Renoprotective effects of *Moringa oleifera* pods in 7,12-dimethylbenz[a]anthracene-exposed mice

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**OBJECTIVE:** To investigate the potential of hydroethanolic extract of *Moringa oleifera* (MOHE) against 7, 12-dimethylbenz[a]anthracene (DMBA)-induced toxicity in male Swiss albino mice.

**METHODS:** Experimental mice were respectively pretreated with 200 and 400 mg/kg of MOHE, and 0.5% and 1% of butylated hydroxyanisole (BHA) for two weeks prior to the administration of 15 mg/kg of DMBA, respectively. Levels of xenobiotic metabolizing enzymes such as cytochrome (Cyt) P450 and Cyt b5, activities of reduced glutathione (GSH) and glutathione-S-transferase (GST) and renal aspartate aminotransaminase (AST), alanine aminotransaminase (ALT) and alkaline phosphatase (ALP), and content of protein and total cholesterol were measured to determine the nephrotoxicity caused by DMBA and to elucidate the ameliorating role of *M. oleifera*.

**RESULTS:** Single oral administration of 15 mg/kg of DMBA resulted in significant increases in Cyt P450 and Cyt b5 (*P* < 0.01). The toxic effect of DMBA was justified by the significant decreases in the activities of GSH and GST in renal tissues (*P* < 0.05). The levels of renal AST, ALT and ALP and protein content which are indicative of renocellular damage were also found decreased along with significant increase in total cholesterol content in DMBA-treated mice (*P* < 0.01). The DMBA-induced alterations in the tissues were significantly reversed after pretreatment with 200 and 400 mg/kg of MOHE orally for 14 d (*P* < 0.01).

**CONCLUSION:** The effects of MOHE in enhancing the levels of antioxidants and enhancing the levels of biochemical assays in DMBA-induced carcinogenesis are by reducing the formation of free radicals. This study rationalizes the ethnomedicinal use of *M. oleifera* for the protection against nephrotoxicity induced by chemical carcinogens.

**KEYWORDS:** *Moringa oleifera*; plant extracts; 9,10-dimethyl-1,2-benzanthracene; carcinogens; cytotoxicity, immunologic; mice
Cancer is a group of diseases that can occur in all living cells in the body. Different cancer types have different natural history. Epidemiological studies have shown that 70% to 90% of all cancers are of environmental origin. Life-style-related factors are the most important and preventable among the environmental exposures. Chemoprevention has evolved as a promising and valuable strategy to inhibit, suppress or control the incidence of carcinogenesis by using specific natural and synthetic agents. Chemopreventive agents may act by multiple pathways to block tumorigenesis. Agents, either natural or synthetic, that exhibit any or a combination of these pharmacological characteristics are qualified as cancer-chemopreventive agents. The most useful cancer-chemopreventive agents should have significant ability to reduce tumor incidence, delay tumor onset and prevent tumor progression.

A major aspect of this drug research, therefore, has targeted natural agents, hoping that they would augment actions or reduce the doses of ordinary chemotherapeutic drugs, thus improving their overall performance. The use of medicinal plants in modern medicine suffers from the fact that although hundreds of plants are used in the world to prevent or to cure diseases, the scientific evidence in terms of modern medicine is lacking in most cases. Herbal remedies are widely used for the treatment and prevention of various diseases and often contain highly active multitudes of chemical compounds. Over three quarters of the world population rely mainly on plants and plant extracts for healthcare. More than 30% of the entire plant species, at one time or other were used for medicinal purposes. It has been estimated that in developed countries such as United States, plant drugs constitute as much as 25% of the total drugs, while in fast developing countries such as China and India, the contribution is as much as 80%.

Many compounds have been tested with proved efficacy against experimentally induced reno-carcinogenesis. The polycyclic aromatic hydrocarbons (PAHs) are reasonably anticipated to be human carcinogens based on sufficient evidence of carcinogenicity in experimental animals. The PAH 7, 12-dimethylbenz[a]anthracene (DMBA) is well known as a cytotoxic, carcinogenic, mutagenic and immunosuppressive agent. The PAH 7, 12-DMBA acts as a potent carcinogen by generating various reactive metabolic intermediates leading to oxidative stress. On the other hand, DMBA induces substantive nephrotoxicity that is characterized by renal tubular necrosis, proteinuria with upregulation of specific signals like tumor necrosis factor-α, chemokines and cytokines. Therefore, the evaluation of antioxidant activity of various plant extracts is considered as an important step in the identification of their ability to scavenge the free radicals.

