

# The Effect of Ingested Kerosene Contaminated Diets on the Liver, Kidney and Lungs of Wistar Albino Rats

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**Abstract:**-The purpose of this study was to investigate the effects of ingested kerosene contaminated diets on the liver, kidney and lungs of wistar albino rats. Twenty (20) wistar albino rats were grouped into four (4) of five (5) rats each. Group A was the control group while group B, C and D were administered 1 ml, 2ml and 4ml respectively to kerosene contaminated diets. The result of the final body weight of the animals showed significant increase ( $p<0.05$ ) in group B ( $150.43\pm 8.785$ ), group C ( $156.30\pm 3.691$ ) and group D ( $135.70\pm 7.503$ ). Compared to the control group ( $98.53\pm 6.678$ ). The results of the biochemical parameters were; The activity of AST was significantly higher in group B ( $104.93\pm 2.555^b$ ), group C ( $129.47\pm 23.700^b$ ) and group D ( $146.13\pm 28.010^b$ ) compared to the control group ( $92.89\pm 0.465$ ). The activity of ALT was significantly higher in group B ( $27.00\pm 7.000^b$ ), group C ( $29.50\pm 1.500^b$ ), group C ( $29.50\pm 1.500^b$ ) and group D ( $44.00\pm 5.000^b$ ) compared to the control group ( $20.00\pm 1.000$ ). ALP activity in the animals were significantly higher ( $P<0.05$ ) in group B ( $67.56\pm 1.450^b$ ), group C ( $69.94\pm 0.170^b$ ) and group D ( $96.91\pm 1.130^b$ ) compared to the control group ( $61.36\pm 0.650$ ). There is a significant reduction ( $P<0.050$ ) in group B ( $61.62\pm 8.390^a$ ), group C ( $62.09\pm 0.680^a$ ), and group D ( $62.01\pm 2.095^a$ ) Glutathione (GSH) activities compared to the control group ( $89.00\pm 0.400$ ). In this study, exposures of the albino rats to kerosene showed significant increase ( $p<0.05$ ) in serum creatinine level of group B ( $64.06\pm 3.045^b$ ), group C ( $60.56\pm 1.450^b$ ) and group D ( $71.66\pm 1.350^b$ ) compared to the control group ( $51.66\pm 1.550$ ). Urea was retained in group D ( $7.11\pm 1.485^b$ ) as indicated by the significant increase ( $p<0.05$ ) in serum urea compared to the control group ( $5.47\pm 0.185$ ). Whereas the level of urea in group B ( $4.08\pm 0.590^a$ ) and C ( $5.43\pm 1.325^a$ ) decreases as compared to the control group ( $5.47\pm 0.185$ ). In this study, sodium increased significantly ( $p<0.05$ ) in group B ( $133.35\pm 3.050^b$ ), group C ( $138.75\pm 5.050^b$ ) and group D ( $141.85\pm 1.250^b$ ) compared to the control group ( $128.90\pm 4.800$ ). In this study, potassium increased significantly ( $p<0.05$ ) in group B ( $10.32\pm 0.825^b$ ), group C ( $9.28\pm 0.310^b$ ) and group D ( $10.94\pm 0.665^b$ ) compared to the control group ( $6.94\pm 0.110$ ). . In this study, chloride increased significantly ( $p<0.05$ ) in group C ( $27.49\pm 2.805^b$ ) compared to the control group ( $25.78\pm 2.140$ ). Whereas the level of chloride in group B ( $24.91\pm 2.460^a$ ) and group D ( $25.73\pm 3.630^a$ ) decrease compare to the control group ( $25.78\pm 2.140$ ). Histological examination of the liver, kidney and the lungs indicated that kerosene contaminated diets induced significant degenerative changes in the structural integrity of both the hepatic, renal and the lungs cells. However, the histological examination of the control group showed normal histological structure. Therefore, the results of this work suggest that kerosene contaminated diets indicated oxidative stress and

could possibly cause adverse effect on the kidney and impaired liver and lungs functions.

**Key words:** Kerosene, contaminated diets, histological examination, liver, kidney and lungs.

## I. INTRODUCTION

Kerosene (synonyms: paraffin, paraffin oil, fuel oil no. 1, lamp oil) is a middle distillate of the petroleum refining process that boils between 145 and 300°C. It is a transparent liquid fuel with a mixture of hydrocarbon chains 6 to 16 carbon atoms in length. Since the mid-19<sup>th</sup> century, when it replaced the more expensive whale oil as a lighting fuel, it has become a major household, commercial, and industrial fuel used for cooking, lighting, and heating. But in the first half of the 20<sup>th</sup> century, the prevalence of household kerosene lighting greatly reduced as electrification and availability of gas fuels spread, particularly in developed countries. However, in the developing countries of Africa, Asia, and Latin America, due to its economic viability and ease of availability, kerosene has continued to be used for several purposes with domestic uses such as cooking, heating and lighting using kerosene lamps or wick lamps, and lately the hurricane and high pressure lamps particularly in the developing countries being among top uses (Lam *et al*, 2012 cited in Maiyoh *et al*, 2015). Globally, an estimated 500 million households still rely on kerosene for lighting, corresponding to 7.6 billion liters consumed annually (Mills 2005).

Kerosene is commonly used in countries where solid fuels-biomass (wood, agricultural residues, animal dung and coal) are major house-hold energy sources. Its use also as cooking fuel is mostly restricted to some portable stoves most especially by students who use kerosene to cook in an enclosed environment without chimneys or smoke hoods. In addition, kerosene has been used to treat pools of standing water to prevent mosquitoes from breeding. It is also used as a solvent and in conjunction with cutting oil as a thread cutting and reaming lubricant. More so, it is also used by motor mechanics for washing of hands and other parts of the car together with petrol. Some cultural practices also drink kerosene as antidots to snake bite (Igboh *et al*, 2001).

Despite the wide acceptability and consumption of kerosene as a cheap cooking fuel, our knowledge on the

toxicological and biochemical effect of exposure to kerosene either by ingestion, inhalation or physical contact is still sparse. Exposure to kerosene, which occurs frequently in Nigeria, can result in toxicity. The sources of exposures include accidental ingestion by children, motor mechanics and non-accredited vendors, occupational inhalation by storage tank workers, transport tank workers, service station workers, petroleum refining workers, and deliberate human activities such as kerosene pipeline leakages as a result of vandalism.

