Indian Journal of Novel Drug Delivery

IJNDD

An Official Publication of Karnataka Education and Scientific Society

Research Article

Hepatoprotective Activity of Methanol Extract of the Seeds of *Sesamum Indicum* in Carbon Tetrachloride Induced Hepatoxicity in Rats

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ARTICLE DETAILS

Article history:
Received on 24 January 2011
Modified on 13 March 2011
Accepted on 24 March 2011

Keywords:
Sesamum indicum,
Methanol extract,
Hepatotoxicity,
Biochemical markers,
CCl_4

ABSTRACT

The hepatoprotective activity of methanol extract of seeds of *Sesamum indicum* was investigated in carbon tetrachloride (CCl_4) induced liver injury in rats. The rats were divided into six (6) groups (A-F) of five each. Graded doses (200, 400, and 800 mg/kg) of methanol extracts of seeds of *Sesamum indicum* (MESI) were administered orally to three different groups(A-C) respectively for seven days prior to CCl_4 injection. Groups D and E received 5ml/kg of physiological saline and 50 iu/kg α-tocopherol respectively using oral gavage while the last group (F) served as the baseline control group. On the 8th day, animals in all the groups except group F were given carbon tetrachloride (2 ml/kg body weight) in olive oil subcutaneously. Twenty four (24) hours later, blood was collected from all the groups by retro-orbital puncture for liver marker enzyme determination. Liver was excised for histopathology. Results showed significant increase in the levels of biochemical markers of hepatic damage like alanine transaminase (ALT), aspartate transaminase (AST), and alkaline phosphatase (ALP) in CCl_4 control group(D) when compared with the baseline control group F ($p < 0.001$). Treatment with MESI (200, 400, and 800 mg/kg) prior to CCl_4 administration, significantly protected rats from injury as evident by moderate changes in liver histoarchitecture in groups A-C and significant reduction(p‧ 0.001) in levels of ALT, AST and ALP when compared to the CCl_4 control group. The CCl_4 control group showed marked vacuolar degeneration, inflammatory cell infiltration, and necrosis of hepatic tissue. The methanol seed extract of *Sesamum indicum* possess hepatoprotective activity.

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INTRODUCTION

Sesamum indicum L (Pedaliaceae) is a flowering plant of the genus Sesamum. Numerous wild relatives occur in Africa and a smaller number in India. It is widely naturalized in tropical regions around the world and is cultivated for its edible seeds, which grow in pods. The seed is used as diuretic, emolient, galactogogue, lenitive[1] and acts as a tonic for the liver and kidneys[2]. It is taken for the treatment of premature hair loss and greying, convalescence, chronic dry constipation, dental caries, osteoporosis, stiff joints, and dry cough. The seed contains flavonoids, cardiac glycosides, anthocyanins, saponins, and reducing sugars[3].

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The liver is the key organ of metabolism, secretion and excretion. It is continuously and variedly exposed to xenobiotics, environmental pollutants and chemotherapeutic agents because of its strategic location in the body. Liver diseases are worldwide problems. Some compounds produce metabolites that cause liver injury in a uniform, dose-dependent fashion[4]. Injury to hepatic tissue results either directly from the disruption of intracellular function or membrane integrity or from damages affecting endothelial or bile duct cells as seen in cholastatis or indirectly from immune mediated membrane damage[5]. Factors promoting the accumulation of hepatocyte toxins include genetic alterations in enzymes that allow the formation of the harmful metabolites of other drugs, and depletion of the substrates required to detoxify the metabolite[6]. Conventional drugs used in the treatment of liver diseases are

sometimes inadequate and can have serious adverse effects. None of the drugs used in allopathic medical practice provide dependable liver protection. Medicinal plants play significant role in the treatment/management of liver disorders and many of them have shown significant hepatoprotective potency[7, 8, 9, 10].

The search for new pharmacologically active agents obtained by screening natural resources such as plant extracts has led to the discovery of many clinically useful drugs that play a major role in the treatment of human diseases. A large number of medicinal plants have been tested and found to contain active principles with curative properties against a variety of diseases[11]. Recent experience has shown that plant drugs are relatively non-toxic, safe and even free from serious side effects[12]. Thus, the need to search for alternative drugs for the treatment of liver diseases to replace currently used drugs of doubtful efficacy and safety. The antioxidant activity or the inhibition of the generation of free radicals is important in providing protection against hepatic damage[13].