*Moringa oleifera* Lam. is well known for its different therapeutic uses. The plant that has been known by regional names such as drumstick tree, sajiwan and sajna, is a natural as well as cultivated variety of the genus *Moringa* belonging to the family Moringaceae. Accordingly, drumsticks exhibited a plethora of health benefits including the protection against hepatorenal disorders and cardiovascular, neurological and neoplastic diseases. The antineoplastic effects of *M. oleifera* that have been evaluated in animal models and relevant human trials, showed that this plant may offer antitumor profiles, and could also enhance the sensitivity of tumor cells to ordinary chemotherapy. *M. oleifera* possesses antitumor, anti-oxidative, anti-inflammatory, anti-ulcer, antispasmodic, diuretic, antihypertensive, cholesterol-lowering, antioxidant, anti-diabetic, and renal- and hepatoprotective activities. Early studies documented the presence of phenolics, flavonoids, saponins, terpenoids, proanthocyanidins, and cardiac glycosides in the pods of *M. oleifera*. This study was to elucidate the alleviation effects of *M. oleifera* on DMBA-evoked lethal renal dysfunction in mice.

**Related Articles**


1 Materials and methods

1.1 Drugs and chemicals All chemicals used in the study were respectively purchased from SRL, Merck, Ranbaxy, Himedia, Qualligens and Suyog, India. DMBA was purchased from Sigma Chemical Co., USA.

1.2 Experimental plant and preparation of hydroethanolic extract of M. oleifera The experimental plant *M. oleifera* was collected from Krishi Vigyan Kendra, Banasthali University, Banasthali, India, in October, 2009. The plant material was taxonomically identified by Botanist of Krishi Vigyan Kendra, Banasthali, India.

For preparation of hydroethanolic extract of *M. oleifera* (MOHE), dried powdered pods were placed in the Soxhlet thimble with 80% ethanol in 250 mL flat bottom flask. Collected solvent was cooled at room temperature and poured into a glass plate. The extract was concentrated under vacuum at 40 °C to yield a semisolid mass, dried in hot air oven below 50 °C for 48 h and stored in a desiccator. The percentage yield of MOHE was found to be 22% and the extract was stored at 4 °C in airtight containers. Suspensions of the extract was prepared in distilled water and used to assess the renoprotective activity.

1.3 Experimental animals Male Swiss albino mice weighing 15 to 30 g were obtained from Haryana Agricultural University, Hisar, India for experimental purpose. The animals were acclimatized for a month prior to experiment. The Institutional Animal Ethical Committee approved the animal studies. All experiments were conducted on the mice when they weighed 25 to 35 g with 3 to 4 months old. Colony bred mice were maintained under standard laboratory conditions at a temperature of (22 ± 3) °C, relative humidity of 50% ± 5% and photoperiod of 12 to 12 h dark and light cycle.

1.4 Treatment regime Mice divided into six groups with 6 in each were treated by oral gavage. The groups were as follows. Group 1 served as normal control which received 1 mL distilled water by oral gavage. Group 2 received distilled water for 14 d prior to a single dose of 15 mg/kg of DMBA served as DMBA control group. Groups 3 and 4 were respectively treated with 200 and 400 mg/kg of MOHE for 14 d before being intoxicated with 15 mg/kg of DMBA dissolved in olive oil. Groups 5 and 6 respectively received 0.5% and 1% of butylated hydroxyanisole (BHA) for 14 d, before being intoxicated with 15 mg/kg of DMBA dissolved in olive oil.

The doses for DMBA, BAH and the plant extract were decided and selected on the basis of lethal dose 50 (LD50) calculated in the laboratory and published reports[5,10,11,14].