The principal toxicological effects arising from the exposure to ingestion of kerosene is often associated with nausea, vomiting and occasionally diarrhoea. Inhalation and/or exposure to kerosene may cause headache, dizziness, drowsiness, in coordination and euphoria. Aspiration into the lungs causes pneumonitis with choking, cough, wheeze, breathlessness, cyanosis, and fever. Skin exposure to kerosene may result in dermatitis through the extraction of endogenous skin lipids. Also acute exposure to kerosene in humans has been associated with a variety of central nervous system (CNS) effects including irritability, restlessness, ataxia, drowsiness, convulsions, coma, and death.

The role of the liver, kidney and lungs in detoxification of exogenous compounds such as kerosene has made it susceptible to damage. The liver contains enzymes which are present in serum in low concentration. Elevated serum levels of these enzymes occur when there is liver injury, damage or necrosis of liver cell. Exposure of animal or humans to kerosene can also increase the level of creatinine, urea and body electrolytes concentrations which are usually used for assessing renal function. In renal disease, serum creatinine values do not increase significantly until renal function has been considerably impaired. Renal dysfunction may be caused by several diseased conditions and exposure to certain reactive or toxic metabolites. Renal dysfunction of any kind affects all parts of the nephron to some extent, although sometimes, either glomerular or tubular dysfunction is predominant. The net effect of renal disease on plasma and urine depends on the proportion of glomeruli or tubules affected, and on the number of nephrons involved.

The low viscosity and surface tension of kerosene allow it to be aspirated into the lungs of people who have ingested it, provoking a chemical pneumonitis, which can be fatal if untreated. Nevertheless, kerosene poisonings make up a significant portion of total poisoning incidents each year, particularly in developing countries. For example, a study of 120 unintentional childhood poisoning cases in Pakistan produced a population-attributable risk of 40% (Ahmed *et al.* 2011). In Nigeria, kerosene poisoning constituted 1.2% of all paediatric admission (not limited to only poisoning cases). Kerosene poisoning mostly affect respiratory, central nervous, and gastro-intestinal systems, rarely are other organs involved. Chemical pneumonitis and bronchitis are the most common features of kerosene poisoning.

So this has prompted the researcher to investigate the effect of ingested kerosene contaminated diets on the liver, kidney, and lungs of albino rats.

## II. MATERIALS AND METHODS

*Test Animals:* Wistar albino rats weighing 80-110g were bought from National Veterinary Research Institute (NVRI), VOM and transported to the animal house, Department of biochemistry, faculty of basic medical sciences, University of Jos for further analysis.

*Test Sample:* The test sample kerosene (0.77-0.82 specific gravity) was purchased from Echepet filling station pankshin, plateau state, Nigeria and was transported to the animal house, Department of biochemistry, faculty of basic medical sciences, University of Jos for further analysis.

*Formulation of Diets:* The contaminated diets were formulated by measuring out various volumes (1, 2, and 4ml) of kerosene and the corresponding measured amount of animal feed which were mixed thoroughly. The formulated diet was made into pellets to feed the rats. The feed for the control group was compacted with water only.

*Experimental Design:* Twenty (20) wistar albino rats weighing 80-110g were randomly selected into 4 groups according to the varying percentage concentrations of kerosene (0%, 1%, 2% and 4%) of five animals each. Control has 0% concentration of kerosene. All the experimental animals were housed in separate metabolic cages measuring about 60cm x 30cm x 45cm in a well-ventilated animal house of Department of Biochemistry, Faculty of Basic Sciences, University of Jos. Jos plateau state. They were given access to water *ad libitum* and fed with normal feed obtained from vital feeds (Grand cereals Ltd, Nigeria), Jos, Nigeria, for seven days of acclimatization before exposure to feed contaminated with kerosene. After a period of seven days, the test groups were exposed to the feed contaminated with different concentrations of kerosene. While the control groups were given access to normal feeds and water.

*Collection of samples:* At the end of the experiment, the rats were anaesthetized with chloroform soaked in swab of cotton wool in desiccators. They were then sacrificed and 5ml sterile syringes with needle were used for collection of blood from vena cava into properly labeled EDTA sample bottles and were taken to biochemistry department, National Veterinary Research Institute (NVRI), VOM for biochemical test analysis. After that, the organs (kidney, Liver and lungs) were collected by pinning the animal in a dissecting tray, and using a scalpel and dissecting scissors for making incisions, the lungs was collected and preserved in 10% formaldehyde and were taken to National Veterinary Research Institute (NVRI), VOM for histological examination.

*Determination of Liver Enzymes:* Plasma enzymes like alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP) were determined using Randox diagnostic Kits, UK

**Determination of kidney function Test:** Plasma urea (UR), Creatinine (CR) was determined using Randox diagnostic Kits, UK. Potassium and Chloride were determined using Teco Diagnostic Kits, U.S.A while Sodium is determined using Spectrum reagent Kits, Egypt.

**Determination of reduced glutathione (GSH):** Reduced glutathione (GSH) was determined using a colorimetric micro method, U.S.A (G-Biosciences.com)

**Histopathological assays:** The histopathological assays were determined by paraffin wax method of tissue processing. Conventional method (avwioro, 2011).

**Data analysis:** Statistical analysis was carried out using one-way analysis of variance (ANOVA). Data were analysed using GraphPAD prism 7 computer software. Data were expressed as the mean  $\pm$  standard error of mean and values of  $P < 0.05$  were considered significant.

### III. RESULTS

Result observed in animals fed with kerosene contaminated diets show marked increase in the final body weight compare to the control group shown in table 1 below. The biochemical parameters results investigated show a significant increase ( $p < 0.05$ ) in the activity level of the enzymes (AST, ALT and ALP) in all groups exposed to kerosene contaminated diets compare to the control group as shown in table 2 below. There is a significant reduction ( $P < 0.050$ ) in Glutathione (GSH) activities compared to the control group shown in table 2. The results from the effects of kerosene contaminated diets on serum creatinine, urea and electrolyte concentration (sodium, potassium and chloride)

