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## Some Biochemical and Hematological Alterations Associated with Lead Exposure in Gasoline Station Workers in Gaza Strip

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## *Dedication*

*To my parents  
my brothers,  
my sister,  
my friends,  
my Islamic University and to  
all I love*

# *Acknowledgement*

It is impossible to convey, in a couple of sentences, my gratitude to many people for helping me to learn and who cooperation made this work possible.

All praises and glory are due to ALLAh for all the bounty and support granted to me.

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Last but not least, I want to thank my beloved father and mother, thank you for always believing in me and loving me unconditionally, without your support and love I would not be where I am today.

# **Some Biochemical & Hematological Alterations Associated with Lead Exposure in Gasoline Station Workers in Gaza Strip**

## **Abstract**

Although occupational lead exposure is one of the major public health problems, no previous published research was conducted on lead exposed gasoline station workers in Gaza Strip. This study aimed to identify risk factors associated with lead exposure and to determine biochemical and hematological parameters in leaded gasoline station workers in Gaza Strip.

**Method:** A total 105 gasoline station workers from the Gaza Strip were asked to fill in a questionnaire on knowledge, attitudes, and practice towards lead, as well as associated toxicity symptoms. Out of the 105, seventy two workers gave blood sample for blood lead level and biochemical analysis and those were compared with 70 controls. Atomic absorption spectrometry was used to determine Blood lead level. Liver and kidney function were determined by Biosystem reagent kits. Cell dyne was used to measure complete blood count and sphygmomanometer for blood pressure. SPSS version 11.0 was employed for data analysis.

**Results:** More than half of workers work more than 5 years in the gasoline station. None of them were previously tested for lead. Although the majority of the workers know that lead is an environmental pollutant and has adverse health impact, small number of them follow the protective measures. The most common self reported symptoms among workers were headache, fatigue, irritability and concentration difficulties, sleep disturbance, hypertension, nausea, constipation and dyspepsia. Neither workers attended training courses nor had health professional visited them.

The mean Blood lead level (BLL) of the followed up workers was  $11.4\mu\text{g}/\text{dl}$  compared to  $5.3\mu\text{g}/\text{dl}$  for the controls. Lower BLL was found in highly educated workers. Positive association was found between BLLs of the workers and work duration. The BLLs in workers who used gloves, respiratory mask or drank milk frequently were significantly lower than those who did not ( $8.6\pm 4.9$ ,  $5.6\pm 1.6$  and  $9.3\pm 5.2\mu\text{g}/\text{dl}$  versus  $13.1\pm 6.0$ ,  $12.4\pm 5.9$  and  $13.3\pm 5.2\mu\text{g}/\text{dl}$ , respectively).

The BLLs were significantly higher in workers reported irritability, headache, sleep disturbance, concentration difficulties, hypertension (12.4±5.4, 12.2±5.8, 14.1±5.8, 12.9±5.8, 13.3±6.4 µg/dl, respectively) and those who had not report such symptoms (9.2±5.6, 9.0±5.9, 8.9±5.0, 9.4±5.6, 8.1±2.9µg/dl, respectively).

A statistically significant increase were found in BLL, hemoglobin, MCV, MCH, MCHC, Alanine Transaminase (ALT), Aspartate Transaminase (AST), Creatinine, systolic and diastolic blood pressure among workers (11.4±6.0µg/dl, 15.4±1.5g/dl, 86.7±5.1, 30.5±3.1, 35.2±2.7, 29.1±11.5U/L, 28.6±7.9U/L, 0.9±0.2mg/dl, 125.7±13.2mmHg and 84.6±9.9mmHg, respectively) compared to controls (5.3±1.0µg/dl, 14.5±0.8g/dl, 84.3±3.7, 28.6±1.6, 33.6±1.2, 23.9±9.7U/L, 24.7±3.6U/L, 0.7±0.1mg/dl, 120.4±3.8mmHg and 81.9±2.7mmHg, respectively). While a statistically significant decrease were found in red blood cells count, Platelets counts, alkaline phosphatase (ALP) and urea in workers (4.9±0.4X10<sup>6</sup>, 249.4±58.2X10<sup>3</sup>cell/µl, 179.3±42.6U/L and 27.1±11.6mg/dl, respectively) compared to controls (5.1±0.23X10<sup>6</sup>, 281.5±56.5 X10<sup>3</sup>cell/µl, 193.6±31.8U/L, 31.3±6.3mg/dl, respectively).

**Key words:** Biochemical & Hematological analysis, Lead exposure, gasoline station workers, Gaza strip.

## المستخلص

### بعض التغيرات البيوكيميائية والدموية المرتبطة بالتعرض للرصاص لدى عمال محطات

#### البنزين في قطاع غزة

بالرغم من كون التعرض المهني للرصاص واحد من اهم قضايا الصحة العامة، فإن قطاع غزة خلا من أي بحث في مجال التعرض المهني للرصاص لدى عمال محطات البنزين. لذا تهدف هذه الدراسة لتحديد العوامل المرتبطة بالتعرض للرصاص بالإضافة إلى الفحوصات البيوكيميائية وفحص محتويات الدم والضغط لدى عمال محطات البنزين. ولقد اشتملت الدراسة على (١٠٥) عامل قاموا بتعبئة الاستبيان لاستخلاص معرفتهم عن الرصاص وماهي احتياطاتهم تجاه التعرض له بالإضافة الى بعض الاعراض المصاحبة للتعرض للرصاص. اثنان وسبعون من العمال الذين قاموا بتعبئة الاستبيان ابدوا استعدادهم طوعيا لاعطاء عينات الدم لفحصها وتم مقارنتهم ب ٧٠ عينة ضابطة.

وبعد تحليل الاستبيانات وعمل الفحوصات اللازمة للدم اظهرت النتائج أن معظم العمال عملوا أكثر من خمس سنوات في محطات البنزين، لم يقم أحد منهم بفحص مستوى الرصاص في الدم من قبل، بالرغم من أن معظم العمال يعرفون بأن الرصاص ملوث للبيئة وله اضرار صحية على الانسان الا ان عدد قليل منهم كان يستخدم الادوات الوقائية ويتبع العادات الصحية السليمة اثناء العمل. ووجد أن الأعراض الصحية الأكثر شيوعا بينهم هي الصداع، الاجهاد، الاثارة العصبية، صعوبات في التركيز، اضطرابات في النوم، ارتفاع ضغط الدم، الغثيان، الامساك و عسر الهضم، كما أن أحد من العمال حصل على دورات تثقيفية او صحية او زارهم أحد المرشدين الصحيين. كما وجد ارتفاع في متوسط مستوى الرصاص لدى هؤلاء العمال (١١,٤ ميكرو جرام لكل ديسيليلتر) مقارنة بالعينة الضابطة (٥,٣ ميكرو جرام لكل ديسيليلتر) . ولوحظ انخفاض مستوى الرصاص لدى العمال ذو الدرجات العلمية الاعلى. وكان هناك علاقة موجبة بين مستوى الرصاص في الدم وفترة العمل بالسنوات. وكان مستوى الرصاص لدى العمال الذين يستخدمون الادوات الوقائية (الكفات، الكمادات) ويتبعون العادات الصحية السليمة (وشرب الحليب بانتظام) أثناء العمل اقل وبدلالة إحصائية من أولئك الذين لا يقومون بها (٤,٩±٨,٦، ١,٦±٥,٦ و ٥,٢±٩,٣ مقارنة ١٣,١±٦,٠، ٤,٩±١٢,٤ و ٥,٢±١٣,٣ ميكرو جرام لكل ديسيليلتر، على التوالي)، كما كانت هناك فروق بدلالة إحصائية ما بين العمال الذين يعانون من الاثارة العصبية، الصداع، اضطرابات النوم، صعوبات في التركيز وارتفاع ضغط الدم (٥,٤±١٢,٤، ٥,٨±١٢,٢، ٥,٨±١٤,١، ٥,٨±١٢,٩ و ٦,٤±١٣,٣ ميكرو جرام لكل ديسيليلتر ، على التوالي) والعمال الذين لا يعانون من مثل تلك الأعراض (٥,٦±٩,٢، ٥,٩±٩,٠، ٥,٠±٨,٩، ٥,٦±٩,٤ و ٢,٩±٨,١ ميكرو جرام لكل ديسيليلتر ، على التوالي). كما أظهرت النتائج زيادة بدلائل إحصائية في مستوى الرصاص ، تركيز الهيموجلوبين، معاملات الدم، إنزيمات الانليني و الاسبرتيت وضغط الدم عند عمال محطات البنزين (٦,٠±١١,٤ ميكرو جرام لكل ديسيليلتر، ١٥,٤±١٥,٤ جرام لكل ديسيليلتر، ٥,١±٨٦,٧، ٣,١±٣٠,٥، ٢,٧±٣٥,٢، ١١,٥±٢٩,١ وحدة لكل لتر، ٧,٩±٢٨,٦ وحدة لكل لتر، ٠,٢±٠,٩ مليجرام لكل ديسيليلتر، ١٣,٢±١٢٥,٧ و ٩,٩±٨٤,٦ مليتر زئبقي ، على التوالي) مقارنة بالعينة الضابطة (١,٠±٥,٣، ٠,٨±١٤,٥ ميكرو جرام لكل ديسيليلتر، ٣,٧±٨٤,٣، ١,٦±٢٨,٦، ١,٢±٣٣,٦،



٩,٧±٢٣,٩ وحدة لكل لتر، ٣,٦±٢٤,٧ وحدة لكل لتر، ٠,١ ±٠,٧ ملليجرام لكل ديسيلتر، ٣,٨±١٢٠,٤ و ٩,٧±٨١,٩ ملليمتر زئبقي، على التوالي). بينما كان هناك انخفاض بدلائل احصائية في عدد كرات الدم الحمراء، الصفائح الدموية، وإنزيم الفوسفات القاعدي و البولينا لدى العمال (٠,٤±٤,٩ × ١٠<sup>٦</sup>، ٤٢,٦±١٧٩,٣ وحدة لكل لتر و ١١,٦±٢٧,١ ملليجرام لكل ديسيلتر ، على التوالي) مقارنة بالعينة الضابطة (٠,٢٣±٥,١ × ١٠<sup>٦</sup>، ٥٨,٥±٢٨١,٥ × ١٠<sup>٣</sup> خلية لكل ميكرو لتر، ٣١,٨±١٩٣,٦ وحدة لكل لتر، ٦,٣±٣١,٣ ملليجرام لكل ديسيلتر، على التوالي).

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Lead is naturally occurring element with unique chemical and physical properties (e.g., low melting point, pliability, and resistance to corrosion). Lead has widespread application in general industry and construction. Industries where lead may be found include gasoline, battery manufacture and recycling, radiator repair, painting, firing range operation, nonferrous foundries and recycling of scrap metal (1, 2).

Lead is an element of risk for the environment and human health and has harmful effects that may exceed those of other inorganic toxicants. Most of the atmospheric lead is emitted from two main sources, motor vehicles and industrial sources, such as gasoline station, lead smelter, battery and auto-radiator repairing (1, 2). In addition, human activities also have spread lead throughout water, soil, plants and animals. Lead can be found in everyone's bodies (3, 4).

The major exposure pathways of lead workers are inhalation and ingestion of lead bearing dust and fumes (5). After absorption, lead transported to the blood. Then it builds up in soft tissue; kidneys, bone marrow, liver and brain, and deposited mainly in bone (6, 7, and 8).

Occupational exposure to lead could induce toxicity (9). Lead adversely affects several body systems. The most sensitive are the nervous, hematopoietic, gastrointestinal, cardiovascular, musculoskeletal, renal and reproductive systems (2, 10, 11, and 12).

In contrast with developed countries where lead exposure is on the decline due to the implementation of environmental and occupational regulations (13), developing countries lead poisoning continues to be one of the most important problems of environmental and occupational origin. In Gaza Strip, there are at least 112 gasoline stations recorded officially in Gaza Strip (14), in addition to other illegal ones that located in residential areas. However, leaded gasoline is



still being used in gasoline stations in the Gaza strip. The complicated socioeconomic situation, the lack of environmental control on the industrial activities and the lack of industrial zones in the Gaza strip allow some people to establish their own stations or workshops such as gasoline station, battery and auto-radiator among houses inside highly dense populated areas.

Lead has been extensively studied worldwide. However, only two studies about blood lead poisoning were conducted in the Gaza strip. One study revealed that the prevalence rate of lead poisoning among children was 17.2 % (15), the other one revealed that the mean of blood lead level (BLL) among battery workers was 48.18 $\mu$ g/dl and among auto radiator workers it was 29.52 $\mu$ g/dl (16). However, to our knowledge no previous published research was conducted in Gaza strip on occupational lead exposure among gasoline station workers. The present study, therefore investigated some of the biochemical and physiological changes associated with lead exposure among gasoline station workers in Gaza Strip as well as their awareness towards lead exposure.

### **Aim of the study**

The overall aim of the study was to identify risk factors associated with lead exposure and to determine some biochemical and physiological parameters associated with occupational exposure among gasoline station workers in Gaza Strip.

### **The specific objectives were:**

1. To identify risk factors associated with lead exposure in gasoline station workers in the Gaza strip.
2. To determine BLL of gasoline station workers and compared it with controls.
3. To determine some biochemical and physiological parameters including, urea, uric acid, creatinine, ALT, AST, ALP and complete blood count (CBC) of the workers.

4. To measure the arterial blood pressure of the workers.
5. To analyze the relationship between BLL and risk factors and Biochemical and hematological parameters.
6. To study awareness of the workers toward lead exposure.

## **2.1 Overview**

Lead is found in the environment (air, water and soil) as well as in most biological systems. Lead can combine with other chemicals to form what are usually known as lead compounds or lead salts. Some lead salts dissolve in water better than others. Some natural and manufactured substances contain lead but do not look like lead in metallic form. Some of these substances can burn for example, organic lead compounds in some gasoline's (17). In addition lead and lead compounds are used in storage batteries, gasoline and metallic products including solder and pipes (18). A wide variety of population is at risk of occupational exposure to lead such as battery and auto radiator workers, gasoline station workers, construction and demolition workers, jewelers, lead miners, lead smelters and refiners, painters, pottery workers, printers and soldering of lead products (2, 19)

## **2.2 Lead exposure, absorption, distribution and excretion**

Lead exposure in the general population occurs primarily through ingestion, although inhalation also contributes to lead body burden and may be the major contributor for workers in lead related occupations. In gasoline stations, the major sources of exposure of leaded gasoline workers are lead fumes generated during filling cars or exposed to cars fumes and when lead from contaminated hands, food, water, cigarettes and clothing is ingested (1, 5, 20).

Lead is absorbed primarily through the respiratory and gastrointestinal systems, with the former being the more important route of entry in occupational exposures. Cutaneous absorption of inorganic lead (food, water and paint products) is negligible. However, organic lead compounds (leaded gasoline), because of their lipid solubility, are readily absorbed through intact skin (21, 22, 23). Respiratory lead absorption is primarily dependant on particle size, solubility, respiratory volume and physiological inter individual variation are less important factors. The percentage of inhaled lead reaching the bloodstream is estimated to be 30 to 40 percent (24).

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Gastrointestinal tract (GI) absorption of lead is greatest in infancy; infants can absorb up to 50 percent of lead ingested from food, water, contaminated dust, or soil, while adults absorb only 10-15 percent (22). Iron is believed to impair lead absorption in the gut, while iron deficiency is associated with increase blood lead concentrations (25). Calcium supplementation studies demonstrate that increased dietary calcium in animals and human result in consistent decreases in the absorption of lead (26, 27)

Following exposure to lead, the element is absorbed into and transported by the bloodstream to other tissues. In blood, most of lead is found in the erythrocytes. The freely diffusible plasma fraction is distributed extensively throughout tissues, reaching highest concentrations in bone, teeth, liver, lungs, kidneys, brain, spleen, muscles and heart (24). Lead in blood has an estimated half life of 35 days (28), in soft tissue 40 days (29), and in bones 20 to 30 years (30). With chronic exposure over a long period of time, most absorbed lead ends up in bone. Lead appears to be substituted for calcium in the bone matrix. This is not known to cause any deleterious effect on bone itself. Bone storage likely acts as a "sink", protecting other organs while allowing chronic accumulation. The lead that accumulates in the bone ultimately provides a source and remobilization and continued toxicity after exposure ceases (31, 32).

The biological half-life of lead may be considerably longer in children than in adults (29). Inorganic lead is not metabolized but is excreted unchanged, primarily in the urine. The mechanisms for fecal excretion of absorbed lead are not clearly understood; however, pathways of excretion may include secretion into the bile, gastric fluid, and saliva, accounting for approximately one-third of total excretion of absorbed lead (33). Organic or alkyl-lead, (leaded gasoline, also identified as tetraethyl- and tetramethyl- lead) undergoes oxidative dealkylation to the highly neurotoxic metabolites; triethyl- and trimethyl- lead (34). In the liver, the reaction is catalyzed by a cytochrome p450-dependent mono-oxygenase system (35). Lead can also be excreted through the nails and sweat; two studies have shown significant losses of lead in the sweat of study subjects undergoing sauna

therapy compared to urine levels (36, 37). Although lead is excreted by several routes (including sweat and nails), only the renal (in urine) and gastrointestinal (through biliary clearance ultimately, in the feces) are of practical importance. In general, lead is excreted quite slowly from the body (with the biologic half-life estimated at 10 years). Since excretion is slow, accumulation in the body occurs easily (5, 24).

### **2.3 Lead Toxicity Symptoms**

The presence of lead in the human body can lead to toxic effects regardless of exposure pathway. In the occupational setting lead poisoning remains one of the most important occupational and environmental health problems. Major symptoms of intoxication with leaded gasoline are referable to the CNS. The victims suffer from Insomnia, nightmares, anorexia, nausea & vomiting, diarrhea, headache, muscular weakness and emotional instability. With continued exposure, CNS manifestations progress to delusion, ataxia, exaggerated muscular movements and finally a maniacal state. In the case of severe exposure death may occur within a few hours or may be delayed for several weeks (38).

The most frequent symptoms among 95 lead workers in Korea were generalized weakness and fatigue (67.3%) and the next were tingling and numbness of arm or leg (43.1%), feeling irritation at the slightest disturbance (41%), and weakness of wrist or ankle joint (32.6%), while the least frequent symptoms was pains in the abdomen (4.2%). They added that the relatively high prevalence of symptoms could be partly explained by increased knowledge of lead related symptoms, because of an ongoing program of health education among lead workers in Korea (39).

Chronic health effects in 37 gasoline station Croatian workers who had exposed to leaded gasoline for more than five years were determined. In 13 out of 37 workers, the symptoms of depression and decreased reaction time and

motor abilities were identified (40).

In the Libyan Arab Jamahiriya, it was reported that, signs and symptoms of lead poisoning may include malaise, anorexia, abdominal pain, vomiting, lethargy, colic, constipation, irritability and apathy. Manifestation of lead poisoning includes effects on the hematopoietic, renal and CNS. Lead encephalopathy is characterized by a sudden onset of cerebral edema, coma, convulsions and death (41).

The effect of BLL in the health of lead related industrial workers in the United Arab Emirates was determined. The reported symptoms among industrial workers strongly associated with BLL were nausea/vomiting, muscular symptoms, dizziness, fatigue, irritability, memory disturbances, insomnia and allergic conjunctivitis (42).

Excessive occupational exposure to lead over a brief period of time can cause a syndrome of acute lead poisoning. Classic clinical findings in this syndrome included abdominal colic, constipation, fatigue and CNS dysfunction. With even greater doses, acute encephalopathy with coma and convulsion may occur. In milder exposure, headache and personality changes may be the only signs of neurologic toxicity (1, 5, 19, and 43).

Chronic lead toxicity is an insidious illness with protean manifestation. Symptoms may include arthralgias, headache, weakness, and depression, loss of libido, Importance and vague gastrointestinal difficulties. Late effects may include chronic renal failure, hypertension, gout and chronic encephalopathy (19, 44).

