

1.1 Introduction

Gasoline is a very volatile liquid widely used as fuels for automobiles and other internal combustion engines. It evaporates rapidly, releasing vapors containing several organic and inorganic constituents into the immediate environment. These vapours, being ubiquitous in the environment, constitute various components of petroleum pollutants in the air, which are of great environmental and human health concern. Exposure to these pollutants by a good proportion of the human population, either intentionally or inadvertently, is very common in the course of their day to day indoor and outdoor activities. Generally, those occupationally exposed tend to constitute the population at greater risk of frequent exposure (carballo et al., 1994). The potential health hazards associated with chronic or sub-chronic exposure to these ubiquitous pollutants in the environment has attracted the attention of the general public and scientific community in particular. It has been reported in animal experiments that exposure to gasoline vapors produced various toxicity effects in many tissues. In our earlier studies, it was reported that exposure to gasoline vapors induced proatherogenic changes in serum lipid profile and signs of hepatic oxidative stress, hematotoxicity, reproductive toxicity and nephrotoxicity, in male and female rats (Rabble et al., 1996).

the liver is largest glandular organ in the body and performs many vital functions to keep the body pure of toxins and harmful substances. Its considered a gland—an organ that secretes chemicals—because it produces bile, a substance needed to digest fats. Bile's salts break up fat into smaller pieces so it can be absorbed more easily in the small intestine (Maton et al., 1993). The applications of kerosene as cooking and lighting fuels in the home have resulted in direct exposure of these products to a good percentage of the populace. Moreover, the

day-to-day use of petrol outside the industrial settings is likely to have the same effect on the users as kerosene since they have been reported to contain most of the same hydrocarbons. However, the most affected are those who occupationally exposed to the fumes (Fischbein, 1998). Despite the high utilization of kerosene and petrol, our knowledge is sparse on the toxicological effects of inhaling the composite fumes evaporating from kerosene and petrol. However, mutagenic, genotoxic, carcinogenic, neurotoxic, immunotoxic and haemotoxic effects of some petroleum and petrochemical products' constituents have been reported in experimental studies on humans and animals(Wilbourn ,1986) . Hydrocarbons and other constituents of petroleum and petrochemical products, like other xenobiotics, are metabolized in the liver to a greater extent reported that exposure of rats to motorcycle exhaust and organic extracts of the exhaust particulate caused a dose- and time-dependent increase in cytochrome P-450-dependent monooxygenases and glutathione-S-transferase in the liver, kidney and lung microsomes(Raza,1995). Since kerosene and petrol contain some of these constituents, chronic or frequent exposure to their fumes may affect the normal liver functions,The expression of toxicity of xenobiotics is usually determined biochemically by the monitoring of some plasma enzymes and lipids. A rise in AST, ALT, ALP, TG and cholesterol are commonly measured as indices of the damage of the liver cells (Maton et al., 1993).

1.2 literature review

1.2.1 Gasoline

Gasoline is the generic term for petroleum fuel complex mixture of aliphatic and aromatic hydrocarbons. It is commonly used as fuel for internal combustion engines and is also used as a thinner, decorative agent, and industrial solvent. Some of its constituents are known to be highly toxic or carcinogenic to humans (Mehlman, 1990). It is complex, volatile, and highly flammable and contains over 500 saturated or unsaturated hydrocarbons having from 3 to 12 carbon atoms. About 110 million people are exposed to gasoline constituents in the course of refueling at gasoline stations (Wixtrom, 1992)

1.2.1.1. Stability of gasoline

Quality gasoline should be stable for six months if stored properly but gasoline will break down slowly over time due to the separation of the components. The addition of a fuel stabilizer to gasoline can extend the life of fuel that is not or cannot be stored properly though removal of all fuel from a fuel system is the only real solution to the problem of long term storage of an engine or a machine or vehicle. Some typical fuel stabilizers are proprietary mixtures containing mineral spirits, isopropyl alcohol, 1,2,4-trimethylbenzene, or other additives. Fuel stabilizer is commonly used for small engines, such as lawnmower and tractor engines, especially when their use is seasonal (low to no use for one or more seasons of the year). Users have been advised to keep gasoline containers more than half full and properly capped to reduce air exposure, to avoid storage at high temperatures, to run an engine for ten minutes to circulate the stabilizer through all components prior to storage, and to run the engine at intervals to purge stale fuel from the carburetor (Werner et al., 2007).

1.2.1.5 Additives of gasoline

Leaded gasoline contains a range of organolead compounds, especially tetraethyl lead, are difficult to eliminate from the CNS, and the injuries induced usually result in permanent neurological deficits that cause medical as well as socio-economical problems (Chang, 1990).

Ethylene glycol is another gasoline constituent and a general fuel additive widely used in automobiles for its ability to absorb water and to prevent overheating or freezing. Ingestion of ethylene glycol produces not only central nervous system depression, but also cardiopulmonary complications, acute renal failure, and delayed neurological sequelae (Lewis, 1997).

Methyl tertiary-butyl ether (MTBE) was the most commonly used oxygenate in the 1990s, but its use was dramatically reduced in the subsequent decade. Inhalation is the most common route of exposure to workers and the general public. Worker exposure may occur during manufacture and transport of MTBE-containing fuels. Inhalation of vapors while refueling automobiles is the predominant route of exposure for the general public (HEI, 2001; Ahmed, 2001). with concentrations measured near the car during approximately 3 min of refueling with gasoline containing MTBE ranging from 0.1 to 38 ppm (0.36 to 138 mg/m³; HEI, 2001). Concentrations of hydrocarbon vapor encountered during refueling have also been measured. In one study, total hydrocarbon vapor from unleaded gasoline measured during 2 min of self-service fill-up was 10–100 ppm (Clayton Environmental Consultants, 1993). In an earlier study, conducted during fueling test cars with unleaded gasoline, no vapor exposure was detected during up to 22 min of refueling (4 cars fueled with a total of 17 gallons of gasoline). A 27-min exposure (6 cars fueled with 25 gallons of fuel) resulted in 4–8 ppm hydrocarbon exposure 12.7–24.3 mg/m³ (Halder et al., 1986).

