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**Effects of Maize Cob (*Zea May*) on Nickel Induced Liver Toxicity in Female Wistar Rat**

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**Abstract**
Corncobs are increasingly being utilized in food production, ground into flour for bread-making and as a stabilizer in edible oils, enhancing their acceptance in global markets. This study aims to assess the impact of corncob ash on liver function in Wistar rats subjected to nickel-induced liver toxicity. Six groups of five Wistar rats each were organized into control and test groups: Nickel only, Nickel + 300 mg/kg Corncob Ash, Nickel + 450 mg/kg Corncob Ash, Nickel + 600 mg/kg Corncob Ash, 450 mg/kg Corncob Ash only, and a control group with no treatment. The treatments were administered orally for 28 days, after which the animals were sacrificed, and blood samples collected. Liver function markers—Aspartate transaminase (AST), Alanine transaminase (ALT), Alkaline phosphatase (ALP), Total and Conjugated Bilirubin, Total Protein, and Albumin—were analyzed using Reitman-Frankel, Phenolphthalein Monophosphate, Jendrassik-Grof, Biuret, and Bromocresol green methods, respectively. Data analysis was conducted using SPSS version 25. Significant differences ($P < 0.05$) were observed in AST activity (U/L) across the groups, with values of 32.00±3.46 (control), 49.33±2.60 (Nickel), 58.66±4.40 (Nickel + 300 mg/kg), 40.66±1.45 (Nickel + 450 mg/kg), 35.33±1.45 (Nickel + 600 mg/kg), and 33.33±0.88 (450 mg/kg Corncob Ash). ALT activity (U/L) also showed significant variation, with values of 13.00±1.15, 20.00±1.15, 18.10±0.49, 23.33±0.88, 17.00±0.58, and 17.00±0.58 across respective groups. Total Protein (g/L) levels of 71.00±0.58, 70.00±1.15, 63.33±1.76, 65.00±1.15, 70.00±1.15, and 74.33±1.20 and Albumin (g/L) of 44.66±0.88, 44.00±1.45, 41.00±0.58, 41.66±0.88, 45.00±1.15, and 46.00±1.52 were significantly different ($P < 0.05$). Total Bilirubin (μmol/L) was also significantly altered with values of 6.73±0.43, 8.46±0.14, 11.43±0.44, 8.00±0.36, 6.96±0.26, and 6.73±0.14, along with conjugated Bilirubin (μmol/L) at 4.23±0.26, 5.43±0.35, 7.46±0.43, 5.03±0.39, 4.93±0.42, and 4.16±0.20 across the groups. No significant difference ($P > 0.05$) was noted in ALP activities, with values of 54.00±0.57, 56.66±0.88, 2.17±0.48, 53.33±1.45, 53.33±0.88, and 54.00±1.15 across the control and treatment groups. These findings indicate that corncob ash may have a modulating effect on certain liver function parameters in nickel-induced toxicity, depending on the administered dose.

**Keywords:** Corncobs, Nickel, liver, toxicity

## Introduction
Corncobs are by-products of maize processing industries and households. They can constitute an environmental menace not only on the farms but on the streets and in homes if not properly disposed. Corncob contains hemicelluloses that upon splitting acid produce appreciable quantities of xylose and arabinose, with lesser quantities of glucose and galactose fermentable sugars. These are webbed up in mass of lignin and other polysaccharides, which gives it characteristic hardness and insolubility in water (Warren, 1996). The fact that corncobs are currently being used as ground flour to produce bread and as a stabilizer for edible oil increases its acceptance in world markets (Anwar *et al*.,

2006). The β (1–4)-D-xylan-type hemicelluloses are the second most abundant biopolymer, after cellulose, in annual plants and hardwoods. They represent an important renewable biopolymer resource whose potential has yet to be completely realized. All parts of corn plants are good sources of a variety of bioactive phytochemical compounds which possess antioxidant potential. Phytochemicals are the non-nutritional bioactive compounds found in various parts of plants. In plants these compounds perform vital functions particularly protection from predators and harsh environmental conditions. These compounds are also important in pharmaceutical and medicinal field due to their antioxidant, antimicrobial, and other biological properties. Flavonoids are bioactive phytochemical compounds which make the plant resistant to the attack of microbes and insects and also protect the animals against various diseases.