Clinical signs and symptoms in the different stages of lead toxicity as a result of exposure to lead were summarized (45). Signs of lead toxicity include in

general: hypertension, decreased nerve conduction velocity, hyper-reflexia, upper extremity weakness, forearm extensor weakness, gingival lead lines, buccal lead staining, papilledema, increased intracranial pressure and macular gray stains. Lead related symptoms include: (1) Earliest Symptoms: diffuse muscle weakness, general fatigue/lethargy, myalgia, joint pain/arthritis, loss of appetite, unusual taste in mouth/, change in taste of food, headache, insomnia, irritability, diminished libido, weight loss of 10 lbs or more without known cause, tremulousness, personality changes.

(2) Symptoms of chronic exposure: abdominal pain/cramping, nausea/vomiting, short-term memory loss, depression, incoordination, numbness and tingling in extremities, constipation, inability to concentrate and impotence.

(3) Severe toxicity: frank paralysis, somnolence/severe lethargy and abdominal colic.

#### **2.4. Blood lead level and associated risk factors**

BLL is the most powerful indicator for occupationally lead exposure. Several authors used BLL as a direct indicator for lead exposure in gasoline station workers as well as an indication of potential for adverse effect on health (46, 47). The BLL rises rapidly within hours after an acute exposure and remains elevated for several weeks (2, 5, 19, 43 and 48). Other measures used for lead exposure include Delta-aminolevulinic acid dehydratase (49, 50) and erythrocyte protoporphyrin (EP) or zinc protoporphyrin (ZPP) (19, 43, 51, and 52). Lead concentration can also be measured in urine, teeth and hair but these measurements are not reliable as BLL (1, 53).

BLLs were studied in 42 gas-station employees, 47 taxi drivers, 47 bus drivers, and 36 controls, all of whom worked in Athens, Greece (54). The BLLs did not differ significantly among the four groups ( $5.64 \pm 1.7 \mu\text{g/dl}$ ,  $5.96 \pm 1.7 \mu\text{g/dl}$ ,  $5.88 \pm 1.3 \mu\text{g/dl}$ , and  $5.76 \pm 1.7 \mu\text{g/dl}$ , respectively). This may be due to the reduction of tetraethyl lead in gasoline and the introduction of unleaded fuel in the past 10 years. Gas-station employees who smoked had higher BLLs than their nonsmoking counterparts.

In the Beirut, it was found that a BLL was associated with eating lunch at work, smoking, years of work, and younger age (47). Also, a significant and positive correlation with duration of employment and BLL among lead exposed group in the Egypt was reported (55).

Many authors demonstrated that the risk factors, together with lack of training programs and lead poisoning awareness campaigns, evidence that lead workers have little or no knowledge of the risk involved in occupational lead exposure (56, 57, and 58). The lack of appropriate hygiene measures in factories included infrequent washing of work clothes, lack of adequate respirators to avoid inhalation of lead, uncommon and improper use of rubber boots and gloves and the high proportion of workers who ate and smoked within the premises.

It was demonstrated that in workers exposed to lead, the Chinese have the lowest BLL, while the higher BLL in Malays and Indians may have been to by eating habits (eating with hands), in addition to the higher prevalence of smoking among Malays workers (59).

BLLs in relation to blood delta-aminolevulinic acid dehydratase (ALAD) activity were evaluated in lead smelters, automobile mechanics and gasoline retailers in the city of Accra, Ghana (60). Relationship between high BLLs (mean: 108 $\mu$ g/dl) and low ALAD activity (mean: 74.3 units) indicating lead over exposure was found in the lead smelters. Non-toxic lead exposure was, however, noted in the automobile mechanics and the gasoline retailers. Their respective mean blood lead levels were 27.8 $\mu$ g/dl (mean blood ALAD activity 212.5 units) and 8.6 $\mu$ g/dl (ALAD: 239.9 units). Personal habits at the work place appear to play a major role in facilitating exposure to lead among all the three groups of workers in addition to lack of control measures at the work place of the lead smelters to protect them against lead exposure.



Several studies demonstrated that male smokers have on the average slightly higher BLL than non-smoker (61, 62). Moreover among "Israelis" lead exposed workers, significant association between smoking and calcium intake with BLL was exhibited (63).

## **2.5. Biochemical and hematological implications of lead**

### **2.5.1. Effect of lead on some liver enzymes**

A study conducted on 37 chronic exposed gasoline workers in Croatia (40) revealed that the workers suffered from liver disorders: lipoid degeneration of liver (14 of 37), chronic functional damages of liver (3 of 37) and liver cirrhosis (1 of 37).

The effect of lead on some liver enzymes in 42 gasoline station employees, 47 taxi drivers, 47 bus drivers, and 36 controls worked in Athens, Greece has been studied (54). AST and ALT were elevated in gasoline station employees, and the former was elevated in taxi drivers.

Some toxicological effects of occupational exposure to petroleum products (especially petrol which contains tetraethyl lead) amongst 25 occupationally exposed and 25 none exposed as controls in Nigeria were evaluated (64). Significantly lower activities were observed for ALP in occupationally exposed subjects were higher compared with controls ( $66 \pm 18.9$  lu/L versus  $78 \pm 22.4$  lu/L,  $p < 0.01$ ). The activities of ALT ( $11.4 \pm 4.0$  lu/L) and AST ( $15.8 \pm 4.4$  lu/L) in occupationally exposed subjects were higher compared with controls (ALT= $6.8 \pm 2.7$  lu/L, AST= $9.6 \pm 3.5$  lu/L,  $p < 0.01$ ).

A group of Egyptian lead workers who were exposed to lead fumes for periods up to 22 years was studied (65). Changes in serum lipids and some of the liver function tests were investigated. Increase in serum AST and ALT was recorded implying that lead poisoning may have hepato-toxic action.

### 2.5.2 Renal effects of lead

The threshold level at which lead has an adverse effect on the kidney remains unknown. Most documented renal effects for occupational workers have been observed in acute high-dose exposures and high-to-moderate chronic exposures. However, some population-based studies showed accelerated increases in serum creatinine or decrements in creatinine clearance at relatively low BLL (66). It was found that early kidney damage is difficult to detect. However, a 10µg/dL increase in BLL has been associated with a 10.4ml/minute decrease in renal creatinine clearance rate (67). These results support the hypothesis that exposure to low-level environmental lead correlates with a significant decrement in renal function. Late effects may include chronic renal failure (19, 44).

The association of low-level lead exposure with impaired renal function was also determined (68). Blood lead concentration was positively and significantly associated with concurrent concentration of serum creatinine ( $P=.005$ ). A 10-fold increase in BLL predicted an increase of 7µmol/L (0.08µg/dL) in serum creatinine concentration, which is roughly equivalent to the increase predicted by 20 years of aging. The age-related increase in serum creatinine level was earlier and faster in the group with the highest-quartile levels of long-term lead exposure than in the group with the lowest-quartile levels.

Assessment of chronic health effects in 37 Croatian workers exposed to gasoline and its constituents for more than five years at gasoline station was made (40). Ultrasound examination indicated that (8 of 37) had chronic kidney damages. These results significantly differed from those of controls ( $P < 0.05$ ).

It was found that blood urea nitrogen (BUN) shows a significant relationship with five lead exposure indicators; blood lead level, blood zinc protoporphyrin, urine coproporphyrin, ALAD and urine ALA (69). The relationship between renal dysfunction and occupational lead exposure was studied in 55

Singapore male workers (70). Recent blood lead concentration was measured. Four-hour creatinine clearance and various other urinary and serum markers of renal dysfunction were used as effect indices. Creatinine clearance decreased significantly ( $P < 0.001$ ) with increasing BLL indicating a positive association between overall lead exposure and renal dysfunction.

### 2.5.3 Hematological effects of lead

Hematological parameters were examined in thirty-seven Croatian workers who had been exposed to gasoline for more than five years (40). Data were compared with controls. Peripheral smear revealed basophilic punctuated erythrocyte and reticulocytosis. Haematological disorders included mild leukocytosis (7 of 37), lymphocytosis (20 of 37), mild lymphocytopenia (3 of 37) and decrease of red blood cells count (11 of 37). These results were significantly differed from those of controls ( $P < 0.05$ ).

Lead levels and related biochemical findings occurring in Ghanaian subjects occupationally exposed to lead were also studied (60). Non-toxic lead exposure was noted in the automobile mechanics and the gasoline retailers. Their respective mean BLLs were 27.8  $\mu\text{g}/\text{dl}$  (mean blood ALAD activity 212.5 units) and 8.6  $\mu\text{g}/\text{dl}$  (ALAD: 239.9 units). However, anemia was found in 12.5% of the gasoline retailers but in none of the automobile mechanics.

Increased BLL associated with decreased blood hemoglobin was recorded in a group of Egyptian lead workers who were exposed to lead fumes for periods up to 22 years (65). The threshold BLL for a decrease in hemoglobin was estimated at 50  $\mu\text{g}/\text{dL}$  for occupationally exposed adults workers and approximately 40  $\mu\text{g}/\text{dL}$  for children, although other studies have indicated a lower threshold (17, 71).

Lead may inhibit the body's ability to make hemoglobin by interfering with

several enzymatic steps in the heme pathway. Specifically, lead decreases heme biosynthesis by inhibiting delta-aminolevulinic acid dehydratase and ferrochelatase activity (71, 72 and 73).

Nine subjects with sever blood lead burden level were evaluated (74). Most of the patients had mild or moderate anemia and moderate basophilic stippling evident in Wright's-stained peripheral smears. Basophilic stippling and microcytic or normocytic, hypochromic anemia only occur after significant levels of exposure as well as Hemoglobin levels do not start to decrease as a result of lead exposure until blood lead levels are 50µg/dL for adults (75).

#### **2.5.4 Lead exposure and hypertension**

Numerous observations have indicated a relationship between moderate or heavy lead exposure and high blood pressure. To determine whether low-level lead exposure is related to blood pressure in the U.S. population, data from the National Health and Nutrition Examination Survey II for persons 12 to 74 years of age were analyzed (76). Significant correlations were found between blood lead and blood pressure for each race-gender group, and blood lead levels were significantly higher in groups with high diastolic blood pressure (greater than 90 mm Hg). It was also suggested that lead may elevate blood pressure in susceptible adults in US population at BLL as low as 14µg/dL.

Several studies support an association between lead exposure (primarily occupational) and elevations in blood pressure (77, 78, and 79). The hypothesis of a positive association between lead exposure and high blood pressure was supported among lead exposed workers in United Arab Emirates (42). Long-term cumulative lead exposure can significantly increase blood pressure in moderately lead-exposed male workers.

The relationship between lead and blood pressure of 303 subjects occupationally exposed to this metal with blood-lead between 10 and 80  $\mu\text{g}/\text{dl}$  and 206 subjects belonging to the general population with blood-lead between 0.5 and 9  $\mu\text{g}/\text{dl}$  was investigated (80). In both groups there was a positive and statistically significant correlation between blood-lead values and systolic and diastolic blood pressure values. In contrast , other published evidence suggests a weak positive association between blood pressure and lead exposure (81).

### **3.1 Study Design**

The present study is a cross sectional study.

### **3.2 Target Population**

The study population was gasoline station male workers in Gaza Governorates during the spring of 2006.

### **3.3 Sampling and sample size**

The estimated number of legal gasoline stations registered in the Gaza Strip in the year 2006 was 81 (personal communication with municipalities of Gaza Governorates, Palestinian National Authority) distributed in the five Governorates of the Strip as follows: Northern (17), Gaza (27), Mid Zone (10), Khan Younis (19), and Rafah (8). The estimated number of workers in Gaza and Khan Younis gasoline stations was 3 workers/station whereas in Northern, Mid zone and Rafah the number was estimated to be 2 workers/station. Therefore, the total number of gasoline station workers in Gaza strip was approximately 208 workers. A stratified sample was used according to the number of workers in each Governorate. Therefore, the sample size of 105 workers was distributed as follows: Northern (18), Gaza (39), Mid Zone (12), Khan Younis (23), and Rafah (13).

### **3.4 Ethical considerations**

The researcher obtained the necessary approval to conduct the study from Helsinki committee in the Gaza strip. Helsinki committee is an authorized professional body for giving permission to researchers to conduct their studies with ethical concern in the area. An official letter of request was sent to Palestinian Ministry of Health to obtain approval to make some biochemical analysis in the central laboratories of AL Remal Clinic. Gasoline station workers were given an explanation about the purpose of the study and assurance about the confidentiality of the information and that the participation was optional.

### **3.5 Questionnaire Design**

A meeting interview was used for filling in the questionnaire. All interviews were conducted face to face by one investigator himself. The questionnaire was based on the review of literature related to occupational lead exposure and on that used in a similar study with some modifications (16). The questionnaire validity was tested by four experts in the field of environment and public health. Most questions were one of two types: the yes/no question, which offers a dichotomous choice; and the multiple choice question, which offers several fixed alternatives (82). The questionnaire includes several areas of questions such as personal data (address, age, marital status and education), work duration, house location, previous measurement of BLL and personal protective equipments in use. Workers were asked to provide information on personal hygiene practices and habits such as: smoking, drinking, eating, chewing gum, milk consumption and taking shower at work site. Workers were also asked about the common toxicity symptoms of lead experienced on the preceding 6 months. Several questions related workers knowledge's were included such as routes of lead entry into human body, and lead as environmental pollutant.

### **3.6 Pilot study**

Pilot study was done prior to beginning of data collection to know the length and clarity of questionnaire and to evaluate the outcome. Ten gasoline station workers were interviewed. At the end of the pilot study, a comprehensive revision to questionnaire was made and modified as necessary. The pilot subjects were not included in the study.

### **3.7 Data collection**

Data were collected over ten months through:

1. Questionnaire interviews which were conducted by the researcher himself as previously mentioned. At the end of every interview, the researcher looked over

the filled questionnaire to check adequate completion of all information and to monitor the flow of the serial numbers of the questionnaire.

2. Blood analysis of both gasoline station workers and controls who agreed to give blood samples.
3. Measurement of arterial blood pressure.

### **3.8 Blood sampling and processing**

Out of 105 gasoline station workers, a total of 72 workers were agreed to give blood sample for analysis. Controls who gave blood sample were 70 subjects selected from general population who almost have no history of being exposed to lead and matching the experimental group in age, sex and residence. Blood samples were collected by the researcher himself from the antecubital area of the workers and controls hands after cleaning with 70% ethanol. About 6 ml blood was drawn from each individual by plastic metallic-free disposable syringe (SANWOO corporation-Korea). Four ml blood was collected into two vacutainer vials containing potassium ethylenediamine tetracetic acid "EDTA (K<sub>3</sub>)" as anticoagulant produced by AFMA-DISPO-Jordan (each containing 2ml blood) for determination of blood lead level and complete blood count. The rest 2ml blood was collected in plastic tube and was left for short time to allow blood to clot. Then clear serum sample was obtained by centrifugation at 3000 rpm for 15<sup>th</sup> min. Serum AST, ALT, ALP, creatinine, urea and uric acid were analyzed in the central laboratories of AL Remal Clinic. In general, whole blood specimens were transported and stored at 4 °C.

### **3.9 Biochemical analysis**

#### **3.9.1 Determination of blood lead level**

##### **Principle**

Lead was measured in blood by atomic absorption spectrometry based on the method described by Miller and his colleagues (83). Quantification was based



on the measurement of light absorbed at 283.3 nm by ground state atoms of lead from either an electrode-less discharge lamp or from a hollow-cathode lamp source. Blood samples human and bovine blood quality control pools, and aqueous standards were diluted with a matrix modifier (nitric acid, Triton X – 100, and ammonium phosphate). The lead content was determined by using a Perkin-Elmer model 5100 atomic absorption spectrophotometer with Zeeman Effect background correction. Lead contamination must be carefully avoided throughout all procedures.

## Reagents

### A. reagent preparation

- Matrix modifier (0.2 % (v/v) nitric acid, 0.5% (v/v) Triton X-100, 0.2% (w/v) ammonium phosphate).
  
- Using Eppendorf pipets, dilute 2mL redistilled concentrated nitric acid and 5000 $\mu$ l (5.0mL) Triton X-100 in approximately 750ml Ultra pure water in an acid-cleaned 1000mL volumetric flask.
  
- Weight out 2.0 g of dibasic ammonium phosphate and add it to the flask by washing down the weighing boat with Ultra pure water delivered from a wash bottle.
  
- Add a magnetic stirring bar and stir the solution on a stirring plate until the Triton X-100 has dissolved.
  
- Remove the stirring bar and bring the solution to volume with Ultra pure water.
  
- After preparation, this solution should be checked for contamination at the beginning of each analytical run and discarded if absorbance greater than 0.005 Abs-sec is observed.
  
- Stored at room temperature and prepare as needed in a flask dedicated to this solution.

**B. Standards preparation**

1. 1000mg/L stock lead standard: If using NIST SRM 2121-2 or 3128, dilute 1.00mL (delivered using either a Class a volumetric pipet or the Micromedic Digiflex) to 10mL with ultra pure water in an acid-cleaned volumetric flask. Stored at room temperature and prepare every six months in a flask dedicated to this solution.
2. 10mg/L intermediate lead standard: using either a Class A volumetric pipet or the Micromedic Digiflex, dilute 1.00mL of the 1000mg/L stock lead standard to 100mL with Ultra pure water in an acid-cleaned volumetric flask. Stored at room temperature and prepare monthly in a flask dedicated to this solution.
3. Working lead standard: Using the Micromedic Digiflex, transfer the following volumes of intermediate standard to 10mL volumetric flasks and dilute to volume with Ultra pure water.

**Table (3.1) Standard preparation.**

Intermediate stock ( $\mu\text{L}$ )	Working standard concentration ( $\mu\text{L}$ )	Sample concentration ( $\mu\text{g/L}$ )
0(*)	0	0
50(*)	50	5
100(*)	100	10
150(*)	150	15
200(*)	200	20
250	250	25
300(*)	300	30
400(*)	400	40
500	500	50
600 (*)	600	60
750	750	75
800 (**)	800	80
1000	1000	100
Store at room temperature and prepare weekly in a flasks dedicated these solutions.		
(*) use these standard concentrations for NHANES3 studies.		
(**) use this standard when the SRM 955 control materials are to be run.		

**Sample preparation and procedure**

1. Using the micromedic Digiflex, dilute the specimens and controls tenfold (1:10) with the matrix modifier solution into clean auto-sampler cups. Use 100 $\mu$ L of specimen and 900 $\mu$ L of matrix modifier in the following procedure: pull up 100 $\mu$ L of blood into the tip, wipe the tip with a Kim-Wipe tissue, and dispense the blood and 900 $\mu$ L of matrix modifier into a cup. Observe the tip and release any air bubbles that may become trapped before dispensing into the auto-sampler cups. To do this, remove the tip from its clamp and hold the tip end to release air. If air becomes trapped in a blood sample in the tip, dispense it into the waste beaker and take another sample.
2. Place the auto-sampler cups containing the specimens in positions 7–34, with an additional blank (100 $\mu$ L water and 900 $\mu$ L matrix modifier) in position 6.
3. Place a cup containing the water used in the preparation of the run on the tray as a sample in position 37.

**Calculation**

1. The method described here is linear up to 4 $\mu$ mol/L, or 80 $\mu$ g/dL. Use the linear regression program in ROSCOE ("PESLEAD") to calculate the calibration curve and integrated absorbance values of the standards and samples. It will then subtract the blank and calculate the concentrations of the controls and specimens. The linear regression program generates slopes, intercepts, correlation coefficients; standardized residuals, and plotted and fitted curves. The correlation coefficient  $r^2$ , for each curve should be 0.990 or better. For optimum sensitivity, slopes should be more than 0.035; intercepts should be less than 0.003.
2. Repeat a specimen analysis. When duplicate integrated absorbencies below 0.03 Abs-sec (mean) differ be more than about 0.005 Abs-sec or when duplicate

integrated absorbencies above 0.03 Abs-sec (mean) differ by more than 0.01 Abs-sec. This corresponds to concentration differences of 0.01µg/dL and 0.025µg/dL, respectively. Reanalyze specimens containing more than 30µg/dL lead for confirmation. When reanalyzing any specimen with concentration greater than 60µg/dL. Prepare a new specimen by a diluting it twenty-fold (1+19), rather than tenfold (1+9). The results output from PESLEAD must then be multiplied by 10 to account for this higher dilution.