1.2.1.6.1 Toxicity of gasoline

Many of the toxicological effects associated with the exposure to gasoline can be attributed to specific components of gasoline, such as benzene, toluene, ethylene and xylene, which are also known as volatile organic compounds (VOCs) [Kirchstetter, 1999]. A number of studies have demonstrated that the occurrences of various health problems are closely associated with occupational exposures to VOCs (Rinsky, 2002). exposure to benzene from petrol vapour caused haematotoxicity among petrol station workers. This study also found significantly diminished pulmonary function that was associated with duration of exposure to gasoline vapour(Uzma, 2008) . Furthermore, other studies have also suggested that there is a causal relationship between industrial exposures to benzene and the incidence of some types of leukaemia and aplastic anaemia(Cronkite, 1989). A number of experimental animal models have been used to determine the toxicological effects of gasoline inhalation (Huff, 1989).Recently, there has been increasing concern for environmental safety and health hazards related to gasoline exposure, and as a result, several strategies have been put in place to remove the potentially toxic compounds from the gasoline mixture. For example, the usage of leaded gasoline was substituted by non-leaded fuel. Likewise, some of the potentially noxious components were also eliminated from the petrol blend, and oxygenates were added to enhance engine performance (Schuetzle, 1994). These strategies have resulted in the production of reformulated gasoline blends without harmful heavy metal additives and with relatively lesser amounts of noxious constituents such as benzene (Costantini, 1993).The Euro II standard recommends that the research octane number (RON), which is a measure of performance of a fuel, should be 97, and the standard recommends a maximum sulphur content of

500 ppm and a maximum volume of benzene of 5% (Tan, 2007).

1.2.2.The liver

The liver is a vital organ of vertebrates and some other animals. In the human it is located in the upper right quadrant of the abdomen, below the diaphragm. The liver has a wide range of functions, including detoxification of various metabolites, protein synthesis, and the production of bio chemicals necessary for digestion. There is currently no way to compensate for the absence of liver function in the long term, although liver dialysis techniques can be used in the short term (Maton et al., 1993).

The liver is a gland and plays a major role in metabolism with numerous functions in the human body, including regulation of glycogen storage, decomposition of red blood cells, plasma protein synthesis, hormone production, and detoxification. It is an accessory digestive gland and produces bile, an alkaline compound which aids in digestion via the emulsification of lipids. The liver's highly specialized tissue consisting of mostly hepatocytes regulates a wide variety of high-volume biochemical reactions, including the synthesis and breakdown of small and complex molecules, many of which are necessary for normal vital functions (Maton et al., 1993). Estimates regarding the organ's total number of functions vary, but textbooks generally cite it being around 500 (Zakim et al., 2002).

The liver is unique in the sense that it is a relatively resilient organ that can regenerate cells that have been destroyed by some short term injury or disease. However, if the liver is damaged repeatedly over a long period of time, it may undergo irreversible changes that permanently interfere with its essential function (Maton et al., 1993).

The liver gets a dual blood supply from the hepatic portal vein and hepatic arteries. Supplying approximately 75% of the liver's blood supply, the hepatic portal vein carries venous blood drained from the spleen, gastrointestinal tract, and its associated organs. The hepatic arteries supply arterial blood to the liver, accounting for the remainder of its blood flow. Oxygen is provided from both sources; approximately half of the liver's oxygen demand is met by the hepatic portal vein, and half is met by the hepatic arteries (Schneider .et al, 2008).

The bile produced in the liver is collected in bile canaliculi, which merge to form bile ducts. Within the liver, these ducts are called intra-hepatic (within the liver) bile ducts, and once they exit the liver they are considered extra-hepatic (outside the liver). The intra-hepatic ducts eventually drain into the right and left hepatic ducts, which merge to form the common hepatic duct. The cystic duct from the gallbladder joins with the common hepatic duct to form the common bile duct. Bile either drains directly into the duodenum via the common bile duct, or is temporarily stored in the gallbladder via the cystic duct (Sheporaitis and Freeny, 1998).

1.2.2.1. Liver Physiology

The various functions of the liver are carried out by the liver cells or hepatocytes. Currently, there is no artificial organ or device capable of emulating all the functions of the liver. Some functions can be emulated by liver dialysis, an experimental treatment for liver failure. The liver is thought to be responsible for up to 500 separate functions, usually in combination with other systems and organs (Schneider et al ., 2008).

The liver performs several roles in carbohydrate metabolism: Gluconeogenesis (the synthesis of glucose from certain amino acids, lactate or glycerol), Glycogenolysis (the breakdown of glycogen into glucose), Glycogenesis (the formation of glycogen from glucose) (muscle tissues can also do this). The liver is responsible for the mainstay of protein metabolism, synthesis as well as degradation. The liver also performs several roles in lipid metabolism: Cholesterol synthesis, Lipogenesis, the production of triglycerides (fats), A bulk of the lipoproteins is synthesized in the liver. The liver also produces coagulation factors I (fibrinogen), II (pro-thrombin), V, VII, VIII, IX, X and XI, as well as protein C, protein S and anti-thrombin. In the first trimester fetus, the liver is the main site of red blood cell production. By the 32nd week of gestation, the bone marrow has almost completely taken over that task.

The liver produces and excretes bile (a yellowish liquid) required for emulsifying fats and help the absorption of vitamin K from the diet. Some of the bile drains directly into the duodenum, and some is stored in the gallbladder. The liver also produces insulin-like growth factor 1 (IGF-1), a polypeptide protein hormone that plays an important role in childhood growth and continues to have anabolic effects in adults. The liver also is a major site of thrombopoietin production. Thrombopoietin is a glycoprotein hormone that regulates the production of platelets by the bone marrow (Sheporaitis and Freeny, 1998).

