



## COMPARISON OF TOXICITIES OF SAMPLES OF CRUDE OIL FROM DIFFERENT SOURCES IN NIGERIA

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### ABSTRACT

The use of crude oil in the treatment of diseases is gaining popularity in several parts of Nigeria. The impact of handling and storage on the toxicity profile of the Nigeria Crude oil has not been determined. A few hepatic and renal parameters were used in this study to assess the toxicity of crude oil samples found in different parts of Nigeria. Three different crude oil samples from Onitsha market, Lagos markets and NNPC refinery Port Harcourt were administered orally for 30 days at three dose levels (750, 1500 and 3000 mg/kg) while the control was given water (10 ml/kg). There was a rise in serum BUN of 1500 mg/kg and 3000 mg/kg for both NNPC (Groups C & D) and Lagos (Groups I & J) samples while for Onitsha, a significant rise was observed only in the 3000 mg/kg dose group (Group G). Similarly, a significant percentage rise in creatinine was observed in 1500 mg/kg and 3000 mg/kg doses for both NNPC (Groups C & D) and Lagos (Groups I & J) samples, while all doses of crude oil from Onitsha showed no significant increase. Furthermore, all doses of the three samples of crude oil showed a significant percentage increase in serum levels of AST, whereas only the 1500 mg/kg dose of crude oil from NNPC showed a significant percentage rise in the serum levels of ALT. The results of the histological studies for the liver and kidney tissues demonstrated more abnormality in NNPC and Lagos samples than in the Onitsha samples. Conclusively, crude oil from the NNPC refinery and Lagos market was more toxic than the samples from the Onitsha market. Factors likely to contribute to differences in toxicity profiles could be exposure, environmental conditions, and time.

**Keywords:** Crude oil, Hepatic function. Medicinal values, Nigerian refineries, Renal function. Toxicity

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## INTRODUCTION

Unarguably, crude oil has been established to be toxic to various organs of both human and animal body (Orisakwe et al, 2004, Best Ordinioha *et al*, 2013). Unfortunately, it is used in several states in Nigeria as medicine against different diseases (Orisakwe, 2000). The impact of handling and storage on the toxicity profile of the Nigeria Crude oil has not been determined.

According to the American Petroleum Institute [API], crude oil contains paraffinic (straight and branched-chain alkanes), naphthenic (cycloalkanes or cycloparaffins), and aromatic hydrocarbons (Almimar *et al*, 2015). Crude oil is classified based on density, as light, medium, heavy, or extra heavy. It can also be classified, based on sulfur content, into a sour and sweet category. Its density classification would tell if the crude oil would float on water or sink. Light crude oils are essentially liquid and comparatively contain components more volatile and hence can flow with ease (Dahhan *et al*, 2019).

Major and minor classes of crude oil emanate from various export terminals in Nigeria. Bonny light was so named because its export terminal is in the city of Bonny in Rivers State, South-South Nigeria. An evaluation of physical and chemical characterization of Nigerian light crude oils obtained from Warri Refinery and Petrochemical Company (WRPC) revealed that the crude oil sample obtained from WRPC contains low level of sulphur and such samples were of light crude oil category (Adetoro *et al.*, 2015). Since crude oil is a complex combination of different sorts of hydrocarbons, the exact contents of a given crude oil sample may be affected by storage or handling conditions.

Crude oil is considered medicinal by some folks and is used and sold in Nigeria as such. Previous studies have reported that crude oil is being ingested by some persons for claim of its anti-poison, anti-convulsive anti-witchcraft, antibiotic, and anti-inflammatory effects (Udoele 1997; Dede *et al.*, 2002; Orisakwe et al.,2004; Dienye *et al.*, 2012).

