oxo acids by transfering of amino groups.

The transamination reaction is important in intermediary metabolism because of its function in the synthesis and degradation of amino acids. The ketoacids formed by the transamination reactions are ultimately oxidized by the tricarboxylic acid cycle to provide a source of energy (Bishop, 1986).

1.3.3. Alanine Transaminase

Alanine transaminase (ALT) is a transferase enzyme with enzymatic activity similar to that of AST. It catalyzes the transfer of an amino group from alanine to α-ketoglutarate with the formation of glutamate and pyruvate. The older termimology still in use is glutamic–pyruvic transaminase (GPT). Pyridoxal phosphate acts as the coenzyme for this enzyme (Bishop, 1986).

1.3.4. Alkaline Phosphatase

Alkaline phosphatase (ALP) belongs to a group of enzymes that catalyzes the hydrolysis of a wide variety of phosphomonoesters at an alkaline pH. Cosquently, ALP is a non-specific enzyme capable of reacting with many different substrates. Specifically, ALP functions to liberate inorganic phosphate from an organic phosphate ester with the concomit production of alcohol (Bishop, 1986)

The optimal pH for the reaction is 9 to 10, but this depends on the substrate used. The enzyme requires Mg++ as a cofactor.

1.3.5. Gamma-Glutamyl Transferase

Gamma-glutamyltransferase (GGT), is an enzyme involved in the transfer of the γ -glutamyl residue from γ -glutmyl peptides to amino acids, H_2O, and other small peptides. In most biological systems, glutathione serves as the γ -glutamyl donor.

The specific physiological function of GGT has not been clearly established, but it has been suggested that GGT is involved in peptide and protein synthesis, regulation of tissue glutahione levels and the transport of amino acids across cell membranes. In addition to the above mentioned important liver function enzymes liver function includes analysis for albumin, total protein and bilirubin (Bishop, 1985).

1.4 CARDIAC ENZYMES

1.4.1. Creatine Kinase(CK)

Its concentration in skeletal muscle and myocardium is very high. Appreciable amounts are found in the brain. Tiny amounts are found in few other organs and are found in the liver. Many studies have shown that CK values are high in patients with myocardial infraction, progressive muscular dystrophy, alcoholic myopathy, and delirium tremens, but normal in patients with hepatitis and other forms of liver diseases. The high values in patients with hypothyroidism reflect the muscle changes in this condition. Although CK is found almost exclusively in myocardium, muscle and brain, and early reports suggested it to be an almost specific index of injury of myocardium and muscle, more CK values can occur in patient with pulmonary infraction and pulmonary edema (Henry, 1984).

1.5. Kidney Function

1.5.1 Urea

Urea is the major end product of protein and amino acid catabolism and is generated in the liver through the urea cycle. From the liver urea enters the blood to be distributed to all intracellular and extracellular fluids, since urea is freely diffusible across most cell membranes. Most of the urea is ultimately excreted by kidneys, but minimal amounts are also excreted in sweat and degraded by bacteria in the intestines (Henry, 1984).

1.5.2. Creatine

Creatine is important in muscle metabolism in that it provides storage of high-energy phosphate through phosphocreatine. Creatine is synthesized in a two-step process involving the initial synthesis of guanidoacetate which take places in the kidneys, small intestinal mucosa, pancreas and probably the liver. This reaction between glycine and argis catalyzed by transaminase which is subject to feedback transported to the liver where it is methylated to be distributed, mainly to muscle cells. The body content of creatine is proportional to the muscles mass. Furthermore, creatinine is a hydride of creatine and is formed by a spontaneous and irreversible reaction (Henry, 1984).

1.6 AMINO ACIDS

1.6.1. Amino Acids

Amino acids are essential compounds for the synthesis of proteins, hormones such as thyroxin, epinephrine and serotonin, nitrogenous compounds such as purines and pyrimidines, heme and nitrogenous bases of phospholipids (Freedland and Briggs, 1980).

Amino acids, that are ingested from dietary sources are broken down and transported to the liver. Some of them are transaminated or deaminated to keto-acids, while others are metabolized to urea and ammonia (Laker, 1996). The highest level of amino acids is found in the intestinal tract, liver, pancreas and serum proteins, while the lowest level of amino acids in plasma is present in skeletal muscles and in the skin (Bender, 1975). The concentration of plasma amino acids varies during the day by about 30%. The highest level of amino acid is shown at midafternoon, while the lowest level is at early morning (Norbert, 1987; Freedland and Briggs, 1980).

1.6.2. Metabolism of Amino Acids

The liver and kidneys are active in interconverting amino acids by transamination and deamination (Norbert, 1987). Most tissues contain 20 amino acids and all are needed for growth and repair (Laker, 1996). Amino acids are classified into two main groups according to the necessity for supply from an external source.

a) Essential amino acids that have to be supplied by dietary intake, because endogenous synthesis is inadequate to meet normal requirements. These amino acids are Valine, Leucine, Isoleucine, Methionine, Threonine, Lysine, Phenylalanine, Histidine and Tryptophan.

b) Non-essential amino acids that could be synthesized in the body through various metabolic pathways. These are Glycine, Alanine, Serine, Cysteine, Aspartic acid, Tyrosine, Glutamine, Glutamic acid, Proline, and Arginine. Some other amino acids occur in the body, but are not found in proteins such as ornithine, citrulline and taurine (Henry, 1984; William, 1995).

There is a pair of amino acids, namely serine and glycine, that are their metabolic pathways are closely related to each other. Serine is formed from the glycolytic intermediate 3-phosphoglyceric acid, requiring oxidation, transamination and dephosphorylation. It is a primary component of many phospholipids. Glycine is synthesized from serine after removal of its hydroxyl group through the transhydroxymethylation process. Glycine is required for synthesis of heme that is contained in haemoglobin of blood and in cytochrome system for oxidative phosphorylation process (Freedland and Briggs, 1980).

Threonine is deaminated non-oxidatively by threonine deaminase or dehydratase reaction. Tryptophan is metabolised by a very unstable enzyme tryptophan purrolase, having a half-life in vivo of only about 2hr, so that very fine control of the rate of tryptophan catabolism can be achieved by regulation of the amount of pyrrolase synthesized. The catabolism of proteins containing the two amino acids phenylalanine and methionine is

necessary for the synthesis of tyrosine and cysteine by hydroxylation in the liver (William, 1995; Henry, 1984).

Cysteine can be metabolized to taurine, to which bile acids are conjugated before excretion. Proline and arginine can be synthesized from glutamate semialdehyde (GSA), which is formed from glutamate by reduction with nicotinamide adenine dinucleotide-phosphate (NADPH). Cyclization and reduction of (GSA) forms proline, whereas transamination of (GSA) forms ornithine, which can be converted to arginine in the urea cycle (Freedland and Briggs, 1980). Aromatic amino acids (AAA), such as, phenylalanine, tyrosine and tryptophan, are metabolized by the liver. Branched chain amino acids (BCAA) such as leucine, isoleucine and valine are excreted by the liver and taken-up by the muscles (William, 1995).

1.6.3. Disorders of Amino Acids Metabolism

Disorders of as a metabolism will result in increased concentration of amino acids in blood and their urinary excretion rates (aminoacidurias) (Norbert, 1987). More than fifty hereditary diseases of amino acid metabolism have been described. Most of them are reported to be in an autosomal recessive mode. These diseases are uncommon or rare but, when present, they can cause severe mental retardation, failure to thrive or some other metabolic abnormalities (Ikizler et. al, 1994).

Aminoacidurias can be classified into the following categories based on their causes:

1) An over-flow aminoaciduria, which shows an increased plasma concentration of one or more amino acids, while the kidneys are functioning properly.

2) Renal aminoaciduria, which is associated with increased urinary excretion of one or more amino acids, while plasma amino acids concentration are normal. The defect is in the renal tubular mechanism that causes decreased reabsorption of amino acids from glomerular filtrate. It is either primary due to hereditary defect in renal tubular transport, e.g, Cystinuria or secondary due to acquired renal tubular disease mostly of toxic causes (William, 1995; Norbert, 1978).

Homocystinuria is due to accumulation of homocystine and methionine in the body fluid. It causes mental retardation, lens dislocation, osteoporosis, ocular, skeletal, vascular effects and thromboembolism (Henry, 1984).

Cystinuria is the most common in born error in amino acid transport. It is characterized by massive urinary excretion of cystine, lysine, arginine and ornithine. Because cystine is the least soluble amino acid, its overexertion leads to the formation of renal calculi along the urinary tract. This will be complicated by obstruction, infection and renal insufficiency.

Maple syrup urine disease is an inherited defect of the decarboxylase enzyme responsible for oxidative decarboxylation of alpha keto acids into acyl coenzyme A (acyl-Co-A). This results in accumulation of branched-chain amino acids and corresponding α-keto acids in blood, urine and cerebrospinal fluid. In affected infants, an acute ketoacedosis develops and causes vomiting, lethargy, seizures, coma and mental retardation (Norbert, 1987).

1.6.4. Factors Affecting Plasma Amino Acids Level

a) Portal Systemic Encephalopathy with severe liver disease, that causes inhibition of the oxidative deamination of amino acids, results in an increased concentrations of aromatic amino acids (phenylalanine, tryptophan and tyrosine) and decreased concentrations of branched chain amino acids (leucine, isoleucine and valine) (Gary, 1992).

b) Most studies have shown abnormal plasma free amino acid (PFAA) in patients with cancer and weight loss (Clarke *et al*, 1978; Ching *et al*, 1984; Norton *et al*, 1985). There is a decrease in the level of gluconeogenic amino acids such as alanine, glycine and threonine in malnourished patients with cancer (Clarke *et al*, 1978; Bennegard *et al*, 1984). In small cell lung cancer the plasma concentration of glutamic acid, glutamine, aspartic acid, asparagine and arginine were found to be increased, while plasma concentrations of ornithine, citrulline and methionine were decreased (Russell *et al*, 1981).

c) In patient with acute leukemia, the plasma concentration of alanine, glutamine, histidine, proline, threonine and methionine are increased (Rudman *et al*, 1971).

d) In patients with hepatocellular carcinoma, the plasma concentration of tyrosine, methionine and phenylalanine are increased (Kubota *et al*, 1992).

e) Hemodialysis causes a decrease in the levels of essential amino acid, while the non-essential amino acid levels are increased (Ikizler *et al*, 1994).

CHAPTER II

AIMS AND OBJECTIVES

2.1. AIM

The aim of the present study to determine the Blood Lead Levels (BLLs) among expatriate industrial (exposed) and non-industrial workers (unexposed) and investigate predictor factors influencing BLLs in UAE population. Additionally, the present study aiming to determine the effect of lead con the plasma levels of amino acids and serum liver enzymes, cardiac enzymes and renal functions protein in industrial workers in Al-Ain, UAE. Finally, to study acute and chronic respiratory symptoms occurred due to lead exposure among industrial workers.

2.2. OBJECTIVES

1. To apply a specifically designed questionnaire to industrial and non-industrial workers to collect information on their nature of job and socio-demographic characteristics of their work.
2. To identify some risk factors associated with working conditions which resulting lead as a cause adverse health effect.
3. To determine the Blood Lead Levels (BLLs) in the industrial workers and non-industrial workers and investigate predictor factors influencing BLLs in the present study.
4. To study relationship between BLLs and levels of liver function enzymes, cardiovascular enzymes, and renal function proteins.
5. To study the amino-acid levels in both sets of workers differing in their exposure to factors likely to cause adverse health effects.

6. To investigate significant increase or decrease in urine with levels of exposed industrial workers and non-exposed subjects.
7. To study acute and chronic respiratory symptoms occurred due to lead exposure among industrial and non-industrial workers.
8. To identify suitable monitoring criteria for screening and early detection of damage to their health.
9. To compare the results and prevalence rate with available studies from Gulf States and other Western Countries.

CHAPTER III

MATERIALS AND METHODS

3.1 LOCATION

No study has yet been conducted to define the epidemiology and characteristics of lead exposure among industrial workers in the UAE and the exact prevalence rate of exposure is not known in UAE population. The subjects were selected from the Department of Preventive Medicine in Al-Ain City.

3.2 STUDY POPULATION AND DETAILS

Male industrial and non-industrial workers were targeted who were attended to the Preventive Medicine Department in Al-Ain for the renewal of their residence visa. The study was conducted between February –June 1999. The purpose of this survey was explained to the industrial workers and non-industrial workers. Those who agreed to participate was asked to sign the consent form and a questionnaire were applied to them through qualified physicians, nurses and investigators.

3.3. STUDY DESIGN

It is vital to obtain an accurate base for considering environmental influences on lead. This study was based on matched study of the lead exposed and non-exposed subject selected from Al-Ain City. This case-control study consisted of 100 exposed and 100 unexposed, matched for age, sex and nationality. This study was conducted during a period between from February to June, 1999.

This research study was performed by the following three steps:

a). An epidemiological study: socio-demographic, lifestyle and Blood Lead level.

b). Laboratory analysis of plasma amino acids.

c). The analysis of serum liver enzymes, cardiac enzymes and renal functions.

3.3.1. An Epidemiological Study: Socio-demographic, Life Style And BLLs

In this part of the study a brief questionnaire was used to collect information on socio-demographic characteristics of the target population. The questionnaire was administered to male industrial workers and manual semi-skilled workers attending the Preventive Medicine Department in Al-Ain City (See Appendix Questionnaire).

Exposed - Unexposed Comparison

Workers were classified as exposed or unexposed according to the following criteria: (Exposed): This group consisted 100 male industrial-workers those using working in heavy industrial production or industrial works.

(Unexposed): The 100 male were working in manual jobs, skilled or unskilled professional workers, but not in industrial works, or similar jobs in urban area. A specifically designed questionnaire was applied to the selected non-industrial workers to elicit the required information on the following topics:

a) Questionnaires about socio-demographic data

b) Work history

c) Job practices during the work shift

d) Patterns of ill-health and any information on medications taken

e) Dietary habits and life style

f) The Symptoms occurring among exposed workers

Information on the frequency of use of chemicals, use of protective equipment and degree of exposure was collected from all exposed subjects. Both exposed and unexposed were drawn from the same geographic region for controlling environmental and other geographical exposures.

Pre-tested, verbally administered questionnaires obtained information about socio-demographic characteristics, work history, occupational chemicals or toxic materials exposure within a week of enrollment. Industrial and non-industrial workers were questioned about domestic and occupational exposure to chemicals or toxic gases. A fairly comprehensive range of symptoms was included in the questionnaire.

3.3.2. Laboratory Analysis

3.4.1. Plasma Amino Acids analysis

Sulphosalicylic Acid 50% was prepared by dissolving 50 grams (gm) of sulphosalicylic acid powder into 100 milliliters (ml) of buffer Lithium-S and stored at 4°C. 2ml of venous blood sample were collected in a lithium habarinised bottle and centrifuged immediately for five minutes at 2000 rotation per minutes (rpm). 200 microlitres (µl) of the plasma was deproteinised by adding 20 µl of sulphosalicylic acid solution (50%) and centrifuged at 10,000 rpm for 10 minutes at + 4 ° C. The supernatant was stored at -70 °C until analysis,

then was diluted (1:1) with Li-S Beckman buffer along with the internal standard. The supernatant was thawed and analysis made for individual free amino acids using Spackman, according to the method of Moore and Stein ion exchange chromatography techniques on Beckman 6300 automatic amino acid analyzer was performed: A three Li-buffer system is used with a cation exchange resin column 10 X 0.4 internal diametres (I.D) centimetres (cms) at around 1200 (psi) and at a flow rate of 20 milliliters per hours (ml/hr). The method is based on the post column derivatisation by ninhydrin colouring reagent at 135°C. Derivatised amino acids were detected at a wavelength of 570 nanometres (nm), while proline and hydroxyproline at 440 nm (Spackman, 1958, Beshwari et. al. 1999a).

This analysis was carried out in the research laboratory of Faculty of Medicine and Health Sciences using the reagents provided by the Faculty of Sciences, at the U.A.E. University. All amino acids were measured by the analyzer. The list of essential amino acids are: Threonine, Valine, Lysine, Methionine, Isoleucine, Leucine, Histidine, Phenylalanine & Tryptophan and the non-essential amino acids are: Serine, Glutamic acid, Glutamine, Proline, Glycine, Alanine, Cysteine, Tyrosine, Arginine, Ornithine, Taurine and Citrulline.

3.4.2. Liver Function Tests, Cardiac Enzymes and Renal Function

4 ml of venous blood sample was collected in a non-coagulate plain tube (10-ml) and centrifuged at 3500 rpm. The serum was stored at -20°C until analysis. The standard liver function tests such as albumin (Alb), total protein (T.P), total bilirubin (T.Bil), direct bilirubin (D.B), alanine aminotransferase (ALT), aspartate aminotransferase (AST), lactate

dehydrogenase (LDH), alkaline phosphatase (ALP), Gamma Gulatmyl transferas (GGT) were analyzed by a SYNCHRONE (CX5, CX7) Analyzer; a fully automated, computerized, discrete, selective, random-access clinical chemistry analyzer with an ion selective electrode mode. This system performs a wide range of analyses using both potentiometric and photometric assays. All reagents, calibrators and quality control materials were supplied by Al Zahrawi Company. This analysis was carried out in Al Ain Hospital, Clinical Chemistry Departments Laboratory.

On SYNCHRONE, the Total Proteins were estimated by the Biuret method and measured at 546 nm, (Weichselbaum *et al*, 1946). Albumin was done by Bromocresol green method and measured at 600 nm, (Dumas *et al*, 1971). Bilirubin is coupled with diazonium compound then measured at 570 nm (Wahleteld *et al*, 1972). Transaminase was measured potentiometrically at 340 nm (Bergmeyer et. al. 1986). Alkaline phospwas measured by 4-nitrophenyl-phosphate method at 405 nm (Expert Panel On Enzymes, 1983). Lactate dehydrogenase was measured by the optimized standard method, (Anon, 1970). GGT is measuredc by SZASZ method at 410 nm, creatine kinase is measured by DGKC method at 340 nm, urea is measured by UV Rate method at 340nm, and creatinin is measured by Colorimetry/Alkaline Picrate at (520-560 nm). In the first part of the analysis, employment as a industrial-worker was taken as an indicator of the potential for lead exposure. Amino acid and liver enzymes levels were examined among industrial workers and the comparison group of non-industrial workers, and their mean amino acid levels and liver enzymes levels were compared.