The principal causes of carbon tetrachloride (CCl_4) induced hepatic damage is lipid peroxidation and decreased activities of antioxidant enzymes and generation of free radicals[14]. The present study was performed to assess the hepatoprotective activity of *Sesamum indicum* seed extract against carbon tetrachloride induced liver damage in rats in order to validate its use in folklore medical practices in treatment of liver diseases.

MATERIALS AND METHODS

Animals

Thirty (30) male albino Wistar rats weighing 180-220 g were obtained from the Animal House of the College of Medicine, University of Nigeria, Enugu Campus. The animals were acclimatized for two weeks under standard condition of temperature {25^0C ±3^0C} with a 12:12 hr Light/Dark cycle. The rats were fed with standard pellets (Guinea feed Nigeria, Plc) and water ad libitum. All animals were handled in this study according to international guidelines for handling experimental animals[15].

Plant collection

Sesamum indicum seeds were obtained from their natural habitat in Nsukka, Enugu State, Nigeria. The sample of the seeds and plant were authenticated by a taxonomist at the Herbarium Section of the Department of Botany, University of Nigeria, Nsukka and a voucher specimen no: UNH/74C was opened for future references.

Plant extraction

The seeds were dried under a shade and 3.5 kg were powdered with a mill grater III (MS 223, Taiwan) and soaked in 5 litres of 80% methanol for 48 hrs with intermittent agitation. After 48 hrs, the methanol extract (ME) was filtered through a Whatman No. 1 filter paper. The filtrate was evaporated to dryness on a rotary evaporator (Model type 349/2 Corning Ltd). The residue obtained was stored in the refrigerator (4^0C ± 2^0C). 20g of the extract was dissolved in 0.85% NaCl and made up to final volume of 100 ml with the same solvent to give a final concentration of 200 mg/ml.

Acute toxicity study was not carried out as the seeds are used as food and therefore considered to be safe. However, the oral LD_{50} of the seed in mice is 50000 mg/kg[3].

Experimental design

Thirty (30) male albino rats weighing 200-220 g were divided into six groups (A-F) of 5 per group. Rats in groups A-E received 200mg/kg ME, 400 mg/kg ME, 800 mg/kg ME, 5 ml/kg of physiological saline (0.85% NaCl), and 50 iu/kg α-tocopherol respectively using oral gavage. Alpha- tocopherol was employed in this study as positive control since several studies have validated its hepatoprotective effect on liver injury induced by several hepatotoxicants[16, 17, 18, 19, 20]. Group F did not receive any treatment and served as the baseline control group. All the animals in the treatment groups (A to E) were given daily doses for 7 days in order to stabilize their plasma drug concentration. On the 8th day, animals in all the groups except group F were given carbon tetrachloride (2 ml/kg body weight) in olive oil subcutaneously[21]. Twenty four (24) hours after CCl_4 injection, blood was collected from all the groups by retro-orbital puncture for liver marker enzyme determination. Serum was separated from cells as soon as possible and stored frozen prior to analysis. After blood collection, the animals were euthanized under ether anaesthesia and their liver exercised, washed in cold saline and fixed in 10% formal saline.

Liver marker enzyme determination

Alanine and aspartate transaminase

Alanine and aspartate transaminase were determined by the endpoint technique of Reitman and Frankel[22].

Alkaline phosphatase
Alkaline phosphatase was estimated using the method of Roy[23]

Histopathological evaluation of liver
The formalin fixed liver tissues were embedded in paraffin wax and microtome sections of 5-6 µm made from them. These thin sections were stained with haematoxylin and eosin for Light microscopy. Photomicrographs were subsequently be made from these sections.

Statistical analysis
The data obtained were analyzed using student's t-test and results expressed as Mean ± standard error of mean. Statistical differences between means were determined by ANOVA. Values of $p<0.05$ was considered significant.