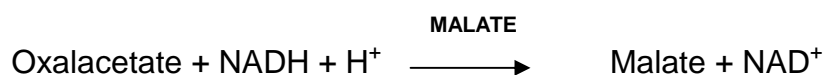
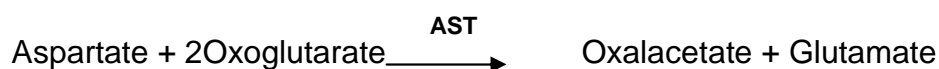
3. The detection limit, based on three times the standard deviation of ten repeat measurements of a sample with low lead concentration, is 0.03µmol/L (or 0.6µg/dL). Results below the detection limit are repeated as non-detectable.

### 3.9.2 Determination of aspartate aminotransferase (AST) activity

The activity of AST was determined according to Gella method (84) using Biosystems Reagent Kits (Spain).

#### Principle

AST catalyzes the transfer of the amino group from aspartate to 2-oxoglutarate, forming oxalacetate and glutamate. The catalytic concentration is determined from the rate of decrease of NADH, measured at 340 nm, by means of the malate dehydrogenase (MDH) coupled reaction.



## Reagents

**A Reagent:** Tris 121mmol/L, L-aspartate 362mmol/L, malate dehydrogenase >460U/L, lactate dehydrogenase >660U/L, Sodium hydroxide 255mmol/L, pH 7.8.

**B Reagent:** NADH 1.3mmol/L, 2-oxoglutarate 75mmol/L, Sodium hydroxide 148mmol/L, sodium azide 9.5g/L.

**C Reagent** (code11666): Pyroxial phosphate 10mmol/l. 5mL.

## Reagent preparation

Working Reagent: Pour the contents of the Reagent B into the Reagent a bottle. Mix gently. Other volumes can be prepared in the proportion: 4mL Reagent A+1mL Reagent B. Stable for 2 months at 2-8<sup>0</sup>C.

Working Reagent with Pyridoxal Phosphate: Mix as follows: 10mL of Working Reagent + 0.1mL of Reagent C (cod 11666). Stable for 6 days at 2-8<sup>0</sup>C.

## Procedure

1. Bring the working reagent and the instrument to reaction temperature.
2. Pipette into a cuvette:

Reagent temperature	37 <sup>0</sup> C	30 <sup>0</sup> C
Working reagent	1mL	1mL
Sample	50μL	100μL

3. Mix and insert the cuvette into the photometer. Start the stopwatch.

4. after 1 minute, record initial absorbance and at 1 minute intervals thereafter for 3 minutes.
5. Calculate the difference between consecutive absorbance, and the average absorbance difference per minute ( $\Delta A/\text{min}$ ).

### Calculation

AST concentration in the sample is calculated using the following general formula:

$$\Delta A/\text{min} \times \frac{V_t \times 106}{\epsilon \times l \times V_s} = \text{U/L}$$

The molar absorbance ( $\epsilon$ ) of NADH at 340 nm is 6300, the light path ( $l$ ) is 1 cm, the total reaction volume ( $V_t$ ) is 1.05 at 37°C and 1.1 at 30°C, the sample volume ( $V_s$ ) is 0.05 at 37°C and 0.1 at 30°C, and 1 U/L are 0.0166  $\mu\text{kat/L}$ . The following formulas are deduced for the calculation of the catalytic concentration.

	37°C	30°C
	x 3333 = U/L	x 1746 = U/L
$\Delta A/\text{min}$	x 55.55 = $\mu\text{kat/L}$	x 29.1 = $\mu\text{kat/L}$

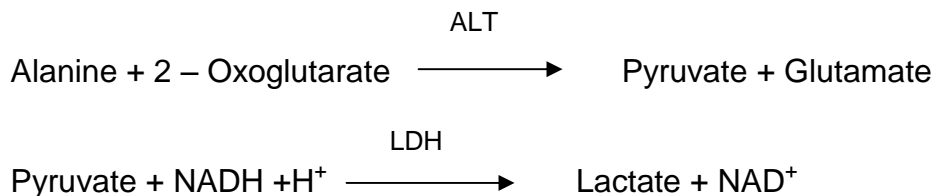
### 3.9.3 Determination of alanine aminotransferase (ALT) activity

The activity of ALT was determined according to Gella method (84) using Biosystems Reagent Kits (Spain).

#### Principle

ALT catalyzes the transfer of the amino group from alanine to 2-oxoglutarate, forming pyruvate and glutamate. The catalytic concentration is

determined from the rate of decrease of NADH, measured at 340 nm, by means of the lactate dehydrogenase (LDH) coupled reaction.



### Reagents

**A Reagent:** Tris 150mmol/L, L-alanine 750mmol/L, lactate dehydrogenase >1350 U/L, pH 7.3.

**B Reagent:** NADH 1.3 mmol/L, 2-oxoglutarate 75 mmol/L, Sodium hydroxide 148 mmol/L, sodium azide 9.5 g/L.

**C Reagent** (code11666): Pyroxial phosphate 10 mmol/l. 5mL.

### Reagent preparation

Working Reagent: Pour the contents of the Reagent B into the Reagent a bottle. Mix gently. Other volumes can be prepared in the proportion: 4 mL Reagent A + 1 mL Reagent B. Stable for 2 months at 2-8 °C.

Working Reagent with Pyridoxal Phosphate: Mix as follows: 10 mL of Working Reagent + 0.1 mL of Reagent C (cod 11666). Stable for 6 days at 2-8 °C.

### Procedure

1. Bring the Working Reagent and the instrument to reaction temperature.
2. Pipette into a cuvette:

Reagent temperature	37 °C	30 °C
Working reagent	1 mL	1 mL
Sample	50 µL	100 µL

3. Mix and insert the cuvette into the photometer. Start the stopwatch.
4. after 1 minute, record initial absorbance and at 1 minute intervals thereafter for 3 minutes.
5. Calculate the difference between consecutive absorbances, and the average absorbance difference per minute ( $\Delta A/\text{min}$ ).

### Calculation

ALT concentration in the sample is calculated using the following general formula:

$$\Delta A/\text{min} \times \frac{V_t \times 10^6}{\epsilon \times l \times V_s} = \text{U/L}$$

The molar absorbance ( $\epsilon$ ) of NADH at 340 nm is 6300, the light path ( $l$ ) is 1 cm, the total reaction volume ( $V_t$ ) is 1.05 at 37°C and 1.1 at 30°C, the sample volume ( $V_s$ ) is 0.05 at 37°C and 0.1 at 30°C, and 1 U/L are 0.0166 µkat/L. The following formulas are deduced for the calculation of the catalytic concentration:

	37 °C	30 °C
	$\times 3333 = \text{U/L}$	$\times 1746 = \text{U/L}$
$\Delta A/\text{min}$	$\times 55.55 = \mu\text{kat/L}$	$\times 29.1 = \mu\text{kat/L}$

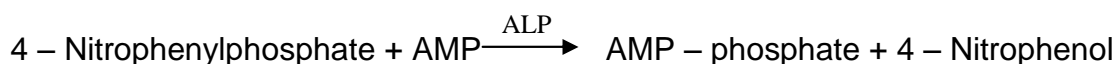
#### 3.9.4 Determination of alkaline phosphatase (ALP) activity

The activity of ALP was measured according to Rosalki method (85) using Blosystems Reagent Kits (Spain).



## Principle

ALP catalyzes in alkaline medium the transfer of the phosphate group from 4-nitrophenylphosphate to 2-amino-2-methyl-1-propanol (AMP), liberating 4-nitrophenol. The catalytic concentration is determined from the rate of 4-nitrophenol formation, measured at 405 nm.



## Reagents

**A Reagent:** 2-Amino-2-methyl-1-propanol 0.4 mol/L, zinc sulfate 1.2 mmol/L, N-hydroxyethyl ethylene diaminetriacetic acid 2.5 mmol/L, magnesium acetate 2.5 mmol/L, pH 10.4.

**B Reagent:** 4-Nitrophenylphosphate 60 mmol/L.

## Reagent preparation

### Working Reagent

- Cod. 11592 and 11593: Transfer the contents of one Reagent B vial into a Reagent A bottle. Mix gently. Other volumes can be prepared in the proportion: 4 mL Reagent A + 1 mL Reagent B. Stable for 2 months at 2-8<sup>0</sup>C.

- Cod. 11598: Transfer 25 mL of one Reagent B vial into a Reagent A bottle. Mix gently. Other volumes can be prepared in the proportion: 4mL Reagent A + 1 mL Reagent B. Stable for 2 months at 2-8<sup>0</sup>C.

**Procedure**

1. Bring the working reagent and the instrument to reaction temperature.
2. Pipette into a cuvette:

Working reagent	1 mL
Sample	20 $\mu$ L

3. Mix and insert the cuvette into the photometer.
4. Record initial absorbance and at 1 minute intervals thereafter for 3 minutes.
5. Calculate the difference between consecutive absorbencies, and the average absorbance difference per minute ( $\Delta A/\text{min}$ ).

**Calculation**

ALP catalytic concentration in the sample is calculated using the following general formula:

$$\frac{\Delta A/\text{min}}{\text{U/L}} \times \frac{V_t \times 10^6}{\epsilon \times l \times V_s} =$$

The molar absorbance ( $\epsilon$ ) of 4-nitrophenol at 405 nm is 18450, the light path ( $l$ ) is 1 cm, the total reaction volume ( $V_t$ ) is 1.02, the sample volume ( $V_s$ ) is 0.02, and 1 U/L are 0.0166  $\mu$ kat/L. The following formulas are deduced for the calculation of the catalytic concentration:

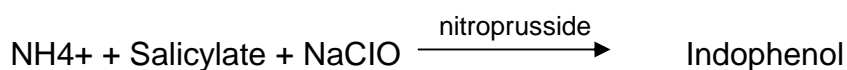
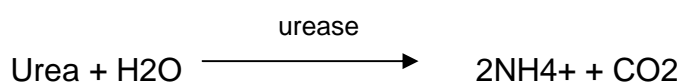
$\Delta A/\text{min}$	$\times 2764 = \text{U/L}$ $\times 46.08 = \mu\text{kat/L}$
-----------------------	--

### 3.9.5 Determination of urea

Urea determination is based upon the cleavage of urea by urease according to Burtis assay (86) using Biosystems Reagent Kits (Spain).

#### Principle

Urea in the sample consumes, by means of the coupled reaction described below, NADH that can be measured by spectrophotometry.



#### Reagents

**A Reagent:** Tris 100mmol/L, 2-oxoglutarate 5.6mmol/L, urease >140U/mL, glutamate dehydrogenase >140U/mL, ethyleneglycol 220g/L, sodium azide 9.5 g/L, pH8.0.

**B Reagent:** NADH 1.5 mmol/L, sodium azide 9.5g/L.

**S** Glucose/Urea/Creatinine Standard. Glucose 100mg/dL, urea 50mg/dL (8.3mmol/L, BUN 23.3mg/dL), creatinine 2mg/dL. Aqueous primary standard.

#### Reagent preparation

Working Reagent: transfer the content of one reagent B vial into a reagent A bottle. Mix gently. Other volumes can be prepared in the proportion: 4mL Reagent A+1mL Reagent B. stable for 2 months at 2-8°C.

**Procedure**

1. Bring the working Reagents and the photometer to 37°C.
2. Pipette into a cuvette:

Working Reagent	1.5mL
Standard (S) or sample	10µL

3. Mix and insert the cuvette into the photometer. Start stopwatch.
4. Record the absorbance at 340 nm after 30 seconds ( $A_1$ ) and after 90 seconds ( $A_2$ ).

**Calculation**

The urea concentration in the sample is calculated using the following general formula:

$(A_1 - A_2)$  Sample

$$\frac{\text{—————}}{\text{—————}} \times C_{\text{Standard}} \times \text{Sample dilution factor} = C_{\text{Sample}}$$

$(A_1 - A_2)$  Standard

If the Urea Standard provided has been used to calibrate:

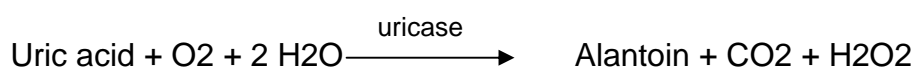
	Serum and plasma
$(A_1 - A_2)$ Sample —————	x 50 = mg/dL urea x 23.3 = mg/dL BUN
$(A_1 - A_2)$ Standard	x 8.3 = mmol/L urea

### 3.9.6 Determination of uric acid

Uric acid was measured using Biosystems Reagent Kits (Spain) and following their instruction manual, described by Fossati and his colleagues (87).

#### Principle

Uric acid in the sample originates, by means of the coupled reactions described below, a colored complex that can be measured by spectrophotometry.



#### Reagents

**A Reagent:** Phosphate 100mmol/L, detergent 1.5g/L, dichlorophenolsulfonate 4 mmol/L, uricase > 0.12U/mL, ascorbate oxidase > 5U/mL, peroxidase > 1U/mL, 4-aminoantipyrine 0.5mmol/L, pH 7.8

**B Uric Acid Standard:** Uric acid 6mg/dL (357µmol/L). Aqueous primary standard.

#### Reagent preparation

Reagent and standard are provided ready to use.

#### Procedure

1. Bring the reagent to room temperature.

2. Pipette into labeled test tubes:

Reagent temperature	Blank	Standard	Sample
Distilled water	25 $\mu$ L	—	—
Uric Acid Standard (S)	—	25 $\mu$ L	—
Sample	—	—	25 $\mu$ L
Reagent (A)	1.0mL	1.0mL	1.0mL

3. Mix thoroughly and incubate the tubes for 10 minutes at room temperature (16-25<sup>0</sup>C) or for 5 minutes at 37<sup>0</sup>C.

4. Measure the absorbance (A) of the Standard and the Sample at 520 nm against the Blank. The color is stable for at least 30 minutes.

### Calculation

The uric acid concentration in the sample is calculated using the following general formula:

$$(A \text{ sample} / A \text{ standard}) \times C_{\text{Standard}} \times \text{Sample dilution factor} = C_{\text{Sample}}$$

If the uric acid Standard provided has been used to calibrate

	Serum and plasma
A sample	x 6 = mg/dL uric acid
A standard	x 357 = $\mu$ mol/L uric acid

### 3.9.7 Determination of creatinine

Serum creatinine was determined kinetically using Biosystems Reagent Kits (Spain) and following their instruction manual described by Fabiny and Ertingshausen (88).

#### Principle

Creatinine in the sample reacts with picrate in alkaline medium forming a colored complex. The complex formation rate is measured in a short period to avoid interferences.

#### Reagents

A Reagent Picric acid 25mmol/L.

B Reagent Sodium hydroxide 0.4mol/L, detergent.

S Glucose / urea / creatinine standard. Glucose 100mg/dl, urea 50mg/dl, creatinine 2mg/dl (177  $\mu$ mol/L). Aqueous primary standard.

#### Reagents preparation

Standard (S) is provided ready to use.

Working reagent: mix equal volumes of reagent A and reagent B. mix thoroughly. Stable for 1 month at 2- 8<sup>0</sup>C.

#### Procedure

1. Bring the Working Reagent and the photometer to 37<sup>0</sup>C.
2. Pipette into a cuvette:

Working Reagent	1.0mL
Standard (S) or Sample	0.1mL

3. Mix and insert cuvette into the photometer. Start stopwatch.
4. Record the absorbance at 500 nm after 30 seconds ( $A_1$ ) and after 90 seconds ( $A_2$ ).

### Calculation

The creatinine concentration in the sample is calculated using the following general formula:

$$\frac{(A_2 - A_1) \text{ Sample}}{(A_2 - A_1) \text{ Standard}} \times C_{\text{Standard}} \times \text{Sample dilution factor} = C_{\text{Sample}}$$

If the creatinine standard provided has been used to calibrate:

	Serum and plasma
$\frac{(A_2 - A_1) \text{ Sample}}{(A_2 - A_1) \text{ Standard}}$	$\times 2 = \text{mg/dL creatinine}$ $\times 177 = \mu\text{mol/L creatinine}$

### 3.9.8 Hematological analysis

A complete system of reagents of control and calibrator, Cell-Dyn 1700 (89) was used to determined complete blood count (CBC) in Balsam laboratory.



### 3.10 Measurement of arterial blood pressure

The researcher used Mercurial Sphygmomanometer (Bokang, china) to measure the arterial blood pressure for all participants (90). The examination sequence was as follows:

- Support the arm comfortably at about heart level.
- Apply the cuff to the arm.
- Identify the brachial pulse.
- Inflate the cuff until the pulse is impalpable. Note the pressure on the manometer which is a rough estimate of systolic pressure.
- Now inflate the cuff another 10mmHg and listen through the stethoscope over the brachial artery.
- Deflate the cuff slowly until regular heart sounds (called Korotkoff sounds) can be just heard. This is the systolic pressure which should be measured to the nearest 2mmHg.
- Continue to deflate the cuff slowly until the sounds disappear.
- Record the point at which the sounds just disappear as diastolic pressure.

### 3.11 Limitation of the study

The researcher in this thesis faced some problems that he could solve some but others he couldn't. **The following are the most obstacles that faced the researcher during the study conduction:**

- The researcher found that some gasoline stations were closed or destroyed because of the circumstances in Gaza strip during the period of the study.
- Some gasoline station owners restrict the researcher ability to get the information he wants.

- Some gasoline station workers, who filled questionnaires and firstly agreed to give blood sample, refused later to give the sample when the researcher returned to collect the blood samples.
- Lack of funds and high cost of some materials.

### **3.12 Statistical analysis:**

Data were computer analyzed using SPSS/PC (Statistical Package for the Social Science Inc., Chicago, Illinois USA, and version 11.0).

#### **Data analyses were carried out as follows:**

- Over viewing field questionnaire.
- Coding of questionnaire.
- Choosing data entry mode and data entry.
- Data cleaning.
- Frequency table for study variables.
- Defining and re-coding of certain variables.

#### **The statistical tests of significance were used depending on the nature as follows:**

- The one-way ANOVA test was used for analysis of variance for average BLL as quantitative dependant variable by qualitative variables such as the relationship between BLL by place of residency, age groups, levels of education and so on.
- The chi-square test ( $X^2$ ) was used to test the relationship between the BLL of the followed up workers by workers house location to lead facility BLL of the followed up workers by protective measures in use BLL of the followed up

workers by knowledge of lead entry into human body, lead health effects or lead as an environmental pollutant.

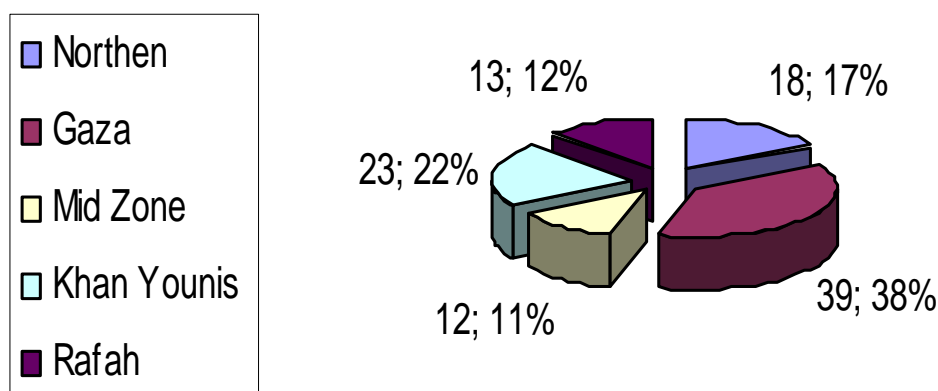
- The independent-sample t-test procedure was used to compare means of quantitative variables by the separated cases into two qualitative groups such as the relationship between workers and controls biochemical parameters.
- The results in all the above mentioned procedures were accepted as statistical significant when the P-value was less than 5% ( $P < 0.05$ ).

