

# Hepatoprotective effect of Quail egg against carbon tetra chloride (CCl<sub>4</sub>) induced hepatic damage in albino rats

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## ABSTRACT

**Introduction:** Quail egg has been used traditionally in the treatment of many ailments. Despite the wide speculations of its involvement in the treatment of liver diseases, very little scientific evidence exist to support this claim. This work investigated the hepatoprotective effect of quail egg against carbon tetrachloride (CCl<sub>4</sub>) induced liver damage in albino rats. **Materials and Methods:** The rats were divided into five groups of five rats per group. Animals of group A (positive control) were fed with vehicle (distilled water) on the first four days and with vehicle and CCl<sub>4</sub> on the fifth, sixth and seventh day. Animals of group B (negative control) were given only vehicle for seven days. Animals of groups C, D and E were respectively administered with 100, 200 and 400 mg/kg body weight of quail egg for the first four days and with vehicle, quail egg and CCl<sub>4</sub> for the fifth, sixth and seventh day. Animals were subsequently anaesthetized, and blood samples were taken for the estimation of albumin, alkaline phosphatase (ALP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), total protein (TP) and bilirubin. The liver was isolated for histopathological studies. **Results:** The levels of ALT, ALP and TP were significantly affected ( $p < 0.05$ ) in CCl<sub>4</sub> fed groups, indicating liver injury. The effects were reduced significantly ( $p < 0.05$ ) after treatment of rats with quail egg. Furthermore, histopathological studies of the liver tissues also supported the hepatoprotective activity of quail egg- photomicrographs of treated groups showed mild reduction in vacuolarisation/ballooning degeneration of the hepatocytes. **Conclusion:** Quail egg showed some potentials of protecting the liver from damage by stabilising the levels of ALP, ALT and TP and reducing the degeneration of the hepatocytes. Thus, this finding has provided information that suggests utilising quail egg for treatment of liver dysfunction.

**Key Words:** Carbon tetrachloride, Hepatotoxicity, Hepatoprotection, Histopathology, Quail egg.

## INTRODUCTION

The liver is one of the most vital organs in the human body, it plays significant role in regulation and maintenance of physiological processes. The detoxification of poison, secretion of bile for digestion, storage of vitamins and minerals, metabolism of macromolecules etc. are some of the primary functions of the liver.<sup>1-3</sup> The high metabolic

demand on the liver, especially detoxification of drugs and toxins consequently places the liver at a greater risk of toxic damage than any other organ in the body.<sup>4</sup>

Liver disease is said to be the fifth most common cause of death after heart disease, stroke, chest disease and cancer. However, unlike other major causes of mortality, liver disease rates are increasing rather than declining.<sup>5</sup> Treatment of liver disease/damage is a leading challenge in modern medicine, as there are no orthodox drugs that confer protection against liver damage or help regenerate damaged hepatic cells. Thus, arrays of medicinal preparations are used for treatment of liver diseases which often present side effects. Most often, liver transplantation

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becomes necessary.<sup>3,6-8</sup> Due to this shortcoming, researches have been directed in recent years, towards discovery and development of novel hepatoprotective drugs from medicinal plants and food items used for trado-medicinal purposes.<sup>9,10</sup>

Quail (*Coturnix coturnix*) is a small to medium sized bird of economic importance. The bird is naturally found in the wild, but can be raised in the farm. The eggs of quail have been reported to be rich in proteins, Vitamin A, Vitamin E, B- Complex Vitamins, Choline, Iron, Potassium, Phosphorus and HDL cholesterol.<sup>11,12</sup> They have also been reported to have folk medicinal potency; they are used to treat an array of diseases such as respiratory and digestive tract disorders, sexual potency, heart diseases, renal insufficiency, cancer and as anti-ageing agents.<sup>12, 13</sup>

In this study, the hepatoprotective activity of Quail eggs on CCl<sub>4</sub> induced liver damaged Albino rats was investigated. Biochemical parameters (aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, total protein, albumin and total bilirubin) were estimated from blood samples collected. The liver of the rats was also isolated for histopathological studies.