After 19 d, the mice were fasted overnight and then sacrificed under light ether anesthesia. The kidneys were dissected out and washed immediately with ice-cold saline to remove blood, and the wet weights were noted and then the kidneys were stored at −80 °C for various oxidative stress and biochemical assays. Post-mitochondrial supernatant (PMS) was prepared using the method of Mohandas *et al*[15] with some modifications.

1.5 Determination of xenobiotic metabolizing enzymes, oxidative stress and biochemical assays

1.5.1 Cytochrome P450 and cytochrome b5 Cytochrome (Cyt) P450 was determined using the carbon monoxide difference spectra. Both the Cyt P450 and Cyt b5 content was assayed in microsomal suspension by the method of Omura and Sato[16], using an absorption coefficient of 91 and 185 cm⁻¹/mmol, respectively.

1.5.2 Glutathione Reduced glutathione (GSH) content was assayed by the method of Jollow *et al*[17]. Briefly, 1.0 mL of 10% PMS was precipitated with 1.0 mL of 4% sulfosalicylic acid. The sample was kept at 4 °C for at least 1 h and then subjected to centrifugation at 12000 × g for 15 min at 4 °C. The assay mixture contained 0.1 mL of filtered aliquot, 2.7 mL of phosphate buffer (0.1 mol/L, pH 7.4) and 0.2 mL of 5, 5'-dithiobis-(2-nitrobenzoic acid) (100 mmol/L) in a total volume of 3.0 mL. The yellow color developed was read immediately at 412 nm on a spectrometer. The calculation formula of GSH is shown as below:

\[
\text{OD} \times \frac{V}{14 \text{ nmol GSH/g tissue}}
\]

V means assay mixture and v means tissue fraction. Optical density (OD) was measured and the factor is 14 nmol.

1.5.3 Glutathione S-transferase Glutathione S-transferase (GST) was determined by the method of Habig *et al*[18]. The 10% (weight/volume) tissue homogenate was prepared in 0.2 mol/L of chilled phosphate buffer (pH 6.5) and the homogenate was centrifuged in cold at 15000 × g for 15 min. Then 0.1 mL of enzyme source, 2.7 mL of 1 mmol/L of GSH solution and 0.2 mL of 1.0 mmol/L of 1-chloro-2, 4-dinitrobenzene (CDNB) were mixed. The change in the absorbance at 340 nm was recorded at room temperature against blank. The value was calculated on the basis of molar extinction coefficient of 9.6 × 10³ mol/(L · cm) for CDNB and the specific activity of enzyme was expressed as nanomole of GSH-CDNB conjugate formed per minute per microgramme protein.

1.5.4 Aspartate aminotransferase and alanine aminotransferase Activities of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were assayed by the method of Reitman and Frankel[19]. Briefly, 0.2 mL aliquot of tissue fraction and 1 mL of substrate were incubated 1 h for AST and 30 min for ALT, respectively. And 1 mL aliquot of 2, 4-dinitrophenylhydrazine solution was added to arrest the reaction and kept for 20 min at room temperature. After incubation,
1 mL of 0.4 mol/L sodium hydroxide was added and the absorbance was read at 540 nm. Unit of their activities was expressed as IU/L.

1.5.5 Alkaline phosphatase Activity of alkaline phosphatase (ALP) was determined according to the protocol described in laboratory manual[20]. Specific activities were determined by using standard curve of p-nitrophenol and expressed as micromoles of p-nitrophenol formed per minute per gram tissue.

1.5.6 Total protein estimation Total protein content was estimated by the method of Lowry et al[21] using bovine serum albumin (BSA) as standard. The protein content of each sample was evaluated from the standard curve made with BSA and expressed as gram per milliliter.

1.5.7 Total cholesterol estimation The cholesterol level was determined by using cholesterol as standard by the method of Zak[22]. Concentration of total cholesterol in samples was calculated with the aid of standard graph and expressed as milligram per gram in tissue extract.

1.6 Statistical analysis The data were expressed as mean ± standard deviation of three replicates. The data were subjected to one-way analysis of variance and differences between samples were determined by Tukey’s multiple comparison test using the SPSS 16.0 program. When P<0.05, it was considered to have significant statistical difference.