The breakdown of insulin and other hormones and also glucuronidates bilirubin, facilitating its excretion into bile. The liver breaks down or modifies toxic substances (e.g., methylation) and most medicinal products in a process called drug metabolism. This sometimes results in toxication, when the metabolite is more toxic than its precursor. Preferably, the toxins are conjugated to avoid

excretion in bile or urine. The liver converts ammonia to urea (urea cycle) (Sheporaitis and Freeny, 1998).

The liver stores a multitude of substances, including glucose (in the form of glycogen), vitamin A (1–2 years' supply), vitamin D (1–4 months' supply), vitamin B12 (1–3 years' supply), vitamin K, iron, and copper.

The liver is responsible for immunological effects the reticuloendothelial system of the liver contains many immunologically active cells, acting as a 'sieve' for antigens carried to it via the portal system. The liver produces albumin, the major osmolar component of blood serum. And also synthesizes angiotensinogen, a hormone that is responsible for raising the blood pressure when activated by renin, an enzyme that is released when the kidney senses low blood pressure (Sheporaitis and Freeny, 1998).

1.2.2.2. Liver disorders

There are more than a hundred kinds of liver disease. Acute and chronic Hepatitis, inflammation of the liver, is caused mainly by various viruses (viral hepatitis) but also by some liver toxins (e.g. alcoholic hepatitis), autoimmunity (autoimmune hepatitis) or hereditary conditions.

Cirrhosis is the formation of fibrous tissue (fibrosis) in the place of liver cells that have died due to a variety of causes, including viral hepatitis, alcohol overconsumption, and other forms of liver toxicity. Cirrhosis causes chronic liver failure.

Tumor of liver is two type; the first is called primary cancer of liver (hepatocellular carcinoma or Hepatoma) and the second is Metastatic liver tumor

arise from other cancerous tissue where the primary site was of lung , pancreas , gastrointestinal tract, or ovary origin .Hereditary diseases that cause damage to the liver include hemochromatosis . Hepatic jaundice ,Gilbert's syndrome, a genetic disorder of bilirubin metabolism found in about 5% of the population, can cause mild jaundice, Crigler-najjar disease :partial or complete deficiency of UDP-glycuronyltransferase , Dubin – Johnson syndrome : defective liver cell excretion of bilirubin. Intrahepatic cholestasis : may be caused by hepatocyte injury such as cirrhosis , bile duct injury such as rotor syndrome.There are also many pediatric liver diseases including: biliary atresia, alpha-1 antitrypsin deficiency, and progressive familial intrahepatic cholestasis (Anna p.ciulla .et al, 2002).

1.2.2.3. Liver function tests

1-Markers for hepato cellular necrosis (ALT; most specific for hepatocyte injury, AST; less specific than ALT significant presence in other tissues, LD least specific and significant presence in other tissues.)

2-Marker that reflect cholestasis (Alkaline phosphatase, Gamma-glutamyltransferase.)

3-tests to assess liver disorders (Total bilirubin , direct bilirubin (conjugated), indirect bilirubin (un conjugated) , Albumin, Ammonia, Alph fetoprotein) (Anna.et al, 2002).

1.2.2.4. Liver enzymes

I. Aspartate transaminase

Enzyme code (EC2.6.1.1) Aspartate transaminase (AST) or aspartate aminotransferase, also known as AspAT/ASAT/AAT or serum glutamic oxaloacetic transaminase (SGOT), is an enzyme belonging to the class of transferases. It's commonly referred to as a transaminase . AST catalyzes the reversible transfer of an α -amino group between aspartate and glutamate and, as such, is an important enzyme in amino acid metabolism. pyridoxal phosphate (PLP) function as coenzyme.

AST is widely distributed in human tissue the highest concentration found in cardiac tissue liver, skeletal muscle, with smaller amount in kidneys, and red blood cells (Maton et al ., 1993).

Two isoenzymes are present in a wide variety of eukaryotes. In humans: GOT1/cAST, the cytosolic isoenzyme derives mainly from red blood cells and heart and GOT2/mAST, the mitochondrial isoenzyme is present predominantly in liver.

These isoenzymes are thought to have evolved from a common ancestral AST via gene duplication, and they share a sequence homology of approximately 45 % (Hayashi. et al, 1990).

The clinical use of AST is limited mainly to the evaluation of hepatocellular disorder and skeletal muscle involvement. In myocardial infarction AST level begin to rise within 6 to 8 hours, peak at 24 hours, and return to normal within 5

day. AST may be elevated also in diseases affecting other organs, such as pulmonary embolism.

AST level are highest in acute hepatocellular disorders. In viral hepatitis level may reach 100 times ULN. In cirrhosis level reach 4 times URL.

Reference range 5 to 30 U/L (37C) (Maton et al ., 1993).

II. Alanine transaminase

(EC2.6.1.2), Alanine transaminase (ALT) is a transaminaseenzyme . It is also called alanine aminotransferase (ALAT) and was formerly called serum glutamate-pyruvate transaminase (SGPT) or serum glutamic-pyruvic transaminase (SGPT).ALT catalyzes the transfer of an amino group from L-alanine to α -ketoglutarate, the products of this reversible transamination reaction being pyruvate and L-glutamate. pyridoxal phosphate (PLP) function as coenzyme.

ALT is found in plasma and in various body tissues, but is most common in the liver. It is considered the more liver- specific enzyme of the transferaseALT is commonly measured clinically as a part of a diagnostic evaluation of hepatocellular injury, to determine liver health. Higher elevations are found in hepatocellular disorder than in extra-hepatic or intra-hepatic obstructive disorder (Maton et al ., 1993).

ALT, in conjunction with an elevated AST, is used to assess liver involvement with diagnosis of AMI. ALT dose not exhibit a significant increase in muscular dystrophy, and it is not affected in case of pulmonary emboli or acute pancreatitis (Anna p.ciulla .et al, 2002).