show that creatinine increase significantly ( $p < 0.05$ ) in all groups exposed to kerosene contaminated diets compare to the control group. Urea increase significantly ( $p < 0.05$ ) in group D exposed to 4ml of kerosene contaminated diets compare to the control group, and decrease in group B and C exposed to 1ml and 2ml of kerosene contaminated diets respectively compared to the control group. Sodium increase significantly ( $p < 0.05$ ) in all groups administered with kerosene contaminated diets compare to the control group. While chloride increase significantly ( $p < 0.05$ ) in group C exposed to 2ml of kerosene contaminated diets compare to the control group and decrease in group B and D administered 1ml and 4ml of kerosene contaminated diets respectively compare to the control group as shown in table 3 below. Results from the histological examination below showed that the kidney of the rat exposed to 0ml of kerosene contaminated diets (plate 4a) has normal kidney architecture. Whereas, rats fed with 1ml, 2ml and 4ml (plate 4b-c) of kerosene contaminated diets revealed defects ranging from severe inflammation, renal degeneration (necrosis) in the histology of the kidney. Results from the histological examination below showed that the liver of the rat exposed to 0ml of kerosene contaminated diets (plate 4d) has normal liver histological architecture. Whereas, liver rats fed with 1ml, 2ml and 4ml (plate 4e-f) of kerosene contaminated diets revealed defects ranging from hepatic cord distortion, inflammation in the histology of the liver. Results from the histological examination below showed that the lungs of the rat exposed to 0ml of kerosene contaminated diets (plate 4g) have normal lung architecture. Whereas, rats fed with 1ml, 2ml and 4ml (plate 4i-j) of kerosene contaminated diets revealed defects ranging from severe pneumonia in the histology of the lungs.

Table 1. Body weight of animals

Group	Treatment	Initial weight (g)	Final weight (g)	Weight difference (g)
A	Control	91.00 $\pm$ 6.747	98.53 $\pm$ 6.678	7.53 $\pm$ 1.865
B	1ml	151.50 $\pm$ 6.497	150.43 $\pm$ 8.785	-1.07 $\pm$ 2.288
C	2ml	147.93 $\pm$ 1.994	156.30 $\pm$ 3.691	8.37 $\pm$ 1.697
D	4ml	139.23 $\pm$ 6.636	135.70 $\pm$ 7.503	-3.53 $\pm$ 6.114
p-values	-	0.0003	0.0015	0.3937

Values are expressed as mean  $\pm$  SEM, n = 5

If p value is less than 0.05, mean values are statistically significant ( $p < 0.05$ )

Table 2. Liver Enzyme assay

Group	Treatment	AST	ALT	ALP	GSH
A	Control	92.89 $\pm$ 0.465	20.00 $\pm$ 1.000	61.36 $\pm$ 0.650	89.00 $\pm$ 0.400
B	1ml	104.93 $\pm$ 2.555 <sup>b</sup>	27.00 $\pm$ 7.000 <sup>b</sup>	67.56 $\pm$ 1.450 <sup>b</sup>	61.62 $\pm$ 8.390 <sup>a</sup>
C	2ml	129.47 $\pm$ 23.700 <sup>b</sup>	29.50 $\pm$ 1.500 <sup>b</sup>	69.94 $\pm$ 0.170 <sup>b</sup>	62.09 $\pm$ 0.680 <sup>a</sup>
D	4ml	146.13 $\pm$ 28.010 <sup>b</sup>	44.00 $\pm$ 5.000 <sup>b</sup>	96.91 $\pm$ 1.130 <sup>b</sup>	62.01 $\pm$ 2.095 <sup>a</sup>
p-values	-	0.3041	0.0510	<0.0001	0.0261

Values are expressed as mean  $\pm$  SEM, n = 5

If p value is less than 0.05, mean values are statistically significant ( $p < 0.05$ )

<sup>a</sup>Values are significantly low when compared with control ( $p < 0.05$ )

<sup>b</sup>Values are significantly high when compared with control ( $p > 0.05$ )

Table 3. Kidney function examination

Group	Treatment	NA <sup>+</sup>	K <sup>+</sup>	CL <sup>-</sup>	UREA	CREATININE
A	Control	128.90±4.800	6.94±0.110	25.78±2.140	5.47±0.185	51.66±1.550
B	1ml	133.35±3.050 <sup>b</sup>	10.32±0.825 <sup>b</sup>	24.91±2.460 <sup>a</sup>	4.08±0.590 <sup>a</sup>	64.06±3.045 <sup>b</sup>
C	2ml	138.75±5.050 <sup>b</sup>	9.28±0.310 <sup>b</sup>	27.49±2.805 <sup>b</sup>	5.43±1.325 <sup>a</sup>	60.56±1.450 <sup>b</sup>
D	4ml	141.85±1.250 <sup>b</sup>	10.94±0.665 <sup>b</sup>	25.73±3.630 <sup>a</sup>	7.11±1.485 <sup>b</sup>	71.66±1.350 <sup>b</sup>
p-values	-	0.2283	0.0246	0.9251	0.3605	0.0090

Values are expressed as mean ± SEM, n = 5

If p value is less than 0.05, mean values are statistically significant (p < 0.05)

<sup>a</sup>Values are significantly low when compared with control (p < 0.05)

<sup>b</sup>Values are significantly high when compared with control (p > 0.05)

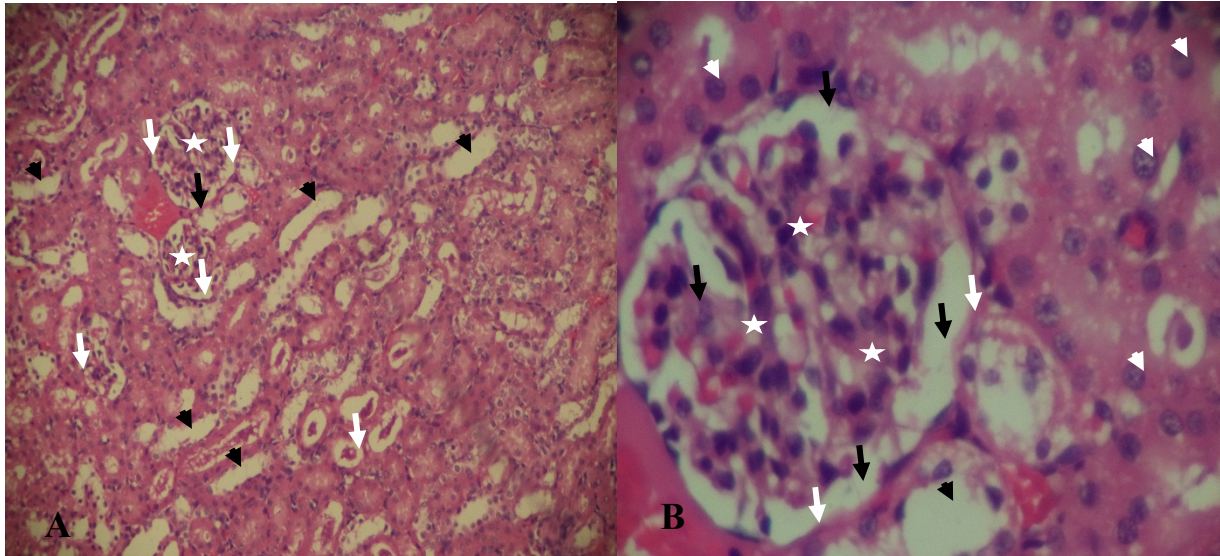


Plate 4a (AB): Kidney of Albino rat exposed to normal atmospheric and nutritional conditions, showing normal kidney architecture. The Glomeruli (white stars) are surrounded by a clear zone of capsular space (black arrows). The glomerular capsule is intact (white arrows) while the tubules (black arrowheads) are clear. White arrowheads= nuclei of cells. H&E A: X100 B: X400