Waste waters holding heavy metals released by industries are discharged into aquatic systems where they bioaccumalate in food chain, their concentration increases and becomes lethal to living organisms (Royer *et al.,* 2006). Nickel and its compounds produce acute and chronic toxicity to aquatic life due to its persistence and bioaccumulation (Vieira *et al*., 2009). Nickel affects liver parameters, body weight and length. Its toxicological, physiological and histopathological alterations in rabbits, mice and minnows have been established (Bersenyi, et al., 2003, Pane *et al*., 2003; Doreswamy *et al.,* 2004; Hoang *et al.,* 2004, Gupta *et al.,* 2006). The effects of acute Nickel poisoning in humans are high blood pressure, kidney damage and destruction of testicular tissue and red blood cells. Nickel is famous as haematotoxic, immunotoxic, neurotoxic, genotoxic, regenerative harmful, aspiratory poisonous, nephrotoxic, hepatotoxic, and cancer-causing operator (Das *et al.,* 2008). Exposure to nickel produced physiological and biochemical disturbances in human and animals' body (Das et al 2018, Buxton *et al* 2019; Genchi *et al* 2020). Human exposure to nickel primarily occurs through inhalation (Das *et al* 2018; Genchi *et al* 2020). Nickel is absorbed through the blood and distributed in various vital organs and excreted mainly through urine. Husna et al. (2016) reported that AST, ALT and ALP levels increased remarkably in experimental fish compared to controls, while the levels of total proteins decreased significantly in all groups when exposed to Nickel.

The aim of this study was to investigate the hepatoprotective effects of maize cob (zea may) on nickel induced liver toxicity in female Wistar rat using aspartate transaminase (AST), Alanine transaminase(ALT), Alkaline phosphatase (ALP), Bilirubin (Total and conjugated ), Total protein and Albumin in the liver as indicator.

## Materials and Methods

### Animals

Thirty (30) female albino rats (*Rattus norvegicus*) of about two months old purchased and acclimatized in the animal house of Madonna University Elele campus Rivers State Nigeria. The initial body weights of the animals were recorded before acclimatization. The animals were divided into 6 groups of 5 rats each in a cage. The rats were housed in wired cages around in rows and kept in the animal house of Medical Laboratory Science in Madonna University. The cages were sanitized and the animal house cleaned every day. The animals were kept under ambient temperature of between 27-30°C under 12 hour day light cycle. The animals (rats) were fed standard Grower's marsh and millet feed with water. The rats gained maximum acclimatization for three weeks (21) days.

### Nickel and corn cob

Nickel and corn cob was obtained from Port Harcourt, River state.

### Preparation of Nickel solution

1g of nickel was dissolved in 100mls of deionized water to obtain a stock solution of 10mg/ml concentration. Animals were administered a dose of 0-5mg/kg body weight. The working solution was stored in an amber colored bottle at room temperature.

### Preparation of ashed corn cob stock Extract

The corn cob were dried and burnt into ashes, the powdered ash of the corn cob was weighed to obtain 90 g and macerated in 300ml of water for forty eight (48) hours. The solutions were filtered

with a cleaned doubled handkerchief and stored. The corn cob was harvested from a farm in Obio-Akpor Local Government, Port Harcourt. There were cleaned and air dried and openly incinerated. Ninety grams (90g) of the ash was collected into a beaker and 300ml of deionized water added to it. The mixture was allowed for 48 hours with constant agitation, triple filtered with Whatman filter paper to get a cleared filtrate. The filtrate yielded a stock solution of 300mg that was used for the study.

**Animal Sacrifice**

This study was carried out for 28 days, at the end of the treatment period, the animals were allowed an overnight fast and the body weight recorded. The animals were sacrificed, and the blood was collected through subclavian puncture under aseptic condition.

**Research Design**

Thirty female wistar rats divided into six groups of five rats each were used for the study. The control group was given 0.2mls of distilled water and rat diet and water *adlibitum*. The rats in group 2 was administered with 0.5mg/kg Nickel, while rats in groups 3,4 and 5 were administered with 0.5mg/kg Nickel with 300mg/kg, 450mg/kg and 600mg/kg of corn cob extracts respectively by oral intubation. The rats in group 6 were fed 450mg/kg corn cob extract. The treatments were done daily for 28 days. The ash of the corn cob extract was administered every morning followed by the nickel. On the 28th day, the rats were sacrificed. Blood was collected into plain tubes for liver function analysis.