The scientific literature is replete with reports of toxic effects of crude oil. It poses significant deleterious effect on the hormonal system (Otitoju *et al*, 2007, Ebokaiwe et al, 2015). Reported symptoms associated with crude oil production and oil spills Niger Delta Nigeria to include headache, sore eyes, sore throat, respiratory problems, itchy skin, rashes on face and neck, sneezing, coughing or congested nose without a cold, nausea, dizziness, chest pain and diarrhea (Campbel et al., 1993; Suarez et al, 2005). Animal studies show that Bonny Light Crude Oil (BLCO) may be hemotoxic and hepatotoxic and could cause infertility and cancer (Ordinioha and Brisibe, 2013).

The Nigerian BLCO which is classified as light has an appreciable quantity of aromatic hydrocarbons. Crude oil can be classified as light or heavy. It is classified as light when it has low carbon to hydrogen content. (Dahhan et al, 2019). Temperature, humidity, exposure to sunlight and atmosphere can affect this oil. Consequently, it makes sense to think that the crude oil sample found in homes and motor parks in different parts of Nigeria may not have exact constituents or proportion of constituents.

Hence this study compared the toxicity of these samples. In this study, we used some hepatic and renal parameters to assess the toxicity of crude oil samples found in different parts of Nigeria.

## MATERIALS AND METHODS

### EXPERIMENTAL ANIMALS

Wister rats of both sexes were obtained from the colony breed of the Animal House of the Faculty of Pharmaceutical Sciences, Nnamdi Azikiwe University, Agulu, Anambra State. All animal experiments were conducted in compliance with the National Institute of Health Guide for care and use of laboratory animals (Pub. No 85-23, Revised 1985).

### CRUDE OIL PROCUREMENT

Bonny light crude oil (BLCO) was obtained from the Department of Petroleum Resources (DPR), Nigerian National Petroleum Corporation (NNPC), Port Harcourt, Nigeria, while the commercial crude oil was obtained from two different sources within Nigeria (Ose market Onitsha and Mile12 market Lagos).

### ACUTE TOXICITY TEST

The LD<sub>50</sub> was determined using the method described by Lorke (1983) which was conducted in two phases.

**Phase 1:** A total of nine rats were divided into three groups of 3 rats each. Groups 1, 2 and 3, received 10, 100, and 1000 mg/kg crude oil orally, respectively. The animals were observed continuously for one hour, and intermittently for the next three hours and then after 24 hours for behavioural changes and mortality

**Phase 2:** A total of four animals were divided into four groups of one rat each. Groups 1, 2, 3, and 4 received 2000, 3000, 4000, and 5000 mg/kg crude oil orally, respectively.

### SUB-ACUTE TOXICITY TEST

#### EXPERIMENTAL DESIGN

The animals were bled by ocular puncture and blood samples were collected for baseline. Thereafter, animals were grouped into ten groups of five rats each. The animals received the BLCO for 30 days.

Group	Treatment	Doses
A (control)	Water	0mg/kg
B	NNPC	750mg/kg
C	NNPC	1500mg/kg
D	NNPC	3000mg/kg
E	Onitsha	750mg/kg
F	Onitsha	1500mg/kg
G	Onitsha	3000mg/kg
H	Lagos	750mg/kg
I	Lagos	1500m/kg
J	Lagos	3000mg/kg

## **SAMPLE COLLECTION**

The rats were sacrificed 24hrs after the last dose of treatment. They were anaesthetized using chloroform and blood samples were collected from the jugular vein and were allowed to clot. Thereafter, the rat was dissected, and the organs isolated. The blood was spun using a centrifuge at 4000 rpm for 10 minutes and the serum was collected using a Pasteur pipette. The serum was stored in the refrigerator until it was used for biochemical analysis.

## **BIOCHEMICAL ANALYSIS**

Biochemical analysis was done for urea and creatinine using Tobacco (1979) and Heinegard and Tiderstrom (1973) methods respectively. Test for alanine aminotransferase (ALT) and aspartate aminotransferase (AST) was by the method of Reitman and Frankel (1957).

## **HISTOLOGICAL STUDIES**

The heart, liver and kidney were fixed immediately in 10% formalin and embedded in paraffin wax and were processed using the method described by Luna (1958).