Reference range of ALT 6- 37 U/L (37C) (Bishop .et al, 2010).

IV. Alkaline phosphatase

(EC3.1.3.1), Alkaline phosphatase (ALP, ALKP, ALPase , AlkPhos) is a hydrolaseenzyme responsible for removing phosphate groups from many types of molecules, including nucleotides, proteins, and alkaloids. The process of removing the phosphate group is called *dephosphorylation*. As the name suggests, alkaline phosphatases are most effective in an alkaline environment. It is sometimes used synonymously as basic phosphatase (Tamaset al., 2002).

In humans, alkaline phosphatase is present in all tissues throughout the entire body, but is particularly concentrated in liver, bile duct, kidney, bone, intestinal mucosa and the placenta. Humans and most other mammals contain the following alkaline phosphatase isozymes (ALPI – intestinal (molecular weight of 150 kDa),ALPL – tissue-nonspecific (liver/bone/kidney) ,ALPP – placental (Regan isozyme) (Anna p.ciulla.et al, 2002).

Clinical significance of ALP a, the increase serum activity seen in heptobiliary disease and bone disorders (with osteoblstic involvement) . in heptobiliary disorders, the increase levels are due to obstructive disease, and the ALP levels are increase more significantly than ALT and AST .(in biliary tract obstruction, synthesis of ALP is induce by cholestasis , which causes serum ALP levels to rise 3 to 10 times the upper reference limit. the elevation is usually greater in case of extrahepatic obstruction in contrast to intrahepatic obstruction, in hepatitis and cirrhosis , which are classified as hepatocellular conditions, ALP levels rise up to 3 times the upper reference limit ,highest elevation of ALP are seen in Paget's disease , ALP levels increase with healing bone fracture) . ALP levels are

normally higher in children than adults because of bone growth. Decrease serum ALP levels are seen in Hypophosphatasia because of lack of ALP bone isoenzyme . This disorder characterized by insufficient bone calcification. ALP levels are normally higher in pregnancy because the placenta is a source of ALP. The source of elevated ALP levels can be deduced by obtaining serum levels of gamma Glutamyl-transferase (GGT). Concomitant increases of ALP with GGT should raise the suspicion of hepatobiliary disease (Anna p. ciulla et al., 2002).

Background study

In study done in Department of Biochemistry, Faculty of Basic Medical Sciences, University of Calabar, Calabar, Nigeria Toxicological implications of exposure to ungraded concentrations of kerosene and petrol fumes in albino Wistar rats were investigated after two weeks of 4 hours daily inhalation. Serum aminotransferases (AST and ALT), alkaline phosphatase (ALP), total cholesterol (Chol), triglyceride (TG) levels and histological analysis of the liver tissues were used as diagnostic markers to assess liver dysfunction. The mean levels of these markers determined for the group of rats exposed to kerosene and petrol fumes (test groups), as compared with the levels for the control group were significantly ($p < 0.05$) higher, increases in the serum levels of AST, ALT, ALP, Chol, TG in the petrol exposed group, when compared with the controls. Histological analysis of the liver tissues of the experimental test groups indicated degenerative changes in the ultrastructural integrity of the hepatic cells. These results showed that frequent exposure to kerosene and petrol fumes may be highly deleterious to the liver cells (University of Calabar, 2005).

The Islamic University-Gaza Deanery of Higher Education Faculty of Science Master of Biological Sciences medical technology More than half of workers work more than 5 years in the gasoline station. 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 compared to controls .

While a statistically significant decrease were found in red blood cells count, Platelets counts, alkaline phosphatase (ALP) and urea in workers (The Islamic University-Gaza, 2008)

2.1 Rationale

Liver enzymes are biocatalytic substance present inside hepatocyte and its concentration are remarkably constant in healthy subject, there are accumulating evidence that cause hepatocyte injury lead to leakag of enzymes and increase activity in serum inhalation of gasoline might have role in liver injury.

Recent research highlight that gasoline have devastating complication on liver and kidney.

This study was performed essentially to investigate the disturbances in the level of serum liver enzymes level in gasoline station workers.

2.2 Objective

2.2.1 General objective

To assess the level of liver enzymes in Sudanese gasoline station workers at Khartoum state

2.2.2 Specific objective

- To measure the liver enzymes level in Sudanese gasoline station workers and healthy individual
- To compare liver enzymes activity between gasoline station workers and healthy non gasoline station workers
- To find out correlation of liver enzymes activity with duration of gasoline exposure in sudanese gasoline station workers
- To find out correlation of liver enzymes activity with age of gasoline station workers

Materials and Method:

3.1 Study approach

This study utilized the quantitative approach in which the serum liver enzymes level is investigated in Sudanese gasoline station workers

3.2 Study type and design

The study is descriptive and design is cross – sectional case control community based study .

3.3 Study population

fifty random blood samples were collected from males Sudanese gasoline station workers In addition to fifty healthy individuals as control group for the comparison of serum liver enzymes level.

3.4 Study variable:

Both quantitative and qualitative variable were included . serum liver enzymes are measured kinetic in U/L

3.5 Inclusion criteria

Person who work at gasoline station as case

And person who non work at gasoline station as control

3.6 exclusion criteria

Person suffring from liver disease(hepatitis)

3.7 sample size

Sampling size was calculated according to the following formula

$$N = \frac{s^2 \cdot z^2}{d^2}$$

N = sample size

z = confidence level

s = standard deviation

d = desired marginal error

due to short time and the financial problem and reagent limitation the sample size was restricted to 100 subjects.

3.8 data collection method and tools

data were collected using structural interviewing questionnaire was designed to collect maintain all valuable information case examined

3.9Collection of sample

2.5 ml of venous blood samples were collected from both test and control subjects, by using sterile disposable plastic syringe and plastic stander non traumatic vein puncture technique. Then sample were emptied in plain container and stand, then centrifuged for 3600 rpm for 5 mins and separated (hemolysed and lipemic sample were excluded) into Eppendorf tube, stored at 2-8 c until analyzed.