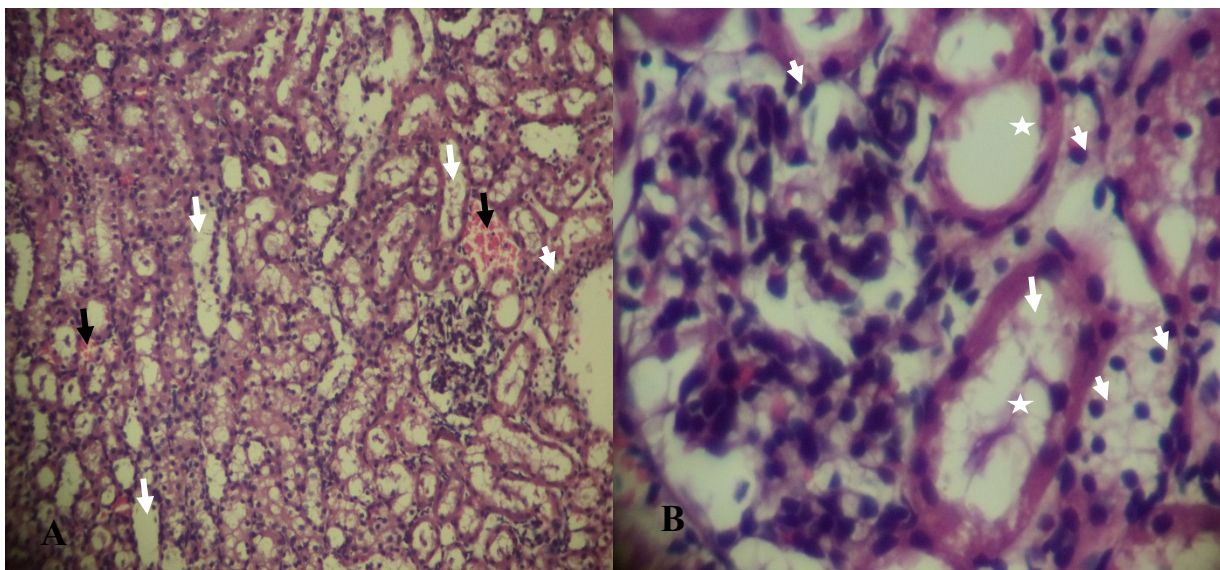


Plate 4b (CBL): Kidney of Albino rat exposed to 2mls of kerosene for two weeks, showing severe renal inflammation evident by the presence of inflammatory cells (white arrowheads) within the tissue. Blood vessels (white stars) appear slightly thickened. White arrows= kidney tubules, black arrows= red blood cells. H&E A: X100 B: X400

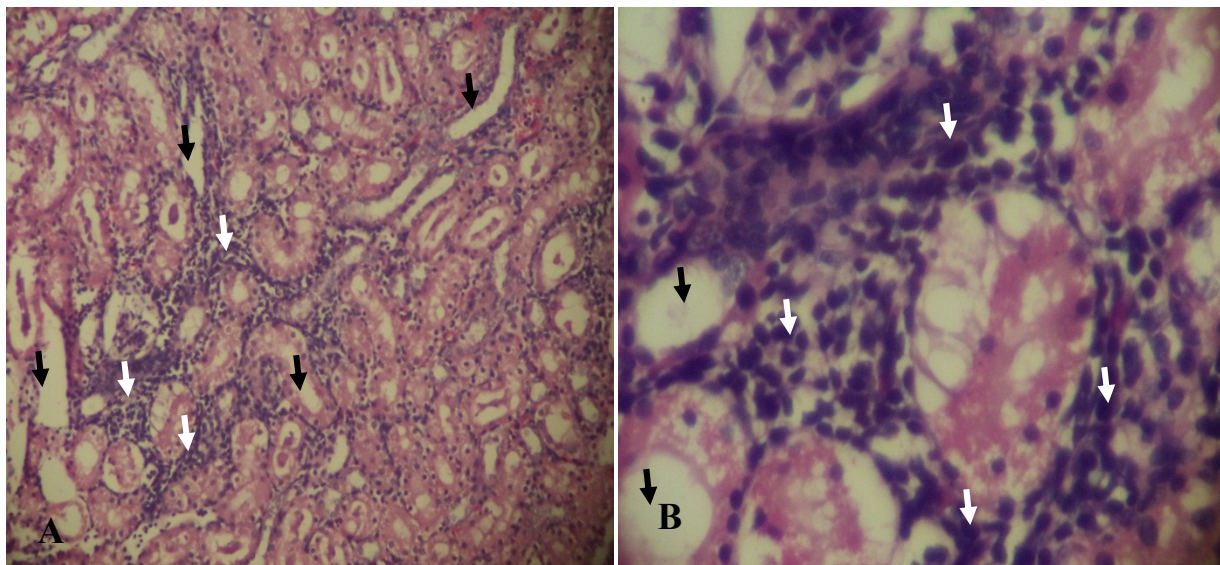


Plate 4c (DB): Kidney of Albino rat exposed to 4mls of kerosene for two weeks, showing severe renal degeneration (necrosis) and inflammation evident by the presence of inflammatory cells (white arrows) within the tissue which has lost its normal morphology. Black arrows= Kidney tubules. H&E A: X100 B: X400