RESULTS
Table 1 shows the levels of liver marker enzymes in all the groups. ALT, AST and ALP values in the methanol treatment groups (A-C) were significantly lower (p· 0.001) compared to those of CCl4 control (D) group. However, these values were significantly higher (p· 0.05) when compared to the baseline control (non-treatment) group (F). In the methanol treatment groups, the effect of the extract on the enzymes appeared to be dose-dependent; mean levels of ALT were: 61.00±5.06, 53.80±6.39 and 47.30±2.20 in groups A, B and C respectively. Mean levels of AST were: 78.50±3.40, 74.60±5.18 and 59.60±3.37 in groups A, B and C respectively while the values for ALP were: 66.60±4.65, 56.20±12.46 and 38.00±2.61 respectively for the three groups.
Table 2 shows that the levels of ALT, AST and ALP in the methanol extract treatment groups were significantly lower (p· 0.001) compared to the antioxidant (α-tocopherol) group E.

Histological evaluation of liver of the animals showed that the histoarchitecture of groups A and B was preserved, though there was mild vacuolation and infiltration of inflammatory cells (figs 1 and 2). Group C treated with highest dose of MESI (800 mg/kg) showed tremendous preservation of hepatic plates (fig. 3), their hepatocytes were normal (fig. 3). The histoarchitecture of the hepatocytes were better preserved in extracts treated groups (A-C) than with the antioxidant control (α-tocopherol) group E (fig.4). Animals in group D treated with vehicle (physiological saline) prior to CCl_4 injection showed marked derangement in hepatocyte architecture as evident by the presence of vacuolation, inflammatory cells at the peri-vascular and peri-central areas, necrosis and fibrosis (fig. 5). Group F served as baseline control and received neither drug nor extract; they showed normal hepato-architecture (fig. 6).

DISCUSSION
Hepatic injury can occur when the liver is exposed to some chemicals and drugs. Carbon tetrachloride-induced hepatic injury is commonly used as an experimental method for the study of hepatoprotective effects of medicinal plant extracts and drugs[24]. The changes associated with CCl_4-induced liver damage are similar to that of acute viral hepatitis[25]. The hepatotoxicity induced by CCl_4 is due to its metabolite CCl_3•, a free radical that alkylates cellular proteins and other macromolecules with a simultaneous attack on lipids, in the presence of oxygen, to produce lipid peroxides, leading to liver damage[26]. Alanine aminotransferase and aspartate aminotransferase are well known diagnostic indicators of liver disease. In cases of liver damage with hepatocellular lesions and parenchymal cell necrosis, these marker enzymes are released from the damaged tissues into the blood stream. Alkaline phosphatase is a membrane bound enzyme and its elevation in plasma indicates membrane disruption in the organ. The liver is the major source of alkaline phosphatase (ALP) although it is not a liver specific enzyme. The level of this enzyme increases in cholestasis[27].

In this study, the levels of these enzymes were found to increase in groups where hepatotoxicity was induced using CCl_4 but were significantly reduced in groups that received methanol extracts of seeds of seamum indicum prior to the induction. Biochemical findings showed that serum ALT, AST and ALP levels were significantly lower in the extract treated groups compared to CCl_4 group. Decreased serum AST and ALT levels suggest that *Sesamum indicum* can prevent liver cell damage[28]. The hepatoprotective effect of extract was superior to that of α-tocopherol, a standard drug used in this study not only because of its known hepatic curative ability but also because it is one of the drugs reported to have modulatory actions on hepatic disorders irrespective of the cause[16, 17, 18, 19,20].

Table 1: Liver marker enzyme levels in the various groups.

GROUP	ALT	AST	ALP
A (200 mg/kg of ME)	61.00±5.06[a,e]	78.50±3.40[a,f]	66.60±4.65[a,f]
B (400 mg/kg of ME)	53.80±6.39 [a,f]	74.60±5.18 [a,f]	56.20±12.46[b,f]
C (800 mg/kg ME)	47.30±2.20 [a,f]	59.60±3.37[b,f]	38.00±2.61[f]
D (CCl4 control)	126.00±7.17[c]	139.00±8.19 [c]	176.40±6.99 [c]
E(50iu/kg α-Tocopherol)	80.60±6.94[d]	99.60±9.81[d]	94.20±8.53[e]
F (Baseline control)	31.40±2.84	48.40±3.39	47.50±4.28

a = p< 0.01 , b = p< 0.05, c = p< 0.001 (w.r.t baseline control)
d = p< 0.05, e = p< 0.01, f = p< 0.001(w.r.t CCl_4 control)