3.4.3 Blood Lead Analysis:

PREPARATION OF STOCK SOLUTION

Dissolve 1.000 g of lead metal in 50 ml of 2M nitric acid. Dilute to 1 liter in a volumetric flask with deionised water. Store in a polythene bottle.

Or dissolve 1.5980 g of lead nitrate $(Pb(No_3))_2$ in 100 ml of deionised water.Dilute to 1 litre in a volumetric flask with deionised water. Store in a polythene bottle.

Organo-Metallic Standard

Lead cyclohexnebutyrate (4-cyclohexylbutyric acid lead salt or lead 4-cyclohexylbutyrate). Stock solution was prepared to draw the standard curve. Blood lead was analyzed by Atomic Absorption Spectrophotometer (AAS) in Department of Chemistry, Faculty of Sciences, UAE University.

About 1 ml of hebarinized samples was collected from both workers, centrifuged and analyzed by AAS.

Philips PU 9100X (Byunkan) Atomic Absorption Spectrophotometer equipped with a graphite furnace and Zeeman background correction system was used in conjunction with a programmable Varian autosampler. The hollow cathode Pb lamp and the pyrolytically coated partition graphite tubes were purchased from Varian. The analytical resonance wavelength at 283.3 nm and a slit width of 0.5 nm were utilized for all analyses. A current of 5 ma was applied to the hollow cathode Pb lamp. The graphite furnace program used for all lead determinations. Extra steps were employed at the termination of the

atomization furnace program to allow the graphite tube to cool slowly to 40 ^{0}C and thus prevent spattering of the subsequent injection. Along with this modification, an extended dispensing rate (with heated injection, 40 ^{0}C) was used to ensure that all the dispensed liquid would be slowly introduced to be contained within the platform cavity for subsequent temperature programming. Interference of excess of Al, Si, Sr, Mg and Ca can be overcome by use of matrix modifier such as ammonium nitrate, lanthanum nitrate or ascorbic acid or platform or probe atomization. The sensitivity may be improved to. 0.3mg/l Slotted Tube Atom Trap.

Theory Of Atomic Absorption Spectrophotometer

A **hollow cathode lamp** consists of two electrode sealed in glass envelope filled with an inert gas, usually argon or neon. The end windows of the lamp must be of an appropriate material in order to transmit the emitted radiation and is either quartz or silica. The cathode of the lamp, usually cup shaped, is either made of the element whose spectrum is required or coated with the element, and the application of potential of between 300 and 400 V is usually required to cause excitation of atoms and discharge of the appropriate radiation.

For certain elements, **Electrodes Discharge Lamps (EDL)** have been designed in which the excitation of atoms is achieved by radio frequencies inducing resonance effects, the energy liberated causing vaporization and excitation of the element. Their use permits more critical control of the emission and the modulation of the impulse can be more easily achieved.

Double beam atomic absorption spectrophotometers are designed to control variations which may occur in the radiation source but are not as valid as double beam molecular absorption instruments because there can be blank sample in flame techniques.

The **flame** as well as containing the unexcited atoms of the element will also emit radiation due to the thermal excitation of a small proportion of the atoms and it is essential that the detector is capable of distinguishing between the identical radiation which is transmitted by the flame and that which is emitted from the flame. This is achieved by introducing a characteristic signal or modulation into the incident radiation by means of rotating segmented mirror or an electrically induced pulse.

High quality monochromating systems are necessary to isolate the required emission line of the element from those emission lines due to the gases, which are also present in the lamp. Owing to the very narrow bandwidth of atomic emission lines, it is not adequate simply to select the required wavelength using the monochromator scale and a procedure known as peaking up has to be undertaken (Holme et. al. 1993).

3.5. STATISTICAL METHODS AND ANALYSIS

3.5.1. Data Processing

All data variables were coded, processed and analyzed on the IBM computer of the Department of Community Medicine, Faculty of Medicine and Health Science at the United Arab Emirates University. Data entry was performed using SPSS. The Statistical Software Package SPSS [Statistical Package for Social Science, Norusis, 1996] analysis was used for performing all statistical analysis. Also, Harvard Graphic Package was utilized for graphing such as bar chart, histogram, line chart and pie chart

3.5.2. Statistical Methods and Analysis

The Statistical Packages for Social Sciences [SPSS], Norusis (1992) used for univariated and multivariate statistical analysis. Data are expressed as mean and standard deviation (SD) unless otherwise stated. Chi-square analysis was performed to test for differences in the proportion of categorical variables between two or more groups. In 2 x 2 tables, the Fisher's exact test (two-tailed) replaced the chi-square test if the assumptions underlying chi-square were violated, namely in case of small sample size and where the expected frequency is less than 5 in any of the cells. Student T-test was used to ascertain the significance of differences between mean values of two continuous variables and confirmed by Mann-Whitney test for non-parametric distribution. One-way analysis of variance (ANOVA) was employed for comparison of several group means and to determine the presence of significant differences between group means of continuous

variables. The Pearsons rank correlation coefficient was used to evaluate the strength association between two continuous variables. Odds ratio (OR) and their 95% confidence intervals (CI) was calculated by using Mantel-Haenzel test (EPI6 INFO Version 6). The level $p < 0.05$ was considered as the cut-off value for significance.

CHAPTER IV

RESULTS

4.0 PRESENTATION OF RESULTS

The study results will be presented as follows:

4.1 - An Epidemiological Study Of Eating Habits, Life Style, Reported Symptoms and Blood Lead Level among Exposed and Unexposed workers.

4.2 - Plasma Amino acids.

4.3 - Liver Function Test & Cardiac Enzymes and Renal Function Protein

4.1 An Epidemiological Study Of Eating Habits, Life Style, Reported Symptoms and Blood Lead Level among Exposed and Unexposed workers.

Table 1. Shows the characteristic of industrial and non-industrial workers among the population surveyed. As can be seen from this table there was no significant difference between industrial and non-industrial workers with respect to age ($p = .592$). Fig.1 presents the percentages of age groups among industrial and non-industrial workers. Over 55% of the industrial workers are above 35 years. The nationality was forming - Indian Subcontinent (78%) and Arabs (22%) - There was statistically significant difference in nationality of both groups ($p = .003$) Indian Subcontinent workers represented the majority among industrial (78%) and non-industrial workers (61%), (Fig. 2).

There was statistically significant differences between industrial and non-industrial workers with respect to level of education ($p = .004$). While the majority of industrial had no or low level of education, the non-industrial workers slightly shifted towards a level of education as can be seen in (Fig. 3).

Also, Table 1 shows the housing condition among industrial and non-industrial workers, there was statistically significant differences between both groups, ($p < .0001$). (Fig. 4) shows that most of the industrial workers (95%), and non-industrial workers (67%) are living in mud\Pre-fabric houses. With regard to marriage status, the majority of both groups were married (81%) in industrial workers and (77%) in non-industrial workers (Fig. 5).

Table 2. Gives dietary habit and life style among industrial and non-industrial workers. Indian food and Arabic food were favored over Western food (Fig. 6). Both industrial (67%) and non-industrial workers (72%) used vegetable oil in cooking (Fig.7).

The majority of both groups are using tap water for drinking, (85%) among industrial workers and (73%) among non-industrial workers (Fig. 8).

Table 3. Shows milk consumption among industrial and non-industrial workers. There was no statistically significant differences between industrial and non-industrial workers in milk consumption ($p = 0.230$) (Fig 9).

(Fig. 10) Shows that (30%) of industrial workers and (32%) of non –industrial workers reported consumption of canned food.

There were no statistically significant differences in both groups according to smoking habits (Fig 11) and drinking alcohol.

Fig.12 presents chemical pesticide usage among industrial workers (23%) and non-industrial workers (1%).

Figures 13-14 show industrial toxic gas and radiation or magnetic exposure among industrial and non-industrial workers, toxic gases exposure was (40%) among industrial workers and (6%) among non-industrial workers, and radiation among industrial workers and (6%) among non-industrial workers, and radiation or magnetic exposure was (13%) among industrial workers and (1%) among non-industrial workers.

Table 4. Gives blood lead level among industrial and non-industrial Workers. It shows that blood lead level in industrial (exposed) workers is higher than non-industrial (unexposed) workers with statistically significant difference ($p<0.0001$).

Reported symptoms among industrial and non-industrial are presented in Table 5.It will be noticed that most symptoms occurred among industrial workers and non-industrial workers with statistically significant differences: Nausea/vomiting [OR = 4.235, 95% CI (1.35-13.25)], (p = 0.008), Kidneys [OR = 3.20, 95% CI (1.20-8.51)],(p = 0.015)], increased anxiety [OR = 2.68, 95% CI (1.16-6.20)],(p=0.017),Gastrointestinal [OR=2.55,95% CI (1.71-5.55),P=(0.016), Muscular symptoms [OR = 3.146, 95% CI (1.46-6.75)], (p= 0.002), Anemia [OR = 3.43, 95% CI (1.30-9.07)], (p = 0.009), Fatigue [OR= 3.439, 95% CI (1.303-9.074)], (p = 0.009),Memory loss [OR = 11, 95% CI (1.38-87.64)], (p = 0.005) , Difficulty breathing [OR=2.72, 95% CI (1.07-6.88),(P=0.030), Mania [OR = 6.62, 95% CI (1.87-23.39)], (p = 0.0016), Difficulty breathing [OR = 2.72, 95% CI (1.07-6.88)], (p = 0.030), Cardiovascular [OR = 3.61, 95% CI (1.27-10.30)], (p = 0.011).

Table 6. Presents respiratory symptoms among industrial and non-industrial workers. There were statistically significant differences between industrial and non-industrial workers in most of the respiratory symptoms. Hay fever was higher in industrial workers (36%) than non-industrial workers (12%), (p=0.0001), Cough up phlegm was statistically significant difference between both groups (p=.025), in industrial workers (19%) and non-industrial workers(8%).

Also asthma in industrial workers (20.0%) was higher than non-industrial workers (5%), (p = 0.001)., eczema in industrial workers (16.0%) was more prevalent than non-industrial (7.0%), (P= 0.046).

4.2. Plasma Amino acids

Table 7. Shows plasma amino acid profiles among industrial and non-industrial workers. It will be observed that most amino acid levels showed higher values among industrial than non-industrial workers. There were statistically very highly significant differences between industrial non-industrial with the respect of some amino acids levels as can be seen as follows: Taurine (112.02 ± 38.70 µmol/l), Mean ± S.D, N= 100 in industrial workers and (55.15 ± 24.97 µmol/l, Mean ± S.D) in non-industrial workers, N= 100, (p = 0.0001), Serine (159.33 ± 34.68 µmol/l), Mean ± S.D in industrial workers and (108.09 ± 26.42 µmol/l, Mean ± S.D) in non-industrial workers, (p = 0.003), Glutamic acid (236.89. ± 149.91 µmol/l, Mean ± S.D) in industrial workers and (82.26 ± 57.09 µmol/l) in non-industrial workers, (p = 0.000), Glycine (236.89 ± 149.91 µmol/l, Mean ± S.D) in

industrial workers and (244.25 ± 72.88 µmol/l, Mean ± S.D) in non-industrial workers,(p = 0.0001), Histidine (107.50 ± 29.61 µmol/l ,Mean ± S.D) in industrial workers and (95.90 ± 18.53 µmol/l, Mean ± S.D) in non-industrial workers, (p=0.008), Ornithine (178.94 ± 59.16 µmol/l, Mean ± S.D) in industrial workers and (104.84 ± 30.64 µmol/l, Mean ± S.D) in non-industrial workers, (p = 0.001), Lysine (218.71 ± 51.73 µmol/l, Mean ± S.D) in industrial workers and (176.77 ± 36.71 µmol/l, Mean ± S.D) in non-industrial workers, (p = 0.006), Tryptophan (69.99 ± 20.06 µmol/l, Mean ± S.D) in industrial worker and in non industrials workers (74.02 ± 26.23 µmol/l, Mean ± S.D), (p = 0.0001), Leucine (162.41 ± 38.73 µmol/l, Mean ± S.D) in industrial workers and (135.57 ± 26.94 µmol/l, Mean ± S.D) in non-industrial workers(p = 0.002), and Threonine (161.67 ± 42.04 µmol/, Mean ± S.D) in the industrial workers and in non-industrial workers Mean ± S.D (130.34 ± 34.55 µmol/l), (p = .055), Lysine Mean ± S. D (218.71± 51.73 µmol/l,(Mean ± S.D) in industrial workers and Mean ± S. D (176.77 ± 36.71 µmol/l , Mean ± S.D) in non industrial workers ,(p=0.006), Valine (264.15 ±65.88 µmol/l , Mean ± S.D) in industrial workers and (234.05 ±58.02 µmol/l, Mean ± S.D) in non industrial workers, (p=0.62), Alanine (526.59 ± 151.35 µmol/l, Mean ± S.D) in industrial workers and (417.64 ± 92.08µmol/, Mean ± S.D) in non industrial workers, (p=0.001), Glutamin (622.86 ±139.85 µmol//, Mean ± S.D) in industrial workers and (583.89 ± 146.50 µmol/ Mean ± S.D) in non industrial workers, (p=.346) and Proline (339.31± 137.29 µmol/ Mean ± S.D) in industrial workers and (261.15 ± 82.464 µmol/, Mean ± S.D) in non industrial workers (p=0.006).

Table 8. Shows the association between Blood Lead Level and amino acid components among industrial and non-industrial workers. As can be seen from this table there is a strong correlation between Blood lead level and amino acid.

Table 9. Shows the association between Blood Lead and amino acids components among industrial workers. It will be observed that only Tryptophan (p=0.013) was statistically significant in association with Blood Lead Level (p=0.013).

4.3 Liver Function Test, Cardiac Enzymes and Renal Functions Protein

Table 10. Gives liver function test, carenzymes and renal function proteins among industrial and non-industrial workers. Liver function tests were performed on industrial and non-industrial workers. Only Alkaline Phosphatase and Lactate Dehedrogenase are statistically significant differences with respect to other enzymes. Lactate Dehydrogenase (211.27 ±57.27 u/l, Mean ± S.D), N = 100; in industrial workers versus (194.77 ± 48.69 u/l, Mean ± S.D), N = 100 in non industrial workers, (p = 0.029). Alkaline Phosphates (84.27 ± 24.64 u/l) for industrial workers and (76.18 ± 20.48 u/l) for non- industrial workers (p=0.012).

Cardiac Enzymes were performed among industrial and non-industrial workers with out any statistically significant differences.

Also Renal Functions Protein were performed among industrial and non-industrial without any statically significant difference.

Table11 shows correlation between Blood Lead Level and liver function test, cardiac enzymes and renal functions protein among industrial and non-industrial workers. Only LDL, TP, and CK showed significant association with Blood Lead Level.

Table 12 presents correlation between Blood Lead Level and liver function test, cardiac enzymes and renal functions protein among industrial workers only. It will be observed only Crea and CK variables are significant in association with Blood Lead Level.

Table 1: The characteristics of Industrial and non-Industrial among population surveyed

Variable	Industrial workers N=100 (%)	Non industrial workers N=100 (%)	P-value significance
Age groups			NS
< 25	17 (17.0)	12 (12.0)	
25-35	46 (46.0)	44 (44.0)	
35-45	26 (26.0)	28 (28.0)	
> 45	11 (11.0)	16 (16.0)	
Nationality			0.003
Indian Subcontinent	78 (78.0)	61 (61.0)	
Arab	22 (22.0)	39 (39.0)	
Education			0.004
Illiterate	38 (38.0)	25 (25.0)	
Primary	32 (32.0)	27 (27.0)	
Secondary/High	30 (30.0)	48 (48.0)	
Housing condition			
Mud/Pre-fabric	95(95)	67(67)	0.0001
Flat	5 (5)	33 (33)	
Marital status			NS
Single	19 (19.0)	23 (23.0)	
Married	81 (81.0)	77 (77.0)	

NS= Not Significant.

Table 2: Dietary habits and life style among industrial and non-industrial workers.

Variable	Industrial workers N=100(%)	Non industrial workers N=100(%)	P-value significance
Type of food			
Arabic food	22 (22.0)	47 (47.0)	.0001
Indian food	78(78.0)	53 (53.0)	
Type of cooking oil used			
Vegetable	67 (67.0)	72 (72.0)	NS
Olive	4 (4.0)	7 (7.0)	
Animal fat/butter	29(29.0)	21 (21.0)	
Smoking cigarette			
Never	64 (64.0)	77 (77.0)	
Ex-smoker	16 (16.0)	12 (12.0)	NS
Current smoker	19 (19.0)	11 (11.0)	
Drinking alcohol			
Yes	2 (2.0)	2 (2.0)	NS
No	98 (98.0)	98 (98.0)	
Eating canned food			
Yes	30 (30.0)	32 (32.0)	NS
No	70 (70.0)	68 (68.0)	
Sources of drinking water			
Tap water	85 (85.0)	73 (73.0)	NS
Well water	3 (3.0)	0 (0.0)	
Natural water	1(1.0)	0 (0.0)	
Bottled spring water	10(10.0)	23(23.0)	

NS = Not -Significant

Table 3: Milk consumption among industrial and non-industrial workers.

Variable	Industrial Workers N=100 (%)	Non-Industrial workers N=100 (%)	P-value Significance
Non drinkers	29 (29 0)	24(24.0)	
Occasionally	40(40.0)	33 (33.0)	NS
More than once week	17 (17.0)	29(29.0)	
Daily	14(14.0)	13(13.0)	

NS = Not- Significant

Table 4: Blood Lead Levels (μg/dl) among industrial and non-industrial workers.