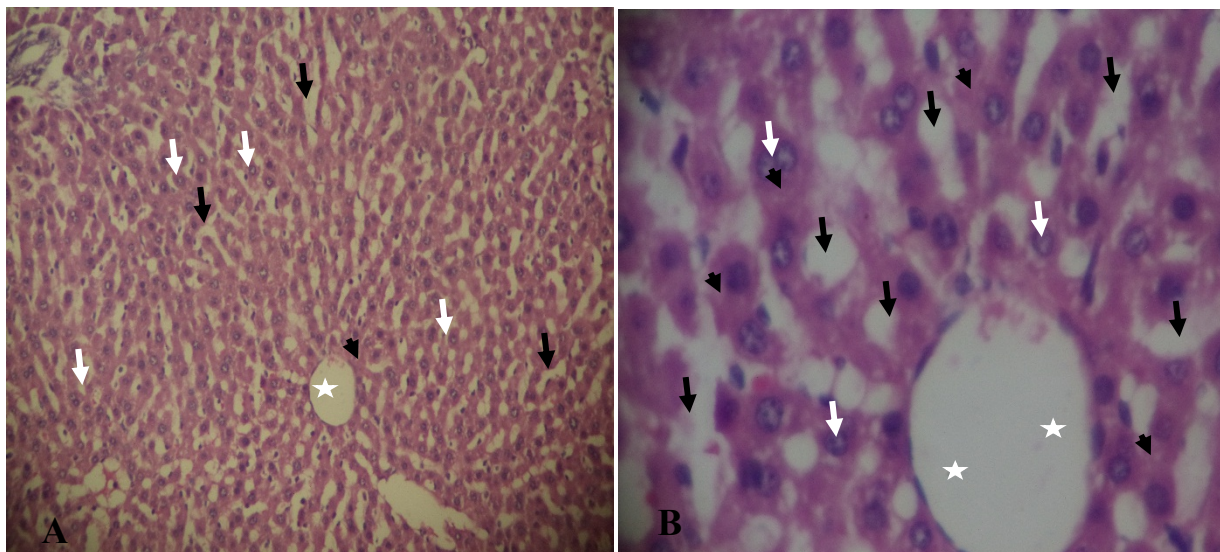


Plate 4d (ABL): Liver of albino rat exposed to normal atmospheric and nutritional conditions, showing normal tissue morphology. The nuclei (white arrows) appear intact and surrounded by intact cytoplasm (black arrowheads). The hepatocytes are interspersed by hepatic sinusoids (black arrows) which are connected to the central vein (white stars). H&E A: X100 B: X400

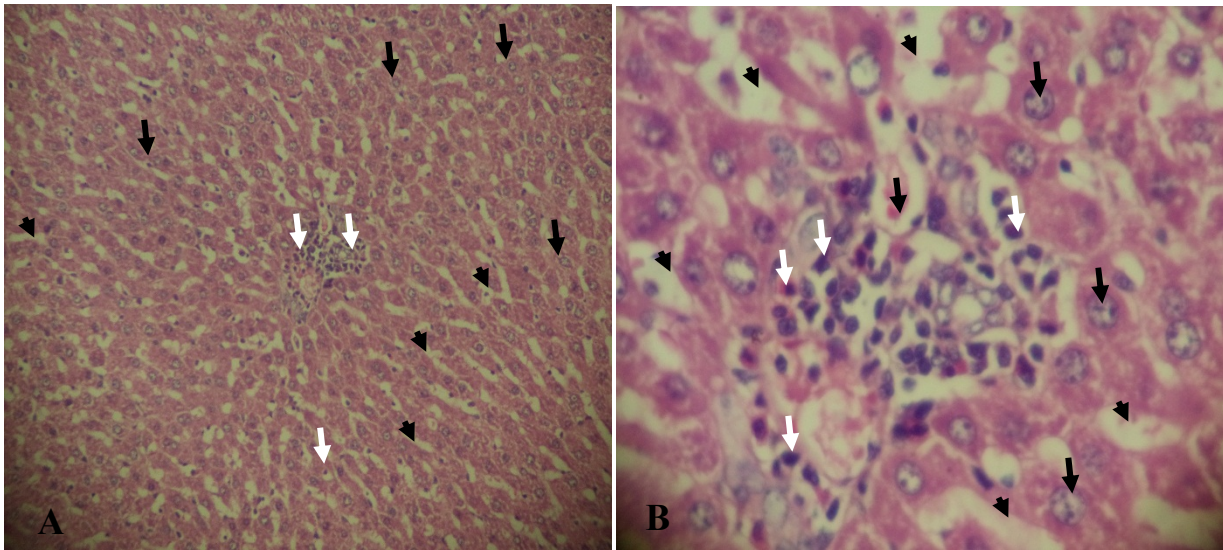


Plate 4e (CB): Liver of Albino rat exposed to 2mls of kerosene for two weeks , showing hepatic cord distortion and inflammation evident by the replacement of hepatocytes with inflammatory cells (white arrows). The inflamed area is surrounded by cells presenting with normal morphology, with intact nuclei (black arrows) surrounded by intact cytoplasm. Normal hepatocytes are seen interspersed by hepatic sinusoids (black arrowheads)H&E A: X100 B: X400

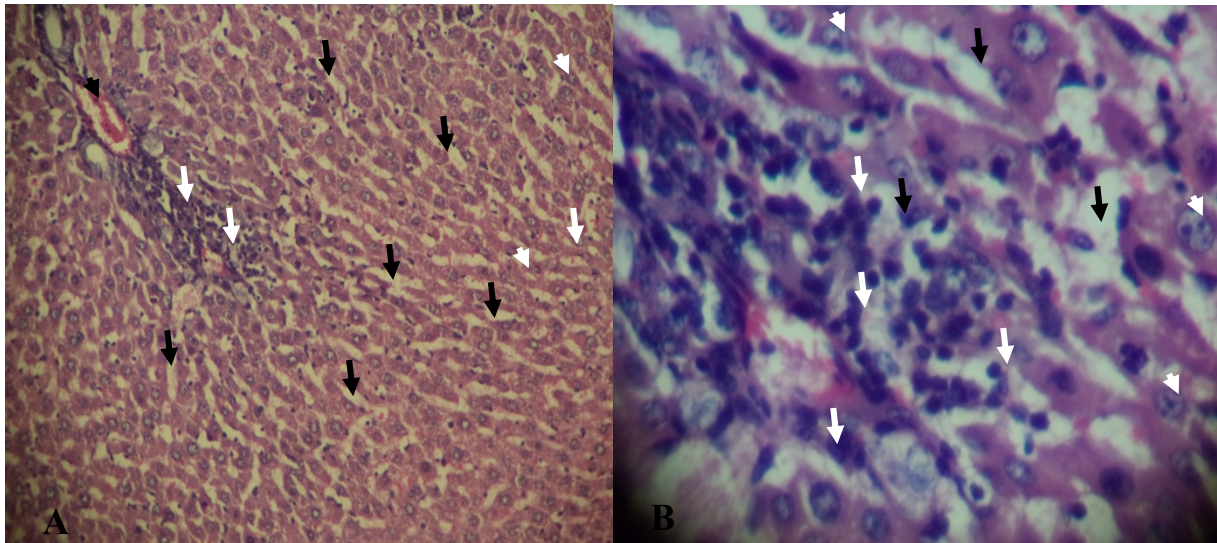


Plate 4f (DB): Liver of Albino rat exposed to 4mls of kerosene for two weeks, showing severe inflammation evident by the replacement of hepatocytes with inflammatory cells (white arrows). The inflamed area is surrounded by normal hepatocytes having intact nuclei (white arrowheads) surrounded by intact cytoplasm and are interspersed by hepatic sinusoids (black arrows). Black arrowhead= blood vessel. H&E A: X100 B: X400