## MATERIALS AND METHODS

### Quail Eggs

Eggs were obtained from a poultry farm (S.G. Adiya) in Sokoto state. A random selection was made and eggs were certified healthy by the farms' veterinary doctor. All eggs used weighed between 8-10 g and their average weight was 9 g.

### Experimental Animals

Thirty healthy (30) adult Albino experimental Rats (of both sexes) with an average weight of 185 g were obtained from the Biological Science Department of Usmanu Danfodiyo University Sokoto, Nigeria. They were allowed to acclimatize to the new environment for 7 days, having access to food and water given *ad libitum*. Chick mash was served as the whole source of feed for the animals throughout the experimental period.

### Experimental Design

The method described by Adebayo *et al.*<sup>14</sup> was adopted with some modifications. The Rats were divided into five groups of five rats each. Animals of group A (positive control) were administered with vehicle (distilled water) on the first four days and with the vehicle and CCl<sub>4</sub> (30% in liquid paraffin) on

the fifth, sixth and seventh day. Animals of group B (negative control) were given only vehicle for the seven days. The animals of group C, D and E were respectively administered with 100, 200 and 400 mg/kg body weight of quail egg and vehicle for the first four days and with vehicle, quail egg and CCl<sub>4</sub> (30% in liquid paraffin) on the last three days. Animals were subsequently anaesthetized (in diethyl ether) and sacrificed after fasting for 12 hours. Blood samples and liver were collected for estimation of biochemical parameters and for histopathological studies respectively.

### Collection and Preparation of Samples

The blood samples were collected by vertical incision made around the neck to cut through the jugular veins into lithium heparin zed bottles. Plasma was obtained by centrifuging the blood at 10,000 rpm for 15 minutes in clean bottles. The plasma was stored at 20°C for biochemical analysis.<sup>15</sup> Liver was collected after dissection and fixed with 10% formaldehyde for histopathological examination.

### Analysis of Biochemical Parameters

Randox laboratory test kits were used for estimation of all the biochemical parameters. Standard operating procedure were used to estimate aspartate aminotransferase (AST),<sup>16</sup> alanine aminotransferase (ALT),<sup>16</sup> alkaline phosphatase (ALP),<sup>17</sup> total protein (TP),<sup>18</sup> albumin (ALB)<sup>19</sup> and total bilirubin (TB).<sup>20</sup>

### Histopathological Analysis

Small piece of liver fixed with 10% buffered neutral formalin were processed for embedding in paraffin. Sections of 5-6 µm thickness were stained with hematoxylin, eosin dye and finally mounted in di-phenyl xylene. The section was examined for histopathological changes under a compound microscope.<sup>21</sup>

### Statistical Analysis

All values were expressed as mean ± standard deviation. Turkey's post hoc test was done to analyse significant difference between different groups using the statistical analysis software SPSS (version 16.0). Values with p < 0.05 were considered as significantly different.