Basic Statistics	Industrial Workers N=100 (%)	Non-Industrial workers N=100 (%)
Mean	77.56	19.84
Median	81.40	11.00
Std. Error of Mean	4.28	2.46
Geometric Mean	62.46	13.21
Variance	18.40	5.9
Minimum	6.0	2.0
Maximum	188	163
Range	182	161
Mean ± Standard Deviation	77.56 ± 42.85	19.84 ± 24.61*

* There is statically significant differences between industrial and non-industrial workers with respect of Blood Lead Levels (μg/dl),($p<0.0001$)

Table 5: Reported symptoms among industrial and non-Industrial workers.

SYMPTOMS	Industrial workers N =100 Yes (%)	Non Industrial workers N=100 Yes (%)	Odds ratio [DR]	confidence interval 95% [CI]	P-value Signf.
NAUSEA/VOMITING	15 (15.0)	4 (4.0)	4.23	1.31-13.25	0.008
KIDNEYS	17(17.0)	6(6.0)	3.20	1.20-8.51	0.015
RED/ IRRITATED EYE/ BLURRED VISION	18(18.0)	12 (12.0)	1.61	0.73-3.55	NS
GASTROINTESTINAL	24(24.0)	11(11.0)	2.55	1.17-5.55	0.016
INCREASED ANXIETY	21 (21.0)	9 (9.0)	2.68	1.16-6.20	0.017
DIZZINESS	16 (16.0)	9 (9.0)	1.92	0.80-4.59	NS
HEADACHE	26 (26.0)	23 (23.0)	1.17	0.61-2.24	NS
MUSCULAR SYMPTOMS	28 (28.0)	11 (11.0)	3.14	1.46-6.75	0.002
CHEST PAIN	11 (11.0)	11 (11.0)	1.0	0.41-2.42	NS
ANEMIA	18(18.0)	6(6.0)	3.43	1.30-9.07	0.009
DIFFICULTY BREATHING	17 (17.0)	7 (7.0)	2.72	1.07-6.88	0.030
MEMORY LOSS	10(10.0)	1(1.0)	11	1.38-87.64	0.005
CARDIOVASCULAR	16(16.0)	5(5.0)	3.61	1.27-10.30	0.011
FATIGUE	18(18.0)	6 (6.0)	3.43	1.30-9.07	0.009
MANIA	17(17.0)	3(3.0)	6.62	1.87-23.39	0.0016
INSOMNIA	14 (14.0)	7(7.0)	2.16	0.83-5.61	NS
ABDOMINAL PAIN	15(15.0)	13(13.0)	1.18	0.53-2.63	NS
MYALGIA AND ANOREXIA	5 (5.0)	4(4.0)	1.26	0.32-4.84	NS

NS = Not -Significant

Table 6: Reported respiratory symptoms among Industrial and Non-Industrial workers.

Respiratory symptoms		Industrial workers N=100 (%)	Non Industrial workers N=100 (%)	P-value Significance
Do you have cough				NS
Never		74 (74.0)	78 (78.0)	
At night		9 (9.0)	8 (8.0)	
Early morning		4 (4.0)	7 (7.0)	
During work		6 (6.0)	7 (7.0)	
Cough up phlegm	Yes	19(19)	8(8)	0.025
	No	81 (81.0)	92 (92.0)	
Throat discomfort				NS
	Yes	10 (10.0)	9 (9.0)	
	No	90 (90.0)	91 (91.0)	
Chest wheezing				NS
	Yes	16 (16.0)	8 (8.0)	
	No	84 (84.0)	92 (92.0)	
Asthma diagnosis by doctor				0.001
	Yes	20 (20.0)	5 (5.0)	
	No	80 (80.0)	95 (95.0)	
Hay fever				0.0001
	Yes	36 (36.0)	12 (12.0)	
	No	64 (64.0)	88 (88.0)	
Eczema				0.046
	Yes	16 (16.0)	7 (7.0)	
	No	84 (84.0)	93 (93.0)	

NS = Not-Significant

Table 7: Plasma amino acid profiles among industrial and non-industrial workers.

Amino Acid Essential	Industrial workers N=100 Mean ± St.Dev Con. μmol/l	Non-Industrial workers N=100 Mean ± St.Dev Con. μmol/l	t-value	p-value significance
Histidine	107.50±29.61	95.90±18.54	3.33	0.008
Isoleucine	85.49±22.61	77.74±16.83	2.749	0.005
Leucine	162.41±38.73	135.57±26.94	5.689	0.002
Lysine	218.71±51.73	176.77±36.71	3.19	0.006
Methionine	34.14±9.40	38.05±8.37	-3.105	NS
Phenylalanine	76.63±17.50	68.74±62.94	1.207	NS
Threonine	161.67±42.04	130.34±34.55	5.758	NS
Valine	264.15±65.88	234.05±58.02	3.429	NS
Arginine	73.27±28.59	77.15±30.54	-.926	NS
Amino Acid Non-Essential				
Tryptophan	48.69±20.91	46.23±11.06	1.037	0.0001
Alanine	526.59±151.35	417.64±92.08	6.149	0.001
Cysteine	27.58±15.62	29.23±9.84	-.892	0.0001
Citrulline	43.68±18.99	40.11±13.38	1.536	0.004
Glutamic acid	236.89±149.91	82.26±57.09	9.639	0.0001
Glutamine	622.86±139.85	583.89±146.50	0.95	NS
Glycine	331.4±86.0	224.24±72.87	7.711	0.0001
Ornithine	178.94±59.16	104.84±30.64	11.123	0.0001
Proline	339.31±137.29	261.15±82.464	4.880	0.006
Serine	159.33±34.68	108.09±26.42	11.753	0.003
Taurine	112.02±38.70	55.15±29.97	12.346	0.0001
Tyrosine	69.99±20.06	74.02±26.03	-1.219	NS

NS = Not - Significant

Table 8: Association between Blood Lead level (μg/dl) and Amino Acids among Industrial and non-Industrial workers

	Pearson Correlation Pb	Sig. (2-tailed) N=200
Taurine	0.368	0.0001
Theronine	0 224	0.001
Serine	0 408	0.000
Glutamic acid	0.448	0.000
Glutamine	-0 147	0 038
Glycine	0.309	0.000
Alanine	0.263	0.000
Valine	0.165	0.020
Cysteine	-0.117	NS
Isoleucine	0.137	NS
Leucine	0.256	0.000
Orinthin	0.396	0.000
Lysine	0.236	0.001
Histidine	0.116	NS
Tryptophan	-0.079	NS
Argnine	0.033	NS
Methionine	-0.132	NS

NS= Not-Significant

Table 9: Association between Blood Lead Level (μg/dl) and amino acids among Industrial workers

	Pearson Correlation Pb	Sig. (2-tailed) N=100
Valine	-0.082	NS
Methionine	-0.160	NS
Isoleucine	-0.061	NS
Leucine	-0.060	NS
Phenylalanine	0.063	NS
Lysine	-0.148	NS
Histidine	-0.104	NS
Arginine	-0.076	NS
Threonine	-0.070	NS
Serine	-0.013	NS
Alanine	-0.011	NS
Taurine	-0.105	NS
Tryptophan	-0.248	0.013
Cysteine	-0.177	NS
Citruline	0.039	NS
Glutaminc Acid	0.183	NS
Glutamine	-0.180	NS

NS = Not-Significant

Table 10: Plasma liver function test, Cardiac enzymes and Kidney Functions Protein among Industrial and non-Industrial workers

Enzymes	Industrial workers N=100 Mean ± St.Dev	Non-Industrial workers N=100 Mean ± St. Dev	t-value	p-value Significance
Total Protein g/l	8.38±.90	8.24±.71	1.220	NS
Albumin g/l	4.90±.52	4.85±.44	0.827	NS
Total Bilirubin mg/dl	.75±.30	.70±.19	1.270	NS
Aspartate Transferase u/l	31.89±12.31	30.72±13.26	0.646	NS
Alanine Transferase u/l	33.25±27.62	33.50±24.22	-.068	NS
Alkaline Phosphatase u/l	84.27±24.64	76.18±20.48	2.525	0.012
Lactate Dehydrogenase u/l	211.27±57.27	194.77±48.68	2.195	0.029
Direct Bilirubin mg/dl	.13±9.58E-02	.12±4.4E-02	1.326	NS
GammaGlutmyl Transferase u/l	36.31±35.27	31.36±17.96	1.251	NS
Blood Urea Nitrogen mg/dl	15.48±4.12	15.16±3.70	.586	NS
Crea mg/dl	1.1±.17	1.1±.15	-1.720	NS
Creatine Kinase u/l	191.62±122.82	189.76±96.92		NS

NS = Not-Significant

Table 11: Correlation between Blood Lead Levels (μg/dl) and LFT among Industrial and non-industrial workers

	Pearson Correlation Pb	Sig.(2-tailed) N=200
BUN	0.109	NS
CREA	0.035	NS
DBIL	0.015	NS
TBIL	0.015	NS
ALB	0.059	NS
TP	0.156	0.028
ALT	0.014	NS
AST	0.070	NS
ALP	0.094	NS
LDL	0.202	0.004
CK	0.157	0.027
GGT	0.043	NS

NS = Not-Significant

Table 12: Correlation between Blood Lead Level (μg/dl) and LFT among Industrial workers

	Pearson Correlation Pb	Sig. (2-tailed) N=100
Bun	0.123	NS
Crea	0.207	0.039
DBIL	-0.037	NS
TBIL	-0.024	NS
ALB	0.001	NS
TP	0.071	NS
ALT	-0.015	NS
AST	0.054	NS
ALP	-0.020	NS
LDL	0.163	NS
CK	0.197	0.050
GGT	-0.095	NS