The present data were collected by face to face interview and each of 105 gasoline station workers filled a questionnaire in different Gaza Strip governorates. Out of the 105 workers, 72 freely accepted to give blood samples for BLL and biochemical analysis.

#### 4.1 Questionnaire Data analysis

##### 4.1.1 Distribution of gasoline station workers by the governorates of the Gaza strip

Figure (4.1) illustrates the distribution of gasoline station workers who filled questionnaire by various governorates of the Gaza Strip. According to the number of workers in each Governorate, a sample size of 105 workers was distributed as follows: Northern 18 (17.1%), Gaza 39 (37.1%), Mid Zone 12 (11.4%), Khan Younis 23 (21.9%), and Rafah 13 (12.4%).



**Figure 4.1 Distribution of gasoline station workers by the governorates of the Gaza Strip**

##### 4.1.2 General characteristics of the study population

All gasoline station workers encountered in our study were males. Table (4.1) summarized the personal profiles of study population. The age of the

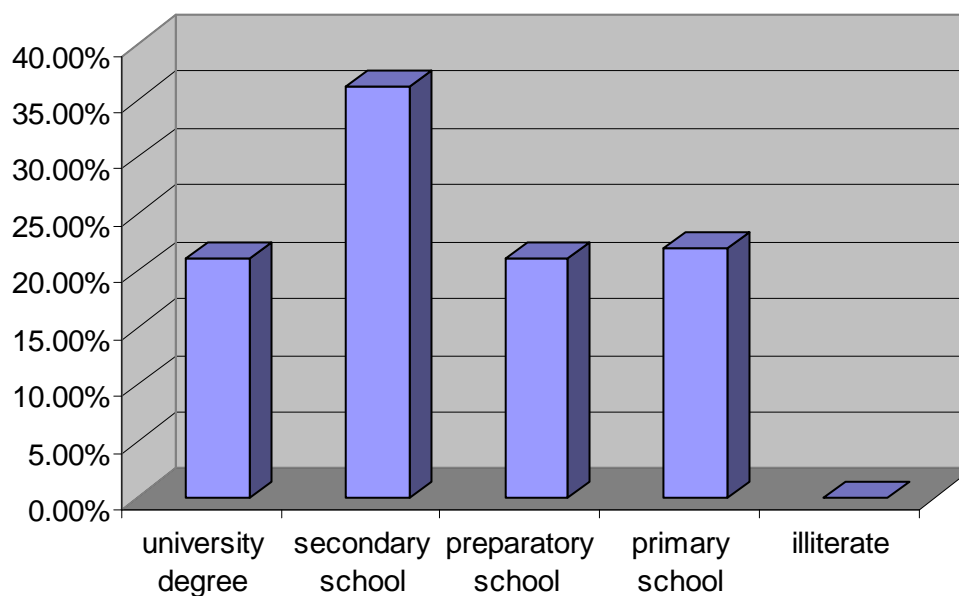
workers ranged between 19 to 73 years old with mean of  $34.4 \pm 10.7$ . The highest number of workers 36 (34.3%) was found in the age group between 27 to 34 years old while the lowest number 7 (6.7%) was found in the age group higher than 51 years old. Eighty eight (83.8%) gasoline station workers were found to be married and 17 (16.2%) were single. Out of 88 married workers, 81 (77.1%) have children; 29 (27.6%) have 1-3 children, 31 (29.5%) have 4-6 children and 21 (20.0%) have more than 7 children. It is worth mentioning that 7 (22.9%) of married workers have no children.

**Table 4.1 Personal profile of the study population (n =105).**

<b>Character</b>	<b>No.</b>	<b>%</b>
<b>Age (year)</b>		
19–26	27	25.7
27-34	36	34.3
35-42	18	17.1
43-50	17	16.2
> 51	7	6.7
<b>Marital status</b>		
single	17	16.2
married	88	83.8
Have no children	7	22.9
Have children	81	77.1
<b>Number of children</b>		
1-3 child	29	27.6
4-6 child	31	29.5
> 7 child	21	20.0

Analysis of the educational status of the study population showed that 22 (21.0%) had a university degree, 38 (36.2%) had finished secondary school, 22

(21.0%) had finished preparatory school and 23 (21.9%) had passed primary school. No workers were found to be illiterate (Figure 4.2).



**Figure 4.2 Educational status for the study population (n=105)**

#### 4.1.3 Work duration

Work duration for gasoline station workers is presented in Table (4.2). More than half of the workers 59 (56.2%) were found to be worked in the gasoline station for more than 5 years, whereas 26 (24.8%) and 20 (19.0%) of them worked for 3 -5 and < 2 years, respectively. It is worth mentioning that, all interviewed workers had no history of previous job related to sources of lead pollution.

**Table 4.2 Work duration in gasoline station for the study population (n =105)**

Work duration (Year)	No.	%
<2	20	19.0
3-5	26	24.8
>5	59	56.2

#### 4.1.4 Previous testing for blood lead level and willing to provide blood sample

As indicated in Table (4.3) none of the workers were found to have previous blood lead testing. However, 90 (85.7%) workers were willing to provide blood sample for lead analysis

**Table 4.3 Previous Blood lead testing and willing to providing blood samples by the study population (n=105)**

Question	Yes	%	No	%
Have you ever tested for blood lead level?	0	0	105	100
Are you willing to give blood sample for lead analysis?	90	85.7	15	14.3

#### 4.1.5 Houses location

House location in relation to some sources of lead pollution is presented in Table (4.4). The numbers of gasoline station workers who mentioned that their houses are located near battery workshop, auto radiator workshop, garage of cars and gasoline station were 3 (2.9%), 3 (2.9%), 16 (15.2%) and 6 (5.7%), respectively. However, none of the workers mentioned that their houses are located near lead smelter.

**Table 4.4 House location in relation to some sources of lead pollution as reported by the study population (n=105).**

Houses near to	Yes	%	No	%
Lead smelter	0	0	105	100
Battery workshop	3	2.9	102	97.1
Auto radiator workshop	3	2.9	102	97.1
Garage of cars	16	15.2	89	84.8
Gasoline station	6	5.7	99	94.3

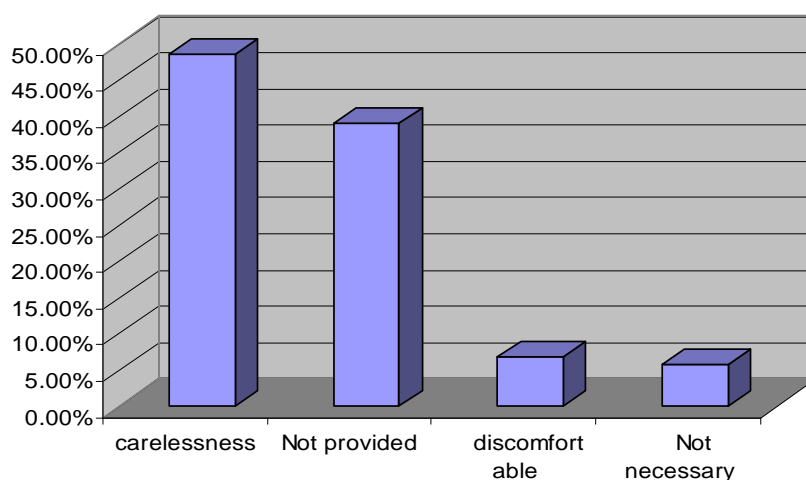
#### 4.1.6 Protective measures in use by the study population

Table (4.5) lists the different protective measures regularly used by gasoline station workers (n=105). In general, the protective measures during work in the station were poorly followed. The highest number of workers (n=30, 28.6%) wore gloves and the lowest number (n=1, 1.0%) wore hat or shoes. The number of workers who mentioned not smoking, not drinking, not eating and not chewing gum were 51 (48.6%), 17 (16.2%), 17 (16.2%), and 10 (9.5%), respectively. Moreover, 47 (44.8%) workers drank milk frequently and 15 (14.3%) had a water bath at work place. Figure (4.3) showed that the main cause of not using safety equipments was carelessness of the workers 52 (48.6%).

**Table 4.5 Protective measures in use among gasoline station workers (n=105).**

Protective measures in use	Yes	%	No	%
<b>Safety equipments</b>				
Gloves	30	28.6	75	71.4
Safety glasses	3	2.9	102	97.1
Hat	1	1.0	104	99.0
Respiratory mask	10	9.5	95	90.5
Shoes	1	1.0	104	99.0
Overall	15	14.3	90	85.7
Not Smoking	51	48.6	54	
Not Drinking	17	16.2	88	51.4
Not Eating	17	16.2	88	83.8
Not Chewing gum	10	9.5	95	83.8
Drinking milk frequently	47	44.8	58	90.5
Have water bath	15	14.3	90	55.2
				85.7





**Figure 4.3 Causes of not using safety equipments**

#### **4.1.7 Knowledge of workers regarding routes of lead entry into the body, lead health effects and lead as an environment pollutant**

Table (4.6) illustrates the knowledge of the gasoline station workers (n=105) on the route of lead entry into the body, health effects of lead and lead as an environmental pollutant. Regarding possible routes of lead entry into the body, 91 (86.7%) workers mentioned that inhalation is the route of entry, followed by 34 (32.4%) who reported that skin is the route of entry, and 31 (29.5%) who claimed that the mouth is the route of entry of lead into the body. A total of 88 (83.3%) workers had knowledge about the health effects of lead on human health. It was also found that 89 (84.8%) knew that lead is an environmental pollutant.

**Table 4.6 Knowledge of the gasoline station workers (n=105) regarding route of lead entry into body, lead health effects and lead as an environmental pollutant.**

Items	Yes	%	No	%
<b>Route of lead entry into body</b>				
Inhalation	91	86.7	14	13.3
Through skin	34	32.4	71	67.6
Through mouth	31	29.5	74	70.5
<b>Effect of lead exposure on health</b>	88	83.3	17	16.2
<b>Lead as an environmental pollutant</b>	89	84.8	16	15.2

#### 4.1.8 Prevalence of self reported symptoms related to lead exposure

Self reported toxicity symptoms associated with lead exposure as recalled by the study population (n=105) who had experienced in 6 months preceding the present interview are listed in Table (4.7). The most common experienced symptoms of lead exposure reported by gasoline station workers were headache 78 (74.3%), fatigue 74 (70.5%), irritability 66 (62.9%) and concentration difficulties 65 (61.9%). Sleep disturbance, hypertension, nausea, constipation and dyspepsia were reported by 55(52.4%), 52 (49.5%), 50 (47.6%), 49 (46.7%) and 45 (42.9%) workers. The least common symptoms were seizures 1 (1.0%), convulsion and miscarriage (f)/infertility 3 (2.9%), and coma 4 (3.8%).

**Table 4.7 Self- reported symptoms related to lead exposure of the study population (n=105).**

<b>Self- reported symptom*</b>	<b>Yes</b>	<b>%</b>	<b>No</b>	<b>%</b>
Fatigue	74	70.5	31	29.5
Irritability	66	62.9	39	37.1
Coma	4	3.8	101	96.2
Convulsion	3	2.9	102	97.1
Headaches	78	74.3	27	25.7
Concentration difficulties	65	61.9	40	38.1
Sleep disturbance	55	52.4	50	47.6
Seizures	1	1.0	104	99.0
Hearing loss	22	21.0	83	79.0
Wrist/foot drop	39	37.1	66	62.9
Loss of libido	33	31.4	72	68.6
Nausea	50	47.6	55	52.4
Dyspepsia	45	42.9	60	57.1
Constipation	49	46.7	56	53.3
Abdominal pain	39	37.1	66	62.9
Lead line in gingival tissue	18	17.1	87	82.9
Renal pain	15	14.3	90	85.7
Hypertension	52	49.5	53	50.5
Miscarriage (f)/infertility	3	2.9	102	97.1

*\*Each worker reported more than two symptoms.*

#### **4.1.9 Attending training courses, seeing lead poisoning cases and health professionals visiting to gasoline station**

Neither worker attended training courses nor they had health professionals visited their station. In addition only one worker reported that he hear about one case of lead poisoning during his work in the gasoline station.

## 4.2 Blood lead level analysis

### 4.2.1 Response rate

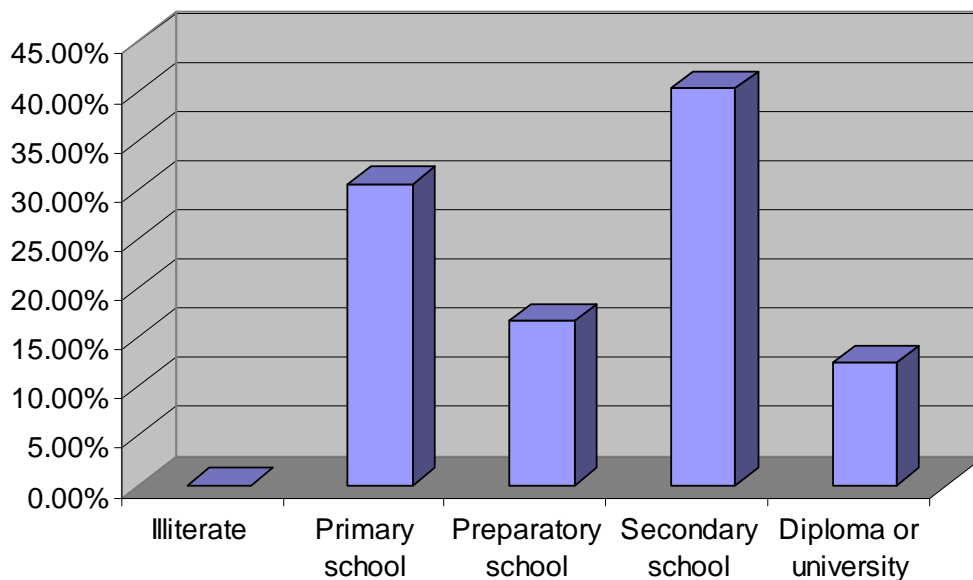
Out of the study population (105 gasoline station workers), a total of 72 workers gave blood samples (follow-up workers) i.e. the total response rate was 68.6 %.

### 4.2.2 General characteristic of the followed up workers

The general characteristics of the followed-up workers i.e. workers who gave blood samples (n=72) are summarized in Table (4.8). The highest number of participants 24 (33.3%) were found in the age group 27-34 years old while the lowest number of respondents 6 (8.3%) were >51 years old. Sixty six (91.7%) gasoline station workers were found to be married and 6 (8.3%) were single. Out of 66 married workers, 65 (98.5%) have children; 22 (33.8%) have 1-3 children, 25 (38.5%) have 4-6 children and 18 (27.7%) have more than 7 children. Analysis of the educational status of the participant workers showed that 9 (12.5%) had a university degree, 29 (40.3%) had finished secondary school, 12 (16.7%) had finished preparatory school and 22 (30.6%) had passed primary school (Figure 4. 4).

**Table 4.8 General characteristic of the followed-up workers (n=72)**

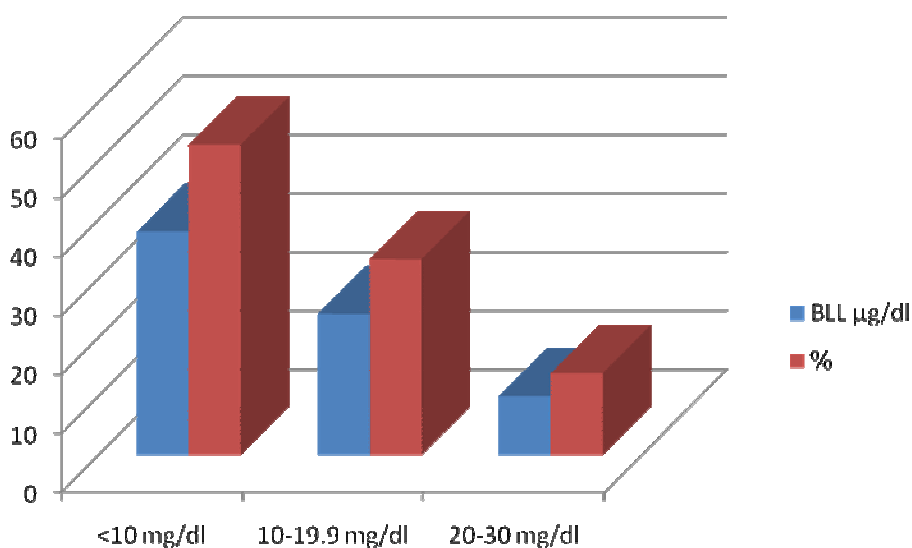
Character	No	%
<b>Age</b>		
19–26 years old	11	15.3
27-34 years old	24	33.3
35-42 years old	16	22.2
43-50 years old	15	20.8
> 51 years old	6	8.3
<b>Martial status</b>		
single	6	8.3
married	66	91.7
Have no children	1	1.5
Have children	65	98.5
<b>Number of children</b>		
1-3 child	22	33.8
4-6 child	25	38.5
> 7 child	18	27.7



**Figure 4.4 Educational status for follow up workers**

#### 4.2.3 Distribution of blood lead level among the gasoline station workers

Figure (4.5) illustrates the distribution of the workers according to their BLL. It is clear that 38 (52.8%) workers had BLL <10  $\mu\text{g}/\text{dl}$  with mean of  $6.79 \pm 1.50$ , 24 (33.3%) workers had BLL 10-19.9  $\mu\text{g}/\text{dl}$  with mean of  $14.12 \pm 2.54$  and 10 (13.9%) workers had BLL 20-30  $\mu\text{g}/\text{dl}$  with mean of  $22.58 \pm 2.42$ .



**Figure 4.5. Distribution of BLL among the following up workers (n=72)**

*BLL was expressed as mean  $\pm$  SD.*

#### 4.2.4 Blood lead level of the followed up workers by age

Table (4.9) showed the relation between mean BLL and age of gasoline station workers. In general there was no significant relationship between BLL and age of the workers ( $F= 1.874$ ,  $P\text{-value}=0.125$ ). However, the highest BLL was found among workers of the age group over 51 years old ( $15.70\pm 6.45$ ) whereas the lowest mean BLL was found among those of the age group 19-26 years old ( $8.67\pm 5.27$ ).

**Table 4.9 Blood lead level of the followed up workers (n= 72) by age.**

Age (years)	No	Mean $\pm$ SD ( $\mu\text{g/dl}$ )	F	P-value
19–26	11	8.67 $\pm$ 5.27		
27-34	24	12.41 $\pm$ 6.73		
35-42	16	9.93 $\pm$ 4.20	1.874	0.125
43-50	15	11.75 $\pm$ 5.81		
> 51	6	15.70 $\pm$ 6.45		

*BLL was expressed as mean  $\pm$  SD,  $P>0.05$ : non significant.*

#### 4.2.5 Blood lead level of the followed up workers by educational status

The mean BLL of workers was found to be decreased with increasing the educational status; where BLL of workers who had diploma or university degree was  $7.98\pm 4.32 \mu\text{g/dl}$  and that of workers who had finished primary school was  $16.24\pm 7.25 \mu\text{g/dl}$  (Table 4.10). This negative relationship was found to be statistically significant ( $P\text{-value}=0.001$ ).

**Table 4.10 Blood lead level of the followed up workers (n= 72) by educational status**

Educational status	No	%	Mean $\pm$ SD ( $\mu\text{g}/\text{dl}$ )	F	P-value
Primary school	22	30.6	16.24 $\pm$ 7.25		
Preparatory school	12	16.7	10.82 $\pm$ 3.84	10.12	0.001
Secondary school	29	40.3	9.10 $\pm$ 3.41		
Diploma or university	9	12.5	7.98 $\pm$ 4.32		

BLL was expressed as mean  $\pm$  SD,  $P < 0.05$ : significant.