Table 2: Comparison of liver marker enzyme levels in methanol group with antioxidant control (α-Tocopherol group)

GROUP	ALT	AST	ALP
A (200 mg/kg of ME)	61.00±5.06**	78.50±3.40***	66.60±4.65***
B (400 mg/kg of AE)	53.80±6.39***	74.60±5.18***	56.20±12.46***
C (800 mg/kg ME)	47.30±2.20***	59.60±3.37***	38.00±2.61***
E(50iu/kg αTocopherol)	80.60±6.94	99.60±9.81	94.20±8.53

*P<0.05 **P<0.01 ***P<0.001.

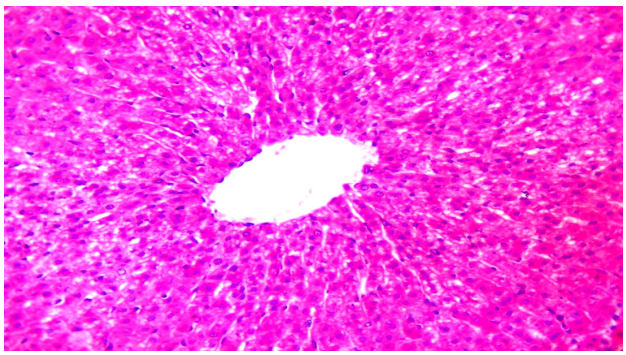

Figure 1: Liver photomicrograph of rats treated with 200mg/kg MESI prior to CCl_4 intoxication showing mild vacuolation and inflammatory cell infiltrates (x 200)

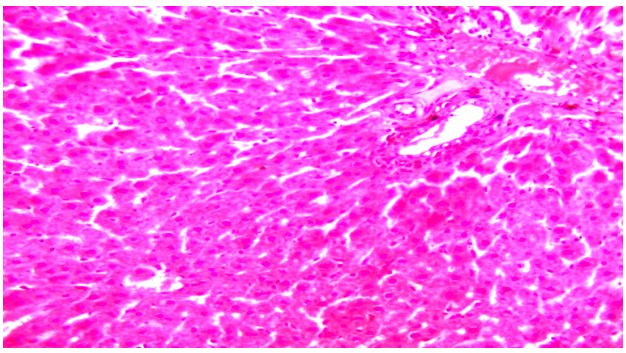

Figure 3: Histological presentation of the liver of rats treated with the 800 mg/kg MESI prior to CCl_4 intoxication showing normal hepatic chords (x 200)

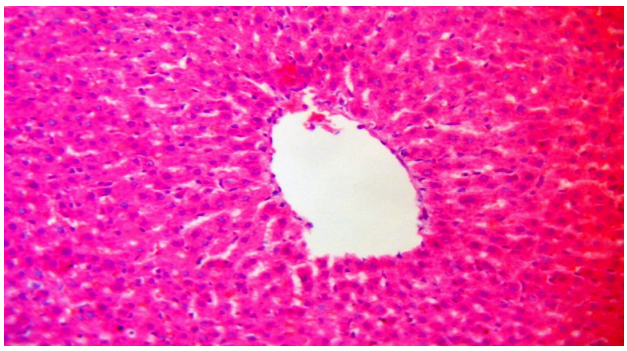

Figure 2: Photomicrograph of the liver of rats treated with the 400mg/kg MESI prior to CCl_4 intoxication showing normal hepatoarchitecture (x 200)

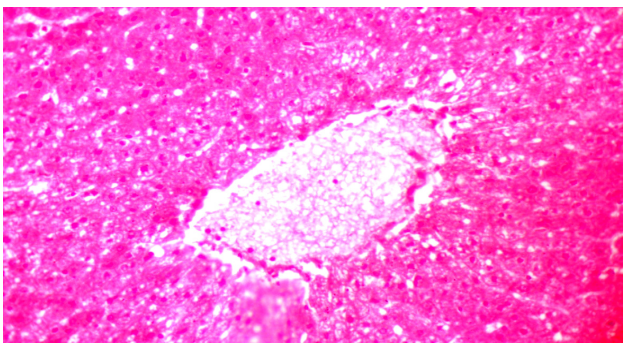

Figure 4: Photomicrograph of the liver of rat in the α-tocopherol group showing vacuolar degeneration, inflammatory cell infiltrates, and haematoma in the central canal (x 200)