NS = Not-Significant

Fig 1: Age groups of industrial & non-industrial workers

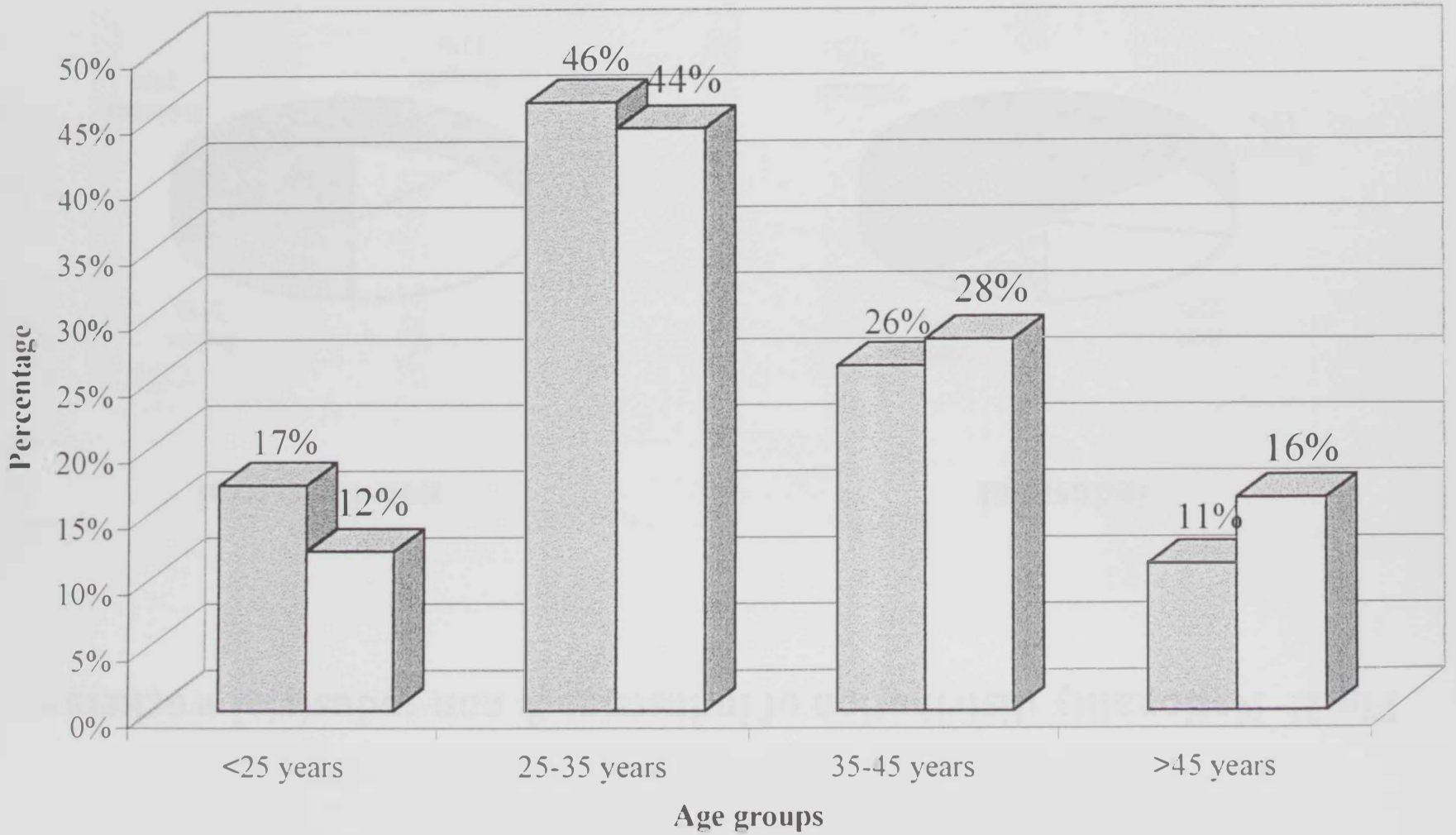


Fig 2: Nationality distribution of industrial & non-industrial workers

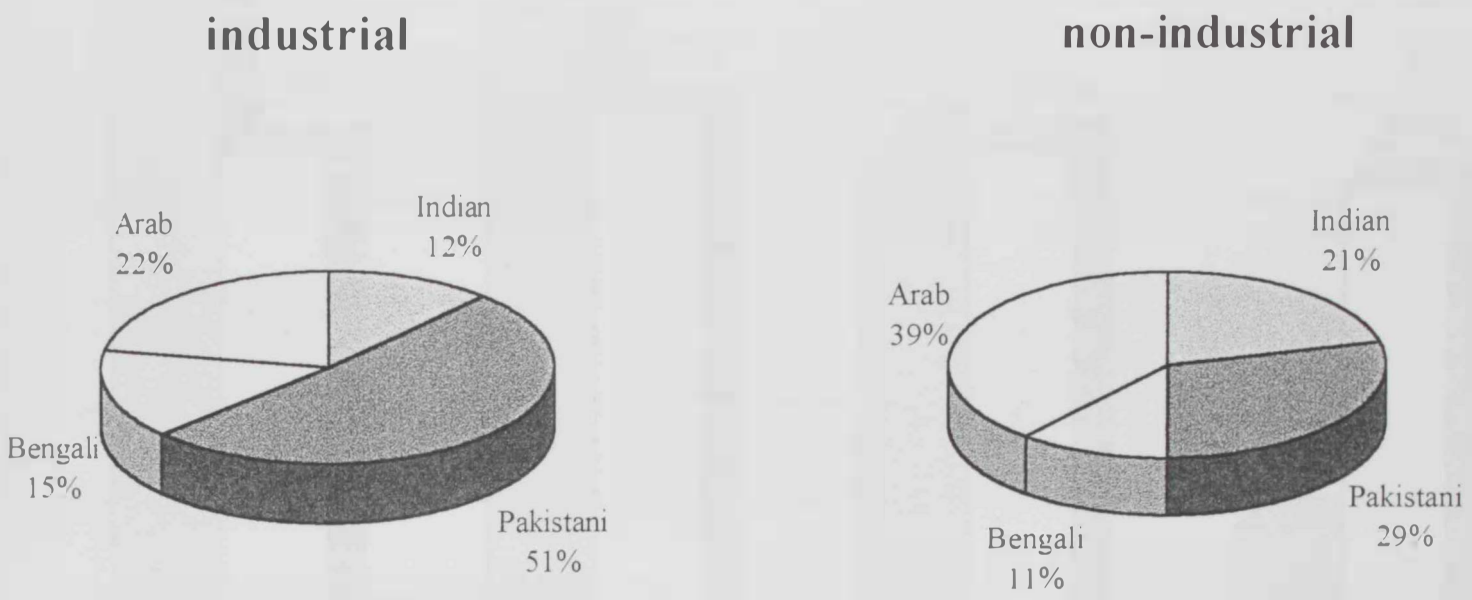


Fig 3: Education level of industrial & non-industrial workers

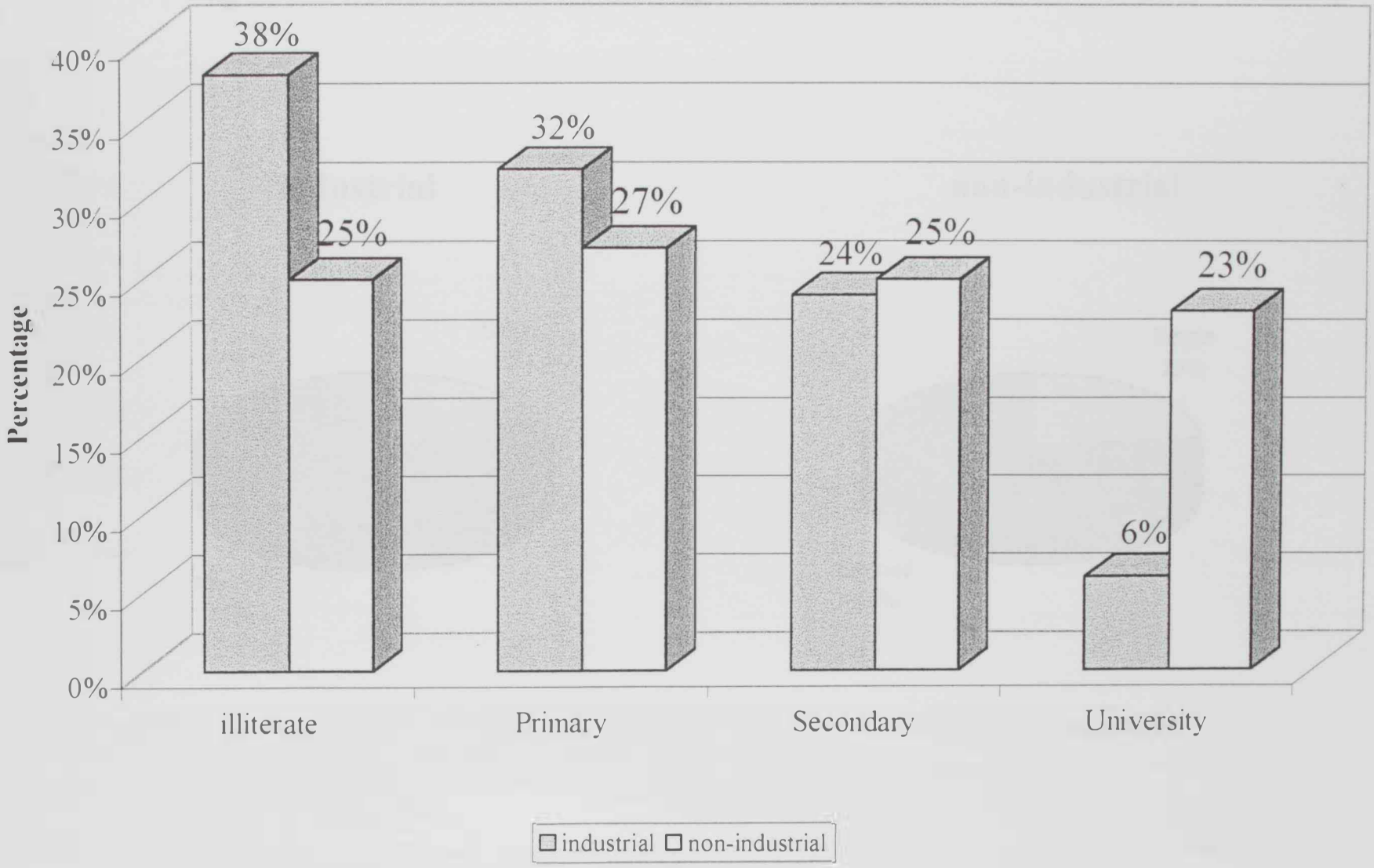


Fig 4: Marital status among industrial & non-industrial workers

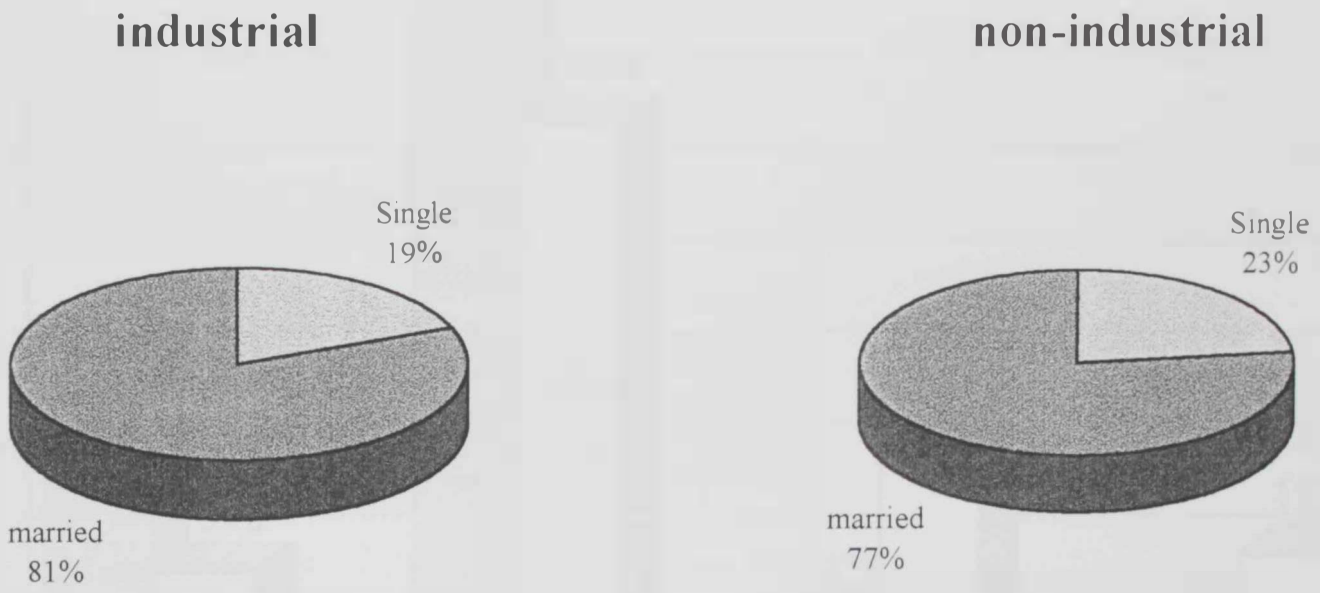


Fig 5: Housing condition among industrial & non-industrial workers

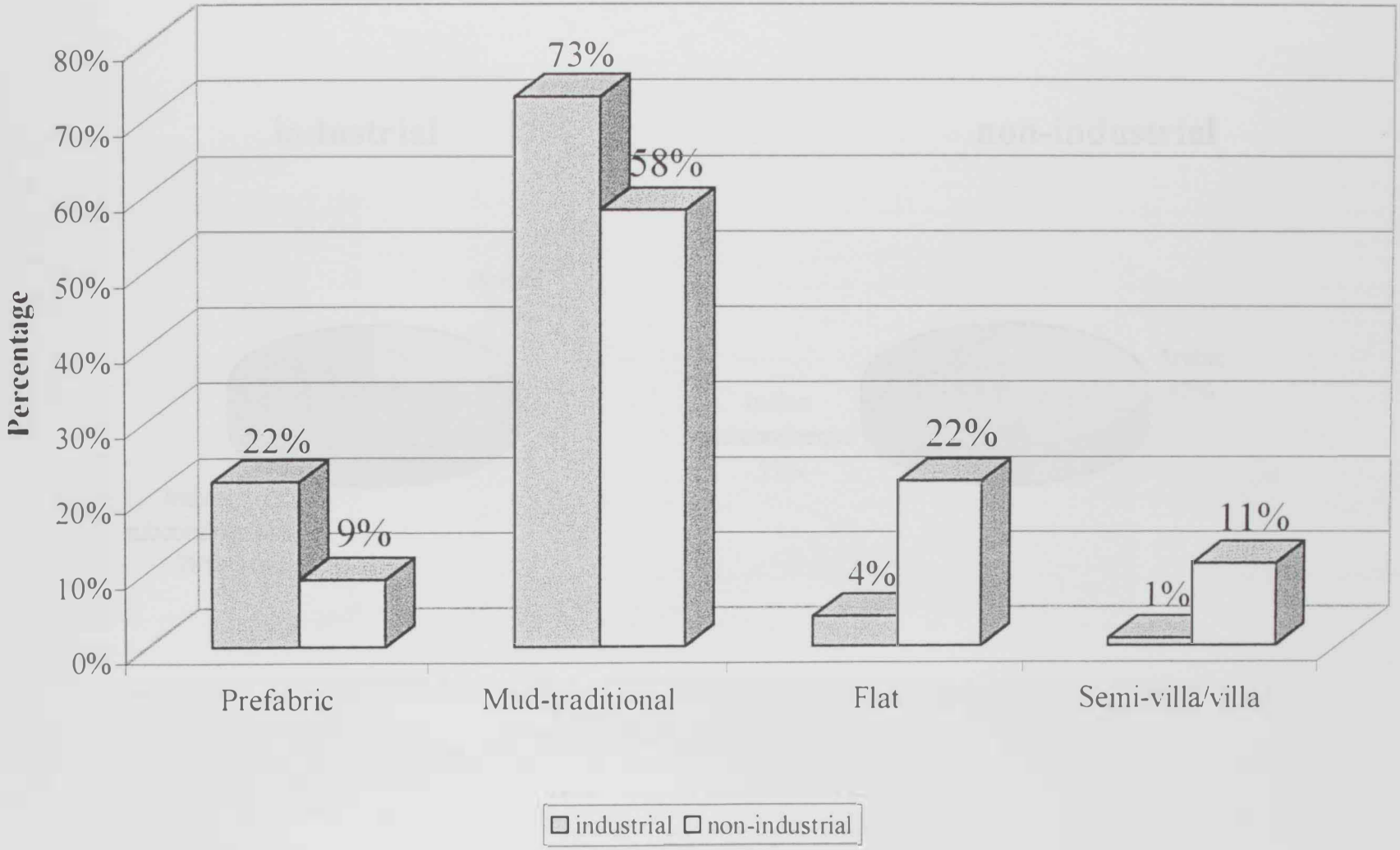


Fig 6: Type of food used among industrial & non-industrial workers

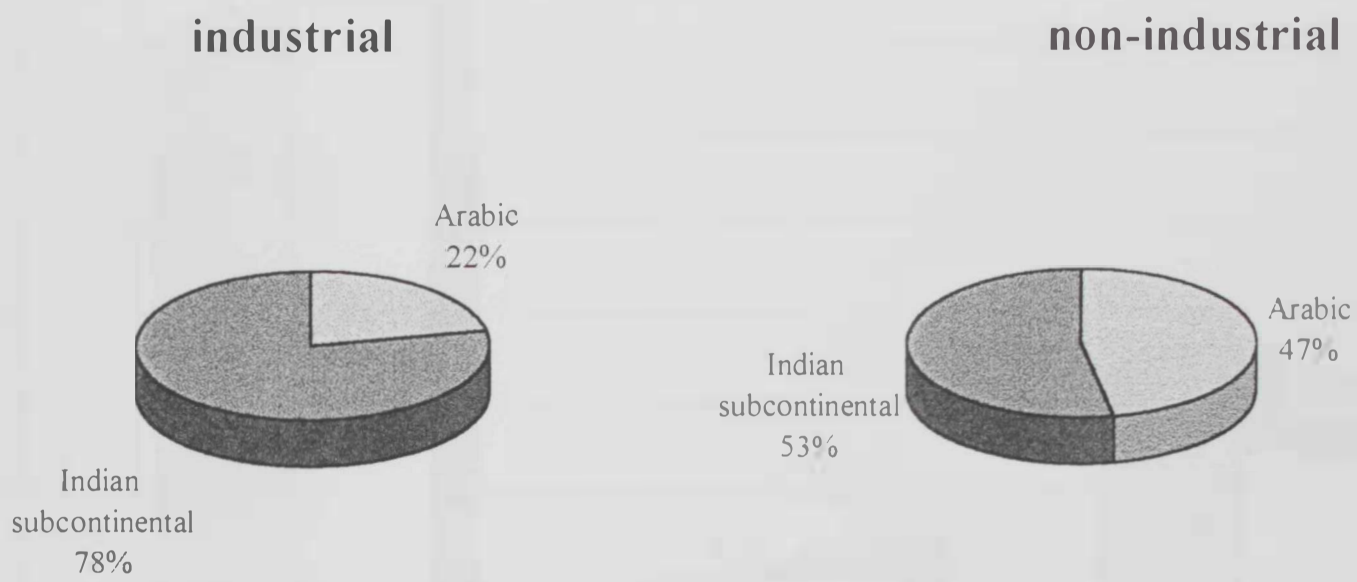


Fig 7: Type of cooking oil used among industrial & non-industrial workers

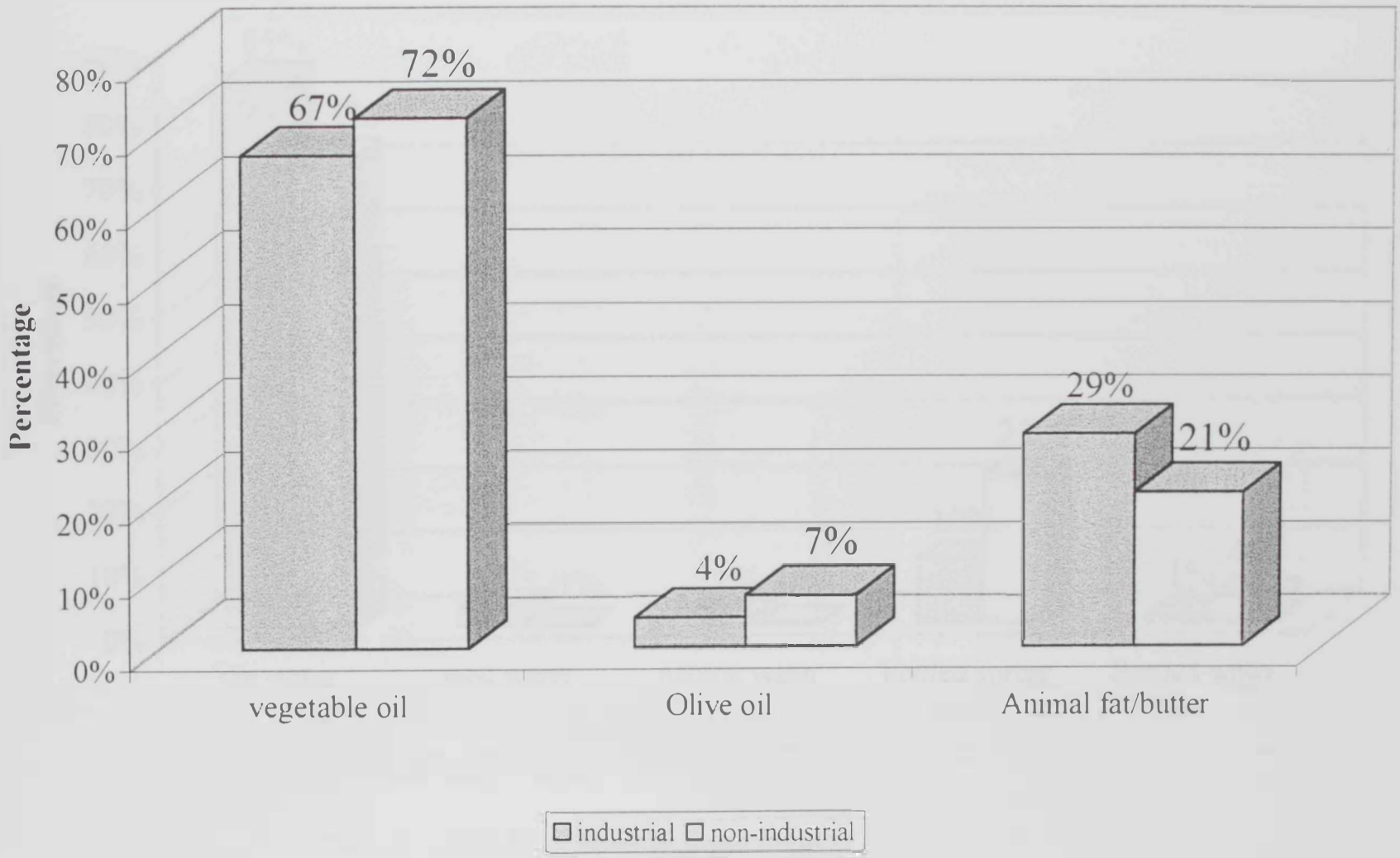


Fig 8: Source of drinking water among industrial & non-industrial workers

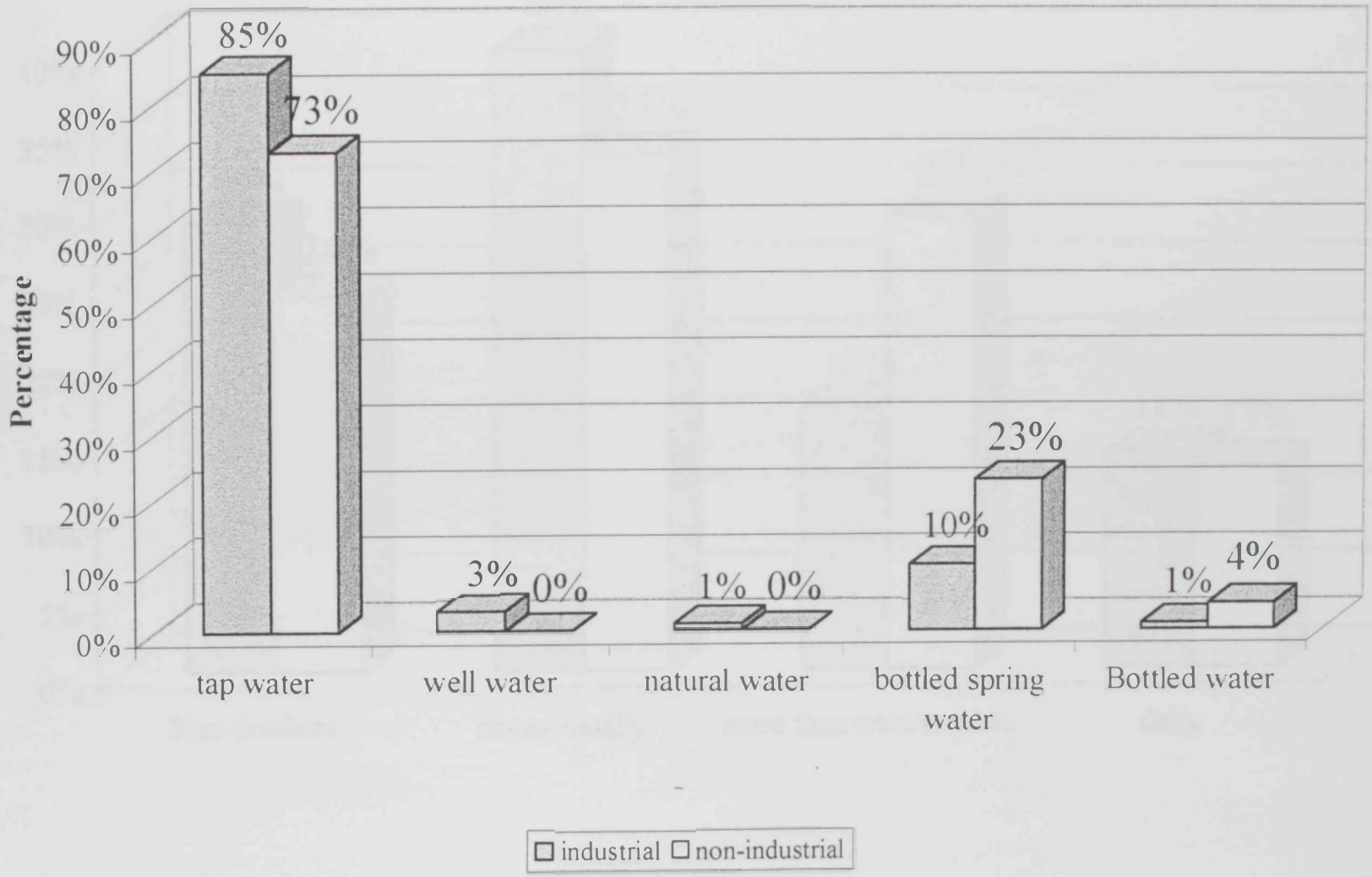


Fig 9: Milk consumption pattern amomg industrial & non-industrial workers

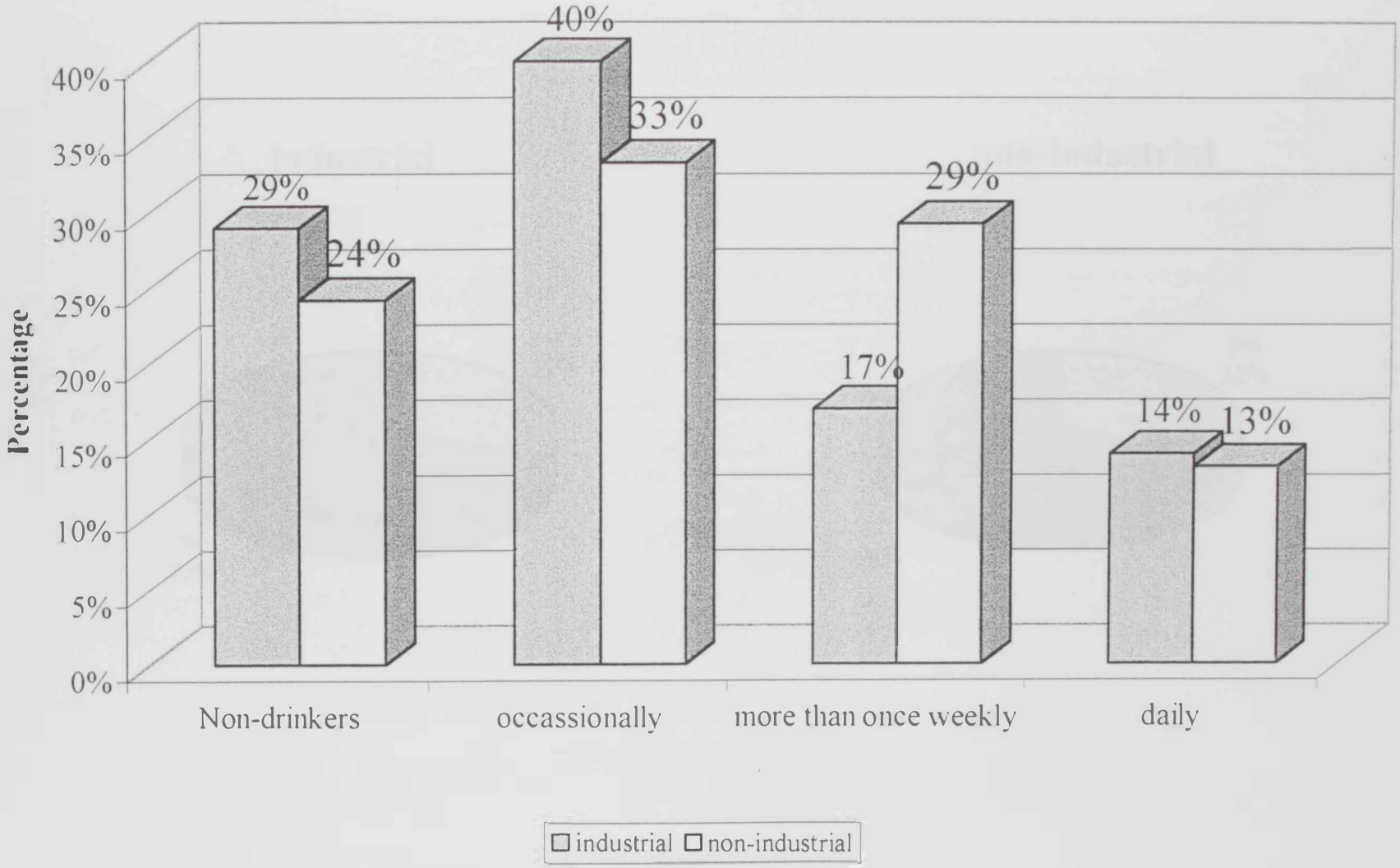