#### 4.2.6 Blood lead level of the followed up workers by work duration

The mean BLL of workers was found to be increased with increasing the work duration; BLL of workers worked for 3-5 years was 7.75 $\pm$ 5.60  $\mu\text{g}/\text{dl}$  and that of workers worked for more than 5 years was 12.48 $\pm$ 5.76 $\mu\text{g}/\text{dl}$  (Table 4.11). This positive relationship was found to be statistically significant (P-value=0.004).

**Table 4.11 Blood lead level of follow up workers (n= 72) by work duration**

Work duration (year)	No	%	Mean $\pm$ SD ( $\mu\text{g}/\text{dl}$ )	T	P - value
3 -5	16	22.2	7.75 $\pm$ 5.60		
> 5	56	77.8	12.48 $\pm$ 5.76	-2.945	0.004

BLL was expressed as mean  $\pm$  SD,  $P < 0.05$ : significant.

#### 4.2.7 Blood lead level of the followed up workers by workers house location to lead facility

Table (4.12) showed that the relationship between BLL and the location of workers house to lead facility including lead smelter, battery workshop, auto radiator workshop, garage of cars or gasoline station was not statistically significant (P-value  $> 0.05$ ).

**Table 4.12 Blood lead level of the followed up workers (n= 72) by workers house location to lead facility**

House location	No.	%	BLL Mean $\pm$ SD ( $\mu$ g/dl)			X <sup>2</sup>	P- value
			>10	10-19.9	<20		
<b>Lead smelter</b>							
Yes	0	0	0	0	0	NA	NA
No	72	100	38	24	10		
<b>Battery workshop</b>							
Yes	3	4.2	2	1	0	0.549	0.760
No	69	95.8	36	23	10		
<b>Auto radiator workshop</b>							
Yes	2	2.8	1	1	0	0.460	0.794
No	70	97.2	37	23	10		
<b>Garage of cars</b>							
Yes	11	15.3	5	5	1	0.919	0.631
No	61	84.7	33	19	9		
<b>Gasoline station</b>							
Yes	4	5.6	3	1	0	1.073	0.585
No	68	94.4	35	23	10		

\* NA= Not available,  $P > 0.05$ : non significant.

#### 4.2.8 Blood lead level of the followed up workers by protective measures in use

The relationship between BLL and protective measures in use is illustrated in Table (4.13). In general, the mean BLL was found to be lower among gasoline station workers who used and /or followed protective measures than among those who did not. However, The differences of mean BLL among workers who used gloves, respiratory mask or drank milk frequently and those who did not were statistically significant (P-value=0.001,  $X^2 = 14.45$ ; P-value= 0.006,  $X^2 = 10.39$  and P-value=0.004,  $X^2 = 10.93$ , respectively).



**Table 4.13 Blood lead level of the followed up workers (n=72) by protective measures in use**

Protective measure	No.	%	Mean $\pm$ SD( $\mu$ g/dl)			X <sup>2</sup>	P-value
			>10	10-19.9	<20		
<b>Gloves</b>							
Yes	27	37.5	22	3	2	14.45	0.001
No	45	62.5	16	21	8		
<b>Glasses</b>							
Yes	3	4.2	2	0	1	2.010	0.366
No	69	95.8	36	24	9		
<b>Hat</b>							
Yes	1	1.4	0	1	0	2028	0.363
No	71	98.6	38	23	10		
<b>Respiratory mask</b>							
Yes	10	13.9	10	0	0	10.39	0.006
No	62	86.1	28	24	10		
<b>Work shoes</b>							
Yes	1	1.4	1	0	0	0.907	0.635
No	71	98.6	37	24	10		
<b>Overall</b>							
Yes	12	16.7	5	5	2	0.717	0.699
No	60	83.3	33	19	8		
<b>Not Smoking</b>							
Yes	38	52.8	24	11	3	4.189	0.123
No	34	47.2	14	13	7		
<b>Not Drinking</b>							
Yes	15	20.8	11	3	1	3.239	0.198
No	57	79.2	27	21	9		
<b>Not Eating</b>							
Yes	15	20.8	11	3	1	3.239	0.198
No	57	79.2	27	21	9		
<b>Not Chewing gum</b>							
Yes	64	88.9	34	21	9	0.073	0.964
No	8	11.1	4	3	1		
<b>Drinking milk frequently</b>							
Yes	36	50.0	26	7	3	10.93	0.004
No	36	50.0	12	17	7		
<b>Have water bath</b>							
Yes	9	12.5	4	4	1	0.573	0.751
No	63	87.5	34	20	9		

*P>0.05: non significant, P<0.05: significant*

#### 4.2.9 Blood lead level of the followed up workers by their Knowledge regarding routes of lead entry into body, lead health effect or lead as an environmental pollutant

Table (4.14) showed no significant differences (P-value >0.05) in BLL by knowledge of workers regarding routes of lead entry to human body, lead adverse health effects or lead as an environmental pollutant.

**Table 4.14 Blood lead level of the followed up workers (n= 72) by knowledge of lead entry into human body, lead health effects or lead as an environmental pollutant.**

Items	No.	%	Mean $\pm$ SD ( $\mu\text{g}/\text{dl}$ )			$\chi^2$	P-value
			>10	10-19.9	<20		
<b>Route of lead entry into body</b>							
<b>Inhalation</b>							
Yes	62	86.1	31	23	8	2.862	0.239
No	10	13.9	7	1	2		
<b>Through skin</b>							
Yes	21	29.2	11	7	3	0.004	0.998
No	51	70.8	27	17	7		
<b>Through mouth</b>							
Yes	18	75.0	7	7	4	2.299	0.317
No	54	25.0	31	17	6		
<b>Effect of lead exposure on health</b>							
Yes	57	79.2	27	21	9	3.239	0.198
No	15	20.8	11	3	1		
<b>Lead as an environmental pollutant</b>							
Yes	59	81.9	31	20	8	0.060	0.970
No	13	18.1	7	4	2		

*P>0.05: non significant.*

#### 4.2.10 Blood lead level of the followed up workers by self reported symptoms

The relationship between BLL and self reported symptoms is demonstrated in Table (4.15). In general, the mean BLL was found to be higher among workers who had self reported symptoms than those who had not report such symptoms. However, the differences of the mean BLL among workers who had reported irritability, headache, concentration difficulties, sleep disturbance

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and hypertension and those who had not report such symptoms were statistically significant ( $X^2 = 7.814$ , P-value=0.020;  $X^2 = -6.531$ , P-value= 0.038;  $X^2 = 7.702$  , P-value= 0.021,  $X^2 = -16.25$ , P-value=0.001; and  $X^2 = 7.820$ , P-value=0.020, respectively).

Table 4.15 Distribution of mean BLL by self-reported symptoms among the followed up workers (n=72)

Self- reported symptom*	No.	%	Mean± SD (µg/dl)			X <sup>2</sup>	P-value
			>10	10-19.9	<20		
<b>Fatigue</b>							
Yes	53	73.6	24	21	8	4.731	0.094
No	19	26.4	14	3	2		
<b>Irritability</b>							
Yes	50	69.4	21	21	8	7.814	0.020
No	22	30.6	17	3	2		
<b>Coma</b>							
Yes	4	5.6	3	1	0	1.073	0.585
No	68	94.4	35	23	10		
<b>Convulsion</b>							
Yes	3	4.3	1	2	0	1.703	0.427
No	69	95.7	37	22	10		
<b>Headache</b>							
Yes	54	75.0	24	22	8	6.531	0.038
No	18	25.0	14	2	2		
<b>Concentration difficulties</b>							
Yes	42	58.3	17	16	9	7.702	0.021
No	30	41.7	21	8	1		
<b>Sleep disturbance</b>							
Yes	35	48.6	10	17	8	16.25	0.001
No	37	51.4	28	7	2		
<b>Seizures</b>							
Yes	1	1.4	0	1	0	2.028	0.363
No	71	98.6	38	23	10		
<b>Hearing loss</b>							
Yes	17	23.6	8	7	2	0.621	0.733
No	55	76.4	30	17	8		
<b>Wrist/foot drop</b>							
Yes	28	38.9	15	7	6	2.835	0.242
No	44	61.1	23	17	4		
<b>Loss of libido</b>							
Yes	27	37.5	13	8	6	2.531	0.285
No	45	62.5	25	16	4		
<b>Nausea</b>							
Yes	37	51.4	23	8	6	4.699	0.095
No	35	48.6	15	16	4		
<b>Dyspepsia</b>							
Yes	29	40.3	14	9	6	1.880	0.391
No	43	59.7	24	15	4		
<b>Constipation</b>							
Yes	30	41.7	13	12	5	1.841	0.398
No	42	58.3	25	12	5		
<b>Abdominal pain</b>							
Yes	25	34.7	12	8	5	1.216	0.544
No	47	65.3	26	16	5		
<b>Lead line in gingival tissue</b>							
Yes	11	18.0	6	2	3	2.576	0.276
No	61	82.0	32	22	7		
<b>Renal pain</b>							
Yes	11	18.0	4	5	2	1.407	0.495
No	61	82.0	34	19	8		
<b>Hypertension</b>							
Yes	46	63.9	20	16	10	7.820	0.020
No	26	36.1	18	8	0		
<b>Miscarriage (f)/infertility</b>							
Yes	0	0	0	0	0	NA	NA
No	72	100	38	24	10		

*P*>0.05: non significant. NA=not available.

### 4.3 Biochemical and hematological Alterations of the followed up workers (n=72) versus controls (n=70)

To assess the effect of lead on some biochemical parameters a total of 70 individuals were selected as controls from the general population who almost had no history of being exposed to lead and compare them with the followed up gasoline station workers.

#### 4.3.1 Blood Lead Level

Figure (4.6) showed that the mean BLL of gasoline station workers ( $11.43 \pm 5.96 \mu\text{g/dl}$ ) was significantly higher than that of controls ( $5.33 \pm 0.99$ ), (t-test = 8.449, P-value = 0.001).

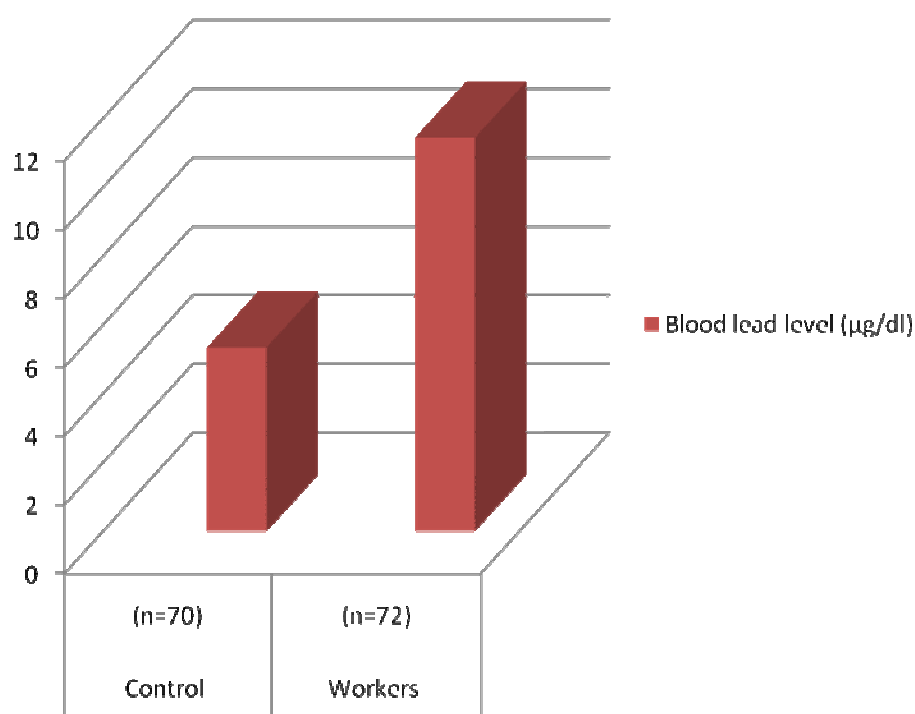


Figure 4.6. BLL of controls and gasoline station workers

BLL was expressed as mean  $\pm$  SD,  $P < 0.05$ : significant

### 4.3.2 Hematological analysis

Complete blood count of controls and gasoline station workers is demonstrated in Figure (4.7). There were no significant differences in the means of white blood cell counts and hematocrit values between controls and workers (t-test = 0.088, P-value =0.930 and t-test =0.803, P-value=0.423, respectively). The means of red blood cell counts and platelets were significantly decreased in workers ( $4.94\pm 0.40$  and  $249.4\pm 58.23$ , respectively) compared to controls ( $5.10\pm 0.26$  and  $281.5\pm 56.49$ , respectively), (t-test = -2.783, P-value=0.006 and t-test = -3.330, P-value=0.001, respectively). In contrast, hemoglobin, MCV, MCH and MCHC were found to be higher in workers ( $15.40\pm 1.48$ ,  $86.73\pm 5.08$ ,  $30.52\pm 3.14$ ,  $35.17\pm 2.69$ , respectively) than controls ( $14.54\pm 0.80$ ,  $84.26\pm 3.68$ ,  $28.55\pm 1.57$ ,  $33.63\pm 1.15$ , respectively). This change was statistically significant (t-test = 4.303, P-value=0.001, t-test = 3.319, P-value=0.001, t-test = 4.701, P-value=0.001 and t-test = 4.423, P-value=0.001, respectively)

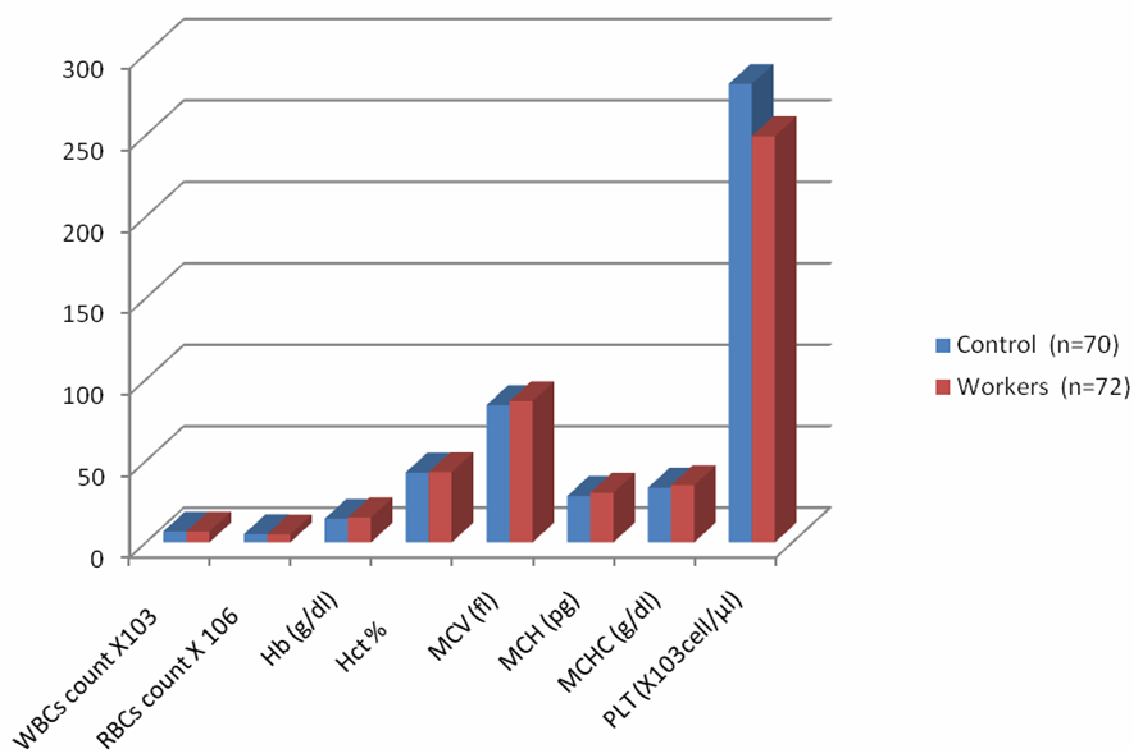


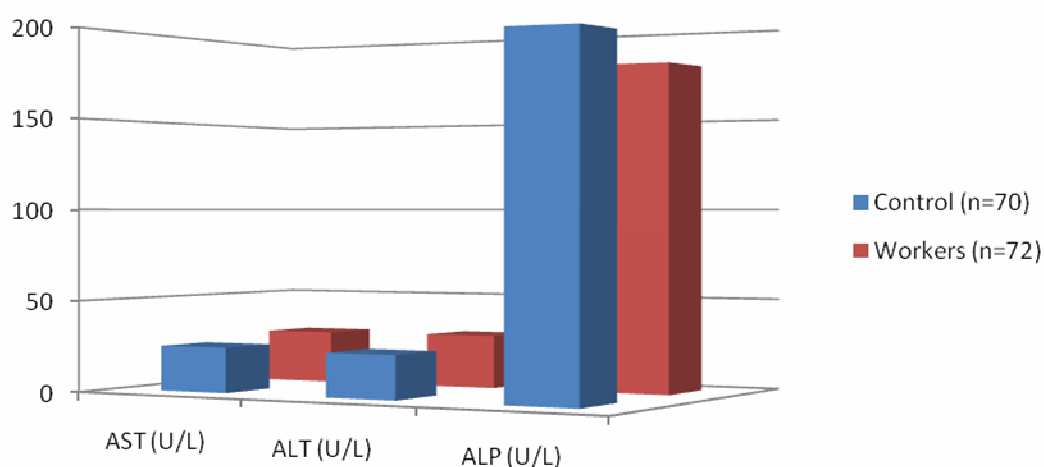
Figure 4.7 . Complete blood count of controls and gasoline station workers

WBCs: white blood cells, RBCs: red blood cells, Hb: hemoglobin, Hct: hematocrit, MCV: mean corpuscular volume, MCH: mean corpuscular hemoglobin, MCHC: mean corpuscular hemoglobin concentration, PLT: platelets count.

All values were expressed as mean  $\pm$  SD,  $P > 0.05$ : non significant,  $P < 0.05$ : significant

### 4.3.3 Liver enzymes activity

Figure (4.8) shows the activities of some liver enzymes in controls and gasoline station workers. The mean levels of serum AST and ALT of workers ( $28.63 \pm 7.87$  and  $29.13 \pm 11.46$ , respectively) were higher than that of controls ( $24.71 \pm 3.60$  and  $23.89 \pm 9.74$ , respectively). These changes were statistically significant (t-test = 3.789, P-value= 0.001 and t-test =2.933, P-value=0.004, respectively). In contrast, the mean level of serum ALP of workers ( $179.3 \pm 42.62$ ) was lower than that of controls ( $193.6 \pm 31.79$ ). Such deference was statistically significant (t-test = -2.267, P-value= 0.025).