**Biochemical Methods**

Determination of ALT and AST was done by monitoring the concentrations of pyruvate hydrazone formed with 2, 4 dinitrophenyl hydrazine. Five hundred microlitre (0.5ml) of buffer solution was dispensed into test tubes labeled blank, sample, control blank and control respectively for AST and ALT respectively. One hundred microlitre (0.1ml) of sample and control was dispensed into their respective test tubes. All the tubes were incubated at 37ºC for 30 minutes. Five hundred microlitre (0.5ml) of 2, 4 dinitrophenylhydrazine was dispensed into all test tubes. One hundred microlitre (0.1ml) of sample and control was dispensed into their respective blank test tubes. The contents of each test tube were mixed and allowed to stand for 20 minutes at 25ºC. 5ml of 0.4N sodium hydroxide was added to each tube, mixed and read at 550nm against the respective blank prepared. The activity of the unknown was extrapolated from the calibration curve already prepared (Reitman, and Frankel, 1957).

Alkaline Phosphatase activity was done by Phenolphthalein Monophosphate method. The test tubes were respectively labeled sample, standard and control. One millilitre (1.0ml) of distilled water was pipetted into each tube followed by a drop of the substrate into each test tube. All the test tubes were incubated at 37ºC for 5 minutes. Ten microlitre (0.1ml) of sample, standard and control were dispensed into their respective test tubes. The test tubes were incubated at 37ºC for 20 minutes. Five milliliter (5ml) of colour developer was added to each test tube, mixed, and read at 550nm using water as blank. The activity of sample was calculated using the absorbance of sample against absorbance of standard multiplied by concentration of standard (Babson *et al*., 1966).

The Serum Total Bilirubin Concentration was determined using Jendrassik-Grof method (1938). The serum total bilirubin concentration is determined in the presence of caffeine, which releases albumin bound bilirubin, by the reaction which diazotized sulphanillic acid. Two hundred milliliters (200ml) of reagent 1 (sulphanillic acid) was dispensed each into two different test tubes labeled sample blank and sample followed by the addition of 1 drop (50µl) of reagent 2 (nitrite) and 1000 µl of reagent 3 (caffeine). 200 µl of the test serum was dispensed into each of the test tubes and the mixtures incubated in a water bath for 10 minutes at 250C. This was followed by the addition of 100 µl of reagent 4 (tartrate) and the mixture incubated again at 250C for 10 minutes. The absorbance of the sample (ATB) was then read against the sample blank using a colorimeter at 578nm wavelength. The total bilirubin concentration (umol/l) was then calculated by multiplying 185 × absorbance of total bilirubin (578nm).

The Serum direct Bilirubin Concentration was determined using Jendrassik-Grof method. The serum Direct/indirect bilirubin reacts with diazotized sulphanillic acid in alkaline medium to form a blue coloured complex (Jendrassik and Grof, 1938; Sherlock, 1951). Exactly, 200ml of reagent 1 (sulphanillic acid) was dispensed each into two different test tubes labeled sample blank and sample followed by the addition of 1 drop (50µl) of reagent 2 (nitrite) and 2000 µl of 0.9 % physiological saline. Two hundred microliter (200 µl) of the test serum was the dispensed into each of the test tubes and the mixtures incubated in a water bath for 10 minutes at 250C. The absorbance of the sample (ATB) was then read against the sample blank using a colorimeter at 546 nm wavelength. The direct bilirubin (umol/l) concentration was then calculated by multiplying 246 × absorbance of Indirect Bilirubin (546nm),

Total Protein concentration was carried out using Biuret method. 5.0ml of Biuret reagent was pipetted into tubes labelled blank, standard, test, and control. 0.1ml of distilled water, standard, sample and control were pipetted into their respective tubes, mixed and incubated for 30minutes at 25ºC. The absorbances were measured against the reagent blank at wavelength of 546nm. The concentration of total protein was calculated by dividing the absorbance of sample against absorbance of standard multiplied by concentration of standard (Henry *et al*., 1974).