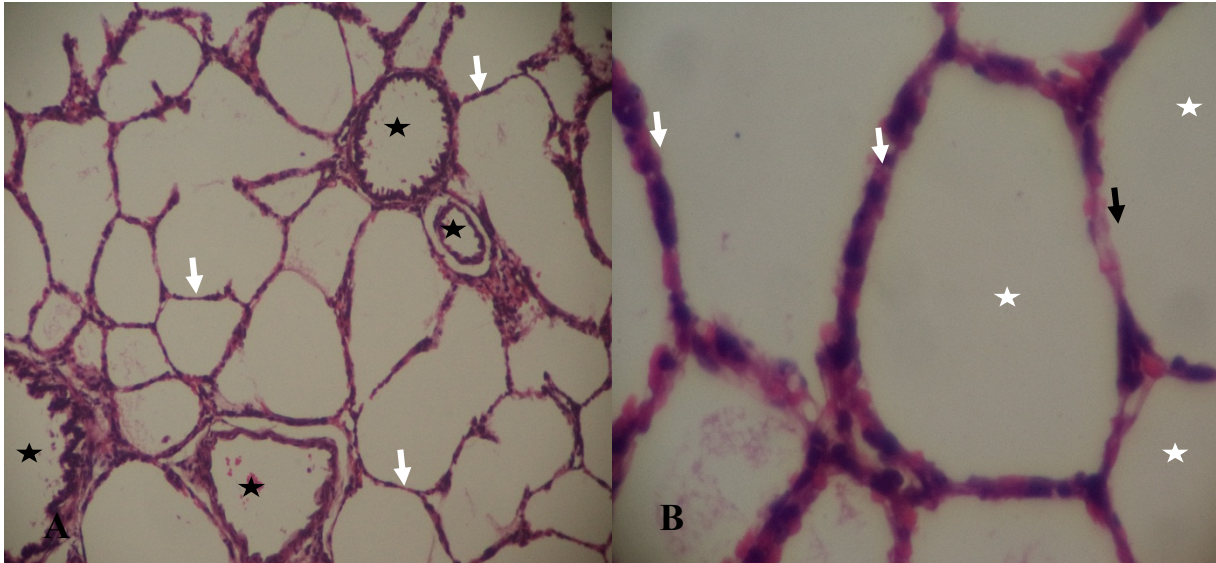


Plate 4g (ABL): Lungs of Albino rat exposed to normal environmental and nutritional condition, showing normal lung architecture. The alveolar septae (white arrows) are intact, surrounding the alveolar sacs (white stars) which appear clear. Black stars= blood vessels. H&E A: X100 B: X40

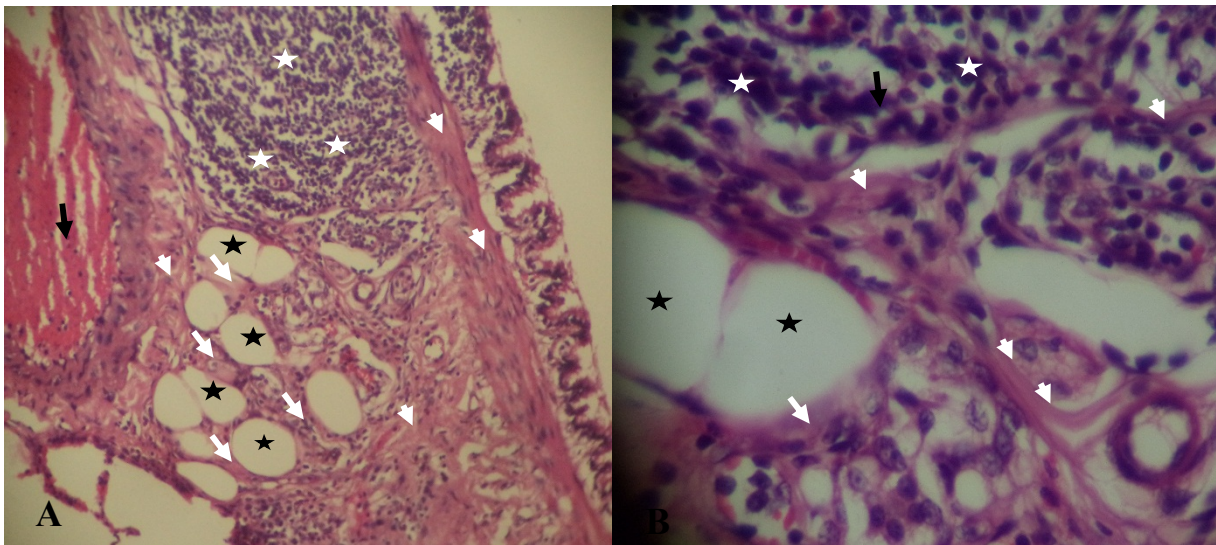


Plate 4h (BBL): Lungs of Albino rat administered 1ml of kerosene for two weeks, showing severe pneumonia as seen by the congestion of the lungs with inflammatory cells (white stars). The alveoli are completely taken over by inflammatory cells while few of the alveoli (black stars) that are free appear with thickened alveolar septae (white arrows) and smooth muscles (White arrowheads). Black arrow= red blood cells in blood vessel. H&E A: X100 B: X400