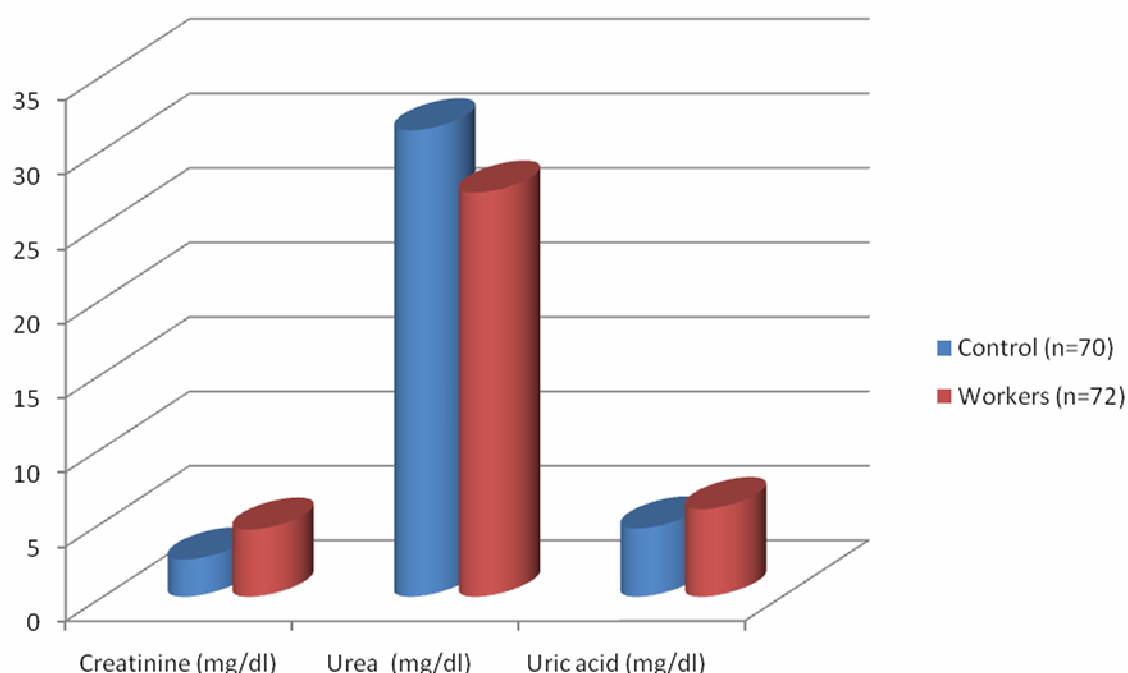


**Figure 4.8.** liver enzymes activities of controls and gasoline station workers

*AST: aspartat aminotransaminase, ALT: alanin transaminase, ALP: alkaline phosphatase.  
All values were expressed as mean ± SD, P<0.05: significant*

#### 4.3.4 Kidney function

The mean levels of non-protein nitrogen constituents (indicator of kidney function) of controls and gasoline station workers were presented in Figure (4.9). Creatinine and uric acid levels were increased in workers compared to controls ( $0.89 \pm 0.17$  and  $5.88 \pm 1.20$  versus  $0.74 \pm 0.11$  and  $5.58 \pm 0.74$ ). Such increase was statistically significant only for creatinine (t-test = 5.962, P-value=0.001). In contrast, the mean level of urea was decreased in workers compared to controls ( $27.13 \pm 11.61$  versus  $31.31 \pm 6.32$ ). Such decrease was statistically significant (t-test = -2.660, P-value=0.009).



**Figure 4.9. non-protein nitrogen constituents in controls and gasoline station workers**

*All values were expressed as mean ± SD, P>0.05: non significant, P<0.05: significant*

#### 4.3.5 Blood pressure

Figure (4.10) illustrates blood pressure among controls and gasoline station workers. Systolic and diastolic blood pressures were increased in workers compared to controls ( $125.7 \pm 13.22$  and  $84.60 \pm 9.93$  versus  $120.4 \pm 3.76$  and



81.94 ± 2.67). These differences were statistically significant (t-test = 3.325; P-value=0.002 and t-test =2.162; P-value=0.032).

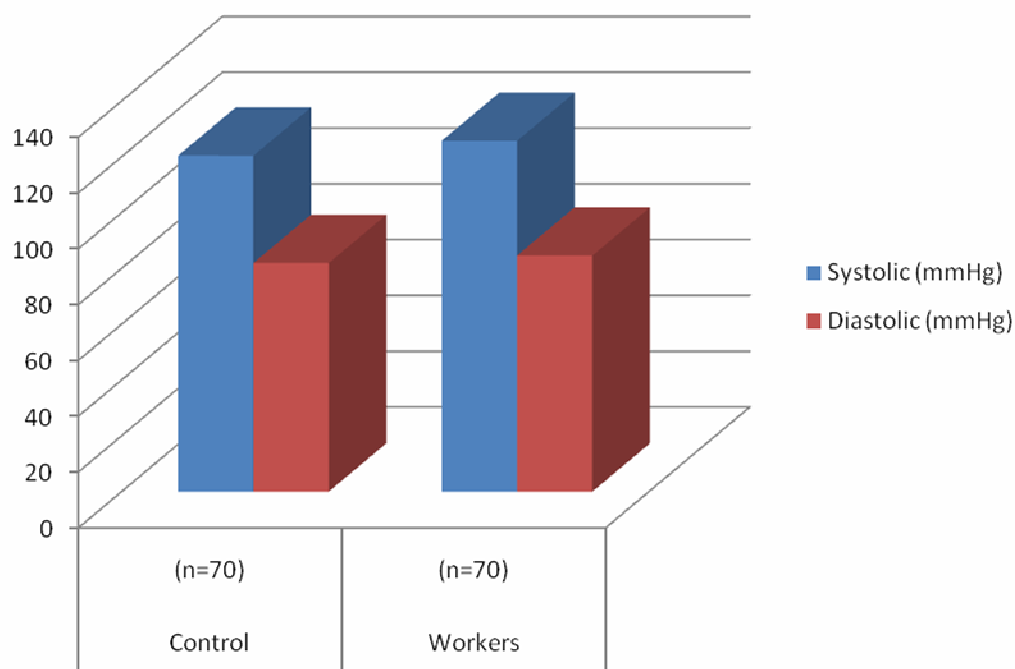


Figure 4.10. Blood pressure among controls and gasoline station workers

All values were expressed as mean ± SD,  $P > 0.05$ : non significant,  $P < 0.05$ : significant

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Occupational lead exposure is a major public health concern and a primary environmental health problem issue. This study is the first one in the Gaza strip to assess awareness of gasoline station workers towards lead exposure to determine their BLL and to identify risk factors associated with lead exposure.

## **5.1 Questionnaire Data**

### **5.1.1 Distribution of gasoline station workers by the governorates of the Gaza strip**

The number of workers in each Governorate was selected according to the number of gasoline stations and the number of employed workers in these stations. The number of studied workers was less than the estimated number. Some gasoline station owners did not allow the researcher to interview the workers because they believe that this research may be considered as alert to workers and may lead to future station inspections. In addition, the political situation contributed to closure of some gasoline stations and leaving their workers. However, the study sample was more or less representative in each Governorate of the Gaza strip.

### **5.1.2 General Characteristic of the study population**

All the study population was male workers. This reflects the Palestinian community tradition, which considered gasoline station work is culturally underpinned for woman. Most of the gasoline station workers engaged in such work were young. This implying fewer alternatives due to employment crises in Gaza strip. Data showed that most of married workers have children. The children could be in a potential risk of lead exposure if workers bring contaminated dust on their skin, clothes and shoes where lead is most harmful to children. Workers can prevent such secondary exposure by showering and changing their clothes before returning home (5, 91). The relationship between worker's BLL and their children and other family members was not investigated in the current study, so it is recommended to conduct such research in this regard. Concerning the

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educational status, the findings that more than half of workers had secondary school and diploma or university certificates and none of workers were illiterate reflect an educated community.

### **5.1.3 Work duration**

More than half of workers were found to be worked in gasoline station for more than 5 years. The working hours ranged from 8 to 12 hours/day with an estimated average of 67 hours/week. The weekly work hours were determined by Palestinian labor law to be 45 hours/week (92). The higher weekly hours among gasoline station workers may be attributed to that such work meet the community needs during the day hours. Also the difficult socioeconomic situation with lack of law enforcement leads to such long working hours. In addition, all interviewed workers were found not to have history of previous job related to sources of lead pollution which may imply that most of lead exposure coming from the work place.

### **5.1.4 Previous testing for BLL and willing to provide blood sample**

None of the gasoline station workers were found to be previously tested for blood lead. This does not match with either international or locally rules for workers health protection. In work places where lead is used, employers must provide an industrial hygiene program and medical surveillance including the monitoring BLL (93). Occupational lead surveillance program have been developed in Taiwan since the mid of 1980s (94). In Singapore, legislation on periodic medical examinations for exposed worker in factories was introduced in 1985 (59). In the United Armenia, the program to use blood lead determinations to investigate lead exposure initiated in 1991 (95). Although Palestinian Ministry of Labor in coordination with Palestinian Ministry of Health suggests formulating rules for health and occupational safety, blood lead monitoring is new and not adequately yet conceptualized by governing bodies. Therefore, frequent blood lead measurement among gasoline station workers is recommended. As shown in this study, the majority of workers were willing to provide blood sample for lead

analysis. This willingness may be stemmed from workers cooperation with the researcher and their curiosity to know about their health.

### **5.1.5 Protective measures in use**

Although the majority of gasoline station workers knew about the adverse health effects of lead exposure on human health, the of protective measures was poor. This means that workers knowledge seems not to reflect their practices. As mentioned by workers, the reasons standing behind such poor practices were carelessness, not providing the protective gear, discomfortable of wearing the gear and believe that the gear is not necessary. It was reported that personal habits at the work place appear to play a major role in facilitating exposure to lead among lead smelters, automobile mechanics and gasoline retailers in Ghana (60). In addition, the appropriate selection and use of personal protective equipments can help prevent or limit exposure to lead hazards (96). It was recommended that appropriate protective work clothing and equipment includes coveralls, gloves, hats, shoes and mask respiratory should be provided to all workers by the employer (97).

### **5.1.6 Knowledge of workers regarding routes of lead entry into body, lead health effect and lead as an environment pollutant**

Results showed that gasoline station workers had knowledge on the routes of lead entry into human body with inhalation were the main one. Dermal absorption of lead compounds was reported to be much less significant than inhalation route (71). In addition, the majority of workers had good knowledge on effect of lead exposure on human health and Lead as environmental pollutant. Without such knowledge, workers can develop serious health problems (98). The literature showed that there is an urgent need for coalition between policy maker, industry, workers, unions, health care providers, and the community to take action to reduce environmental lead pollution (99, 100).

### **5.1.7 Prevalence of self reported symptoms related to lead exposure**

The most common self reported symptoms of lead exposure recalled by gasoline station workers in 6 months preceding the present interview were headache, fatigue, irritability, concentration difficulties, sleep disturbance, hypertension, nausea and constipation. These findings required urgent intervention from MOH and other Non-Governmental organizations (NGOs). The major symptoms of intoxication with leaded gasoline in USA are referable to the CNS. The victims suffer from Insomnia, nightmares, anorexia, nausea & vomiting, diarrhea, headache, muscular weakness and emotional instability. (38) In Libyan Arab Jamahiriya, it was reported that, signs and symptoms of lead poisoning may include malaise, anorexia, abdominal pain, vomiting, lethargy, colic, constipation, irritability and apathy (41). It was found that the most frequent symptoms among lead workers in Korea were generalized weakness and fatigue and the next were tingling and numbness of arm or leg, feeling irritation at the slightest disturbance, and weakness of wrist or ankle joint. (39). In Croatian gasoline station workers who had exposed to leaded gasoline for more than five years, the symptoms of depression and decreased reaction time and motor abilities were identified (40).

### **5.1.8 Attending training courses and health professional's visiting to gasoline station**

Neither workers attended training courses nor they had health professional visited their station. This is an alarming issue to different governor and non governor bodies that necessitates urgent campaign represented by introducing training courses and frequent health professionals visiting to the gasoline stations. Such action would alleviate lead exposure and poisoning among workers. It was reported that workers should receive training courses including instruction about the use and care of appropriate protective equipment and on the manner of wearing them (101, 102).

## **5.2 BLL analysis for the followed up workers**

### **5.2.1 Response rate**

The total response for giving blood samples among the study population was 68.6 %. This was relatively higher than that recorded in a similar study conducted in Beirut (47) where the response rate was 54.4 %. This indicates that workers in Gaza Strip feel the danger of their work and they are keen to know about their health. In addition, workers considered their participation as a benefit to them to get free blood lead test.

### **5.2.2 Distribution of BLL among the workers**

Blood lead level is the most widely used measure and powerful indicator for occupationally lead exposure. Several authors used BLL as a direct indicator for lead exposure in gasoline station workers as well as an indication of potential for adverse effect on health (46, 47). Our findings showed that the mean BLL of gasoline station workers was 11.43 $\mu$ g/dl. The distribution of workers according to their BLL were 38 (52.8%) workers had BLL <10 $\mu$ g/dl with mean of (6.79 $\pm$ 1.50), 24 (33.3%) workers had BLL 10-19.9  $\mu$ g/dl with mean (14.12 $\pm$ 2.54) and 10 (13.9%) workers had BLL 20-30 $\mu$ g/dl with mean (22.58 $\pm$ 2.42). Our findings were higher than that reported in Athens, Denmark and in Ghana where mean BLL of gasoline station workers were 5.6 $\mu$ g/dl, 3.5 and 8.6 $\mu$ g/dl, respectively. (46, 54, 60). The higher blood lead level recorded in Gaza Strip workers may be attributed to lack of regulation, awareness, and health monitoring and promotion and surveillance programs which implemented in other countries. On the other hand, in Greater Beirut the mean BLL of gasoline station workers was found to be 18.4 $\mu$ g/dl (47), which may be attributed to frequent sniffing of cars in such traffic busy city

### **5.2.3 BLL of workers by age and educational status**

The result revealed non-significant relation between BLL and age of gasoline station workers. The highest BLL was found among workers at aged >51

years old. It was reported that the mean BLL concentration increased with age (46). Concerning educational status, results showed that BLL of workers decreased with increasing education level. This mean that educated workers were aware of risk of lead exposure. Our results are in agreement with other studies (103, 104) who found that education level was inversely related to BLL among lead workers in Swiss and Taiwan.

#### **5.2.4 BLL of workers by work duration**

The mean BLL of workers was found to be increased with increasing the work duration. This positive relationship was found to be statistically significant. This means that increasing work duration led to increase workers exposure to lead and put their health at risk. Similar results were reported in workers exposed to lead in occupational settings including gasoline station (47), battery and auto-radiator workshops (16, 55, 104, 105, 106).

#### **5.2.5 BLL of the followed up workers by protective measures in use**

The mean BLL was generally found to be lower among gasoline station workers who used and /or followed protective measures than among those who did not. However, the differences of mean BLL among workers who used gloves, respiratory mask or drank milk frequently and those who did not were statistically significant. Personal protective equipment was consider as an essential component in any occupational health and safety program and as a mean of preventing occupational lead absorption (95, 96). The finding that gloves and respiratory mask protect against lead exposure confirmed that inhalation and skin are important routs of lead entry into human body. Increasing of BLL was found in occupational lead workers who neglect to use face masks (105). In addition, it was reported that milk drinking by workers who occupationally exposed to lead reduced BLL (16, 106). The degree of lead absorption is increased in person, whose diet is deficient in calcium, since calcium competes with lead for intestinal absorption (63, 107, 108). Thus, the milk consumption is recommended as a dietary supplement for lead exposed workers in Gaza Governorates to minimize

lead absorption. Positive association was found between BLL and smoking, eating or chewing gum at work place (47, 56, and 104). Also, the greatest reduction in exposure to lead was achieved by banning smoking and eating at work place (109). The association between BLL and smoking, eating, drinking and chewing gum may be attributed to the ingestion of lead (contamination from hands during work) or increased absorption of inhaled lead (110, 111).

### **5.2.6 BLL of workers by their Knowledge regarding routes of lead entry into body, lead health effect or lead as an environmental pollutant**

There was no association between BLL and knowledge of workers regarding routes of lead entry to human body, lead as an environment pollutant or lead adverse health effects. This indicates that knowledge on lead hazards alone may be not enough to significantly decrease BLL among workers. In agreement of other studies that lead awareness knowledge doesn't necessarily guarantee positive behavior change (112). Knowledge of lead hazards together with good hygiene and education of correct work practice may be the preferential way to reduce lead exposure (113).

### **5.2.7 BLL of workers by self reported symptoms**

In general, the mean BLL was found to be higher among workers who had self reported symptoms than those who had not reported such symptoms. However, The differences of mean BLL among workers who had reported irritability, headache, sleep disturbance, concentration difficulties, hypertension and those who had not report such symptoms were found to be statistically significant. Such result necessitates health professional visiting to those workers. Data reported in many countries including the neighboring ones showed that workers with high BLL have a higher prevalence of most of the symptoms of lead toxicity than did workers with lower BLL (5, 38, 39, 42, 105, and 114).



### **5.3 Biochemical & Hematological analysis of the followed up workers versus controls**

The present study showed that BLL of the followed up gasoline station workers were significantly higher than controls. Lead is known to have toxic effects on several biologic systems in particular nervous system, kidney, liver, hematological cardiovascular and reproductive systems (11, 115).

#### **5.3.1 Hematological analysis**

The study revealed that red blood cells count was significantly decreased whereas hemoglobin level was unexpectedly increased in workers compared to controls. However, increase in Hb level was not significant. Decrease in RBCs count in lead exposed workers was reported by other studies (72, 116). However, the discrepancy in such result may be related to other factors rather than to lead ability at our low level (11.4 µg/dl) to initiate peroxidative damages to the RBC membranes (45, 117). In addition, no significant difference was reported in hemoglobin concentration among controls and gasoline station employees with BLL 5.6µg/dL (54). Elevation in BLL causes decreasing in CBC parameter (40, 60, 64, and 72). Environmental protection agency estimated that the threshold BLL for a decrease in hemoglobin is 50µg/dL for occupationally exposed adult's workers (71).

Our finding revealed that platelets counts were decreased. Pb<sup>2+</sup> stimulated [<sup>3</sup>H]-glutamate binding in human platelets. Hg<sup>2+</sup>, Cd<sup>2+</sup> and Pb<sup>2+</sup> increased lipid peroxidation levels and reactive oxygen species measurement in platelets (118).

#### **5.3.2 Liver enzymes activity**

Data presented here showed that serum AST and ALT were significantly higher in workers than controls while ALP was significantly decreased in workers than controls. This result is in agreement with the other findings (54, 64, 65).

Transaminase enzymes are synthesized mainly in the liver and their levels in serum are low in normal subjects. Lead may accumulate in liver and exert its toxic effect via peroxidative damage to hepatic cell membrane causing transaminases to liberate into the serum (119). Impaired secretion of hepatic ALP may be also due to lead exposure. However, It was reported that the gasoline station workers suffered from liver disorders: lipoid degeneration of liver, chronic functional damages of liver and liver cirrhosis (40).

### 5.3.3 Kidney function

This study showed that creatinine and uric acid levels were generally increased in workers compared to controls. In contrast, urea was significantly decreased in workers than controls. The association of low-level lead occupation exposure with impaired renal function was determined by other studies (68). This change would be prominent in acute high-dose exposures (69). Serum urea was reported to be decreased in gasoline station employees compared to controls (54). This decrease may be referred to impairment of protein metabolism by lead exposure as the urea is the end product of protein catabolism. Uric acid is the end product of the catabolism of tissue nucleic acid, i.e. purine and pyrimidine bases metabolism. The observed increase in uric acid concentration may be due to degeneration of purines and pyrimidines or to an increase of uric acid levels by either overproduction of the liver or inability of excretion (120). It was shown that occupational exposure of humans subjects to lead in petrol increases concentrations of uric acid in exposed subjects compared to unexposed subjects (64). Creatinine is the last variable nitrogenous constituent of the blood; it is more readily excreted by the kidneys than urea and uric acid. The elevation of serum creatinine concentration observed in our study is not high to the extent to reflect end stage renal disease. It is known that about 50% of kidney function must be lost before a significant rise in the serum concentration of creatinine can be detected (121).

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#### **5.3.4 Blood pressure**

Data presented here showed that there is a significant increase in workers systolic and diastolic blood pressures compared to controls. It was reported that lead may elevate blood pressure in susceptible adults in United States population at BLL as low as 14 $\mu$ g/dL (76). Moreover, Bener and his colleagues confirmed this hypothesis by finding a positive association between lead exposure and high blood pressure among lead exposed workers in United Arab Emirates. Long-term cumulative lead exposure can significantly increase blood pressure in moderately lead-exposed male workers (42). In addition, several studies support an association between lead exposure and elevations in blood pressure (78, 79).