3.10 determination of serum liver enzymes level

3.10.1 principle

Aminotransfer reaction between an amino acid and an alpha-keto acid. The amino (NH₂) group and the keto (=O) group are exchanged

Alkaline phosphatase hydrolyse p_nitrophenylphosphate into p_nitrophenol(yellow color) and phosphate

3.10.2 Procedure

automated method used centronic device

Tube	Working reagent	Serum
AST	1ml	0.1ml
ALT	1ml	0.1ml
ALP	1ml	0.02ml

Mixed and incubate for 30sec and measure the change of absorbance for at least 3min determine the difference of absorbance/min.

3.10.3 Calculation

The Enzyme activity in the test and control sample is calculated using the following formula

difference in absorbance/min * factor

3.11 ethical consideration:

approval for this study was taken from the department of clinical chemistry verbal consent was taken from both test and control subjects .

3.12. Quality control

Sample representing the normal and pathological level of serum liver enzymes , was used for assessment of the quality control. Result \pm 2SD of the target values of the control sera were accepted .

3.13.Statistical analysis

Data was analyzed by computer soft ware, by using SPSS program manual master sheet. The mean and standard deviation of liver enzymes level was obtained, and the test was used for the comparison of liver enzymes level between the test and control group, and the mean difference is significant at $p \leq 0.05$, Correlation(r) between liver enzymes level with both age and duration of works is considered to be statistically significant at $P \leq 0.05$.

Results

The study population comprised of 100 individual in Khartoum state 50 test subject works at gasoline station , with age range from 16-55 years and duration from 1- 17 years, In addition to 50 healthy volunteers were age and gender matched with their corresponding test groups.

As illustrated in table 4.1 there is significant increase in serum liver enzymes level in test group when compared with corresponding control group (30 ± 10.6 U/L, $p \leq 0.001$, 22 ± 11.9 U/L, $p \leq 0.01$, 132 ± 40 U/L, $p \leq 0.001$ for AST, ALT and ALP respectively). As illustrated in figure 4.1 correlation between serum AST activity (IU/L) and duration of work (years) in Gasoline stations ($r = 0.28$, p .value = 0.04) ,

Figure 4.2: correlation between serum ALT activity (IU/L) and duration of work (years) in Gasoline stations ($r = 0.28$, p .value = 0.045) and Figure 4.3: correlation between serum ALP activity (IU/L) and duration of work (years) in Gasoline stations ($r = 0.44$, p .value = 0.001)

Also as illustrated in figure 4.4 correlation between serum AST activity (IU/L) and age of workers at Gasoline stations in Khartoum state ($r = 0.67$, p .value = 0.0001),

Figure 4.5: correlation between serum ALT activity (IU/L) and age of workers at Gasoline stations in Khartoum state ($r = 0.45$, p .value = 0.0001)

And figure 4.6 correlation between serum ALP activity (IU/L) and age of workers at Gasoline stations in Khartoum state ($r = 0.23$, p .value = 0.02).

Table 4.1: Comparison of Liver enzymes activity between Gasoline station workers and healthy individuals

	Gasoline station's workers (n = 50)	Healthy individuals (n=50)	p.value
AST (IU/L)	30.3 ± 10.6	24.3 ± 5.6	0.001
ALT (IU/L)	22.3 ± 11.9	17.6 ± 6.2	0.01
ALP (IU/L)	132 ± 40.4	110 ± 20.4	0.001

Data represent mean ± SD, p.value was calculated using Student T. test

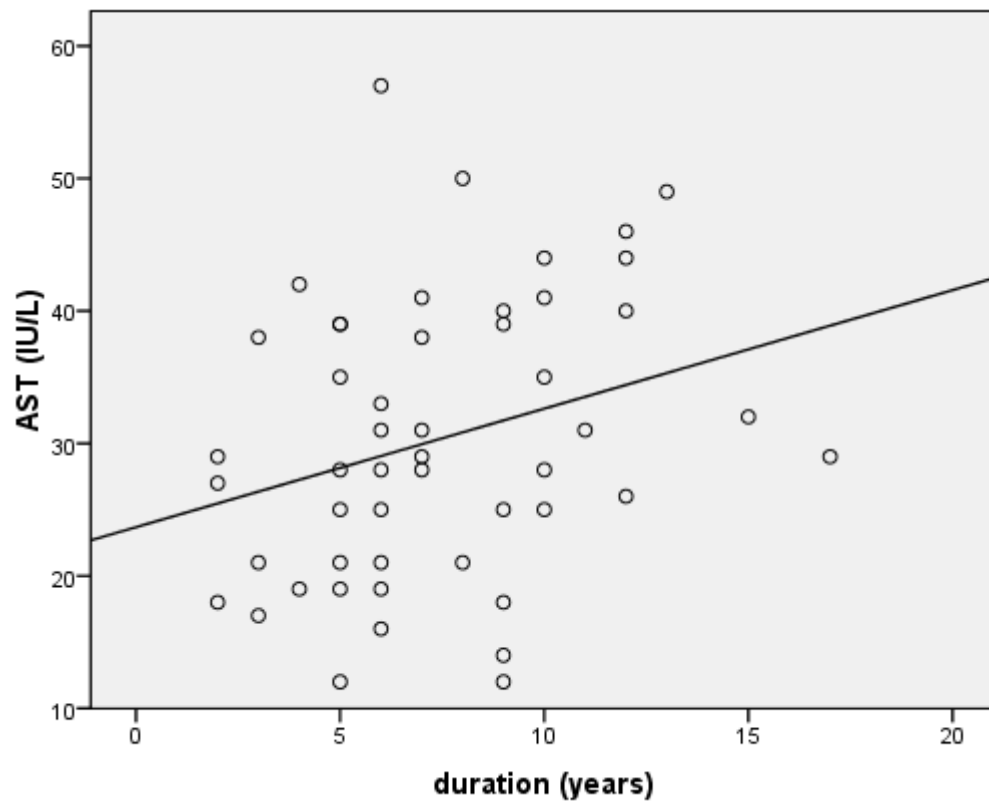


Figure 4.1: correlation between serum AST activity (IU/L) and duration of work (years) in Gasoline stations ($r = 0.28$, $p.\text{value} = 0.04$)

