

## Histopathological Changes in Rat Organ following Ingestion of Crude Oil Contaminated Diet

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### Abstract

The possible effects which crude oil polluted foods may have on specific organs of mammals like the kidney, liver, small intestine, spleen and heart were investigated using white male wistar rats. Four formulated diets (including a control) depicting different levels of crude oil contaminated cassava/fish meal diets were used as experimental meal in an animal and bioassay work. The result revealed a considerable negative effect that crude oil had on the organs of the experimental animals as against that of control. Histopathological investigation showed the level of damage done to the organs which ranged from shrunken villi, oedema in the small intestine to the glomeruli and tubules in the kidney.

**Key words:** Histopathology, crude oil, Hydrocarbon, contamination

### Introduction

Concern over soil contamination by crude oil or hydrocarbon products is high. This has been shown to trickle down to affect both water and food for human and animal consumption. Similarities exist between inland and offshore crude oil spills; these similarities include hazards to life in all its forms, contamination of valuable fresh water resources from aquifers or desalination plants and an uncertain long term environmental impact (Benyahia *et al.*, 2005). The food and water that humans consume are thus subjected to multiple pathways of toxic exposure. The exposure ranges from natural sources to man-made sources. It directly or indirectly contaminates or pollutes our sustenance. Our sustenance can be subjected to acute exposure i.e. heavy contamination substance ingested in a day or two, or chronic exposure i.e. consumption of contaminated substance over a long period of time. Some of the toxic substances ingested are neutralized by the body's detoxification mechanism that involves the liver detoxification systems. Some of the toxins may pass out from the body undetoxified and some may remain in the body. Mercury and lead are among the most notable contaminants, which accumulate and cause degeneration of the cells of human body including brain (Botkin *et al.* 1998). Histopathological studies yield basic information on tissue disorders related to the general state of organisms, and assess the host's susceptibility to infectious diseases and parasitic infestation. Some of these parameters may serve as indicators of the effects of xenobiotic contamination in marine animals (Sindermann 1980).

## Materials and Methods

Four formulated diets (including a control) depicting different levels of crude oil contaminated cassava/fish meal diet obtained from a village in the Niger Delta were used as experimental meal in an animal and bioassay work to investigate the effects of crude oil contamination as earlier described by (Ologunde *et al.*, 1991). Animals were killed by decapitation following which internal organs were sectioned, stained with haematoxylin and eosin for light microscopic examination and an autopsy test.

**Table 1: Diet formulation**

| Diet/Formulation           | Diet I | Diet II | Diet III | Diet IV |
|----------------------------|--------|---------|----------|---------|
| Processed Cassava (kg)*    | 2.56   | -       | -        | -       |
| Processed Cassava (kg)**   | -      | 2.56    | -        | -       |
| Processed Cassava (kg)***  | -      | -       | 2.56     | -       |
| Processed Cassava (kg)**** | -      | -       | -        | 2.56    |
| Fish (kg)                  | 0.8    | 0.8     | 0.8      | 0.8     |
| Groundnut Oil (kg)         | 0.56   | 0.56    | 0.56     | 0.56    |
| Vitamin Premix (g)         | 52     | 52      | 52       | 52      |
| Mineral Premix (g)         | 28     | 28      | 28       | 28      |

\*Gari (Control)

\*\*Gari (kokori)

\*\*\*Gari (Ogulagha)

\*\*\*\*Gari (SPDC)

## Results

Results obtained upon histopathological investigation carried out on the organs of the experimental (diet II, III, IV) and control rats (diet I) are presented in Table 2. The livers of the control animals (diet I) were essentially normal, showing prominent vessel, healthy cells and normal circulation with no pooling of blood. The liver in diets II, III, and IV, were relatively few, if any viable cells left. Hence, the inflammatory response seemed not as localized as that of diet III.

The kidneys of the control animals (diet I) showed cells that were essentially normal with the tubules and glomeruli empty. Mild degenerative changes were noticed in kidneys of animals fed with diet II, with nuclear degeneration in a few cells. In diet III, there was marked pooling of blood with a lot of inflammatory cells and generation of tubules. In diet IV, the degenerative changes were more pronounced and wide spread inflammatory cells were noticeable.

Similar degenerative changes were observed in the intestine. The villi, which are the absorptive surface of the small intestine showed inflamed mucosa and rising out of controlled absorption of toxic chemical (diet II-IV). Epithelia layers of the villi were distended and in some cases, showed lyses and other abnormal signs indicative of toxic chemical consumption. These features were not evident in control animal (diet I).

**Table 2: Histopathological changes in internal organs of animal**

| DIET | ORGAN           | OBSERVED CHANGES  |
|------|-----------------|---|
| I    | Small Intestine | Mild Congestion, Normal Villi   |
|      | Kidney          | Mild congestion occasionally with the venous stasis and thrombosis, come swelling in some cells.  |
|      | Liver           | Normal, prominent vessels, healthy cells and normal circulation, no pooling of blood  |
| II   | Small Intestine | Shrunk Villi, slight mucosal hypertrophy oedema cells.  |
|      | Kidney          | Capillary venous congestion, Lysis of erythrocytes cloudy swelling and cellular degeneration of proximal and distal tubules, cytoplasmic inclusions, lower incidence of renal tubular changes |
|      | Liver           | Swelling of individual hepatic cells, picnosis of nuclei. Darkening staining nuclei and dense chromatin bodies which are indicative of hyperplastic rectum                                    |
| III  | Small Intestine | Marked capillary venous congestion Mucosal hypertrophy with shrunk villi. Damaged epithelial surface with fragmented nuclei in some areas.  |
|      | Kidney          | Oedema of glomeruli and tubules. Areas of tubular necrosis. Tubular debris in glomerular space  |
|      | Liver           | Mild central lobular vacuolation. Picnosis of Kupffer cells. Area of sinusoidal congestion, area of cloudy swelling   |
| IV   | Small intestine | Capillary venous congestion, shrunk villi, oedema, mucosal hypertrophy.   |
|      | Kidney          | Fatty degeneration of tubules, Capillary congestion with haemorrhage in the region of Henle's loop especially about the descending loop.  |

## Discussion

Histopathological changes in the present study may be due to the varying levels of contaminants present in the diet of the rats. Earlier studies have also identified degenerative changes in the cells of somniferous tubular germinal layers. (Arcana N.2007; Pant *et al.* 1995, 1996; Auffret 1988 and Gills *et al* 1999) found that tissue abnormalities were related to chemical contamination. The quantity of food intake is also reflected in the histology of the experimental animals as shown in Table 2. The implication of this observation is that, the diets being fed to the experimental animals contained crude oil contaminants in different proportions that were capable of causing damage to the organ at different levels. The duration of consumption had appreciable effect on the size of the organs and the lipid content of the organs and hence, the degenerative changes observed in the organs (Birch *et al.*, 1986).

## Conclusion

Pollution of the environment had detrimental effects on the organs through contaminated food that was consumed. The contaminants present in crude oil such as the heavy metals and hydrocarbons are capable of damaging the organs if ingested. Histopathological investigation revealed that the extent of damage done to the organ is dependent on the quality of contaminants in diet. Diet IV which had more of the contaminants caused most of the damage followed by diet II, while diet III caused the least damage.

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