## **METHOD OF DATA ANALYSIS**

Results gathered from the study were presented as mean  $\pm$  standard error of the mean (SEM). For tables 1&4, the values of the test samples obtained were corrected relative to the control by subtracting the control from them. Statistical package for social science (SPSS-20, for windows) was the software used for data analyses.

## **RESULTS**

### **Acute toxicity:**

There was no observed sign of toxicity or death in both phases of the LD<sub>50</sub> test.

### **Effects of crude oil on blood urea nitrogen (BUN):**

From table 1, there is no significant difference in the percentage rise (when compared to control) in serum BUN before and after treatment for groups B, E, F, and H. Of these groups listed, all were 750 mg/kg doses except F (Ose, Onitsha) which is 1500 mg/kg dose. On the other hand, all 1500 doses (except F {Ose, Onitsha}) and 3000 mg/kg doses showed a significant percentage rise in their respective after-treatment values. This result showed a significant percentage rise in serum BUN of 1500 mg/kg and 3000 mg/kg for NNPC (Groups C & D) and Lagos (Groups I & J) samples. A significant rise for Onitsha is for only the 3000 mg/kg dose group (Group G).

### **Effects of crude oil on creatinine:**

From table 2, there is no significant difference in the percentage rise (when compared to control) in serum creatinine before and after treatment for groups B, E, F, G, and H. Of these groups listed, all were 750 mg/kg doses and all the doses from Ose, Onitsha (E, F, G). On the other hand, all 1500 doses (except F {Ose, Onitsha}) and 3000 mg/kg doses showed a significant percentage rise in their respective after-treatment values. Also, a significant percentage rise in creatinine was observed in 1500mg/kg and 3000mg/kg doses for the NNPC (Groups C & D) and Lagos (Groups I & J) samples.

### **Effects of crude oil on serum alanine aminotransferase (ALT):**

From table 3, only the 1500 mg/kg dose of crude oil from NNPC showed a significant percentage increase in the serum levels of ALT in the after-treatment group compared to standard. On the other hand, other doses of BLCO from other locations showed no significant difference in the after-treatment groups, compared to the standard.

### **Effects of Crude Oil on Serum Aspartate Aminotransferase (AST):**

From table 4, all the test samples showed a significant percentage increase in the serum levels of AST in the after-treatment groups compared to standard.

### **Effects of Crude Oil on Histology of Liver and Kidney**

The results for the histological studies for the liver tissue for the Onitsha sample showed a dose dependent deterioration from mild inflammation (750 mg/kg) to mild onset of cirrhosis (1500 mg/kg) to expansion of sinusoidal space by edema fluid (3000 mg/kg). The NNPC samples at 750 mg/kg started from mild necrosis to steatosis (1500 mg/kg) to edema (3000 mg/kg). The Lagos samples at 750 mg/kg showed architectural distortion and hydropic cells, then edema with mild necrosis and lymphatic infiltration at 1500 mg/kg and necrosis and hepatocyte degeneration at 3000 mg/kg.

The results for the histological studies for the kidney tissue for the Onitsha sample showed a normal glomerulus and tubules (750 mg/kg), lymphatic infiltration, congested glomeruli (1500 mg/kg), and edema (3000 mg/kg). The NNPC samples at 750 mg/kg started from normal glomerulus and tubules (750 mg/kg), architectural distortion and mild edema (1500 mg/kg) and interstitial inflammation and necrosis (3000 mg/kg). The Lagos samples started with mild necrosis (750 mg/kg), cell degeneration and nephritis (1500 mg/kg) and ended with congested tubules with fibrosis (3000 mg/kg).

## **DISCUSSION**

This study compared the toxicity of different samples of Bonny Light Crude oil on hepatic and renal function parameters in Wistar rats. Acute toxicity test revealed no death and toxic signs at 5000 mg/kg suggesting that crude oil samples from the three locations may not kill within 24 hours (Erhirhie et al, 2018).