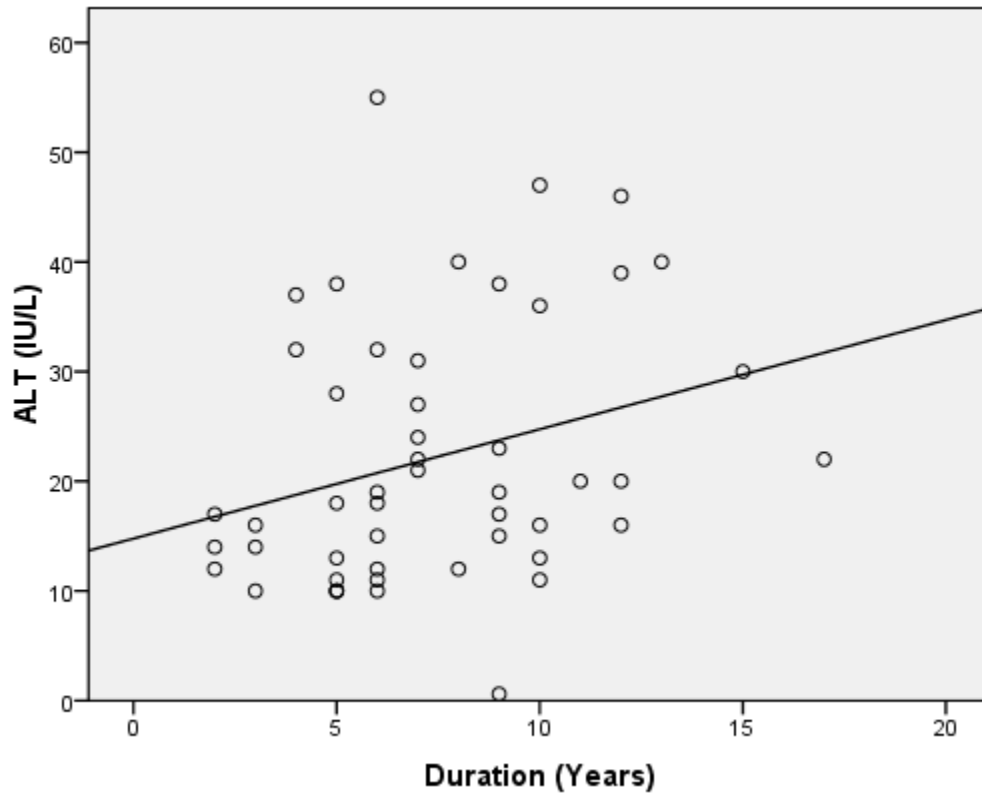


Figure 4.2: correlation between serum ALT activity (IU/L) and duration of work (years) in Gasoline stations ($r = 0.28$, $p.\text{value} = 0.045$)

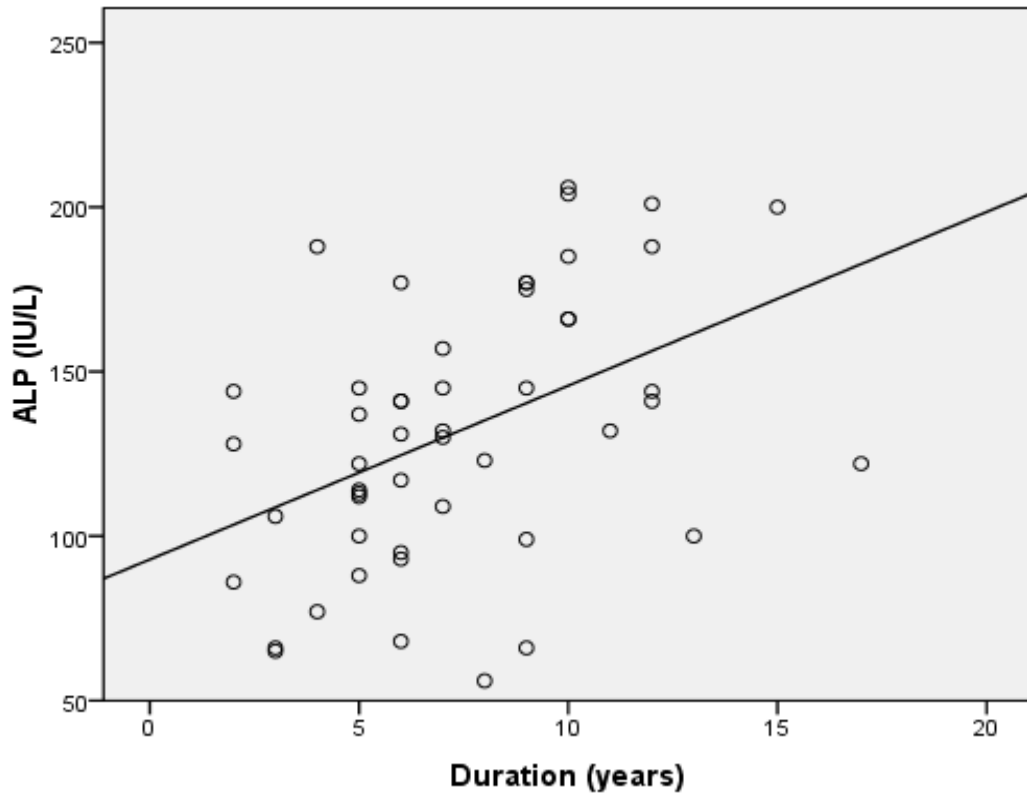


Figure 4.3: correlation between serum ALP activity (IU/L) and duration of work (years) in Gasoline stations ($r = 0.44$, $p.\text{value} = 0.001$)