## 6.1 Conclusion

Data were collected by questionnaire interview from 105 gasoline station workers (study population) in Gaza strip. Out of the 105 workers, 72 gave blood samples (followed up workers) for blood lead level and biochemical analysis.

### A. Study population (n=105)

1. The mean age of the study population was  $34.4 \pm 10.7$  years. Most of them were married and educated. More than half of the workers work more than 5 years in the gasoline station. None of them were previously tested for blood lead.
2. Although the majority of worker know that lead entry into human body by inhalation, through skin, through mouth, have an environmental pollutant and has adverse health impact, small number of them use the protective measures. Carelessness and not provided are the most common reason of not use protective measures.
3. The most common self reported symptoms among workers were headache, fatigue, irritability and concentration difficulties. Sleep disturbance, hypertension, nausea, constipation and dyspepsia.
4. Neither worker attended training courses nor had health professionals visited them.

### B. Follow up workers (n=72).

1. The mean BLL of the followed up workers was  $11.43 \mu\text{g}/\text{dl}$ . Lower BLL was found in highly educated workers compared to less educated ones. Mean BLL of workers increased with increasing work duration.
2. The mean BLL was found to be lower among gasoline station workers who used protective measures than among those who did not. However, the differences of mean BLL among workers who used gloves, respiratory mask or drank milk frequently and those who did not were statistically significant.

respiratory mask or drank milk frequently and those who did not were statistically significant.

3. The mean BLL was found to be higher among workers who had self reported symptoms than those who had not report such symptoms. The differences of mean BLL among workers who had reported irritability, headache, sleep disturbance, concentration difficulties, hypertension and those who had not report such symptoms were found to be statistically significant.

4. The mean BLL of gasoline station workers was significantly higher than that of controls.

5. The means of red blood cell counts and platelets were significantly decreased in workers compared to controls. In contrast, hemoglobin, MCV, MCH and MCHC were found to be significantly higher in workers than controls.

6. Serum AST and ALT were significantly higher in workers than controls while ALP was significantly decreasing in workers than controls.

7. Creatinine and uric acid levels were generally increased in workers compared to controls. In contrast, urea was significantly decreased in workers than controls.

8. There is a significant increasing in workers systolic and diastolic blood pressures compared to controls.

## **6.2 Recommendations**

1. Appropriate protective work clothing and equipment includes gloves, hat, shoes, and masks or respirators should be providing to all workers.

2. The gasoline station owners should provide hygiene facilities in their stations such as; change area, showers, and eating facilities. Also, workers invited to give up smoking and eating at work sites.

3. Workers should receive training courses and awareness programs that include information about the potential adverse health effects of lead exposure, early recognition of lead intoxication, the importance of personal hygiene practices in

reducing lead exposure, instruction about the use of care of appropriate personal protective equipment and information about specific work practices for safely with gasoline.

4. The concerned ministries with labor unions have to form a special committee consisting of legal and technical members in medical, occupational and environmental health professionals in order to develop and enforce strict regulations, instructions, guidelines of occupational health and safety to protect workers. These measures should include workplace air lead level monitoring, blood lead screening, and use of safer technologies, in addition to implement pre-employment and periodic medical examination for workers.

5. MOH is recommended to provide their laboratories with BLL test.

6. Lead poisoning health care and appropriate treatment should be included as integral part of any form of occupational health services to be provided in Gaza Strip.

7. Further research is recommended to include other occupations that are susceptible to cause lead exposure to build up a comprehensive picture of lead occupational lead exposure in Gaza Governorates.

8. Further studies about workers BLL and their children BLL also are recommended.

9. Unleaded gasoline should be imported.

1. CDC-Center for Disease Control and Prevention. (2002). Adult blood lead epidemiology and surveillance, United States, 1998-2001. *MMWR Morb Mortal Wkly Rep*; 51(11); 1-10.
2. OLPPP-occupational lead poisoning prevention program. (2002). Blood lead level in California workers, 1995-1999. California Department of health Services, USA
3. Flegal A.R., Smith D.R. (1992). Blood lead concentrations in pre-industrial humans. *N Engl J Med* 326:1293-1294.
4. Flegal A.R., Smith D.R. (1995). Measurement of environmental lead contamination and human exposure. *Rev Environ Contam Toxicol* 143:1-45.
5. ATSDR-Agency for Toxic Substance and Disease Registry. (2000). Case studies in environmental medicine: lead toxicity. U.S. Department of Health and Human Services.
6. Gorey R.A. (1994). Toxic effects of metal. Lead. The basic science of poisons. New York Pergamon Press/Maxwell Macmillan Publishing Corporation. 4<sup>th</sup> edition; 639-646.
7. Sanin L.H., Gonzalez T., Romieu I., Hernandez M. (1998). Acumulacion de plomo en hueso y sus efectos en la salud. *Salud Publica Mex*; 40: 359-368.
8. Aguilar G., Piacitelli G.M., Juarez C.A., Vazquez J.H., Hu H., Hernandez M. (1999). Exposicion ocupacional a plomo inorganico en una imprenta de ciudad de Mexico. *Salud Piblica Mex*; 41:42-54.
9. Rodriguez A.I., Rocha J.B., Pereira M.E., and Souza D.O. (1996). Delta aminolevulinic acid dehydrates activity in weanling and adult rats exposed to lead acetate. *Bull. Environ. Contam. Toxicol*; 57:47-53.
10. Kosnett, M.J. (1994). Lead poisoning and drug over dose. 2<sup>ad</sup> ed. Norwalk, CT. Appleton and Lange, pp. 196-200.
11. Stollery, B. T. (1996). Reaction time changes in workers exposed to lead. *Neurotox and Teratol*; 18 (4): 477-483.
12. Alexander B.H., Checkoway H., Van Netten C., Muller C.H., Ewers T.G., Kayfman J.D., Mueller B.A., Vaughan T.L., Faustman E.M. (1996). Serum quality of men employed at lead smelter. *J Occup Environ Med*; 53: 411-416.
13. Howson P.C., Hernandez-Avila M., Rall D.P. (1995). Lead in the Americas. A call for action. Institute of medicine, USA.
14. Ministry of Transportation, 2005

15. Safi J.M., Abu-Hashish M., Soliman H., Safi N.M., El-Nahal Y., El-Madhoun S. (2003). Childhood lead poisoning prevention in Gaza Strip. Submitted for publication.
16. El-Madhoun F.I. (2003). Occupational lead exposure in battery and auto-radiatr workers in Gaza Governorates. Thesis, School of public health, AL-Quds University.
17. ATSDR-Agency for Toxic Substance and Disease Registry. (1999). Toxicological profile for lead. Atlanta, GA. U.S. Department of Health and Human Services, Public Health Services.
18. Pirkle J.L., Kaufmann R.B., Brody D.J., Hichman T., Gunter E.W., Paschal D.C. (1998). Exposure to the U.S. population to lead. 1991-1994. Environ Health perspect; 106: 745-750.
19. Landrigan P.J. (1994). Lead in: Rosen stock L, Cullen MR, eds.. Textbook of occupational and Enviromental medicine. Philadelphia: Saunders: 745-54.
20. CDC- Center for Disease Control and Prevention. (1991a). control of excessive lead exposure in radiator workers. MMWR Morb Mortal Wkly Rep 1; 40 (8): 139-41.
21. Fischbein A. (1994). Occupational and environmental lead exposure. In: ROM WN. Environmental and environmental medicine. Philadelphia: Saunders: 745-54.
22. Markowitz M. (2000). Lead Poisoning. *Pediatr Rev*, 21: 327–35.
23. Papanikolaou N.C., Hatzidaki E.G., Belivanis S., et al. (2005). Lead toxicity update. A brief review. *Med Sci Monit*, 11: RA 329-RA336
24. Saryan L.A., Zenz C. (1994). Lead and its compounds. In: Zenz C, Dickerson OB, Horvath EP Jr, and eds. Occupational medicine. St, Louis: Mosby, 3d ed: 506-41
25. Ziegler E.E., Edwards B.B., Jensen R.L., et al. (1978). Absorption and retention of lead by infants. *Pediatr Res*, 12: 29-34
26. Bogden J.D., Gertner S.B., Christakos S., et al., (1992). Dietary calcium modifies concentrations of lead and other metals and renal calbindin in rats. *J Nutr*, 122: 1351-1360.
27. Mahaffey K.R., Gartside P.S., Glueck C.J. (1986). Blood lead levels and dietary calcium intake in 1- to 11-year old children: the Second National



- Health and Nutrition Examination Survey, 1976 to 1980. *Pediatrics*, 78:257-262
28. Roberts J.R., Reigart J.R., Ebeling M., Hulsey T.C. (2001). Time required for blood lead levels to decline in nonchelated children. *J Toxicol Clin Toxicol*, 39: 153–60
29. Guidelines for drinking water quality. (1996). Health criteria and other supporting information. Geneva, World Health Organization, 2nd ed, (2): 254–75
30. Kosnett M.J., Becker C.E., Osterloh J.D., et al., (1994). Factors influencing bone lead concentration in a suburban community assessed by noninvasive K X-ray fluorescence. *JAMA*, 271: 197-203
31. Keogh J.R., Sullivan J.B., Frieger G.R. (1992). Hazardous materials toxicology: clinical principles of environmental health. Baltimore: Williams & Wilkins: 837-844.
32. Fleming D.E., Boulay D., Richard N.S., et al., (1997). Accumulated body burden and endogenous release of lead in employees of a lead smelter. *Environ Health Perspect*, 105:224-233
33. Rabinowitz M.B., Wetherill G.W., Kopple J.D. (1976). Kinetic analysis of lead metabolism in healthy humans. *J Clin Invest*, 58:260-270
34. Bolanowska W. (1968). Distribution and excretion of triethyllead in rats. *Br J Ind Med*, 25:203-208
35. Kimmel E.C., Fish R.H., Casida J.E. (1977). Bioorganotin chemistry. Metabolism of organotin compounds in microsomal monooxygenase systems and in mammals. *J Agric Food Chem*, 25:1-9
36. Hohnadel D.C., Sunderman F.W., Nechay M.W., McNeely M.D. (1973). Atomic absorption spectrometry of nickel, copper, zinc, and lead in sweat collected from healthy subjects during sauna bathing. *Clin Chem*, 19:1288-1292.
37. Omokhodion F.O., Crookford G.W. (1991). Lead in sweat and its relationship to salivary and urinary levels in normal healthy subjects. *Sci Total Environ*, 103:113-122
38. Hardman J.G., Limbird L.E., Molinoff P.B., Ruddon R.W., Goodman A.G. (1996). Goodman and Gilman's the pharmacological basis of therapeutics. New York, NY: McGraw-Hill, 9<sup>th</sup> ed: 1654

39. Lee B.K., Ahn K.D., Lee S.S., Lee G.S., Kim Y.B., Schwartz B.S. (2000). A comparison of different lead biomarkers in their relation with lead-related symptoms. *Int Arch Occup Environ Health*, 73: 298-304.
40. Pranjić N, Mujagić H, Pavlović S. (2003). Inhalation of gasoline and damage to health in workers at gas stations. Article in Croatian. *Med Arh*, 57(1):17-20.
41. Essa K.A. (1999). Lead, the ugly trace element: occurrence, effects, screening and treatment. *Eastern Mediterranean Health Journal*, 5 (4): 798-802.
42. Bener A, Almehdi Am, Alwash R, Al-Neamy FR. (2001). A pilot survey of BLL in various types of workers in the united of Emirates. *Environ Int*, 27(4): 311-314.
43. Staudinger KC, Roth VS. (1998). Occupational lead poisoning. *American Family Physician*, 57 (4): 719-26, 731-732.
44. Lewis R. (1990). Metals. In: La Dou J, ed. *Occupational medicine*. Norwalk, Conn: Appleton and Lange, 306-310.
45. Patrick L. (2006). Lead Toxicity, a Review of the Literature. Part I: Exposure, Evaluation, and Treatment *Alternative Medicine Review u Volume 11, Number 1*.
46. Nielsen JB, Grandjean P, Jørgensen PJ. (1998). Blood lead concentration in the Danish population after introduction of lead-free gasoline. Article in Danish. *Aug 10,160(33):4768-71*
47. Nuwayhid I., McPhaul K., Bu-Khuzam R., Duh S.H., Christenson R.H., Keogh J.P. (2001). Determinants of elevated blood lead levels among working men in Greater Beirut
48. IPCS-International program on chemical safety. (1995). *Inorganic lead*. Geneva, Switzerland: World Health Organization, Environmental Health Criteria, vol 165.
49. Sakai T., Morita Y. (1996). Delta-Aminolevulinic acid in plasma or whole blood as a sensitive indicator of lead effects, and its relation to the other heme-related parameters. *Int Arch Occup Environ Health*, 68(2):126-32.
50. Barauskiene D., Naginiene R., Kregzdyte R., Ryselis S., Abdrakhmanovas O. (2004). Application of Delta-aminolevulinic acid dehydratase test for the assessment of occupational long-term lead exposure. *Trace elements and electrolytes*, 21; (4): 232-235.

51. NCCLS-National committee for clinical laboratory standard. (1996). Procedures for the collection of diagnostic blood specimens by venipuncture-Third edition. Villanova, PA. NCCLS document H3-A3.
52. Froom P., Kristal-Boneh E., Benbassat J., Ashkanazi R., Ribak J. (1996). Zinc protoporphyrin. *INT J. OCC. Health*, (1):181-186.
53. Hu H., Aro A., Payton M., Korrick S., Sparrow D., Weiss S.T., Rotnitzky A. (1996). The relationship of bone and blood lead to hypertension. *JAMA*, 275 (15): 1171-1176.
54. Kapaki E.N., Varelas P.N., Syrigou A.I., Spanaki M.V., Andreadou E., Kakami A.E., Papageorgiou C.T. (1998). Blood lead levels of traffic- and gasoline-exposed professionals in the city of Athens. *Arch Environ Health*. Jul-Aug, 53(4):287-91.
55. Mortada W.I., Sobh M.A., El-Defrawy M.M., Farahat S.E. (2001). Study of lead exposure from automobile exhaust as a risk of nephro-toxicity among traffic policeman. *Am J Nephrol*, 21 (4):274-9.
56. Richter E., Fischbein A. (1992). Lead poisoning: II. Biological standards for occupational lead exposure-where do we stand now. *Isr J Med Sci*, 28:572-577.
57. Gittleman J.L., Engelgau M.M., Shaw J., Wille K.K., Seligman P.J. (1994). Lead poisoning among battery reclamation workers in Alabama. *J Occup Med.*, San Mateo, CA: Appleton and Lange.
58. Chan J., Sim M., Golec R., Forbes A. (2000). Predicators of lead absorption in children of lead workers. *Occup Med*, 50(6):398-405.
59. Phoon W.H., Lee H.S., Ho C.K. (1990). Biological monitoring of workers exposed to inorganic lead in Singapore. *Singapore Med J*, 31(2):127-130.
60. Ankrah N.A., Kamiya Y., Appiah-Opong R., Akyeampon Y.A., Addae M.M. (1996). Lead levels and related biochemical findings occurring in Ghanaian subjects occupationally exposed to lead. *East Afr Med J*, 73(6):375-9.
61. Brockhaus A., Freier I., Ewer U., Jermann E., Dolgner R. (1983). Levels of cadmium and lead in blood in relation to smoking, sex, occupation and other factors in adult population of the FRG. *Int Arch Occup Environ Health*, 52(2):167-175.

62. Schumacher M., Domingo J.L., Libet J.M., Corbella J. (1993). Variability of blood lead levels in an urban population in relation to drinking and smoking habits. *Sci Total Environ*, 138(1-3):23-29.
63. Kristal-Boreh E., Froom P., Yerushalmi N., Ashkanazi R., Pardo A., Shine R., Ribak J. (1998). Effect of dietary calcium on blood lead concentrations in occupationally exposed and nonexposed workers. *Am J Ind Med*, 34(5):512-516.
64. Dioka C.E., Orisakwe O.E., Adeniyi F.A., Meludu S.C. (2004). Liver and renal function tests in artisans occupationally exposed to lead in mechanic village in Nnewi, Nigeria. : *Int J Environ Res Public Health*, 1(1):21-5.
65. Mikhail T.H., El-Sawaf H.A., Ibrahim K.M., Awadallah R., El-Dessoukey E.A. (1980). Evaluation of the effect of lead exposure on the liver in Egyptian lead tank welders. *Z Ernährungswiss*, 19(1):50-6.
66. Staessen J.A., Lauwerys R.R., Buchet J.P., Bulpitt C.J., Rondia D., Vanrenterghem Y., Amery A. (1992). Impairment of renal function with increasing blood lead concentrations in the general population. The Cadmibel Study Group. *N Engl J Med*, 16; 327(3):151-6.
67. Payton M., Hu H., Sparrow D., Weiss S.T., (1994). Low level lead exposure and renal function in the normative aging study. *Am J Epi*, 140(19): 821-829.
68. Kim R., Rotnitsky A., Sparrow D., Weiss S., Wager C., Hu H. (1996). A longitudinal study of low-level lead exposure and impairment of renal function. The Normative Aging Study. *JAMA*, 17; 275(15):1177-81.
69. JUNG K.Y., LEE S.J., KIM J.Y., HONG Y.S, KIM S.R., KIM D., SONG J.B. (1998). Renal dysfunction indicators in lead exposed workers. *J Occup Health*, 40: 103-109.
70. Lim Y.C., Chia K.S., Ong H.Y., Ng V., Chew Y.L. (2001). Renal dysfunction in workers exposed to inorganic lead. *Ann Acad Med Singapore*, 30(2):112-7.
71. EPA-Environmental Protection Agency. (1986a). Air quality criteria for lead. Research Triangle Park, office of health and environmental assessment, Environmental Criteria and assessment office. EPA 600/8-83-028F.
72. Masci O., Carelli G., Vinci F., and Castellino N. (1998). Blood lead concentration and biological effects in workers exposed to very low lead levels. *J Occup Environ Med*, 40(10):886-94.

73. Baranowska-Bosiacka I, Hłyńczak AJ, Machaliński B. (2000). The impact of lead ions on metabolism of erythrocytes. *Med Pr*, 51(1):59-65.
74. Paglia D.E., Valentine W.N., Fink K. (1977). Lead poisoning. Further observations on erythrocyte pyrimidine nucleotidase deficiency and intracellular accumulation of pyrimidine nucleotides. *J Clin Invest*, 60:1362-1366.
75. ATSDR-Agency for Toxic Substances and Disease Registry (2005). Toxicological profile for lead. (Draft for Public Comment). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service:63
76. Harlan W.R. (1988). The relationship of blood lead levels to blood pressure in the U.S. population. *Environ Health Perspect*, 78:9-13.
77. Victory W., Throler H.A., Volpe R., et al., (1988). Summary of discussion sessions: symposium on lead blood pressure relationships. *Environ Health Perspect*, 78:139-55.
78. Schwartz J. (1995). Lead, blood pressure, and cardiovascular disease in men. *Arch Environ Health*, 50(1):31-7.
79. Hu H., Aro A., Payton M., Korrick S., Sparrow D., Weiss S.T., Rotnitzky A. (1996). The relationship of bone and blood lead to hypertension. *JAMA*, 275(15):1171-1176.
80. Apostoli P. (2005). Trend in lead exposure in the workplace and the environment. *Ann Ist Super Sanita*: 34 (1).
81. Staessen J.A., Bulpitt C.J., Fagard R., Lauwerys R.R., Roels H., Thijs L., Amery A. (1994). Hypertension caused by low-level lead exposure: myth or fact. *J Cardiovasc Risk*, 1(1):87-97.
82. Backstrom C., Hursh-Cesar G. (1981). Survey research. London, New York: Macmillan Publishing Company, Collier Macmillan Publishers, (2): 53–81
83. Miller D.T., Paschal D.C., Gunter E.W., Stroud P.E., D' Angelo J. (1987). Determinations of blood lead with electrothermal atomic absorption using a L'vov platform and matrix modifier. *Analyst*, 55:712A-24A.
84. Gella F.J., Olivella T., Cruz Pastor M., Arenas J., Moreno R., Durban R. and Gomez J.A. (1985). A simple procedure for routine determination of Aspartate aminotransferase and Alanine aminotransferase with pyridoxal phosphate. *Clin Chem Acta*, 153: 241-247.