From the study, the observed elevation in plasma or serum urea and creatinine levels is an indication of kidney damage (Adamu *et al.*, 2016). There was no significant difference between the control and Onitsha samples, suggesting Onitsha samples were safe. The serum BUN and creatinine data suggest Onitsha samples may be safer than NNPC and Lagos samples. These abnormal changes are indications of considerable renal damage due to hydrocarbon content in crude oil samples (Odinga et al., 2015). Only 1500 mg/kg dose of crude oil from NNPC increased the serum ALT.

An elevation in plasma or serum liver biomarkers, ALT and AST beyond the normal range is an indication of liver damage (Debelo *et al.*, 2016). Although ALT is liver-specific (cytosolic), AST is in several tissues such as skeletal

muscle, heart, and brain tissues (Debelo *et al.*, 2016). Findings from this study revealed a significant percentage rise in serum ALT levels of animals that received a 1500 mg/kg dose of crude oil from NNPC when compared to the control, although it was not dose dependent. On the other hand, there were no changes in serum ALT levels in crude oils sampled from Lagos and Onitsha. As for AST, there was a significant percentage rise in serum levels of AST in all the doses of crude oil from the three locations, when compared to the control, suggesting liver damage. Histology of the liver architecture of animals treated with various doses of crude oil revealed necrosis, steatosis, periportal inflammation, and edema.

This was substantiated by the histopathology result which showed necrosis, steatosis, edema, lymphatic infiltration, and inflammation in the liver architecture of animals treated with various doses of crude oil. This is a confirmatory indication of liver damage. In line with this study, liver damage characterized by predominant histopathologic lesions was reported following exposure to crude oil spilt from the oil tanker Exxon Valdez (Lipscomb *et al.*, 1993). Dose-dependent elevation in serum ALT and AST as well as severe pathologic changes in the forms of necrosis and oedema was also reported following exposure of animals to crude oil (Orisakwe *et al.*, 2005). Studies by Odinga *et al.*, (2017), Ngokere *et al.*, (2014) and Gashev *et al.*, (2012) revealed that crude oil produced liver damage observed.

Generally, crude oil from Lagos and NNPC refineries produces more obvious toxicity than crude oil from Onitsha. It is possible that hawkers of the so-called medicinal crude oil could influence its content. They could expose or sun the oil or perhaps add other ingredients, capable of mitigating its toxicity. The observed changes in hepatic and renal function would be due to the various constituents of crude oil. Crude oil contains polyaromatic hydrocarbons that can exert a toxic effect on a biological system. Constituents such as paraffin, naphthalenes, aromatic hydrocarbons, several nitrogen, oxygen, and sulphur-containing compounds as well as other inorganic and organometallic compounds in crude oil have been established to be toxic to various organs (Edward, 2003; Igwe *et al.*, 2016). Harmful metallic ions were found to be present in the water-soluble fraction of crude oil (Noyo *et al.*, 2007).

Oxidative stress due to free radicals' generation from crude oil earlier reported by Achuba and Osakwe (2003), could also be a possible mechanism of crude oil toxicity observed in this study. It seems the Onitsha sample is the least toxic of the three samples of crude oil. Like earlier suggested, the exact contents of the 3 samples may not be the same since there are slight differences in their respective toxicity profiles. Factors likely to contribute could be exposure, environmental conditions, and time. Exposure of the samples to air will cause a reduction in volatile contents of the oils. Exposure to sunlight will result in degradation of the contents. Environmental conditions of temperature and humidity would also affect them. It is also plausible that the longer the exposure to these environmental conditions, the wider their variations.

## CONCLUSION

Crude oil from the three sources caused a significant percentage rise in serum levels of renal and hepatic biomarkers in Wistar rats. Histological studies of the organs revealed pathological changes and distortion of the integrity of the liver and kidney. Crude oil from the NNPC refinery and Lagos market was more toxic than the sample from the Onitsha market. Factors likely to contribute to the difference in toxicity profiles could be exposure, environmental conditions and time.