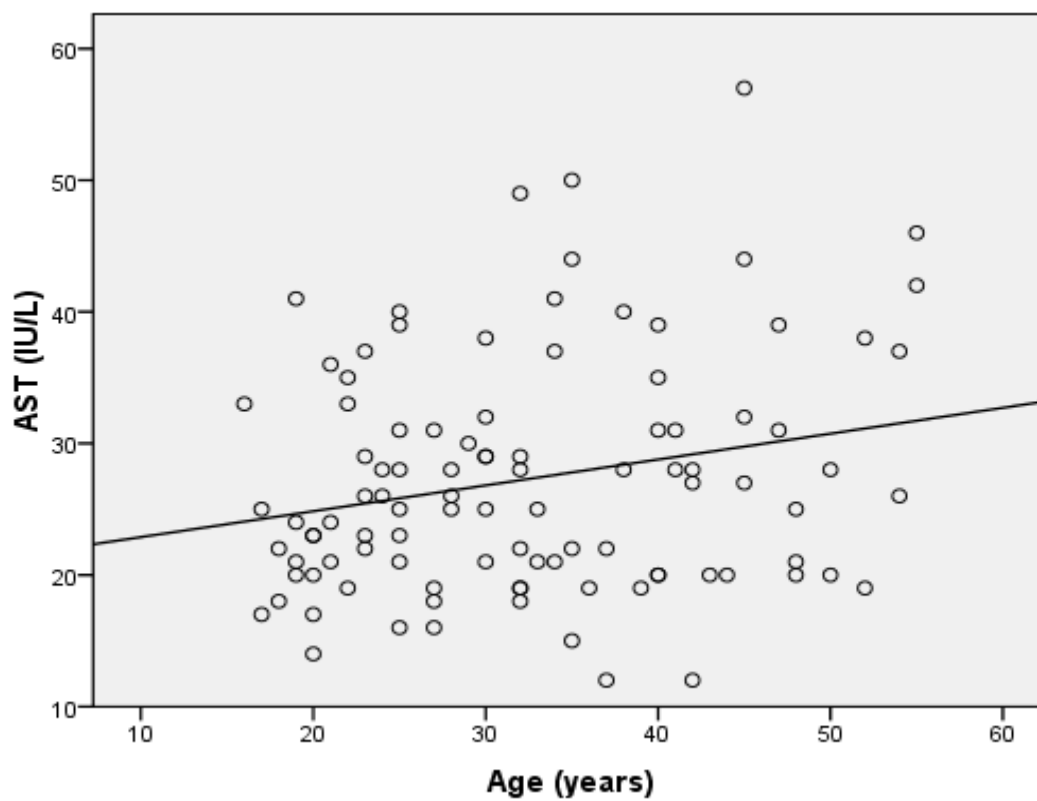


Figure 4.4: correlation between serum AST activity (IU/L) and age of workers at Gasoline stations in Khartoum state ($r = 0.67$, $p.value = 0.0001$)

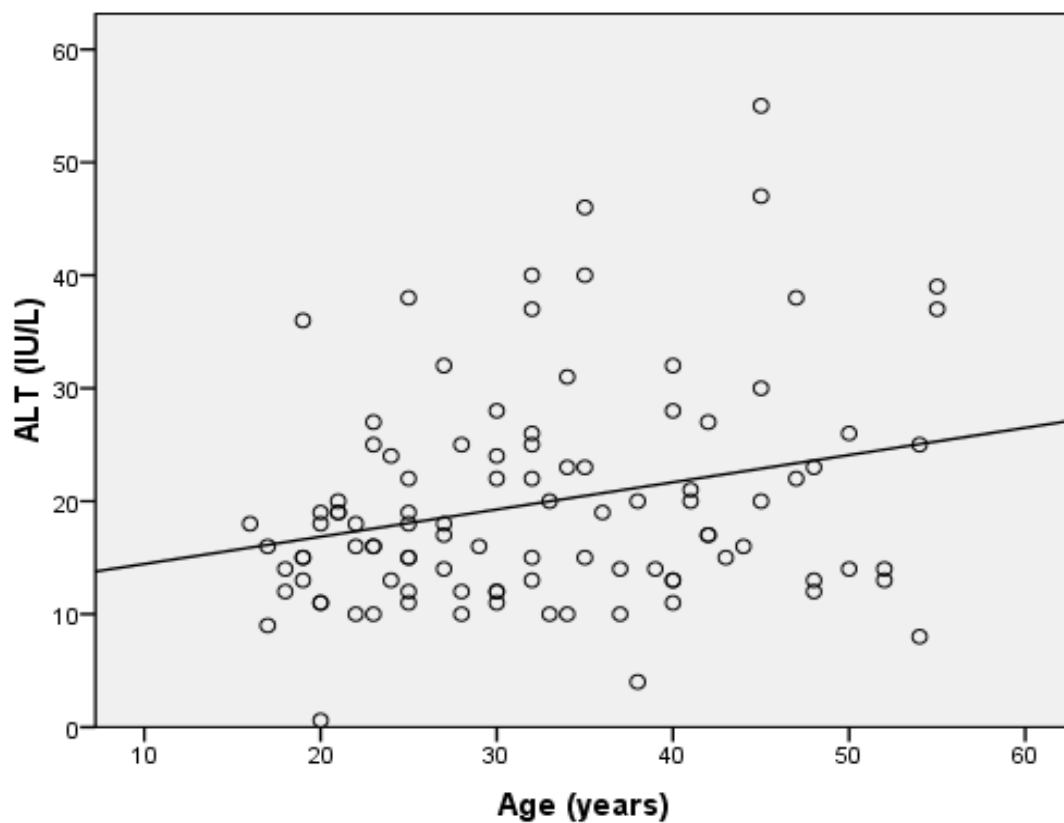


figure 4.5:correlation between serum ALT activity (IU/L) and age of workers at Gasoline stations in Khartoum state ($r = 0.45$, $p.\text{value} = 0.0001$)