Fig 10: Canned food consumption among industrial & non-industrial workers

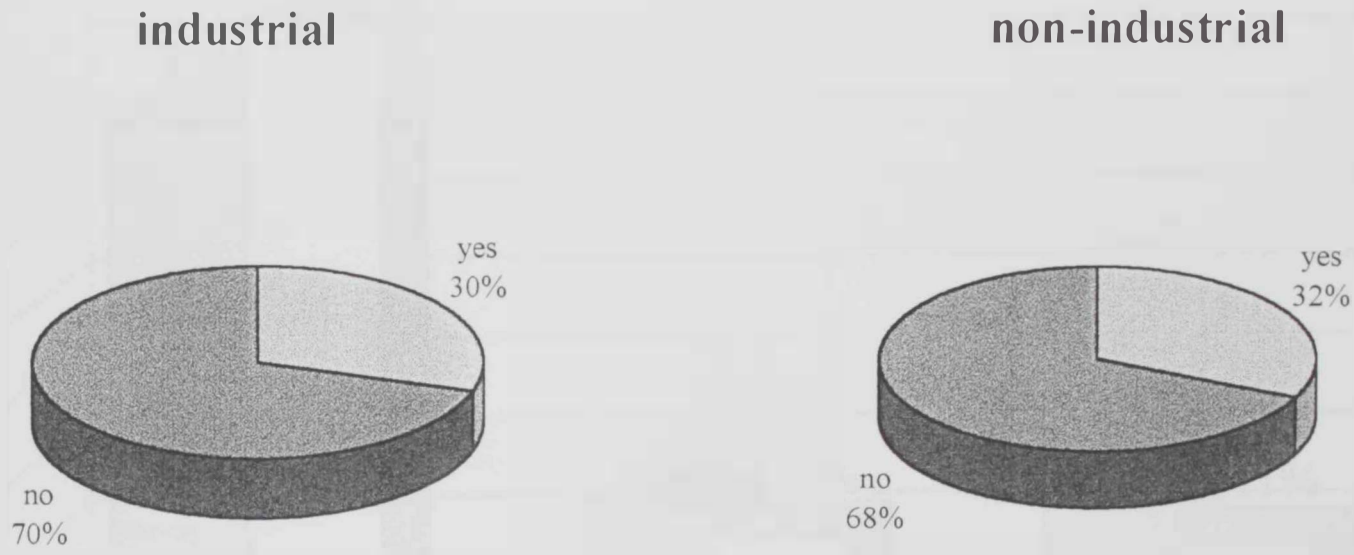


Fig 11: Smoking cigarettes among industrial & non-industrial workers

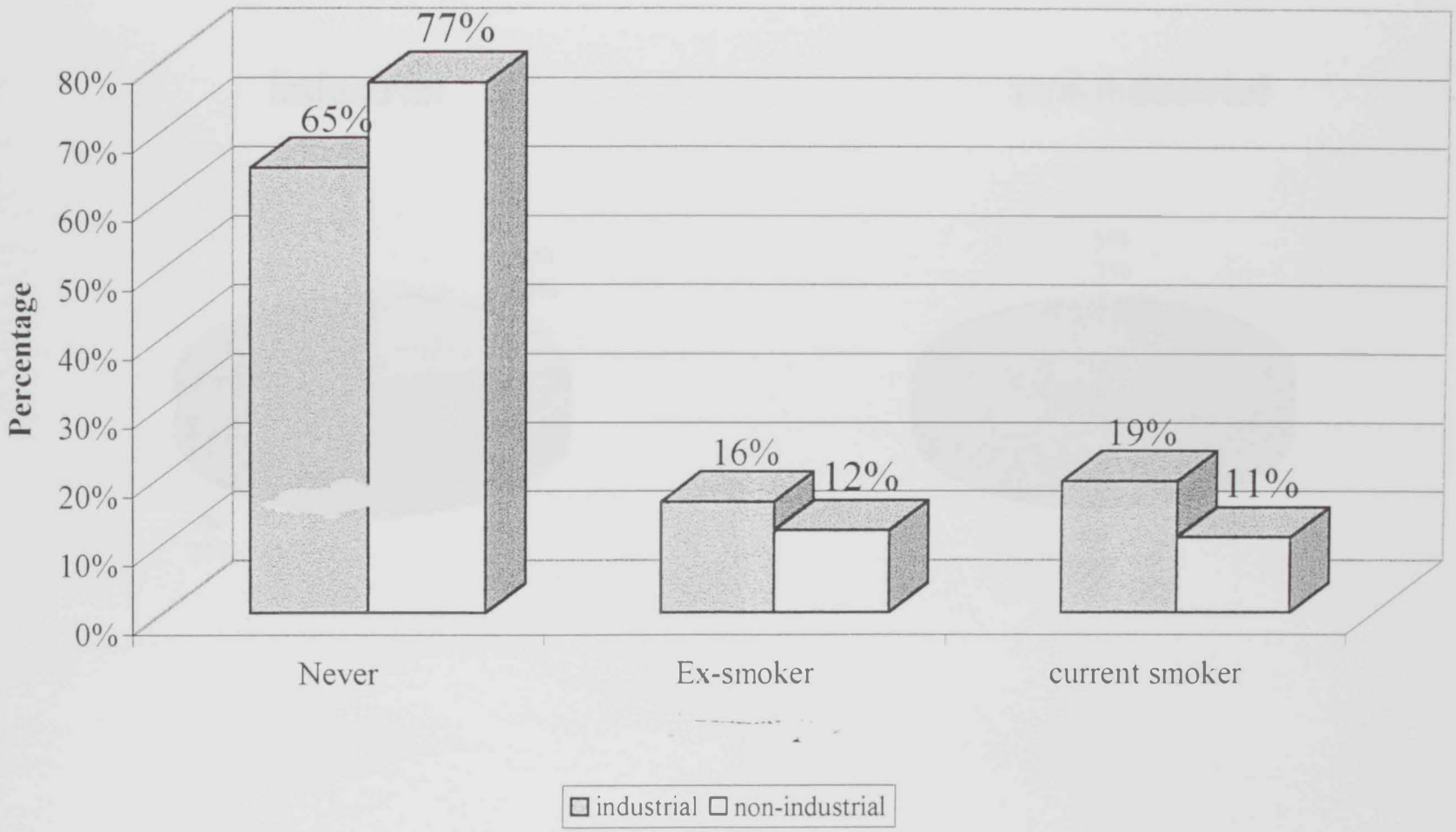


Fig 12: Chemical pesticide usage among industrial & non-industrial workers

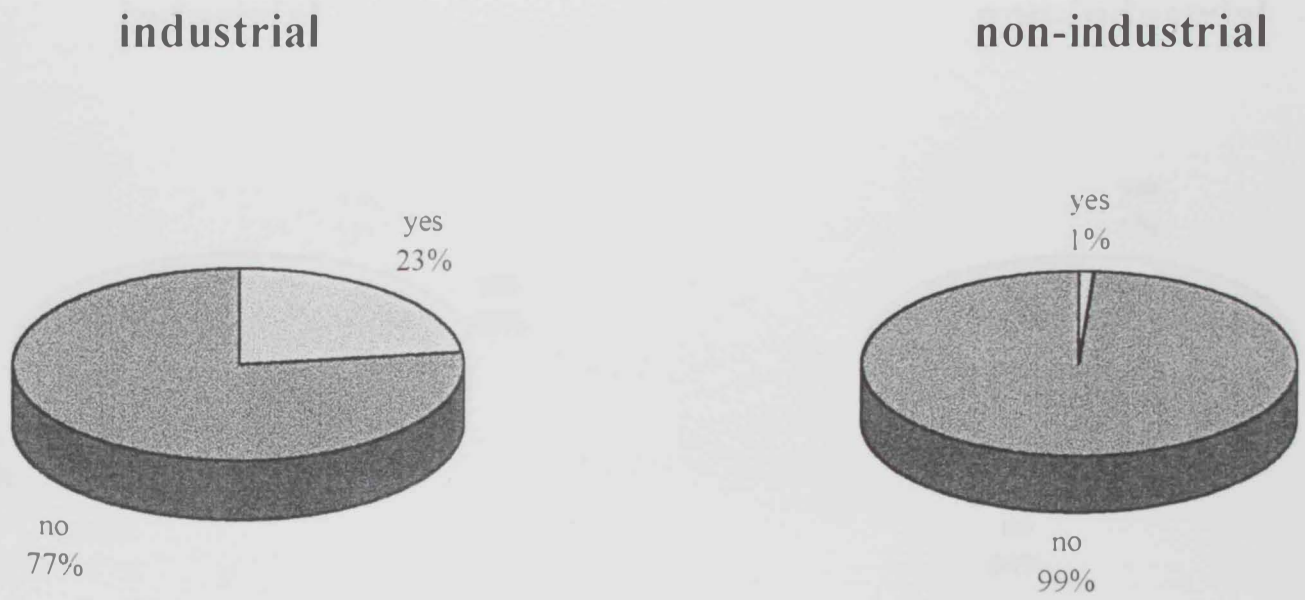


Fig 13: Toxic gas presence in industrial & non-industrial workers

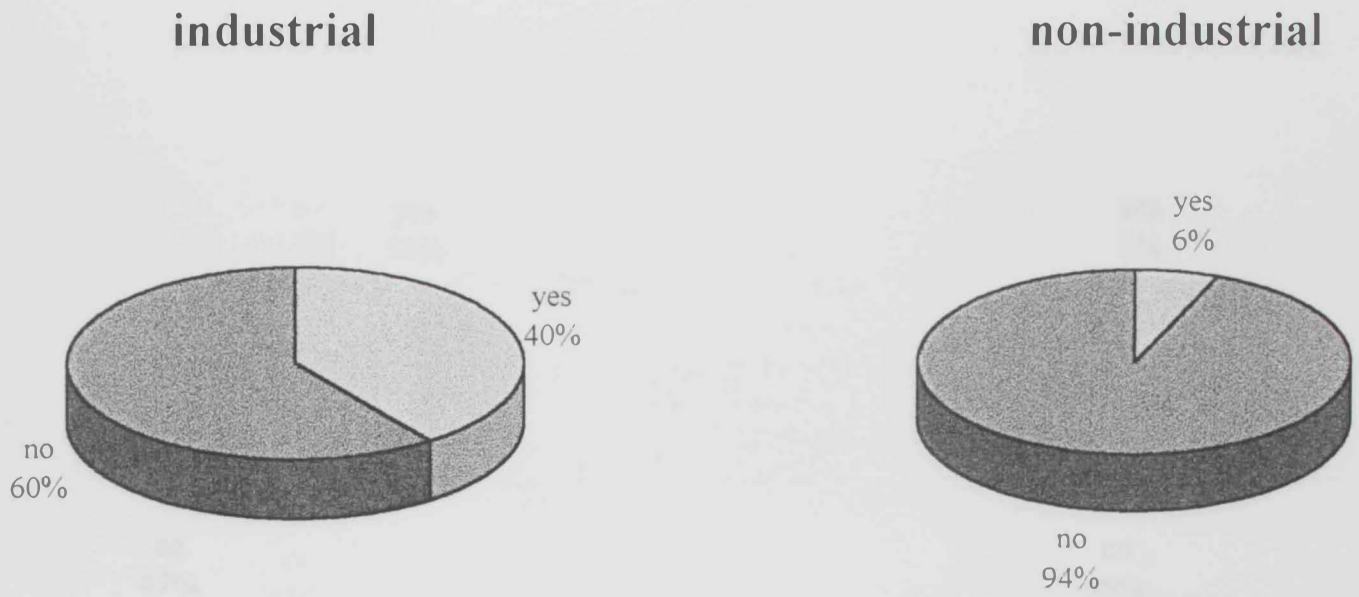


Fig 14: Radiation or magnetic presence in Industrial & non-industrial workers

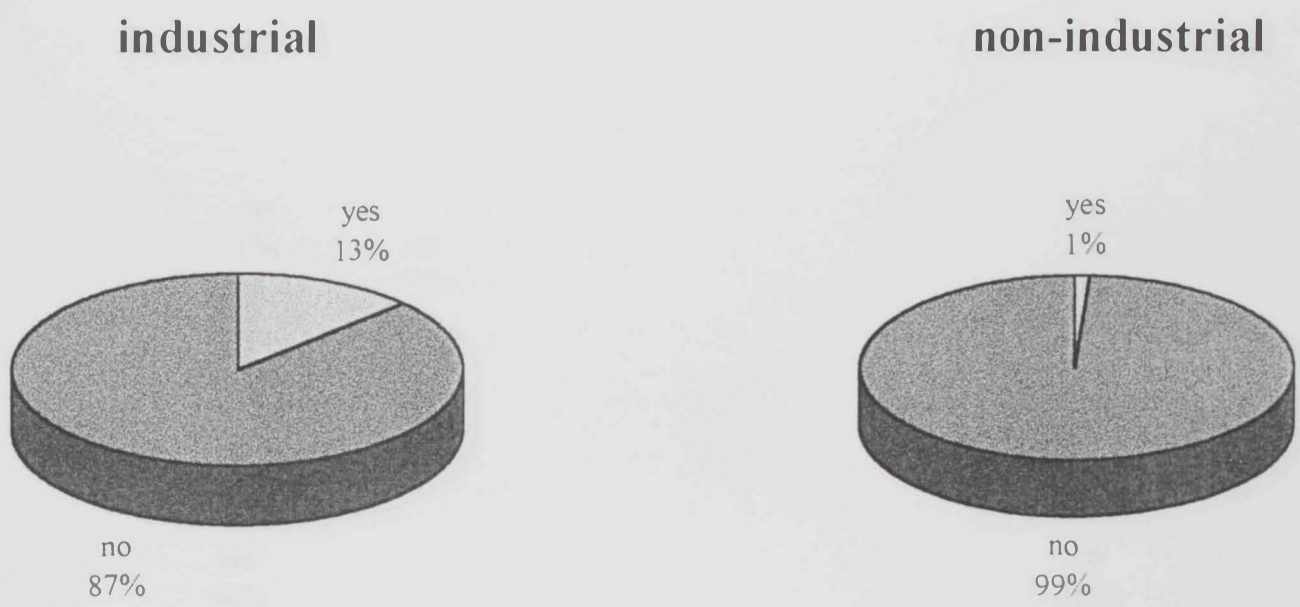


Fig 15: Cooking facility among industrial & non-industrial workers

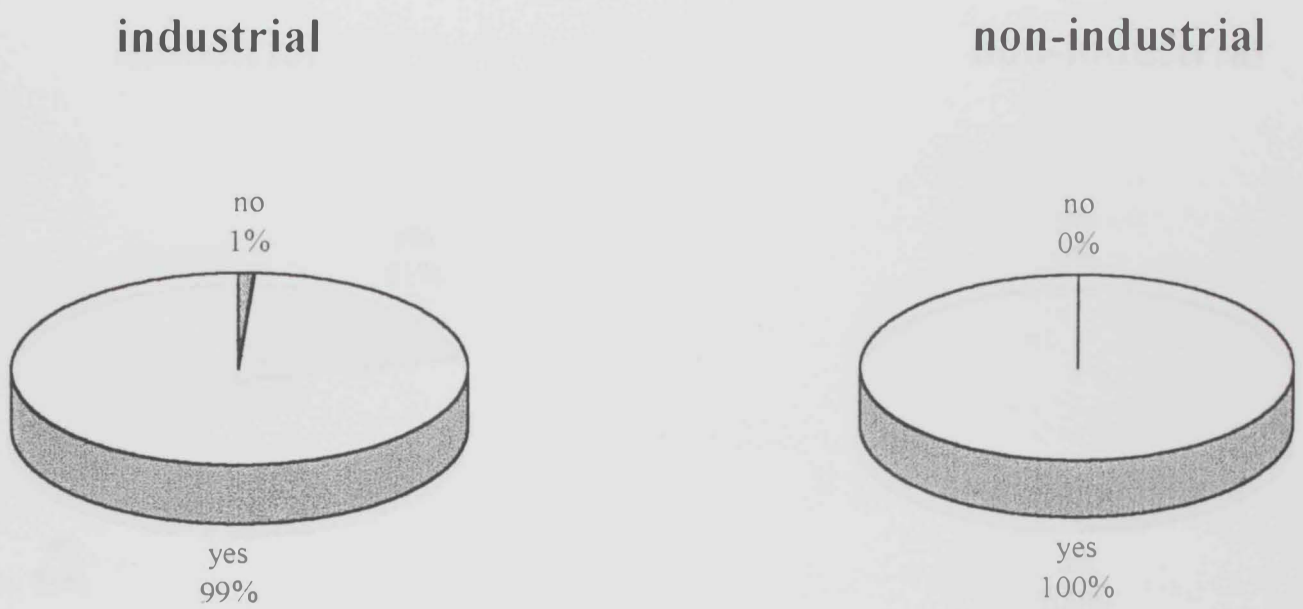


Fig 16: Mask protection equipment among industrial & non-industrial workers

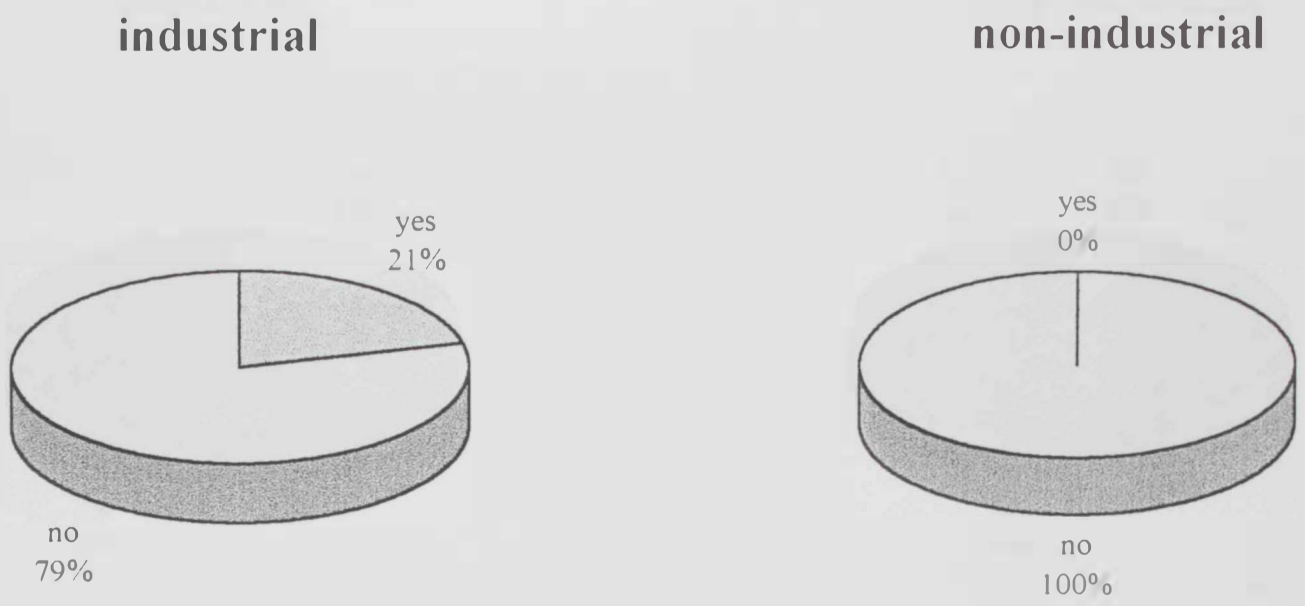


Fig 17: Lead smelter or battery recycling among industrial & non-industrial

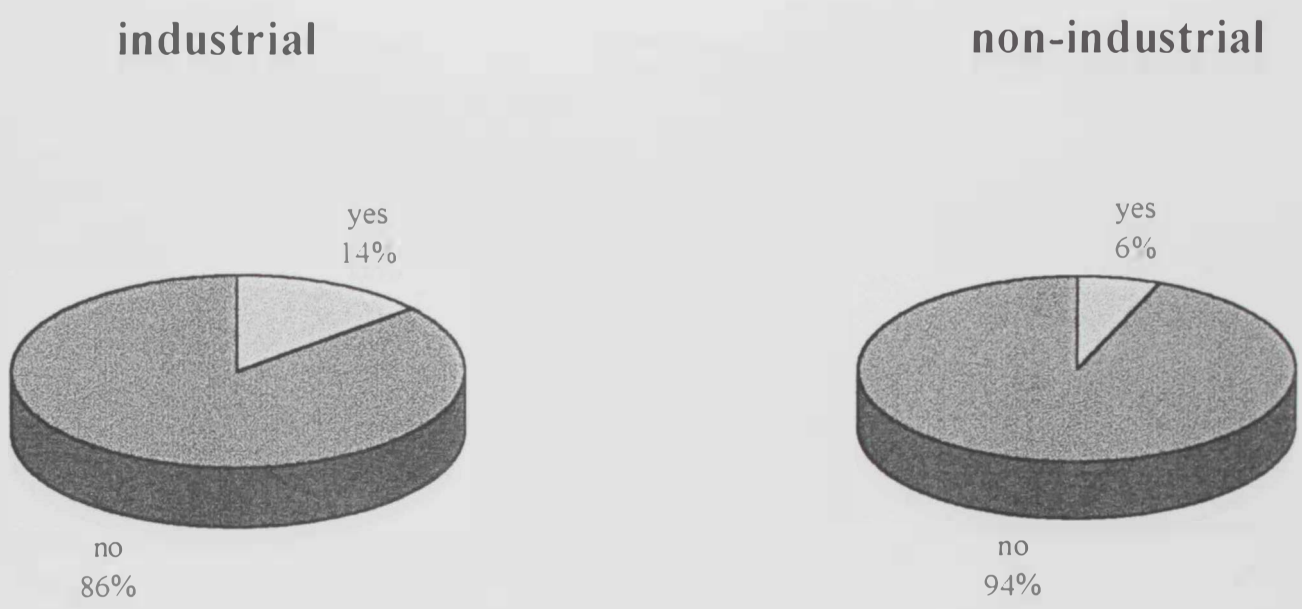


Fig 18: Industrial area likely to release lead among industrial & non-industrial workers