## CONFLICT OF INTEREST

Authors declare that no conflict of interest exists among them

## ACKNOWLEDGEMENT

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**Table 1:** Effects of Crude Oil from Three Locations (NNPC refinery, Onitsha and Lagos) on Serum Blood Urea Nitrogen (BUN) Level of Wistar Albino Rats (BUN (mmol/L))

Treatment	Doses mg/kg	Standard	After treatment	Difference	% Difference	% Difference (corrected)	% Rise in Serum BUN	P Value
<b>GROUP A</b> (control)	0	3.94	4.28	-0.43	-10.9	0.0	0.0	0.581
<b>GROUP B</b>	750	4.2	6.20	-2	-47.6	-36.7	36.7	0.100
<b>GROUP C</b>	1500	4.38	8.38*	-4	-91.3	-80.4	80.4	0.004
<b>GROUP D</b>	3000	3.9	7.76*	-3.86	-99.0	-88.1	88.1	0.013
<b>GROUP E</b>	750	3.78	4.80	-1.02	-27.0	-16.1	16.1	0.092
<b>GROUP F</b>	1500	4.02	5.10	-1.08	-26.9	-16.0	16.0	0.152
<b>GROUP G</b>	3000	4.14	5.74*	-1.6	-38.6	-27.7	27.7	0.026
<b>GROUP H</b>	750	4.18	6.88	-2.7	-64.6	-53.7	53.7	0.100
<b>GROUP I</b>	1500	3.84	9.38*	-5.54	-144.3	-133.4	133.4	0.004
<b>GROUP J</b>	3000	4.34	10.52*	-6.18	-142.4	-131.5	131.5	0.013

GROUP A: 0 mg/kg (control)

GROUP B: 750 mg/kg of crude oil from NNPC

GROUP C: 1500 mg/kg of crude oil from NNPC

GROUP D: 3000 mg/kg of crude oil from NNPC

GROUP E: 750 mg/kg of crude oil from Onitsha

GROUP F: 1500 mg/kg of crude oil from Onitsha

GROUP G: 3000 mg/kg of crude oil from Onitsha

GROUP H: 750 mg/kg of crude oil from Lagos

GROUP I: 1500 mg/kg of crude oil from Lagos

GROUP J: 3000 mg/kg of crude oil from Lagos

**Table 2:** Effects of Crude Oil from Three Locations (NNPC refinery, Onitsha and Lagos) on Serum Creatinine

Level of Wistar Albino Rats.

Creatinine (mmol/L)								
Treatment	Doses mg/kg	Standard	After treatment	Difference	% Difference	% Difference (corrected)	% Rise in Serum Creatinine	P Value
<b>GROUP A</b>	0	60.6	63.8	-3.2	-5.3	0	0	0.32
<b>GROUP B</b>	750	62.2	71.2	-9	-14.5	-9.2	9.2	0.235
<b>GROUP C</b>	1500	58.4	62.8*	-4.4	-7.5	-2.2	2.2	0.022
<b>GROUP D</b>	3000	59	77.6*	-18.6	-31.5	-26.2	26.2	0.039
<b>GROUP E</b>	750	60	63.6	-3.6	-6	-0.7	0.7	0.178
<b>GROUP F</b>	1500	62.8	68.6	-5.8	-9.2	-3.9	3.9	0.061
<b>GROUP G</b>	3000	64.2	70.4	-6.2	-9.7	-4.4	4.4	0.75
<b>GROUP H</b>	750	59.4	61.2	-1.8	-3	2.3	-2.3	0.657
<b>GROUP I</b>	1500	62	91.8*	-29.8	-48.1	-42.8	42.8	0
<b>GROUP J</b>	3000	61.2	95.0*	-33.8	-55.2	-49.9	49.9	0.004

GROUP A: 0 mg/kg (control)