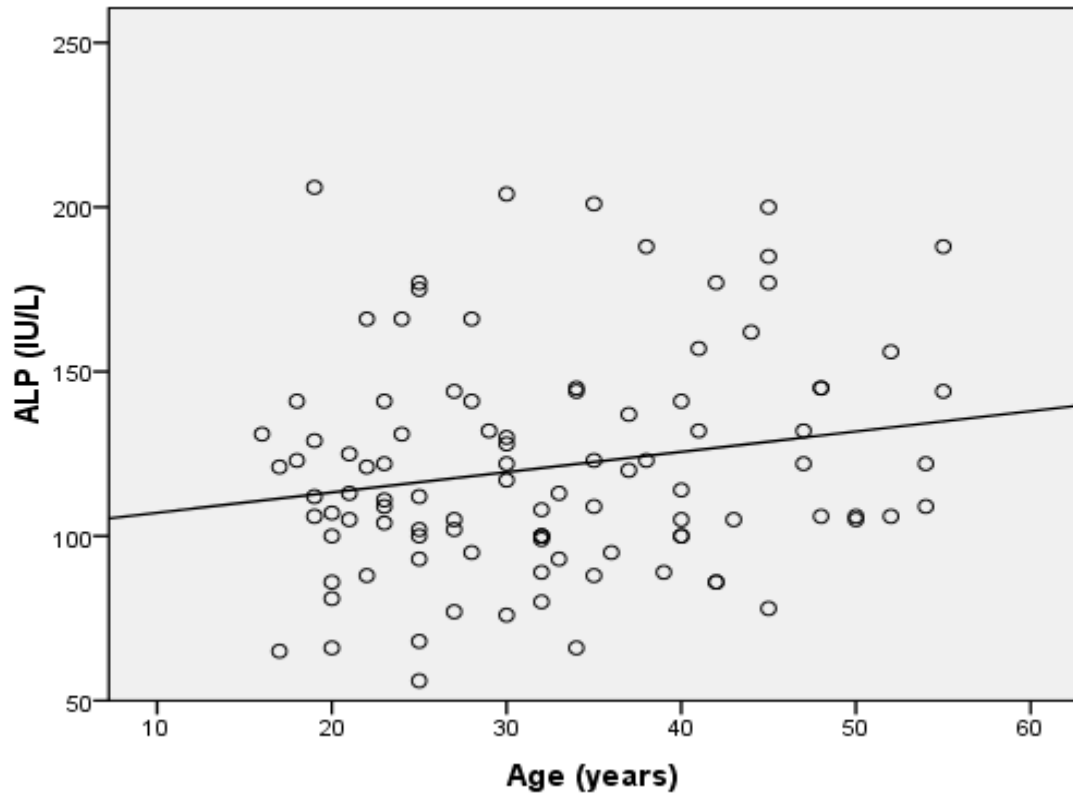


Figure 4.6: correlation between serum ALP activity (IU/L) and age of workers at Gasoline stations in Khartoum state ($r = 0.23$, $p.value = 0.02$)

5.1 Discussion

This study was done to evaluate serum liver enzymes level among Sudanese gasoline station workers a 50 workers included in this study and 50 healthy people The result of this study showed that there is a significant increase between study group and control group in serum liver enzymes level among gasoline station workers group (30 ± 10.6 U/L, $p \leq 0.001$, 22 ± 11.9 U/L, $p \leq 0.01$ 132 ± 40 U/L, $p \leq 0.001$ for AST, ALT and ALP respectively). And there are correlation between serum AST activity (IU/L) and duration of work (years) in Gasoline stations ($r = 0.28$, $p.value = 0.04$) , correlation between serum ALT activity (IU/L) and duration of work (years) in Gasoline stations ($r = 0.28$, $p.value = 0.045$) and correlation between serum ALP activity (IU/L) and duration of work (years) in Gasoline stations ($r = 0.44$, $p.value = 0.001$). Also there are correlation between serum AST activity (IU/L) and age of workers at Gasoline stations in Khartoum state ($r = 0.67$, $p.value = 0.0001$), correlation between serum ALT activity (IU/L) and age of workers at Gasoline stations in Khartoum state ($r = 0.45$, $p.value = 0.0001$) And correlation between serum ALP activity (IU/L) and age of workers at Gasoline stations in Khartoum state ($r = 0.23$, $p.value = 0.02$). this study is agree with another study done by The Islamic University-Gaza Deanery of Higher Education Faculty of Science Master of Biological Sciences Medical Technology 2008 found Serum liver enzymes level were significantly increase in gasoline station workers when compared to control subjects (28.6 ± 7.9 U/L , 24.7 ± 3.6 U/L for AST) (29.1 ± 11.5 U/L , 23.9 ± 9.7 U/L for ALT) (The Islamic University-Gaza, 2008).

And also agree with other study done in Department of Biochemistry, Faculty of Basic Medical Sciences, University of Calabar, Calabar Nigeria Toxicological

implications of exposure to ungraded concentrations of kerosene and petrol fumes in albino Wistar rats were investigated after two weeks of 4 hours daily inhalation that found increases in the serum levels of AST, ALT, ALP, Chol, TG in the petrol exposed group were 177%, 140%, 191%, 100% and 97%, respectively, when compared with the controls (University of Calabar, 2005).

5.2 Conclusion

This study revealed that exposure to gasoline cause significant increase in serum liver enzymes level .also there is correlation between the duration, age of works and level of the enzymes.

5.3 Recommendation

- Investigate other liver function tests
- Regular examination of liver function tests
- Use safety gloves and face mask during handling gasoline