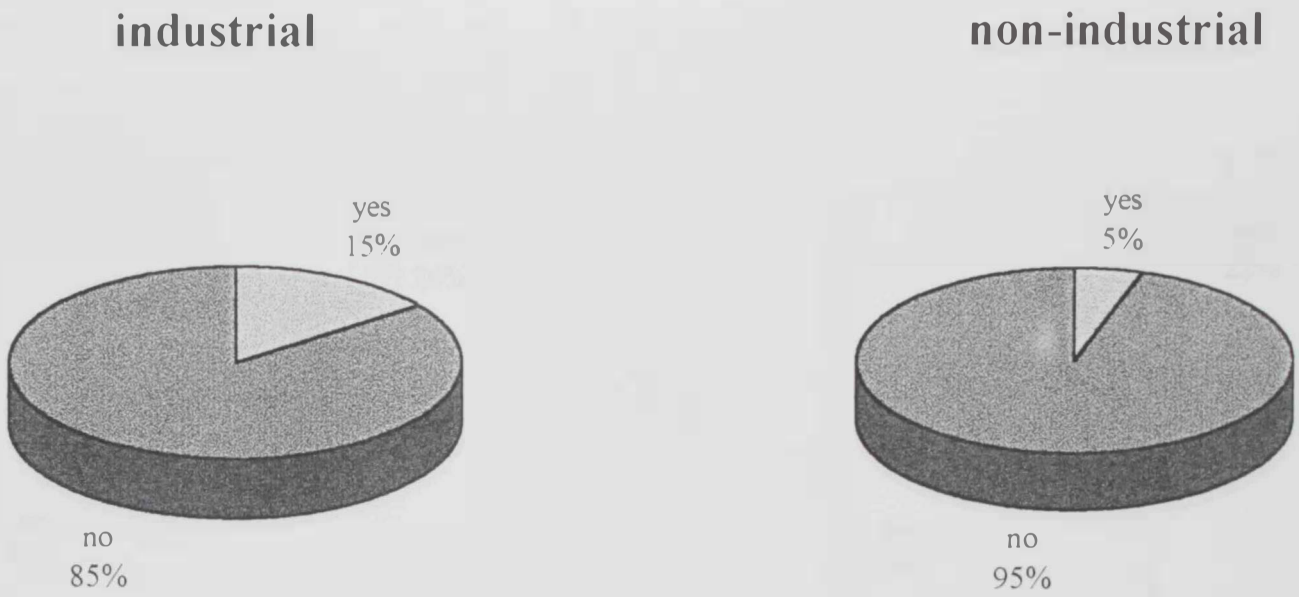


Fig 19: Cough presence among industrial & non-industrial workers

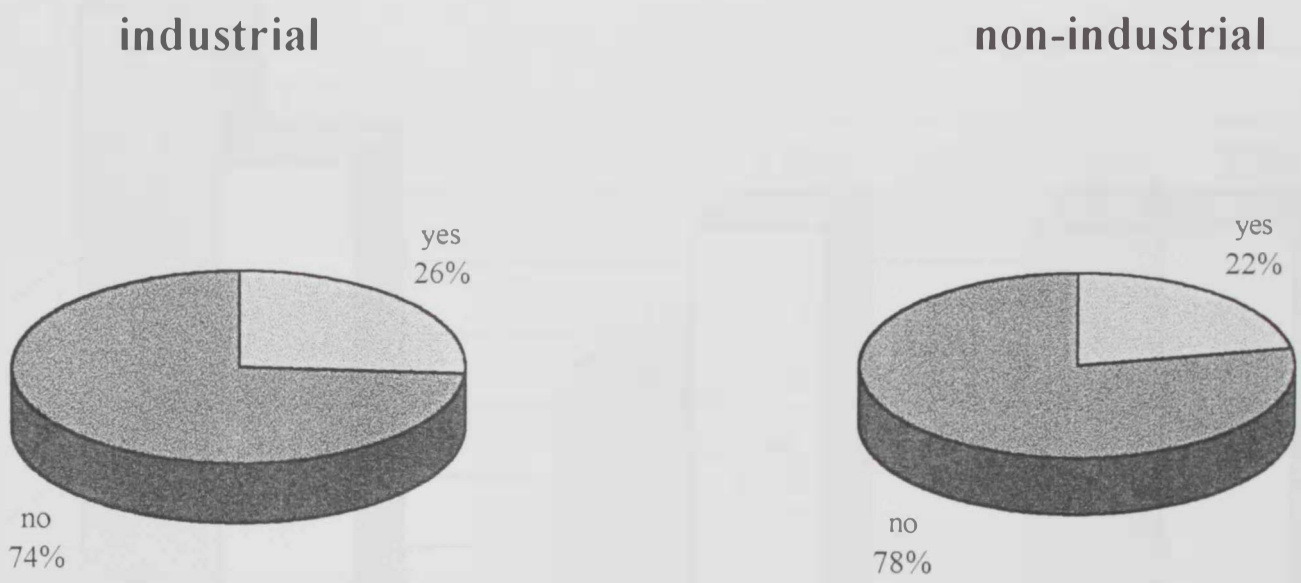
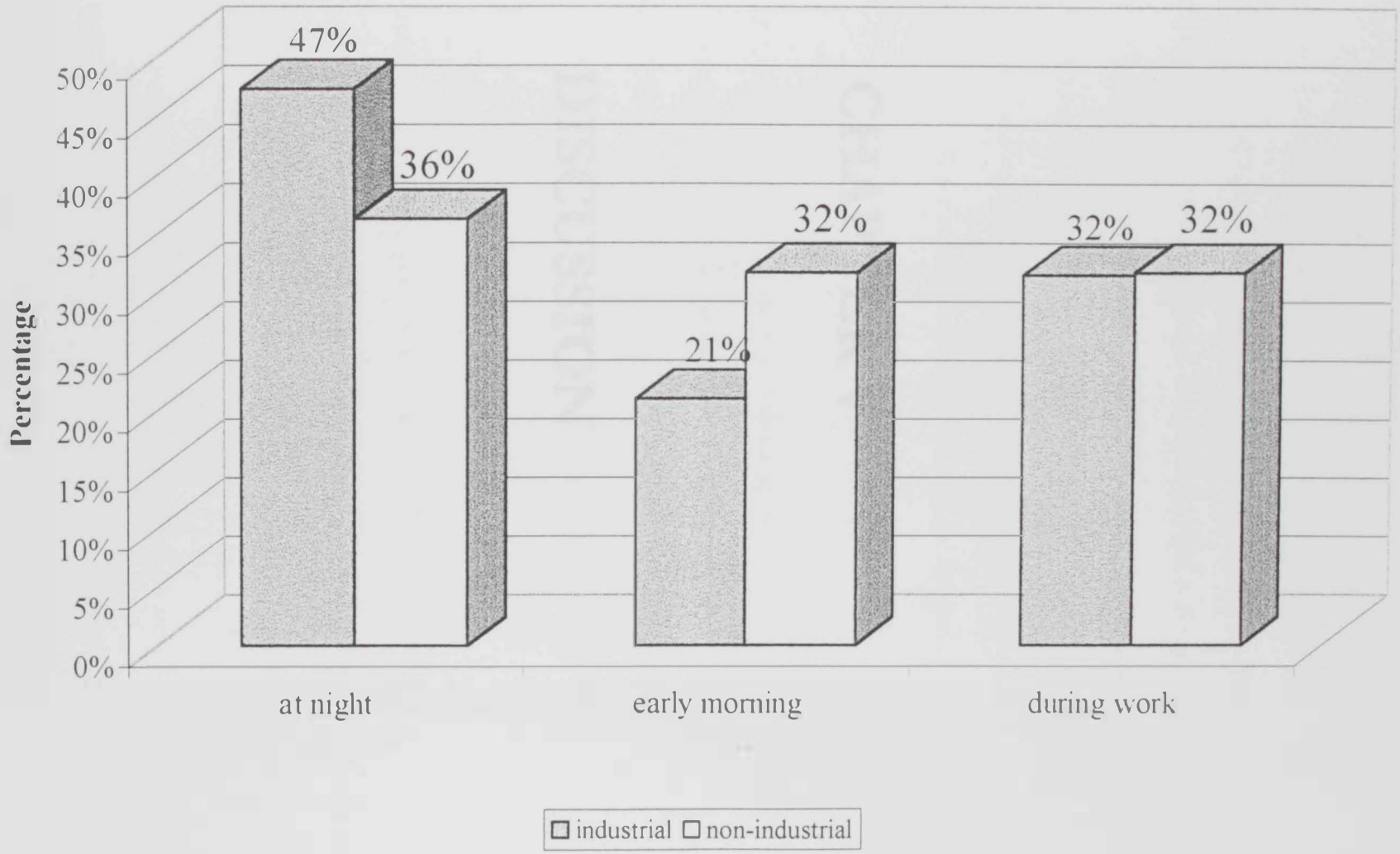


Fig 20: Coughing period among industrial & non-industrial workers



CHAPTER V

DISCUSSION

5.0 DISCUSSION

In recent years, steps have been taken to minimize exposure of the general population, especially children, to lead. Unleaded fuels were introduced in Canada 1975. Airborne lead concentrations dropped dramatically (76% on average) from 1973 to 1985 as the use of unleaded fuels increased. In 1990, the use of leaded gasoline in motor vehicles was prohibited in Canada (Health Canada 1990). Dietary intake of lead has also been reduced since the use of lead solder food canning processes and plumbing of drinking water supplies has been restricted. In 1976, the Hazardous Products Act limited the amount of lead permissible in interior paint to 0.5 percent by dry weight (Health Canada 1990).