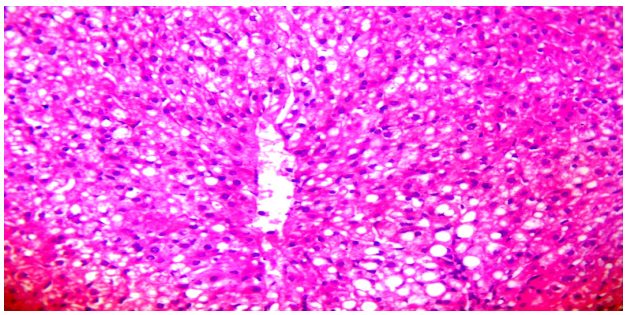

Figure 5: Liver micrograph of rats in the CCl_4 control group showing marked vacuolar degeneration, inflammatory cell infiltrates, and necrosis of hepatic tissue (x 200)

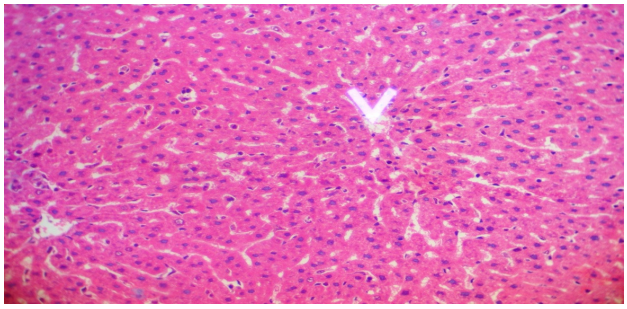

Figure 6: Liver micrograph of animals in the baseline control group E (x200)

Seeds of *sesamum indicum* contain flavonoids, glycosides, saponins, anthocyanins and reducing sugars[3]. Flavonoids have been found to possess antioxidant activity by scavenging for free radicals[29]. Reduction in the levels of AST and ALT towards the normal value is an indication of regeneration process. Reduction of ALP levels suggests the stability of the biliary function during injury with CCl_4. Comparative histopathological study of the liver from different groups of rats supported the hepatoprotective potency of crude methanolic extract of the seeds of *Sesamum indicum*. Various pathological changes like steatosis, centrilobular necrosis and vacuolation seen in toxicant rats may be due to oxidative damage caused by free radical generation[30]. These pathological changes were prevented to a great extent in extract treated groups.

Sesame lignans have antioxidant and health promoting activities[31].

High amounts of both sesamin and sesamolin have been identified in sesame[32]; they were reported to have increased both the hepatic mitochondrial and the peroxisomal fatty acid oxidation rate[33]. Sesame seed consumption appears to increase plasma gamma-tocopherol and enhanced vitamin E activity[34]. This increase in antioxidant status may account for the hepatoprotective activity of the extract. Sesame seed also contains lecithin which has antioxidant and hepatoprotective activity[35]. Lecithin is also effective for reducing hepatic steatosis in long term parenteral nutrition patients[36]. Sesame seeds and its constituents possess very potent antioxidant activity[37,38,39]. Sesamol (3,4-methylenedioxyphenol), a coumarin derivative present in sesame seeds is known to efficiently scavenge hydroxyl, one-electron oxidizing organo-haloperoxyl, lipid peroxyl, and tryptophanyl radicals in-vitro and in-vivo, and has been found to inhibit lipid peroxidation, hydroxyl radical-induced deoxyribose degradation and DNA cleavage[39]. The other constituents of sesame- sesaminol, sesamolinol, and sesamolin (lignans) reduce lipid peroxidation in the liver and kidney[40]. It is also known that both coumarins and lignans reach the systemic circulation after oral ingestion[41,42]. Consequently, it could be suggested that the hepatoprotective activity of sesame seed extract after oral administration could be partly due to its potent antioxidant activity.

This study has shown that the methanol seed extracts of *Sesamum indicum* possess hepatoprotective potency, thus there is scientific justification in the use of this seed for the treatment of liver disorders as found in local communities in Nigeria.

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