## RESULTS

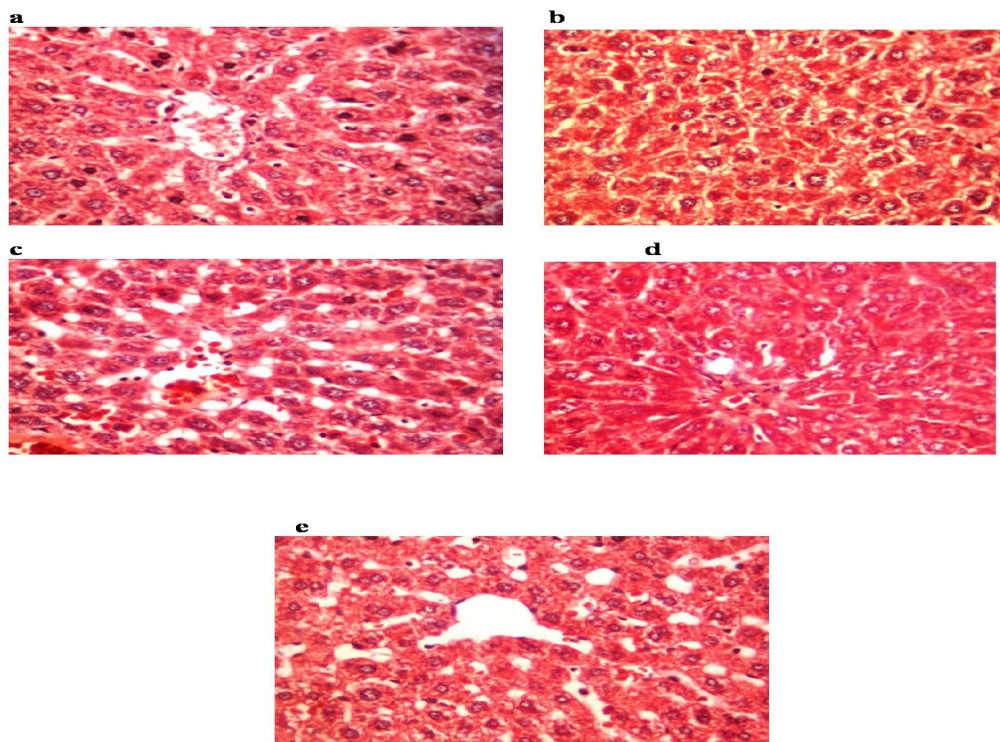
### Estimated Biochemical Parameters

The results of estimated biochemical parameters of CCl<sub>4</sub>

**Table 1: Effect of Quail Egg on CCl<sub>4</sub> Induced Hepatotoxicity in Albino Rats.**

Parameters	Group A (Positive Control)	Group B (Negative Control)	Group C (100 mg/kg)	Group D (200 mg/kg)	Group E (400 mg/kg)
TP (g/dl)	3.27±0.34 <sup>a</sup>	7.32±1.65 <sup>b</sup>	6.18±1.12 <sup>b</sup>	6.53±0.76 <sup>b</sup>	7.25±0.68 <sup>b</sup>
ALB (g/dl)	2.98±0.26 <sup>a</sup>	3.37±0.28 <sup>a</sup>	3.08±0.08 <sup>a</sup>	3.17±0.36 <sup>a</sup>	3.27±0.34 <sup>a</sup>
TB (mg/dl)	0.48±0.31 <sup>a</sup>	0.40±0.20 <sup>a</sup>	0.34±0.10 <sup>a</sup>	0.37±0.13 <sup>a</sup>	0.39±0.21 <sup>a</sup>
ALP (U/l)	500.59±57.81 <sup>a</sup>	200.56±22.59 <sup>b</sup>	274.77±20.78 <sup>b</sup>	300.39±85.70 <sup>b</sup>	300.47±94.29 <sup>b</sup>
ALT (U/l)	127.17±40.29 <sup>a</sup>	77.50±25.51 <sup>b</sup>	75.21±10.31 <sup>b</sup>	75.17±10.37 <sup>b</sup>	83.50±10.93 <sup>b</sup>
AST (U/l)	55.63±10.39 <sup>a</sup>	51.94±12.82 <sup>a</sup>	43.83±18.52 <sup>a</sup>	49.88±8.20 <sup>a</sup>	49.99±10.76 <sup>b</sup>

Data were expressed as Mean ± Standard deviation, n=5 : Mean values with different superscripts in a row indicate significant difference (p<0.05).