Data collected from provincial and municipal studies carried out in Ontario suggest that blood lead levels are decreasing in adults and children representative of the general population (Health Canada 1994). Much of this observed decrease in blood lead levels has been attributed to the elimination of lead-based additives in gasoline and lead solder in commercial food canning.

There is little information regarding the impact of chronic exposure to lead over the lifetime of an individual. While animal studies have shown that lead acetate and lead phosphate may be carcinogens, there is no conclusive evidence linking lead to cancer in humans (Agency for Toxic Substances and Disease Registry 1993). Zeisler et al. (1984) performed multielemental analysis, including the determination of lead, on 36 liver tissue samples collected as part of a pilot project on specimen banking. Lead concentrations were found to range from approximately 0.1 t 1.3 µg/g. Keinonen (1992) compared the

isotopic composition of lead found in human liver, lung and bone to that found in various environmental indicators of sources of lead pollution. Emissions from incinerators and lead smelters were suggested to be the major factors influencing the levels of lead found in the human tissue samples which were analyzed. Bona and co-workers (1992) examined the levels of the heavy metals chromium, manganese, nickel and lead in liver samples obtained from 44 victims of sudden traumatic death. The average hepatic lead concentration was found to be 4.43 ppm (dry weight) with a standard deviation of 3.12 ppm. Concentrations of lead, cadmium, and zinc were determined in various reproductive organs, liver and kidneys from 41 men who had died suddenly (Oldereid et al. 1993). There was no significant correlation between blood lead levels and organ concentrations nor between concentrations and age. Gerhardsson and co-worders (1995) determined the concentration of lead in various tissue samples collected from deceased lead smelter workers. Lead concentrations in liver, lung, kidney, brain, hair, and nail samples collected from 32 long-term smelter workers and were compared to those obtained from a group of 10 control males. Of the four organs sampled, the highest lead were found in the liver for both groups of deceased men. The organs of the smelter workers were found to contain higher levels of lead than the corresponding organs of the control group, the largest relative difference being observed for the brain.

Lead has long been recognized as a potential hazard to human health. The effects of lead appear to be the same regardless of the mechanism by which it enters the body. Lead affects almost every organ and system in the body, in particular the central nervous system (Agency for Toxic Substances and Disease Registry 1993). Fetuses, through maternal exposure, and young children are especially susceptible to the harmful effects of lead.

Although the scientific and medical communities have long realized that lead is particularly harmful to unborn and young children, there is recent evidence to suggest that measurable and possibly even irreversible damage may occur at much lower levels of exposure. For this reason, the U.S. Center for Disease Control recently lowered its "threshold of concern" blood lead level to 10,000 µg/dl (Agency for Toxic Substances and Disease Registry 1993). Health Canada (1994) similarly recommended a blood lead intervention level of 0.5 µmol/l (10,400 µg/dl). As a result of the discovery of oil in the UAE, all aspects in the country had been developed including industry. Many of the industries depend largely on lead and lead compounds, which considered as a big risk effect on the health of industrial workers. Also the increase of population from 1,110,300 (1981) to 2,377,453 (1994) was associated with the rapidly increasing number of cars from 192,031 (1981) to 428,149 (1994) (Bener et al, 1994), and these cars are the main source of lead which causes environmental pollution and resulting health problem.

This study can be considered as the largest epidemiological and occupational medicine study concerning on industrial workers exposure to lead conducted to date in the United Arab Emirates. A community based design was chosen to maximize inclusion of morbidity in order to examine the biological relation between illness, plasma levels of amino acids and levels of liver enzymes, cardiac enzymes and renal function. The methodological difficulties in the epidemiological study of the health of expatriate industrial workers relates primarily to workers transience and workplace conditions. Most of the industrial workers were not sure about lead that they exposed to from their work. Both groups, industrial and non-industrial workers were demographically similar with regard to age groups, nationality and marital status. The majority of industrial workers

(38.0%) were illiterate, or with primary educational certificates (32.0%) which was the expected result. The majority of non-industrial workers were educated with secondary or high school educational certificates.

Many of the industrial workers reported occupational exposure to lead. 40% of them reported exposure to toxic gases and chemicals in workplace and the surrounding environment. 13% of industrial workers reported working in radiation areas or magnetic fields. 14% of them reported living near lead smelted or battery recycling areas (Sanaiya). And this means that the exposure to lead is 100% either from the workplace or from the polluted place where they live.

In a study done in Singapore (1996) among lead-acid storage battery manufacturing and PVC compounding industries, showed an indirect correlation between lead blood level and air lead levels. It also showed that lead contribution from smoking, consumption of specific foods, Chinese herbs and the use of glazed crockery which were reported to contain lead were found to be insignificant compared to other studies. This study reported that over absorption of lead is highly correlated to personal habit of eating with bare hands and environmental exposure which are important influencing factors of over absorption of lead (Ho et al, 1998).

Also in a study done among general population in South Germany in 1984-85, investigated the quantitative associations of demographics, lifestyle-related and anthropometric determinations with Pb in men and women. The study showed that Pb level differs according to sex, the median Pb in men was 70mg/l and was 60 mg/l among

women. Also age had some influence on pbB levels and was greater for women and not withstanding the consistently higher in men. It had been found that heamatocrit and blood pressure was found in a number of populations to be strongly and positively correlated to pbB concentrations. This study concluded that alcohol consumption and cigarette smoking are linearly associated with Pb levels among male and female subjects, but the association is stronger among men than women (Hense, 1992).

In a study performed on large organo-lead manufacturing in 1990, showed that organic lead did not have any effect on blood lead (Mc Grail et al, 1995). After dealkylation to inorganic lead which is stored in the bone may become a more important long-tsource of lead entry into blood and urine. As with the previous studies, this study showed that age, cigarette and alcohol consumption are positively associated with blood lead level.

Another type of exposure is related to potentially protective behaviors such as using masks. Only 21% of the industrial workers reported using mask as a method of protection. The frequent chronic sypmtoms among industrial workers was examined with respect to lead including nausea/vomiting (15.0%), muscular symptoms, weakness, cramps (28%), cardiovascular (16.0%), gastrointestinal (24.0%) wheeze (16.0%) and hay fever (36.0%). These results are consistent with Beshwari et. al. (1999-b).

In the present study, we have found that industrial workers reported more chronic symptoms and respiratory symptoms than non-industrial workers. Respiratory symptoms such as asthma, attacks of breathlessness and throat discomfort could be related to lead but also to other risk factors associated with respiratory symptoms such as exposure to

other chemicals, air conditioning and kerosene or gas. These results are consistent with Bener et al (1996, 1997, 1998 and 1999). Plasma amino acids analysis showed higher values in industrial groups than non-industrial groups. This increase was highly significant in most of amino acids, while not significant in others. Out of 21 amino acids analyzed only valine, leucine, lysine and theronin as non essential amino acids, and serine, alanine, taurine, glutamic acid, glutamine, glycine, orinthine and proline as essential amino acids showed significantly higher values in industrial workers when compared with non-industrial workers. Increase in the mean value of these amino acids in industrial workers group might be due to exposure to lead. This might affect the renal tubular disease, kidney dysfunction or chronic liver affects. The present study results are consistent with previous results, Beshwari et.al. (1999-b). Studies by Ikizler et. al. (1994), Zunic et al (1996), Vanter Jagt et al (1997) and Beshwari et. al. (1999-a) confirm higher amino acid levels obtained in exposed group as confirm with unexposed group. In the present study, these increases in the mean value of many of the amino acids in inustrial workers groups might be due to exposure to toxic materials, lead, chemical gases, chemical components, pesticides and motor vehicles exhaust. This might affect the industrial workers' health if they are exposed for a long term and could cause renal diseases, kidney dysfunction or chronic liver effects.

This study showed that (TP) Total protein and (LD) Lactate Dehydrogenas as liver function test are significantly correlated with Blood Lead Level and creatine kinase is also significantly correlated with Blood Lead Level as cardiac enzymes. Although there is some increase in the level of Lactate Dehydrogenase in both industrial workers (211.27 u/l) and non-industrial workers (194.77 u/L) but are within the normal range of 0-480u/l. Also

there is increase in level of Alkaline Phosphatase in induworkers than non industrial workers in industrial workers (84.27 u/l) and (76.18 u/l) in non industrial workers.

I think the two subjects might be due to exposure of industrial workers to lead and might increase if they were exposed for long period of time which might affect liver and heart function. The realities of industrial work atmosphere, the lack of legal protection and severe weakness in the existing laws, combined with the toxic gases chemicals and radiation that are ubiquitous in the industrial workers' environment make their work especially hazardous.

Industrial workers may be exposed to lead from many sources, factories where they work, cars and other means of transport, canned food they eat and drinking water. Most of the industrial workers live in "Sanaiya" which mean that they live in homes surrounded by these toxic chemicals and radiation.

The following conclusions have been drawn from a community based study on industrial workers and non-industrial workers. This study determined possible exposure and risk factors. There is evidence that some of the health problems like nausea/vomiting, muscular symptoms, weakness, cramps, cardiovascular, gastrointestinal, wheeze and hay fever are found.

The result showed that industrial workers had higher prevalence of chronic respiratory symptoms than non-industrial workers. A separate analysis of the prevalence of chronic respiratory symptoms from smoking in industrial workers was performed. There were no

statistically significant differences in the prevalence of chronic respiratory symptoms between smokers and non-smokers. Industrial workers reported more acute symptoms than non-industrial workers. This study suggested that the industrial workers suffered from an excess of respiratory symptoms including asthma, cough, chronic cough, chest tightness and abnormal lung function.

This study showed that the concentration of essential amino acids was increased particularly with regards to serine, alanine, taurine, glutamic acid, glutamine, glycine, orinthine and proline and non-essential amino acids such as valine, leucine, lysine and theoronine. These results might be related to lead and might have effect on liver and kidneys. These results confirm the earlier reported studies by Ikizler et al (1994), Zunic el al (1996), VandenJogt et al (1997) and Beshwari et al (1999 b).

The liver function enzymes of the industrial workers could be affected because of exposure to lead for a long-term and we found activity of lactate dehydrogenase being higher among industrial workers than non-indusrial workers. These results are in confirmation with the earlier reported studies by Ikizler et al (1994), Zunic et al (1996), VandenJogt el al (1997) and Beshwari et al (1999 b).

We think that the increased concentration of some amino acids and the liver enzyme lactate dehydrogenase activity in industrial workers was due to a large amount of lead exposure which human health and could cause kidney dysfunction and chronic liver diseases.

Lead and compounds containing lead are widespread throughout the environment. Aside from specific instances of occupational exposure (for example, workers in smelters, radiator repair shops, battery manufacturing plants), there are several routes by which the general population is exposed to lead. Humans are exposed to trace quantities of lead on a daily basis through inhalation of air, consumption of drinking water, and ingestion of food. Children who tend to have increased hand-to-mouth activity, ingest larger quantities of lead-containing dust and dirt than adults.

Although direct contact is made daily with dust, dirt, and painted surfaces containing lead, very little lead is absorbed through skin. Lead generally enters the body via the lungs or gastrointestinal tract and subsequently enters the bloodstream. It has been estimated that young children absorb about 42% of the lead that reaches the gastrointestinal tract as compared to the 5 to 15% absorbed by adults (Goyer 1991). Children also tend to retain a much higher proportion of absorbed lead than adults, typically greater than 30% versus less than 5%, respectively. Most of the lead which is stored by the body is found in bone and teeth.

In a recent study by Castilla (1995), the relationship between blood and liver lead levels and liver function in patients with liver disease were investigated. As with the earlier study by Oldreid (1993), no statistically significant relationship was determined to be present for lead concentrations in blood and liver samples. While blood lead levels could be linked to alcohol consumption and alcohol related liver disease, the same relationship was not observed for hepatic lead concentrations. Liver function did not appear to influence either

blood or hepatic lead levels. These are consistent with the present study conducted in Al Ain, UAE.

It was the intention of this study to obtain preliminary data in order to establish a normal range of blood lead concentrations from individuals representative of the general population among industrial and non-industrial workers of UniArab Emirates.

The public health task is clear, not only must there be more resources and priority given to biological monitoring and epidemiological studies of industrial workers, but also support given to the efforts of industrial workers to make their workplace safer. Overall we hope that our data will be useful for the establishment of an occupational exposure limit to lead in future.

CHAPTER VI

CONCLUSIONS

6.0 CONCLUSIONS

The realities of industrial work context, the lack of legal protection and severe weaknesses in the existing laws, combined with the toxic gases, chemicals and radiation that are ubiquitous in the industrial workers' environment make their work especially hazardous.

Industrial workers are exposed to lead from many sources:
Factories where they work, cars and other transports, canned food they eat, drinking water etc., Most of the industrial workers live in "Sanaiya" which means that they live in homes surrounded by these toxic chemicals and radiation.

This study determined possible exposure and risk factors associated with lead among industrial workers. There is evidence that some of the health problems which are found to be related increased blood lead concentrations include nausea/vomitting, muscular symptoms, weakness, cramps, cardiovascular, gastrointestinal, wheeze and hay fever.

The result showed that industrial workers had higher prevalence of chronic respiratory symptoms than non-industrial workers. A separate analysis of the prevalence of chronic respiratory symptoms by smoking in industrial workers was performed. There were no statistically significant differences in the prevalence of chronic respiratory symptoms between smokers and non-smokers. Industrial workers reported more acute symptoms than non-industrial workers. This study suggested that the industrial workers suffered from an excess of respiratory symptoms including asthma, cough, chronic cough and plegm, chest tightness and abnormal lung function.

This study showed that the concentrations of essential amino acids were increased particularly in regards to serine, alanine, taurine, glutamic acid, glutamine, glycine, orinthine and proline and non-essential amino acids such as valine, leucine, lysine and theronine. These results might be related to lead and might affect liver and kidneys.

The liver function enzymes of the industrial workers could be affected, due to exposure to lead for a long-term and we found that the activity of lactate dehydrogenase and Alkaline Phosphatas were higher among industrial workers than non-industrial workers.

We think that the increased concentration of some amino acids and the liver enzyme Lctate Dehydrogenase and Alkaline Phosphotase activities in industrial workers was due to a large amount of lead exposure which might affect human health and kidney dysfunction or chronic liver diseases.

The public health task is clear, not only must there be more resources and priority given to biological monitoring and epidemiological studies of industrial work, but also support given to the efforts of industrial workers to make their workplace safer.

Overall, we hope that our data will be useful for the establishment of an occupational exposure limited to lead in future.

CHAPTER VII

RECOMMENDATIONS

7.0 RECOMMENDATIONS

1. Advesice industrial workers to take basic preventive measures when they are contact with lead or radiation such as masks, gloves, protective glasses.

2. Periodic medical screening of industrial workers for the blood lead level.

3. Investigate other clinical parameters such as haematocrit among industrial and non-industrial workers.

4. Arrange medical surveillance of affected industrial workers in a lead-free environment to determine if the side effects are reversible.

5. We strongly recommend that Primary Health Centers (PHC) develop routine lead levels in blood or laboratory routine checkup to improve diagnosis of lead poisoning and the description of the nature of lead related illness among industrial workers.

6. Furthermore, industrial workers' behavior suggests that they are motivated to reduce lead exposure, but that their ability to do so is constrained by occupational conditions beyond their control.

7. It is clear that education and training of industrial workers in handling dealing with lead in an appropriate way and the use of protective equipment are needed to reduce the exposure and therefore long term risks to their health.

8. A government law should be enforced for the limitation and control of the uses of lead.

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Appendix 1

Lead exposure and physiological enzymes among industrial versus non-industrial workers in Al-Ain, U.A.E.