GROUP B: 750 mg/kg of crude oil from NNPC

GROUP C: 1500 mg/kg of crude oil from NNPC

GROUP D: 3000 mg/kg of crude oil from NNPC

GROUP E: 750 mg/kg of crude oil from Onitsha

GROUP F: 1500 mg/kg of crude oil from Onitsha

GROUP G: 3000 mg/kg of crude oil from Onitsha

GROUP H: 750 mg/kg of crude oil from Lagos

GROUP I: 1500 mg/kg of crude oil from Lagos

GROUP J: 3000 mg/kg of crude oil from Lagos



**Table 3:** Effects of Crude Oil from Three Locations (NNPC refinery, Onitsha and Lagos) on Serum Alanine Aminotransferase (ALT) level of Wistar Albino Rats ALT (mmol/L)

Treatment	Doses mg/kg	Standard	After treatment	Difference	% Difference	% Difference (corrected)	% Rise in Serum ALT	P Value
<b>GROUP A</b>	0	27.32	26.54	-0.78	-2.9	0	0	0.434
<b>GROUP B</b>	750	26.36	26.92	-2.18	-8.3	-5.4	5.4	0.283
<b>GROUP C</b>	1500	28.02	31.32*	-3.48	-12.4	-9.5	9.5	0.034
<b>GROUP D</b>	3000	27.4	31.52	-2.3	-8.4	-5.5	5.5	0.201
<b>GROUP E</b>	750	26.7	27.5	-0.8	-3	-0.1	0.1	0.145
<b>GROUP F</b>	1500	28.36	27.64	-0.72	-2.5	0.4	-0.4	0.514
<b>GROUP G</b>	3000	27.32	30.06	-2.74	-10	-7.1	7.1	0.192
<b>GROUP H</b>	750	27.1	29.28	-0.56	-2.1	0.8	-0.8	0.075
<b>GROUP I</b>	1500	26.24	29.56	-3.3	-12.6	-9.7	9.7	0.11
<b>GROUP J</b>	3000	27.78	30.1	-4.12	-14.8	-11.9	11.9	0.106

GROUP A: 0 mg/kg (control)  
 GROUP B: 750 mg/kg of crude oil from NNPC  
 GROUP C: 1500 mg/kg of crude oil from NNPC  
 GROUP D: 3000 mg/kg of crude oil from NNPC  
 GROUP E: 750 mg/kg of crude oil from Onitsha  
 GROUP F: 1500 mg/kg of crude oil from Onitsha  
 GROUP G: 3000 mg/kg of crude oil from Onitsha  
 GROUP H: 750 mg/kg of crude oil from Lagos  
 GROUP I: 1500 mg/kg of crude oil from Lagos  
 GROUP J: 3000 mg/kg of crude oil from Lagos

**Table 4:** Effects of Crude Oil from Three Locations (NNPC refinery, Onitsha, and Lagos) on Serum Aspartate Aminotransferase (AST) Level of Wistar Albino Rats (AST (mmol/L))

Treatment	Doses mg/kg	Standard	After treatment	Difference	% Difference	% Difference (corrected)	% Rise in Serum AST	P Value
<b>GROUP A</b>	0	69.56	72.74	-3.18	-4.6	0	0	0.109
<b>GROUP B</b>	750	70.36	92.66*	-22.1	-31.4	-26.8	26.8	0
<b>GROUP C</b>	1500	69.92	92.50*	-27.46	-39.3	-34.7	34.7	0
<b>GROUP D</b>	3000	72.14	94.56*	-34.16	-47.4	-42.8	42.8	0.001
<b>GROUP E</b>	750	70.52	91.48*	-20.96	-29.7	-25.1	25.1	0
<b>GROUP F</b>	1500	68.54	91.08*	-22.54	-32.9	-28.3	28.3	0
<b>GROUP G</b>	3000	69.84	93.12*	-23.28	-33.3	-28.7	28.7	0
<b>GROUP H</b>	750	70.52	92.62*	-22.3	-31.6	-27	27	0.001
<b>GROUP I</b>	1500	67.62	95.08*	-22.58	-33.4	-28.8	28.8	0
<b>GROUP J</b>	3000	69.32	103.48*	-22.42	-32.3	-27.7	27.7	0