**Figure 1:** Photomicrograph of (a): Liver section of rat treated with CCl<sub>4</sub> show hepatocytes arranged in cords separated by sinusoid, some hepatocytes show sinusoidal dilation and ballooning degeneration, H&E x 400 (b): liver architecture showing normal features of control group; hepatocytes arranged in cords separated by sinusoid, H&E x 400. (c): liver of rat treated with CCl<sub>4</sub> and 100 mg/kg bw of quail egg showing mild centrilobular fatty degeneration, H&E x 400. (d): liver of rat treated with CCl<sub>4</sub> and 200 mg/kg bw of quail egg showing moderate sinusoidal dilation and fatty degeneration, H&E x 400. (e): liver of rat treated with CCl<sub>4</sub> and 400 mg/kg bw of quail egg showing reduced dilation of the sinusoids and ballooning degeneration, H&E x 400.

induced hepatotoxicity in Albino Rats administered with quail egg are presented in Table 1.

## DISCUSSION

The efficacy of any hepatoprotective agent depends on its ability to either reduce damage done on liver or maintain its normal physiology.<sup>22</sup> Most experiments involving the induction of liver damage by CCl<sub>4</sub> revealed elevated level of liver enzymes (AST, ALT and ALP), this is because it

is metabolized in hepatocytes by cytochrome P<sub>450</sub>, which leads to the formation of trichloromethyl radical that facilitates a chain of lipid per oxidation reactions, thus, causing liver fibrosis. Also, CCl<sub>4</sub> reduces tissue catalase and superoxide dismutase activities, which can result to oxidative modification of the liver tissue.<sup>23-26</sup>

The results of this study in Table 1 revealed significant elevation (p<0.05) in the levels of ALP and ALT, while AST elevation was not significant in CCl<sub>4</sub>-treated group (positive control). This is understandable as AST is less

specific than ALT as a liver function index.<sup>27</sup> However, TP was significantly ( $p < 0.05$ ) decreased in the  $\text{CCl}_4$ -treated group. Elevated levels of these biochemical indices is a direct reflection of a compromise in hepatic structural integrity, as these enzymes are situated in the liver and injury by toxicants cause cellular leakage and loss of functional integrity.<sup>28</sup> Conversely, decrease in TP indicates loss of hepatic synthetic capacity as it measures albumin and globulins found in blood serum. This is an indication that  $\text{CCl}_4$  induced damage to the liver and altered hepatic structural and functional integrity. The result confirms the study of Adebayo *et al.*<sup>29</sup> which showed that  $\text{CCl}_4$  has the ability to induce hepatic injury. Histopathologically, the photomicrograph revealed some of the hepatocytes of positive control group (Figure 1a) showing ballooning degeneration and sinusoidal dilation.

Upon treatment of rats with quail egg, the elevated ALP and ALT levels in  $\text{CCl}_4$ -treated groups was significantly reduced ( $p < 0.05$ ), indicating hepatoprotection. Also, total protein level was significantly increased ( $p < 0.05$ ) in treated groups. Photomicrograph of liver sections showed that the vascularisation/ ballooning degeneration of treated groups (C, D and E) was reduced, but still observable. These findings corroborate with a similar study by Ozbek *et al.*,<sup>13</sup> who stated that quail egg effectively reduced elevated levels of liver enzyme markers and from histopathological studies, quail egg was not seen to have absolute hepatoprotective activity, but increased body resistance and decreased severe weight loss were achieved after administration of quail egg to  $\text{CCl}_4$  induced liver damaged Rats. Significant change in albumin was not observed in this study. This is because serum albumin does not change in mild liver injury, the

half-life of albumin is 19-21 days which makes it not to reflect acute changes in liver synthetic ability.<sup>30</sup>

The hepatoprotective property of quail egg may be due to antioxidant activity of the individual or combined effects of the vitamins (A and E) it contains. The exact metabolite responsible for hepatoprotection needs investigation.

## CONCLUSION

From the result of this study, it was observed that quail egg showed some potentials of protecting the liver from damage by stabilising the levels of ALP, ALT and TP and reducing the vascularisation/ ballooning degeneration of the hepatocytes. Thus, this finding has provided information on the possibility of utilising quail egg for treatment of liver dysfunction.

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## CONFLICT OF INTEREST

The authors wish to declare that there is no conflict of interest.

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