1. Study Number :_____ A).Case [ ] B).Control [ ]

2. Age :_____ Years Height: _____Cm Weight: _____Kg

3. Systolic blood Pressure (mm Hg):_____

4. Diastolic Blood Pressure (mm Hg):_____

5). Nationality: 1).Indian [ ] 2). Pakistani [ ] 3).Bengali [ ] 4). Arab[ ]

6). Education: 1).Illiterate[ ] 2.Primary [ ] 3).Secondary [ ] 4.High/Univer. [ ]

7). Marital status: 1).Single[ ] 2).Married [ ]

8). Housing condition: 1).Pre-fabric [ ] 2).Mud [ ] 3.Flat[ ] 4.Villa[ ]

9). Occupation: 1).Taxi driver[ ] 2).Garage worker [ ] 3.Heavy Industry worker[ ]
4.Gasoline filler[ ] 5.Painter[ ] 6.Chemical dealer/mixer[ ]

10). How long have you been working in your job? Years

11). What type of foods do you have usually:
1.Arabic foods [ ] 2.Indian / Pakis /Bengali foods [ ] 3.Western foods [ ]

12). Do you use any of the following in your cooking :
1.Vegetable oil [ ] 2.Olive oil [ ] 3.Animal fat / butter [ ]

13). Source of drinking water: 1.Tap water [ ] 2.Well water [ ] 3.Natural [ ]
4.Bottled spring water [ ] 5.Bottled water [ ]

14). Frequency of milk consumption? 1.Non-drinkers [ ] 2).Occasionally [ ]
3.More than once weekly [ ] 4.) Daily [ ]

15) Do you usually eat canned food? 1).Yes [ ] 2).No[ ]

16) Have you ever smoked cigarette? 3.Never [ ] 2.)Ex-smoker [ ] 3.Current [ ]

17). How many cigarettes do you smoke?
1.Less than 5 cigarettes/day 1).Yes [ ] 2).No[ ]
2.About 1/2 a packet of cigarette/day 1).Yes [ ] 2).No[ ]
3.About 1 or more than 1 packet /day 1).Yes [ ] 2).No[ ]

18). Do you usually drink alcoholic beverages ? 1).Yes [ ] 2).No[ ]

19). Do you spray crops with chemical pesticides ? 1).Yes [ ] 2).No[ ]

20). Do you work in in toxic gas, chemical odor or smeel ?1).Yes [ ] 2).No[ ]

21). Do you work in radiation areas or magentic fields1).Yes[ ] 2.No[ ]

22). Which type of cooking facilities is used inside your home:
1.electrical cooker 1.Yes [ ] 2.No[ ]
2.kerosene or gas 1.Yes [ ] 2.No [ ]
3.charcoal 1.Yes [ ] 2.No[ ]

23). Do you use mask as an protection equipment in in your job 1).Yes [ ] 2.No [ ]

24). Do you live near an active lead smelter or,battery recycling plant?1.Yes [ ] 2.No [ ]

25). Do you live in industrial area likely to release lead? 1.Yes [ ] 2.No [ ]

REPORTED SYMPTOMS

26. Do you usually have the following symptoms / signs:

1.Nausea / Vomiting	1).Yes [ ]	2).No[ ]
2.Red / irritated eye / Blurred vision	1).Yes [ ]	2).No[ ]
3.Increased anxiety	1).Yes [ ]	2.No[ ]
4.Dizziness	1).Yes [ ]	2).No[ ]
5.Headache	1).Yes [ ]	2).No[ ]
6.Muscular symptoms.[weakness, cramps	1).Yes [ ]	2).No[ ]
7.Difficulty breathing	1).Yes [ ]	2).No[ ]
8.Chest pain	1).Yes [ ]	2).No[ ]
9.Fatigue	1).Yes [ ]	2).No[ ]
10.Memory loss	1).Yes [ ]	2).No[ ]
11.Insomnia	1).Yes [ ]	2).No[ ]
12. Mania (Psychiatric disorder)s	1).Yes [ ]	2).No[ ]
13.Abdominal pain and constipation	1).Yes [ ]	2).No[ ]
14.Myalgia and anorexia	1).Yes [ ]	2).No[ ]
15.Anemia	1).Yes [ ]	2).No[ ]
16.Cardivascular	1).Yes [ ]	2).No[ ]
17.Kidneys	1).Yes [ ]	2).No[ ]
18.Gastrointestinal	1).Yes [ ]	2).No[ ]

RESPIRATORY SYMPTOMS:

27). Do you usually have cough: 1).Yes [ ] 2).No[ ]

28). When do you usually have cough:

1.at night [ ] 2.early morning [ ] 3.during work [ ] 4.after work [ ]

29). Do you usually have cough up phlegm from your chest:

1.at night [ ] 2.early morning[ ] 3.during work [ ] 4.after work [ ]

30). Did you ever had attacks of breathlessness or tightness in your chest? Yes [ ] 2.)No [ ]

31). Do you usually have any throat discomfort? 1).Yes [ ] 2.)No [ ]

32). Have your ever had wheeze ? 1).Yes [ ] 2.)No [ ]

33). Have you ever been diagnosed as having asthma 1).Yes [ ] 2.)No [ ]

34). Have your ever had attacks of hay fever? 1).Yes [ ] 2.)No [ ]

35). Have your ever been diagnosed having allergy? 1).Yes [ ] 2.)No [ ]

Appendix 2

Abbreviations

LDH	Laktate Dehydrogenase
GOT	Glotamic Oxoacetatic Transaminase
RBC	Red Blood Cells
U.A.E	United Arab Emirates
BC	Before Christ
EPA	Environmental Protect Agency
LD50	Lethal Dose of Toxicity that kill 50 percent of the test animals
WHO	World Health Organized
MRL	Maximum Residue Limits
NADH	Nicotinamide Adenine Dinucleotides
AAA	Aromatic Amino Acids
BCAA	Branched Chain Amino Acids
PKU	Phenylketonuria
gm	grams
ml	mililiter
rpm	rotation per minutes
µl	microlitre
km	kilometer
I.D	internal diametres
Cm	Centimetres

ml/hr	milliliters per hours
nm	nanometres
Tau	Taurine
Ser	Serine
Glu	Glutamic acid
Glun	Glutamine
Pro	Proline
Gly	Glycine
Ala	Alanine
Val	Valine
Cys	Cystine
Met	Methionine
Ile	Isoleucine
Leu	Leucine
Tyr	Tyrosine
Phe	Phenylalanine
Orn	Ornithine
Lys	Lysine
His	Histidine
Arg	Arginine
Try	Tryptophan
Thr	Threonine
Cit	Citrulline

Cyt	Cysteine
Alb	Albumin
T.P	Total Protein
T.Bil	Total Bilirubin
ALT	Alanane aminotransferase
AST	Aspartate aminotransferase
LDH	Lactate Dehydrogenase
ALP	Alkaline Phosphatase
SD	Standard Deviation
LFT	Liver Function Test
U/L	Unit per Litere
gm/l	gram per litere
C I	Confidence Interval
r	regression
OR	Odd Ratio
Gpt	Glutamic-pyruvic Transaminase
NADPH	Nicotinamicde Adenine Dinucleotide-Phosphate
PK4	Phenylketonuria
μ g/dl	Microgram per decilitere

إن إنجاز هذا العمل ونشر النتائج التي توصلنا إليها في المجلات والدوريات العلمية سوف يكون له تأثير كبير على مؤسسات الخدمات الصحية المهتمة بمثل هذه المشاكل. ويؤمل أن يؤدي نشر نتائج هذه الدراسة إلى مزيد من البحوث في هذا المجال في العديد من مجتمعات دولة الإمارات العربية المتحدة ودول الخليج العربي الأخرى.

بينما كانت نسبة اللاكتيت ديهايدروجين Lactate Dehydrogenase في فئة العاملين في المجال الصناعي كانت (211.27 ملغم / للتر + 57.27 ، الوسط الحسابي + الانحراف المعياري) و في فئة العاملين في المجال غير الصناعي كانت (194.77 ملغم / للتر + 48.69 ، الوسط الحسابي + الانحراف المعياري) وكان معامل الارتباط كما يلي:

Total Protein (p=0.023)
Total Bilirubin (p=0.002)
Indirect Bilirubin (p =0.0001)
Gamma Glutamyl Transferase (p=0.008)

كذلك قمنا بعمل فحوص وظائف الكلى Urea Nitrogen, Creatinine وبينت النتائج الإحصائية أنه لا يوجد فرق يذكر بين العينات التي فحصت للفئتين من العاملين في المجال الصناعي و غير الصناعي.

إن قليل من العاملين في القطاع الصناعي أشاروا إلى تعرضهم مباشرة للرصاص. إن هناك فئة منهم تتعرض للرصاص لأن عملهم مرتبط مباشرة بالرصاص. بينما أشار 40% من العاملين في هذا المجال إلى تعرضهم للغازات الكيماوية السامة. بينما أشار 13% منهم إلى أن عملهم يحتم عليهم التعامل مع الأشعة و المجال المغناطيسي. و هناك 14% أشاروا إلى أن عملهم مرتبط بالتعامل مع إعادة استخدام البطاريات الكهربائية المستهلكة.

إن واقع العاملين في القطاع الصناعي يشير إلى ضعف التشريعات الخاصة بحمايتهم مقارنة بالمخاطر التي يتعرضون لها حيث توجد المواد والغازات الكيماوية السامة في كل مكان في البيئة التي يعملون بها.

لقد تم قياس مدى التعرض للرصاص بين العاملين في المجال الصناعي وغير الصناعي. إن غالبية العاملين في القطاع الصناعي لا يستخدمون الأقنعة الواقية والتي يجب أن تستخدم في الصناعة. وأن غالبيتهم أبلغ عن كثير من الأعراض الصحية. ولوحظ أن الحمى وألام العضلات والضعف العام والتشنج هي من الأعراض الشائعة بين العاملين في المجال الصناعي. ولوحظ وجود أعراض صحية أخرى بينهم مثل الغثيان والقيء التهيج الجلدي وضبابية الرؤيا وزيادة القلق والدوار والإرهاق والصداع واللهاث وأمراض الجهاز التنفسي مثل وجود التهابات الجيوب الأنفية والحلق وضيق التنفس والربو.

كذلك قمنا بتحليل الأحماض الأمينية للفئتين (العاملين في المجال الصناعي وغير الصناعي). إن غالبية التحاليل للأحماض الأمينية والبلازما أظهرت نسب أعلى لدى العاملين في المجال الصناعي مقارنة بالعاملين في غير المجال الصناعي. وبينت النتائج الإحصائية إلى أن هناك فروق واضحة بين الفئتين في مجال نسب تركيز بعض الأحماض الأمينية في البلازما كما هو مبين على النحو التالي. :-

الأحماض الأمينية الهامة مثل الهستدين Histidine والثيرونين Threonine واللسين Lysine التربتوفان Tryptophan . الأحماض الأمينية الأقل أهمية مثل الترونين Taurine والسيرين Serine والجلوتاميك Glutamic والأرجنين Arginine والجيليسين Glycine والأورنثين Ornithine .

كذلك قمنا بعمل فحص لوظائف الكبد لكلا الفئتين من العاملين في المجال الصناعي وغير الصناعي فقد فحصنا مجموع البروتينات Total Protein و الألبومين Albumin و البيليروبين Bilirubin والأسبيرتات Aspartate transferase والألنين alanine transferase و فوسفات الألكالين alkaline phosphatase وجاما جلتاما Gamma Glutamyl . ولم تظهر التحاليل الإحصائية وجود أي فروق تذكر بين الفئتين.

إن نتائج هذه الدراسة حددت بالضبط نسبة الرصاص في الدم في مجتمع الإمارات. كذلك أدت إلى تحديد العوامل البيئية التي تؤدي إلى زيادة نسبة التعرض للرصاص مما يساعد في التخطيط المستقبلي ووضع البرامج الوقائية من قبل وزارة الصحة في مدينة العين والجامعات والمؤسسات البيئية الوطنية ومنظمة الصحة العالمية والبلدية والمؤسسات البحثية الأخرى.

إن هذه الدراسة أجريت على ثلاث مراحل :-

أ) الدراسة الوبائية

ب) تحليل نسبة الرصاص في الدم

جـ) التحليل المخبري للأحماض الأمينية وأنزيمات الكبد والقلب ووظائف الكلى لكلا الفئتين.

إن العوامل الاجتماعية - الديمغرافية للفئتين (العاملين في المجال الصناعي وغير الصناعي) موضع الدراسة متشابهة. غالبية العاملين في المجال الصناعي غير متأكدين من تعرضهم للرصاص. إن كلا الفئتين (العاملين في المجال الصناعي وغير الصناعي) متشابهين من ناحية الفئات العمرية والجنسية والحالة الاجتماعية. إن غالبية العاملين في القطاع الصناعي (38%) من الأميين وهي نتيجة كانت متوقعة. إن غالبية العاملين في غير القطاع الصناعي حاصلين على شهادة الثانوية العامة أو أعلى من ذلك. إن غالبية العاملين في القطاع الصناعي غير مكترثين لنسبة الرصاص التي يمكن أن يكونوا قد تعرضوا لها.

تركز الدراسة على مقارنة نسبة الرصاص في الدم بين العاملين في القطاع الصناعي وغير الصناعي في مدينة العين كمثال لسكان دولة الإمارات العربية المتحدة. إن الدراسة لا تهدف إلى تعميم مشكلة التعرض للرصاص في مجتمع الإمارات ككل ولكن مما لا شك فيه بأنها ستكون دراسة مساعدة لعينة ممثلة لمشكلة انتشار الرصاص في مجتمع الإمارات ككل. إن الجزء الأول من الدراسة هو قياس مستوى الرصاص في الدم لعينة من العاملين في القطاع الصناعي ومقارنتها مع العاملين في غير القطاع الصناعي مع الأخذ في الاعتبار عوامل العمر والحالة الاجتماعية والعرق والتدخين. أما الجزء الثاني من الدراسة فيتركز على العلاقة بين مستوى الرصاص في الدم وعمل أنزيمات الكبد والقلب ووظائف الكلى.

إن هذا البحث قائم على دراسة مقارنة بين فئتين من سكان مدينة العين يشكلان عينة ممثلة لمجتمع الإمارات: فئة معرضة للرصاص وفئة غير معرضة للرصاص. تتكون كل فئة من مائة حالة وكلا الفئتين متماثلتين في الجنس والعمر والجنسية . لقد أجريت الدراسة العملية في الفترة ما بين ١ إبريل و 31 مايو من عام 1999.

وقد تم دراسة وتحليل العوامل التي يتوقع أن تكون ذات أثر كبير على مستوى الرصاص في الدم بين العاملين في القطاع الصناعي بطريقة إحصائية. ومن ثم تم قياس مدى حساسية وتأثير كل عامل من هذه العوامل على مستوى الرصاص في الدم بطريقة التحليل الإحصائي الارتدادي (العنقودي).

ملخص

إن الرصاص من أكثر المعادن الثقيلة استخداما في الحياة العملية وهو من المعادن السامة والتي ليس لها أي فائدة وظيفية أو كيماوية للجسم البشري. ومنذ بداية التقدم الصناعي الحديث والرصاص يدخل في التركيب الكيماوي للكثير من الآلات والبضائع المستخدمة. فهو موجود في البنزين والأصباغ واللحام الخاص بالمعلبات والآنية الزجاجية والخزفية. والتبغ كمثال هو أحد مصادر الرصاص في الجسم. لهذا لازال الرصاص يعتبر مشكلة صحية عامة ذات بعد عالمي.

وفى أيامنا هذه يستحيل أن تجد شخص تقل نسبة الرصاص في دمه عن 0.48 ملغم / لتر وخاصة بين سكان المدن. ولقد وجد أن التغير في عمل النظام العصبي للجسم والأوعية الدموية والشرايين والكلى مرتبطة ارتباطا كبيرا بمعدل وجود الرصاص في الدم حتى ولو كان هذا المعدل منخفض نسبيا. وفى المعدلات المنخفضة يتسبب الرصاص بأضرار للأنزيمات المتعددة وكذلك لترتيب النظام الخلوي في الجسم.

إن الهدف من الدراسة هو قياس نسبة الرصاص في الدم لعينة من مجتمع الإمارات، وكذلك البحث عن العوامل التي من المتوقع أن تكون مؤثرة في زيادة نسبة الرصاص في الدم. بالإضافة إلى ذلك تهدف الدراسة إلى قياس تأثير التعرض للرصاص على مستوى الأحماض الأمينية ووظائف الكبد والقلب والكلى والأنزيمات الحيوية للعاملين في القطاع الصناعي في مدينة العين بدولة الإمارات العربية المتحدة.

شكر

أود أن أقدم جزيل شكري للمشرف الرئيسي على الرسالة الدكتور عبد الباري بيتر والذي لم يدخر جهدا في إبداء النصح والتشجيع والمساعدة على إتمام هذا العمل.

كذلك أود تقديم الشكر الجزيل للدكتور أحمد ميرزا المهدي المشرف المساعد على الرسالة للمساعدة والتشجيع الذي لم يبخل بها علي. كما أوجه شكري لجميع العمال المشاركين في هذه الدراسة سواء كانوا من القطاع الصناعي أو غير الصناعي لتعاونهم معنا في إتمام هذه الرسالة. وكذلك أوجه شكري الجزيل لوالدي ووالدتي وجميع أفراد أسرتي على جهودهم ودعمهم لي لإتمام هذا العمل.

كما وأقدم شكري الخاص لسمو الشيخ نهيان بن مبارك آل نهيان وزير التعليم العالي والبحث العلمي والرئيس الأعلى لجامعة الإمارات العربية المتحدة، وكذلك لمعالي حمد عبد الرحمن المدفع وزير الصحة لاتاحتهم الفرصة لي لإكمال دراستي العليا.

كذلك أود تقديم شكري الجزيل لكل من الأستاذ الدكتور عبد الرحمن الشرهان عميد كلية العلوم والأستاذ الدكتور دوجلوس فوث عميد كلية الطب والعلوم الصحية لتعاونهم ومساعدتهم في إكمال هذا العمل.

كما أود أن أشكر السيد أحمد إبراهيم لمساعدته لي في تحليل الأحماض الأمينية للعينات، وكذلك السيد ساجر والسيد جمال محمد والسيد جون شيريان من قسم طب المجتمع بكلية الطب والعلوم الصحية للمساعدات التي قدموها لي في إتمام هذا العمل.

المشرفون

المشرف الرئيسي

د. عبد الباري بينر

أستاذ مساعد

قسم طب المجتمع

كلية الطب والعلوم الصحية

جامعة الإمارات العربية المتحدة

المشرف المساعد

د. أحمد ميرزا المهدي

مدرس

قسم الكيمياء

كلية العلوم

جامعة الإمارات العربية المتحدة

تأثير التعرض للرصاص على الإنزيمات الفيسيولوجية بين العاملين في المجال الصناعي وغير الصناعي في مدينة العين دولة الإمارات العربية المتحدة

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