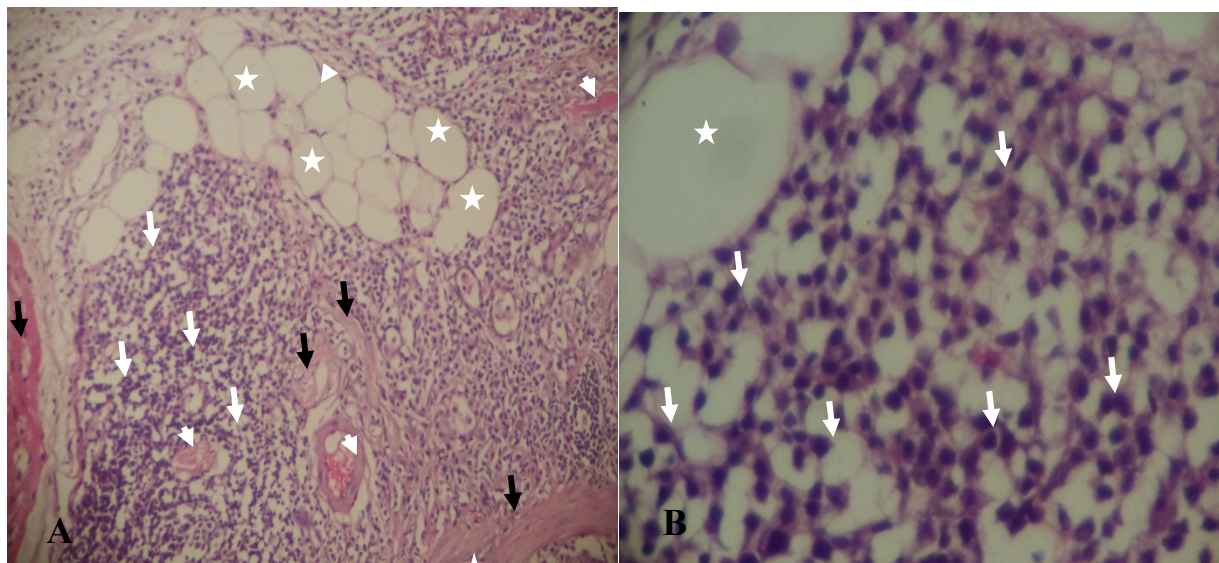


Plate 4i (CB): Lungs of Albino rat administered 2mls of kerosene for two weeks, showing severe pneumonia evident by the replacement of lung tissue with inflammatory cells. The inflammation is so severe that the tissue parenchyma is been replaced by the inflammatory cells (white arrows) thereby losing its morphology. There is also an increase in smooth muscles (black arrows) components. Blood vessels (white arrowheads) within the inflammatory cells appear thickened. Few alveoli sacs (white stars) are seen within the inflamed area. H&E A: X100 B: X400

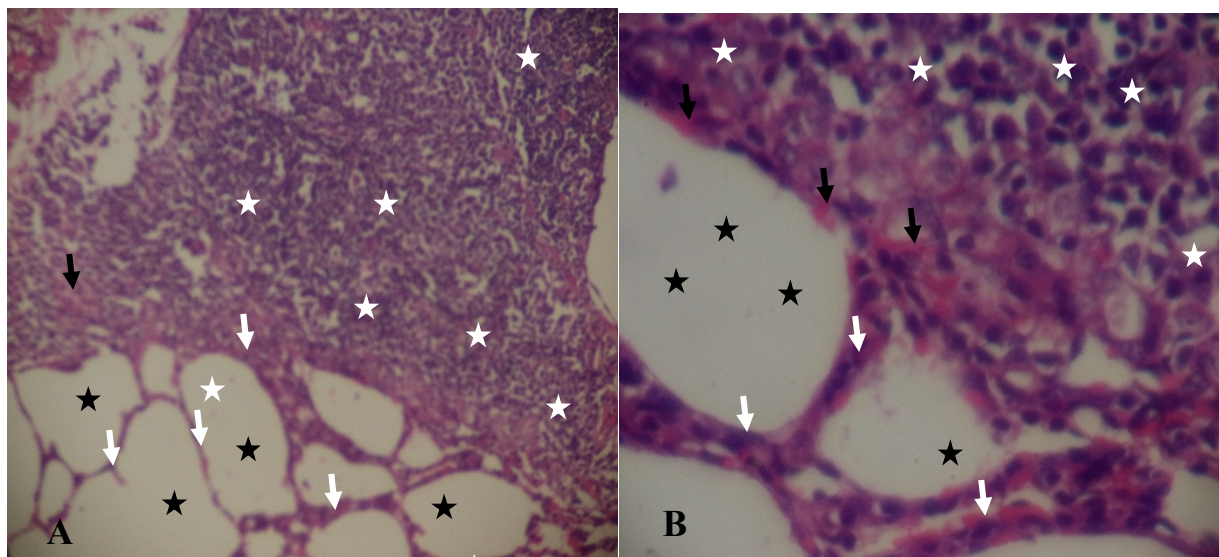


Plate 4j (DT): Lungs of Albino rat administered 4mls of kerosene for two weeks, showing severe pneumonia evident by the replacement of lung tissue with inflammatory cells thereby presenting a mass of inflamed area (white stars). The inflammation is so severe that the tissue parenchyma is been replaced by the inflammatory cells leaving only small areas presenting with normal alveoli (black arrows) surrounded by alveoli septae (white arrows). Black arrows= red blood cells. H&E A: X100 B: X400

#### IV. DISCUSSIONS

Exposure of humans and animals to kerosene, which is increasing in terms of the environmental levels and different usage of this petroleum product, may be toxic. Kerosene is

used in various homes to run many types of engines, lamps and heaters.

From table 4.1, the final body weight of the animals fed with kerosene contaminated diets were as follows. Group A has (98.53±6.678), group B (150.43±8.785), group C



(156.30±3.691) and group D (135.70±7.503). The results observed from the animals fed with kerosene contaminated diets showed marked increase ( $P<0.05$ ) in final body weight compared to the control group. This result is in agreement with the findings of Uboh et al (2008). This can involve an increase in muscle mass, fat deposits, excess fluids such as water or other factors. It has been reported that metabolism of aliphatic and aromatic hydrocarbons petroleum and petroleum derivatives, as well as other xenobiotics generates a significant increase in the level of free radical species in various tissues (Iam et al, 1994). This maybe an indication that the animals could not convert to the feed consumed into useful nutrients required by the body, thus accounting for the increased final body weight when compared to the control.

Exposure of humans and animals to kerosene which is increasing in terms of the environmental levels and different usage of this petroleum product may be toxic. The table 4.2.1 showing the biochemical parameters (AST, ALT and ALP) investigated showed a significant increase ( $P<0.05$ ) in all group exposed to kerosene contaminated diets when compared to the control group. Treatment of rates with kerosene contaminated diets resulted to a significant hepatic damage as elicited by the elevated levels of serum marker enzymes: AST, ALT and ALP. These marker enzymes are cytoplasm in origin and are released into the circulation after cellular damage (Lin et al, 2000).