Bromocresol green (BCG) method by Doumas *et al*. (1971) was used for albumin estimation. Three milliliters (3ml) of Bromocresol green reagent was pipetted into tubes labeled blank, standard, sample and control. 0.01ml of distilled water, standard, sample and control was pipetted into their respective tubes, mixed and incubated at 25 ºC for 5 minutes. The absorbances were measured at 578nm against the reagent blank. The concentration of Albumin was determined by dividing the absorbance of sample against absorbance of standard multiplied by concentration of standard.

**Statistical analysis**

Data obtained were subjected to statistical analysis using statistical package for social science version 21 using statistical tools such as t-test and analysis of variance (ANOVA). Results were expressed as mean ± standard Deviation|(X± SD). The values of P<005 were considered significant.

**Results**

There was significant difference (P<0.05) in AST activities (U/L) of 32.00±3.46, 49.33±2.60, 58.66±4.40, 40.66±1.45, 35.33±1.45 and 33.33±0.88 and ALT activities (U/L) of 13.00±1.15,20.00±1.154, 18.10±0.49, 23.33±0.881, 17.00±0.58 and 17.00±0.58 in control, Nickel treated, Nickel + 300mg/kg Corn cob Ash, Nickel + 450mg/kg Corn cob Ash, Nickel + 600mg/kg Corn cob Ash, and 450mg/kg Corn cob Ash. There was significant difference (P<0.05) in Total Protein (g/L) of 71.00±0.58, 70.00±1.15, 63.33±1.76, 65.00±1.15, 70.00±1.15,and 74.33±1.20 and albumin concentration (g/L) of 44.66±0.88, 44.00±1.45, 41.00±0.58, 41.66±0.88, 45.00±1.15, 46.00±1.52 in control, Nickel treated, Nickel + 300mg/kg Corn cob Ash, Nickel + 450mg/kg Corn cob Ash, Nickel + 600mg/kg Corn cob Ash, and 450mg/kg Corn cob Ash. There was significant difference (P<0.05) in total bilirubin concentration (Umol/l) of 6.73±0.43, 8.46±0.14, 11.43±0.44, 8.00±0.36, 6.96±0.26 and, 6.73±0.14 and conjugated bilirubin (Umol/l) of 4.23±0.26, 5.43±0.35, 7.46±0.43, 5.03±0.39, 4.93±0.42 and 4.16±0.20 in control, Nickel treated, Nickel + 300mg/kg Corn cob Ash, Nickel + 450mg/kg Corn cob Ash, Nickel + 600mg/kg Corn cob Ash, and 450mg/kg Corn cob Ash while alkaline phosphatase activities (U/L) of 54.00±0.57, 56.66±0.88, 2.17±0.48, 53.33±1.45, 53.33±0.88 and 54.00±1.15 in control, Nickel treated, Nickel + 300mg/kg Corn cob Ash, Nickel + 450mg/kg Corn cob Ash, Nickel + 600mg/kg Corn cob Ash, and 450mg/kg Corn cob Ash showed no significant difference(P>0.05) as shown in table 1 below.

**Table 1: Effect of corn cob ash on liver function of nickel induced liver toxicity**

| Treatment | AST (U/L) | ALT (U/L) | ALP (U/L) | Total Protein (g/L) | Albumin (g/L) | Total bilirubin (Umol/L) | Conjugated bilirubin (Umol/L) |
|---|---|---|---|---|---|---|---|
| Control | 32.00±3.46 | 13.00±1.15 | 54.00±1.15 | 71.00±0.57 | 44.66±0.88 | 6.73±0.43 | 4.23±0.26 |
| Nickel | 49.33±2.60[a] | 20.00±1.15[a] | 54.00 0.58 | 70.00±1.15 | 44.00±1.45 | 8.46±0.15[a] | 5.43±0.35[a] |
| Nickel+300mg/kg Corn cob ash | 58.66±4.40[a,b] | 18.10±0.49[a] | 56.66±0.88 | 63.33±1.76[a] | 41.00±0.58 | 11.43±0.45[a,b] | 7.46±0.43[a,b] |
| Nickel+450mg/kg Corn cob ash | 40.66±1.45[a,b] | 23.33±0.88[a] | 52.17±0.43 | 65.00±1.15[a] | 41.66±0.88 | 8.00±0.36[a] | 5.03±0.39[a] |
| Nickel+600mg/kg corn cob ash | 35.33±1.45[b], | 17.00±0.57 | 53.33±1.45 | 70.00±1.15 | 45.00±1.15 | 6.96±0.26[b] | 4.93±0.42 |
| 450mg/kg Corn cob ash | 33.33±0.88[b] | 17.00±0.57 | 53.33±0.88 | 74.33±1.20 | 46.00±1.52 | 6.73±0.14[b] | 4.16±0.20[b] |
| F | 15.31 | 16.37 | 1.00 | 11.14 | 3.15 | 31.03 | 11.56 |
| P | 0.000 | 0.000 | 0.450 | 0.000 | 0.040 | 0.000 | 0.000 |