GROUP A: 0 mg/kg (control)

GROUP B: 750 mg/kg of crude oil from NNPC

GROUP C: 1500 mg/kg of crude oil from NNPC

GROUP D: 3000 mg/kg of crude oil from NNPC

GROUP E: 750 mg/kg of crude oil from Onitsha

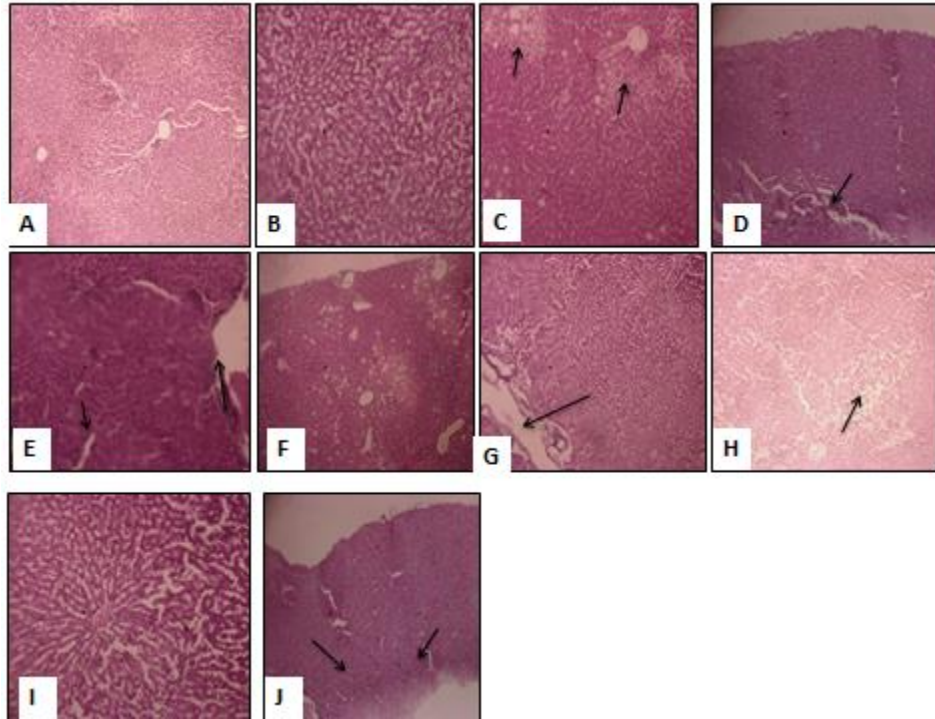
GROUP F: 1500 mg/kg of crude oil from Onitsha