AST is an enzyme found mostly in the heart muscle, liver cells, skeletal muscles and kidneys. The activity of AST was significantly higher in group B (104.93±2.555<sup>b</sup>), group C (129.47±23.700<sup>b</sup>) and group D (146.13±28.010<sup>b</sup>) exposed to kerosene contaminated diets compared to the control group (92.89±0.465). This agrees with the work of Patrick-Iwunanyanmu *et al*, (2011) and deviated from the work of Ogara *et al*, (2016). Increase activity of AST has also been reported in CCL4-induced toxicity in rats (Patrick-Iwunanyanmu et al, 2007; Patrick Uwuanyanm and Wegwu, 2008). This increase may be due to the abnormal dynamic properties of cellular membranes following exposure to hydrocarbons fractions present in kerosene and petrol (Uboh et al, 2005). This increase in activity of this enzymes indicated cellular leakage and failure of functional integrity of Liver cell membranes (Mukherkere, 2003).

The activity of ALT was significantly higher in group B (27.00±7.000<sup>b</sup>), group C (29.50±1.500<sup>b</sup>), group C (29.50±1.500<sup>b</sup>) and group D (44.00±5.000<sup>b</sup>) exposed to kerosene contaminated diets compared to the control group (20.00±1.000). Such elevation is an indicative of liver injury in patients developing severe hepatotoxicity. The result in this study is also in agreement with the findings of Salie et al (1999), who discovered that the rise in the enzyme AST is usually accompanied by an elevation in the levels of ALT, which plays a vital role in the conversion of Amino acids to Keto- acids. This increase maybe due to the abnormal dynamic properties of cellular membranes following exposure to hydrocarbon fractions present in kerosene.

ALP activity in the animals exposed to kerosene contaminated diet were significantly higher ( $P<0.05$ ) in group B (67.56±1.450<sup>b</sup>), group C (69.94±0.170<sup>b</sup>) and group D (96.91±1.130<sup>b</sup>) exposed to kerosene contaminated diets compared to the control group (61.36±0.650). ALP is involved in the transport of metabolites across membrane, synthesis of certain enzymes, protein synthesis, secretory activities and glycogen metabolism. However, the remarkable increase in the level of ALP in rats fed with kerosene contaminated diets may imply that damages occurred in the liver cells. However, the increase in this enzyme activity may not be unconnected with a disturbance in the transport of metabolites or alteration in the synthesis of certain enzymes as in other hepatotoxic conditions (Sharma et al, 1995).

In this study, there is a significant reduction ( $P<0.050$ ) in group B (61.62±8.390<sup>a</sup>), group C (62.09±0.680<sup>a</sup>), and group D (62.01±2.095<sup>a</sup>) Glutathione (GSH) activities compared to the control group (89.00±0.400). This is a result of hepatic injury from oxidative stress caused by the administration of kerosene. In system, organ and tissue damage, GSH makes up the first line of defense against free radicals resulting from Xenobiotic ingestion. The drop in the concentration of liver GSH indicates hepatocytes damage.

Creatinine retention in the blood is a significance of kidney impairment (okpala *et al*, 2014). In this study, exposures of the albino rats to kerosene showed significant increase ( $p<0.05$ ) in serum creatinine level of group B (64.06±3.045<sup>b</sup>), group C (60.56±1.450<sup>b</sup>) and group D (71.66±1.350<sup>b</sup>) compared to the control group (51.66±1.550). This shows that creatinine was actually retained as a result of the kerosene ingested by the animals, thereby signifying indication of kidney impairment. Impairment of the kidney function may be caused by exposure to nephrotoxic substances such as petroleum products.

Diseases of the kidney which diminish the glomerular filtration lead to the retention of urea. Urea was retained in group D (7.11±1.485<sup>b</sup>) administered with 4ml of kerosene as indicated by the significant increase ( $p<0.05$ ) in serum urea compared to the control group (5.47±0.185). This increase maybe as a result of disease in Glomerular Filtration Rate (GFR) which is suggestive of kidney failure, dehydration, congestive heart failure, stress etc. whereas the level of urea in group B (4.08±0.590<sup>a</sup>) and C (5.43±1.325<sup>a</sup>) decreases as compared to the control group ((5.47±0.185). This is an indicative to low protein diet.

The balance of this electrolyte sodium in the blood in an indicatives of how well the heart and kidneys are functioning. Sodium is associated with blood pressure. A reduction in the concentration of sodium intake lowers the blood pressure. In this study, sodium increased significantly ( $p<0.05$ ) in group B (133.35±3.050<sup>b</sup>), group C (138.75±5.050<sup>b</sup>) and group D (141.85±1.250<sup>b</sup>) exposed to kerosene contaminated diets compared to the control group (128.90±4.800). This increase in sodium level indicates hypernatremia which involves dehydration and can have many

causes, including not drinking enough fluids, diarrhea, kidney dysfunction, and diuretics.

Potassium, which is an electrolyte in the intracellular fluid, is reported to be one of the protective electrolytes against hypertension (Nurminen *et al*, 1998). In this study, potassium increased significantly ( $p < 0.05$ ) in group B ( $10.32 \pm 0.825^b$ ), group C ( $9.28 \pm 0.310^b$ ) and group D ( $10.94 \pm 0.665^b$ ) exposed to kerosene contaminated diets compared to the control group ( $6.94 \pm 0.110$ ). This is in agreement with Uhegbu *et al*, (2015) who said very high concentrations of potassium may be as a result of kidney disease or substances that can decrease potassium excretion from the body. Hyperkalemia (elevated potassium levels) are usually associated with kidney failure, adrenal insufficiency or dehydration shock.

Chlorides are important electrolyte in the maintenance of the anion/cation balance between the extra-cellular and intra-cellular fluids. Chloride is essential to the control of osmotic pressure, proper hydration and acid/base equilibrium. In this study, chloride increased significantly ( $p < 0.05$ ) in group C ( $27.49 \pm 2.805^b$ ) administered with 2ml of kerosene contaminated diet compared to the control group ( $25.78 \pm 2.140$ ). Elevated serum chloride concentrations may be seen in conditions of urinary obstruction, dehydration and congestive heart valve (Tietz, 2000). It is also an indicative of renal impairment. Whereas the level of chloride in group B ( $24.91 \pm 2.460^a$ ) and group D ( $25.73 \pm 3.630^a$ ) decrease compare to the control group ( $25.78 \pm 2.140$ ). This is an indicative of problems such as vomiting and dehydration, congestive heart failure, hyperaldosteronism etc

In conclusion, the results generated from this study showed that the ingestion of kerosene contaminated diets into the body of animals is dangerous, having the potential of causing oxidative stress and histological damage in vital organs that are detrimental to the health of the animal.

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