a= Significant when compared with Control
b= Significant when compared with Nickel

**Discussion**

This research is an experimental study designed to estimate AST, Bilirubin, ALT, and Total Protein and Alkaline phosphatase in female Wistar rats exposed to Nickel, following supplementation with ash corn cob extract. Nickel caused significant increases in AST, ALT, Total and conjugated bilirubin in Nickel treated rats compared with its respective control. This is similar to the report by Husna *et al.* (2016) and Bozorgzadeh *et al.* (2023). Husna *et al.* (2016) reported that AST, ALT and ALP levels increased remarkably in experimental fish as compared to the control one, while the levels of total proteins decreased significantly in all groups when exposed to Nickel. Bozorgzadeh *et al.* (2023) reported that nickel within the range used in their experiment appears to be somewhat toxic to fish. Aspartate aminotransferases (AST), Alanine aminotransferases (ALT) and alkaline phosphatase (ALP) are sensitive indicators of liver cell injury. Aminotransferases are normally present in the serum at low concentrations and these enzymes are released into the blood in greater amounts when there is damage to liver cell membrane resulting in increased permeability. High levels of liver enzymes show hepatocellular injury (Winter *et al*., 2005). Most serum proteins are synthesized in liver, and therefore total serum protein is used as an indicator of liver dysfunction. Rivarola and Balegno (1991) reported that pesticides can decrease plasma protein owing to changes in protein and free amino acid metabolism and synthesis. Generally, a decrease in blood protein may be due to loss of protein through decreased protein synthesis or increased proteolytic activity or degradation as mentioned above. Decreasing in total protein can be partly attributed to the effects of the metal on liver cells, which is confirmed by increasing the serum AST and ALT activities observed in this study. Changes in the activity of hepatic enzymes indicate liver cell damage or a disruption in the metabolic process. Therefore, the study of enzyme activity as an important biochemical indicator is considered an important strategy to assess environmental conditions and the presence of toxic compounds (Baghshani and Shahsavani, 2013). ALT, AST, and ALP play very important roles in the metabolic processes of the body and fish health and are

introduced as appropriate biomarkers in toxicological studies (Benincá *et al.,* 2012; Kaviani *et al.,* 2018; Kaviani *et al.,* 2020). These enzymes are present in cells of various tissues, such as liver, heart, kidneys, muscles, and brain. Some physiological conditions, such as liver damage, the activity of these enzymes (Bogé *et al*., 1992). Similarly, Öner *et al*. (2008) observed that levels of ALT and AST in the blood increased due to cell damage in liver and concluded that high levels of these enzymes in serum usually indicate disease and necrosis in animals' liver.

Administration of corn cob ash caused dose dependent reduction in inducible enzymes like AST, ALT and ALP with dose dependent increase in proteins. The alanine amino transaminase (ALT) and aspartate amino transaminase (AST) values reduced as the levels of enzyme treated corncob increased which agrees with findings by Adeniyi and Ogunmodele (2006) who reported that with sufficient energy, transaminase enzyme promoted the removal of amino acid group to yield corresponding acid which enters tricarboxylic acid cycle for additional. Das *et al.* (2006) reported that nickel sulfate, a toxic heavy metal, causes degenerative histopathological changes in rat liver. Simultaneous treatment with L-ascorbic acid partially improved nickel-induced hepatocellular damage.

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