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85. Rosalki S.B., Foo A.Y., Burlina A., et al., (1993). Multicenter evaluation of iso ALP test kit for measurement of bone alkaline phosphatase activity in serum and plasma. *Clin Chem*, 39:648-652
  86. Burtis C.A., Ashwood E.R., Saunders W.B. (1994). *Tietz Textbook of clinical chemistry*, 2<sup>nd</sup> edition.
  87. Fossati P., Prencipe L., Berti G. (1980). Use of 3, 5-dichloro-2-hydroxybenzenesulfonic acid/4aminophenazone chromogenic system in direct enzymic assay of uric acid in serum and urine. *Clin Chem*, 26:227-231.
  88. Fabiny D.L., Ertingshausen G. (1971). Automated reaction-rate method for determination of serum creatinine with Cantrifi Chem. *Clin Chem*, 17:696-700
  89. ABBOTT laboratories. (2001). CELL-DYN 1700 system. Abbott Park, IL 60064, ABBOTT LABS, IL/USA.
  90. Munro J.F. and Campbell I. W. (2000). *Macleod's clinical examination*. 10<sup>th</sup> edition: 86.
  91. Roscoe R.J., Gittleman J.L., Deddens J.A., Petersen M.R., Halperin W.E., (1999). Blood lead levels among the children of lead exposed workers: ameta-analysis. *Am J Ind Med*, 36:475-481.
  92. Palestinian Labor Law. (2001). Labor law no. 7 year 2000. *Dewan El-Fatwa Wa-tashrei. El-Waqaei El-Falastenia*; 39:7-52.
  93. OSHA-Occupational Safety and Health Administration. (1998). Safety and health regulations for construction. Occupational health and environmental control. Lead. U.S. department of labor. Code of Federal Regulations, 29 CFR 1926.62.
  94. CDC- CDC-Center for Disease Control and Prevention. (1996). Occupational lead surveillance-Taiwan, July-December 1993. *MMWR Morb Mortal Wkly Rep*, 44(10); 181, 187-189.
  95. CDC- CDC-Center for Disease Control and Prevention. (1993). Occupational blood lead survey-Armenia, 1991 and 1993. *MMWR Morb Mortal Wkly Rep*, 45(4):85-88.
  96. Blayney M.B. (2001). The need for empirically derived permeation data for personal protective equipment: the death of Dr. Karen E. Wetherhahn. *Appl Occup Environ Hyg*, 16(2):233-236.

97. OLPPP-occupational lead poisoning prevention program. (2000). Cal/Osha General industry safety orders, lead section 5198 (forming section 5216), USA.
98. Kenneth D., Rosenman, Amy S., Sims, Douglas J., Kalinowski. (2003). Annual report on blood lead levels in Michigan, 2001. Michigan State University, Michigan department of consumer and industry services, Bureau of safety and regulation; and Michigan department of community health. Michigan, USA.
99. Romieu I., Lacasana M., McConnell R. (1997). Lead exposure in Latin America and Caribbean. Lead research group of the Pan-American health organization. *Environ Health Perspect*, 105(4):398-405.
100. Wu Y., Zhou X., Hu G., Wang Z., Li H., Bao R., Yan H., Lic, Wu L., He F. (2002). Study on the effects of lead from small industry of battery recycling on environment and children's health. *Zhonghua Liu Xing Bing Xue Za Zhi*, 23(3):520-529.
101. NIOSH-National Institute of Occupational Safety and Health. (1992). Preventing lead poisoning in construction workers NIOSH ALERT; NO. 9-116a.
102. Mayer A. and Korhonen E. (1999). Assessment of the protection efficiency and comfort of personal protective equipment in real condition of use. *Int J Occup saf ergon*, 5(3):347-360.
103. Berode M., Wietlisbach V, Rickenbach M., and Guillemin M.P. (1991). Lifestyle and environmental factors as determinants of blood lead levels in Swiss population. *Environ Res*, 55(1):1-17.
104. Chuang H.Y., Lee M.L., Chao K.Y., Wang J.D., Hu H. (1999). Relationship of blood lead levels to personal hygiene habits in lead battery workers: Taiwan, 1991-1997. *Am J Ind Med*, 35(6):595-603.
105. Mehdi J.K., Al-Imarah F.J.M., and Al-Suhail A.A. (2000). Levels of some trace metals and related enzymes in workers at storage-battery factories in Iraq. *Eastern Mediterranean Health J*, 6(1):66-82.
106. Wang V.S., Lee M.T., Chiou J.Y., Guu C.F., Wu T.N., Lai J.S. (2002). Relationship between blood lead levels and renal function in lead battery workers. *Int Arch Occup Environ Health*, 75(8):569-579.
107. Sargent J.D. (1994). The role of nutrition in the prevention of lead poisoning in children. *Pediatr Ann*, 23:637-642.

108. Odland J.O., Nieboer E., Romanova N., Thoassen Y., Lund E. (1999). Blood lead and cadmium and birth weight among sub-arctic and arctic populations of Norway and Russia. *Acta Obstet Gynecol Scand*, 78:852-860.
109. Hsiao C.Y., Wu H.D.I, Lai J.S., Kuo H.W. (2001). A longitudinal study of the effects of long-term exposure to lead among lead battery factory workers in Taiwan (1989-1999). *The Science of the Total Environment*, 279:151-159.
110. Grandjean P. (1993). International perspectives of lead exposure and lead toxicity. *Neurotoxicity*, 14(2-3):9-14.
111. Zaki A., El-shazly M., Abedel Fattah M., El-said K., and Curtale. (1998). Leads toxicity among working children and adolescent in Alexandria, Egypt. *Eastern Mediterranean*, 4(3):520-529.
112. Serwint J.R., Dias M., and White J. (2000). Effects of lead counseling for children with lead level  $\geq 20\mu\text{g}/\text{dl}$ : impact on parental knowledge, attitude and behavior. *Clinical Pediatrics*, 39(11):643-650.
113. Lai J.S., Wu T.N., Liou S.H., Shen C.Y., Guu C.F., Ko K.N., Chang P.Y. (1997). A study of the relationship between ambient lead and blood lead among battery workers; *Int Arch Occup Environ Health*, 9(4):295-300.
114. Awad El-karim M.A., Hamad A.S., Elhaimi Y.A., Osman Y. (1986). Effects of exposure to lead among lead based-acid battery factory workers in Sudan. *Arc Environ Health*, 41(4):261-265.
115. Ghorbe F., Boujelbene M., Makni-Ayadi F., Guermazi F., Kammoun A., Murat J., Croute F., Soleihavoup J.P>, And El-Feki A. (2001). Effect of chronic lead exposure on kidney function in male and female rats, determination of lead exposure biomarker. *Arch Physiol. Biochem*, 109(5):457-63.
116. Solliway B.M., Schaffer A., Pratt H., Yannai S. (1996). Effects of exposure to lead on selected biochemical and haematological variables. *Pharmacol Toxicol*, 78(1): 18-22.
117. Gurer-Orhan H., Handan U., Sabir and Hilal Özgüne. (2003). Toxicology Correlation between clinical indicators of lead poisoning and oxidative stress parameters in controls and lead-exposed workers. National Institute of Occupational Safety and Health, Etimesgut, Ankara, Turkey. *Toxico*, 195 :( 2-3). 15: 147-154.



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118. Borges V. C., Santos F. W., Rocha J. B. T., and Nogueira C. W. (2007). Heavy Metals Modulate Glutamatergic System in Human Platelets. *Neurochemical Research*, 32(6):953-958.
  119. Sivaprasad R., Nagaraj M., and Voralakshmi P. (2003). Combined efficacies of lipoic acid and meso-2,3-dimercaptosuminic acid on lead-induced erythrocyte membrane lipid peroxidation and antioxidant status in rats. *Hum Exp Toxicol*, 22(4):183-92.
  120. Wolf P.L., Williams D. Tsudaka T., and Acosta L. (1972). *Methods and Techniques in clinical chemistry*, Wiley-Inter-science a division of John Wiley and Sons. New York, London, Sydney, Toronto.
  121. Kaptan A. and Szabo L.L. (1983). *Clinical chemistry interpretation and techniques*. Second edition.

# Appendices

## Some Biochemical & Hematological Alterations Associated with Lead Exposure in Gasoline Station Workers in Gaza Strip

Serial No              
Date:    /    / 2005

1. Name of worker.....
2. Address:.....  
work.....  
Home.....  
Tel:.....

3. Age (Years):

4. Marital status:

- (1) Married    (2) Single    (3) Widowed    (4) Divorced.

5.1. In case of marriage do you have children?

- (1) Yes                      (0) No

5. Education:

- (1) Illiterate (2) Primary (3) Preparatory (4) Sec. School (5) Dip. University

6. How long have you worked in the station? (Years of work)

7. What was your previous job(s) and for how long?

Job title	From	To

8. Is your house/apartment near any of the following works?

8.1 Smelter (1) Yes (0) No

Distance from house  | | | |  meters.

8.2 Lead battery Workshop (1) Yes (0) No

Distance from house  | | | |  meters.

8.3 Auto-Radiator Workshop (1) Yes (0) No

Distance from house  | | | |  meters.

8.4 Garage workshop (1) Yes (0) No

Distance from house  | | | |  meters.

8.5 Gas or petrol station (1) Yes (0) No

Distance from house  | | | |  meters.

8.6 Others (specify.....) (1) Yes (0) No

Distance from house  | | | |  meters.

9. Have you ever tested your blood lead level?

(1) Yes (0) No

10. If blood lead test is available by this study, are you willing to participate and give blood sample for analysis?

(1) Yes (0) No

10.1. If yes BLL.....

11. Which of the following do you wear at your work place?

11.1 Gloves (1) Yes (0) No

11.2 Hat (1) Yes (0) No

11.3 Respirator/Mask (1) Yes (0) No

11.4 Special shoes (1) Yes (0) No

11.5 Overall (1) Yes (0) No

11.6 Other, specify..... (1) Yes (0) No

12. If you have not worn any of the equipment listed above what is the reason?

- (1) Not provided (2) Discomfortable (3) Not necessary
- (4) Carelessness (5) other, specify.....

13. During work are you doing the following?

- 13.1. Smoking (1) Yes (0) No
- 13.2. Drinking (1) Yes (0) No
- 13.3. Eating (1) Yes (0) No
- 13.4. Chewing gum (1) Yes (0) No

14. Do you drink milk frequently?

- (1) Yes (0) No

15. Do you take a shower at work site before going home?

- (1) Yes (0) No

16. According to your knowledge, by which of the following pathway do you think lead enters into the human body?

- 16.1 Inhalation (1) Yes (0) No
- 16.2 Skin (1) Yes (0) No
- 16.3 Mouth. (1) Yes (0) No

17. According to your knowledge, does lead consider as an environmental pollutant?

- (1) Yes (0) No

18. Do you know that, exposure to lead has an adverse health effect?

- (1) Yes (0) No

19. Are you suffering any of the following symptoms/effects in the previous 6 months?

- |                                    |         |        |
|------------------------------------|---------|--------|
| 19.1 Fatigue                       | (1) Yes | (0) No |
| 19.2 Irritability                  | (1) Yes | (0) No |
| 19.3 Coma                          | (1) Yes | (0) No |
| 19.4 Convulsion                    | (1) Yes | (0) No |
| 19.5 Headaches                     | (1) Yes | (0) No |
| 19.6 Concentration difficulties    | (1) Yes | (0) No |
| 19.7 Sleep disturbance             | (1) Yes | (0) No |
| 19.8 Seizures                      | (1) Yes | (0) No |
| 19.9 Hearing loss                  | (1) Yes | (0) No |
| 19.10 Wrist/foot drop              | (1) Yes | (0) No |
| 19.11 Loss of libido               | (1) Yes | (0) No |
| 19.12 Nausea                       | (1) Yes | (0) No |
| 19.13 Dyspepsia                    | (1) Yes | (0) No |
| 19.14 Constipation                 | (1) Yes | (0) No |
| 19.15 Abdominal pain               | (1) Yes | (0) No |
| 19.16 Lead line in gingival tissue | (1) Yes | (0) No |
| 19.17 Renal pain                   | (1) Yes | (0) No |
| 19.18 Hypertension                 | (1) Yes | (0) No |
| 19.19 Miscarriage (f)/infertility  | (1) Yes | (0) No |

20. Have you been attend any training course(s) in health hazards of lead exposure?

(1) Yes (0) No

20.1 If yes, please specify the institution(s) or any other sector, which conducted the course(s)?

.....

21. Have you seen or hear about lead poisoning or death cases?

(1) Yes (0) No

22. Does any health professional come to visit your workplace periodically?

(1) Yes                      (0) No

22.1 If yes, please, specify the health professional sector, who conducted the visit.....

Thank you very much for your cooperation.

**Researcher**

**Abed Al Rahman I. Hamad**

## بعض التغيرات البيوكيميائية والدموية المرتبطة بالتعرض للرصاص لدى عمال محطات البنزين في قطاع غزة

الرقم المسلسل: \_\_\_\_\_

التاريخ: ٢٠٠٥ / /

١. اسم العامل (أختياري): .....
٢. العنوان (أختياري): محافظة مدينة/قرية/مخيم شارع هاتف #  
 (عنوان العمل): .....
- (عنوان السكن): .....

٣. العمر بالسنوات: .....

٤. الحالة الاجتماعية: (١) متزوج (٢) أعزب (٣) أرمل (٤) مطلق  
 إذا كانت الإجابة متزوج فهل لديك أولاد وما عددهم؟

٥. مستوى التعليم:  
 (١) غير متعلم (٢) ابتدائي (٣) إعدادي (٤) ثانوي (٥) دبلوم أو جامعي

٦. ما هي المدة الزمنية التي عملت بها في محطات الوقود؟

٧. ما هي الأعمال السابقة الأخرى التي قمت بها وما هي مدة العمل؟

نوع العمل	من	الى

٨. هل يقع بيتك/شقتك بالقرب من الأعمال التالية؟  
 ٨,١ مصهر للرصاص (١) نعم (٠) لا

يبعد عن السكن .....متر

٨,٢ ورشة بطاريات رصاصية (١) نعم (٠) لا

يبعد عن السكن .....متر

٨,٣ ورشة رديتر (١) نعم (٠) لا

يبعد عن السكن .....متر

٨,٤ ورشة (كراج تصليح سيارات) (١) نعم (٠) لا

يبعد عن السكن .....متر

٨,٥ محطة وقود (١) نعم (٠) لا

يبعد عن السكن .....متر

٨,٦ أخرى (حدد.....) (١) نعم (٠) لا

يبعد عن السكن .....متر

٩. هل سبق لك وقمت بفحص تركيز الرصاص بالدم؟

(٠) لا

(١) نعم

٩,١ اذا كان الجواب نعم فكم كانت نسبة BLL .....

١٠. إذا كان فحص الرصاص في الدم متاحا من خلال هذه الدراسة، فهل ترغب بالمشاركة و إعطاء عينة

دم للفحص؟ (١) نعم (٠) لا

١١. أي من الادوات التالية تستخدم اثناء العمل؟

١١,١ كفات (١) نعم (٠) لا

١١,٢ نظارات واقية (١) نعم (٠) لا

١١,٣ قبعة (١) نعم (٠) لا

١١,٤ كامات (١) نعم (٠) لا

١١,٥ حذاء للعمل (١) نعم (٠) لا

١١,٦ اللباس الواقي (١) نعم (٠) لا

١١,٧ أخرى (حدد.....) (١) نعم (٠) لا

١٢. إذا لم تقم باستعمال أي من الادوات المذكورة اعلاه فما هو السبب؟

(١) غير متوفرة (٢) غير مريحة (٣) غير ضرورية (٤) اللامبالاة (٥) أخرى (حدد.....)

١٣. هل تقوم باحدى هذه الاشياء اثناء العمل؟

١٣,١ التدخين (١) نعم (٠) لا

١٣,٢ الشرب (١) نعم (٠) لا

١٣,٣ الاكل (١) نعم (٠) لا



- ١٣,٤ مضغ قطعة اللين (١) نعم (٠) لا
١٤. هل تشرب الحليب بانتظام (١) نعم (٠) لا
١٥. هل تقوم بالاستحمام في مكان العمل قبل ذهابك الى بيتك؟ (١) نعم (٠) لا
١٦. حسب معرفتك بأي من الطرق التالية يمكن للرصاص أن يدخل جسم الانسان؟
- ١٦,١ التنفس (١) نعم (٠) لا
- ١٦,٢ الجلد (١) نعم (٠) لا
- ١٦,٣ الفم (١) نعم (٠) لا
١٧. حسب معرفتك هل يعتبر الرصاص ملوثا للبيئة؟ (١) نعم (٠) لا
١٨. حسب معرفتك ، هل التعرض للرصاص له تأثيرات صحية ضارة؟ (١) نعم (٠) لا
١٩. هل تعاني أو عانيت من أي من الاعراض / التأثيرات الصحية التالية خلال ال ٦ اشهر الماضية؟
- ١٩,١ إجهاد (١) نعم (٠) لا
- ١٩,٢ إثارة عصبية (١) نعم (٠) لا
- ١٩,٣ غيبوبة (١) نعم (٠) لا
- ١٩,٤ اضطرابات تشنجية (١) نعم (٠) لا
- ١٩,٥ صداع (١) نعم (٠) لا
- ١٩,٦ صعوبات في التركيز (١) نعم (٠) لا
- ١٩,٧ اضطرابات في النوم (١) نعم (٠) لا
- ١٩,٨ نوبة مرضية (١) نعم (٠) لا
- ١٩,٩ ضعف في السمع (١) نعم (٠) لا
- ١٩,١٠ ارتخاء في الاطراف (١) نعم (٠) لا
- ١٩,١١ ضعف الشهوة الجنسية (١) نعم (٠) لا
- ١٩,١٢ دوران او غثيان (١) نعم (٠) لا
- ١٩,١٣ عسر هضم (١) نعم (٠) لا

- ١٩,١٤ إمساك (١) نعم (٠) لا
- ١٩,١٥ الام في البطن (١) نعم (٠) لا
- ١٩,١٦ خطر رصاص في انسجة اللثة (١) نعم (٠) لا
- ١٩,١٧ الام في الكلية (١) نعم (٠) لا
- ١٩,١٨ ارتفاع ضغط الدم (١) نعم (٠) لا
- ١٩,١٩ عدم القدرة على الانجاب (١) نعم (٠) لا

٢٠. هل تلقيت اي دورات تدريبية عن المخاطر الصحية للتعرض للرصاص؟  
(١) نعم (٠) لا

٢٠,١ اذا كانت الاجابة نعم، يرجى تحديد المؤسسة أو الجهة التي قامت بعقد هذه الدورات؟  
.....

٢١. هل سبق ان رايت أو سمعت عن حالات تسمم بالرصاص أو حالات وفاة نتيجة التسمم بالرصاص؟  
(١) نعم (٠) لا

٢٢. هل هناك زيارات توعية من قبل مثقفين صحيين لمكان عملك بشكل دوري؟  
(١) نعم (٠) لا

٢٢,١ إذا كانت الاجابة نعم، من فضلك اذكر المؤسسة التي يتبعون لها؟  
.....

في الختام لا يسعني الا ان اشكر لكم حسن تعاونكم معنا ونرجو من الله أن يسلمكم من كل اذى

الباحث  
عبد الرحمن ابراهيم حمد