GROUP G: 3000 mg/kg of crude oil from Onitsha

GROUP H: 750 mg/kg of crude oil from Lagos

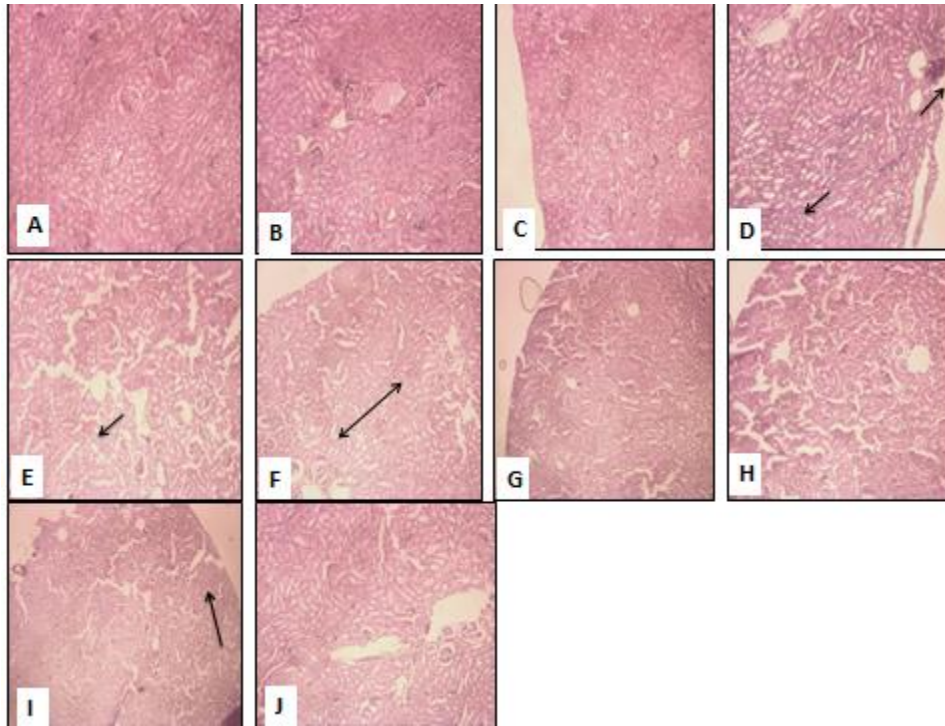
GROUP I: 1500 mg/kg of crude oil from Lagos

GROUP J: 3000 mg/kg of crude oil from Lagos



**Figure 1:** Photomicrograph of liver tissue. Stained by H&E 200x.

**Group A** (normal architecture of the liver), **Group B** (750 mg/kg Onitsha crude oil, showing mild inflammation), **Group C** (750 mg/kg NNPC crude oil, showing mild necrosis), **Group D** (750 mg/kg Lagos crude oil, showing architectural distortion and hydropic cells (arrowhead)), **Group E** (1500 mg/kg Onitsha crude oil, showing the mild onset of cirrhosis), **Group F** (1500 mg/kg NNPC crude oil, showing steatosis), **Group G** (1500 mg/kg Lagos crude oil, showing edema with mild necrosis (arrow), lymphatic infiltration (arrowheads), **Group H** (3000 mg/kg Onitsha crude oil, showing the expansion of sinusoidal space by edema fluid), **Group I** (3000 mg/kg NNPC crude oil, showing edema), **Group J** (3000 mg/kg Lagos crude oil, showing necrosis and hepatocyte degeneration).



**Figure 2:** Photomicrograph of kidney tissue. Stained by H&E 200x.

**Group A** (Photomicrograph of kidney tissue in control showing with normal glomeruli and tubules), **Group B** (Photomicrograph of kidney tissue of rats administered with 750 mg/kg Onitsha crude oil, showing normal glomerulus and tubules), **Group C** (Photomicrograph of kidney tissue of rats administered with 750 mg/kg NNPC crude oil, showing with normal glomerulus and tubules), **Group D** (Photomicrograph of kidney tissues of rats administered with 750 mg/kg Lagos crude oil, showing mild necrosis), **Group E** (Photomicrograph of kidney tissue of rats administered with 1500 mg/kg Onitsha crude oil, showing lymphatic infiltration, congested glomeruli), **Group F** (Photomicrograph of kidney tissue of rats administered with 1500 mg/kg NNPC crude oil, showing architectural distortion and mild edema), **Group G** (Photomicrograph of kidney tissue of rats administered with 1500 mg/kg Lagos crude oil, showing cell degeneration and nephritis), **Group H** (Photomicrograph of kidney tissue of rats administered with 3000 mg/kg Onitsha crude oil, showing edema), **Group I** (Photomicrograph of kidney tissue of rats administered with 3000 mg/kg NNPC crude oil, showing interstitial inflammation and necrosis (arrowhead)), **Group J** (Photomicrograph of rats kidney tissue administered with 3000 mg/kg Lagos crude oil, showing congested tubules with fibrosis).

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