2 Results

2.1 Effects of MOHE on xenobiotic metabolizing enzymes in DMBA-treated mice Cyt P450 and Cyt b5 were significantly increased above the basal levels in DMBA model group as compared with the normal control group (P<0.01). In comparison to the DMBA model group, the administration of 200 and 400 mg/kg of MOHE, and 0.5% and 1% of BHA significantly decreased the enzyme levels (P<0.01). See Table 1.

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>GSH (nmol/g)</th>
<th>GST (nmol CDNB/(min • mg protein))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>6</td>
<td>1.73±0.20</td>
<td>93.1±1.03</td>
</tr>
<tr>
<td>DMBA (15 mg/kg)</td>
<td>6</td>
<td>1.23±0.13</td>
<td>61.2±0.09</td>
</tr>
<tr>
<td>MOHE (200 mg/kg) plus DMBA (15 mg/kg)</td>
<td>6</td>
<td>1.64±0.15</td>
<td>81.67±0.06</td>
</tr>
<tr>
<td>MOHE (400 mg/kg) plus DMBA (15 mg/kg)</td>
<td>6</td>
<td>1.75±0.05</td>
<td>89.11±0.08</td>
</tr>
<tr>
<td>BHA (0.5%) plus DMBA (15 mg/kg)</td>
<td>6</td>
<td>1.55±0.07</td>
<td>69.43±0.15</td>
</tr>
</tbody>
</table>

Table 2 Effects of MOHE on oxidative stress parameters in DMBA-treated mice (Mean ± standard deviation)

2.2 Effects of MOHE on oxidative stress parameters in DMBA-treated mice The toxic effect of DMBA was justified by the significant decreases in the activities of GSH and GST in the kidneys when compared with the normal control (P<0.01, P<0.05). The antioxidant effect of 200 and 400 mg/kg of MOHE, and 0.5% and 1% of BHA was observed by significant increases in the activities of GSH and GST when compared with the DMBA control group (P<0.01). See Table 2.

2.3 Effects of MOHE on biochemical parameters in kidneys of DMBA-treated mice DMBA produced significant depletion of AST, ALT, ALP and total protein levels in kidney tissue as compared with the normal control group (P<0.01), and the total cholesterol level in kidneys of DMBA-exposed mice showed a significant elevation when compared with the control group mice (P<0.01). The levels of AST, ALT and ALP, and total protein content were significantly elevated by pre-administration of 200 and 400 mg/kg of MOHE, and 0.5% and 1% of BHA (P<0.01), and the cholesterol level was significantly reduced when compared with the DMBA model group (P<0.01). See Table 3.

3 Discussion

The cancer-inhibitory action by a variety of human nutrients derived from plants as well as of nonnutritive plant-derived constituents (phytochemicals) has been confirmed in different animal tumor models[23] and has led to an increased emphasis on cancer prevention strategies in which these dietary factors are utilized. The present data revealed the modest protective effects of MOHE against DMBA-induced toxicity in kidney.

Oxidative stress arises when the balance between pro-oxidants and antioxidants is shifted towards the pro-oxidants[24]. DMBA is a synthetic PAH, which has been used extensively as a prototype carcinogen. The main target sites for the potent carcinogenicity of this agent in rodents are the
Table 3  Effects of MOHE on renal biochemical parameters in DMBA-exposed mice

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>AST (IU/L)</th>
<th>ALT (IU/L)</th>
<th>ALP $\mu$mol PNP/ (min \cdot g tissue)</th>
<th>Total protein (g/mL)</th>
<th>Total cholesterol (mg/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>10</td>
<td>75.23±0.03</td>
<td>47.34±0.02</td>
<td>95.23±0.05</td>
<td>5.32±0.06</td>
<td>75.34±0.04</td>
</tr>
<tr>
<td>DMBA (15 mg/kg)</td>
<td>10</td>
<td>36.55±0.07**</td>
<td>21.54±0.06**</td>
<td>52.33±0.12**</td>
<td>3.98±0.09**</td>
<td>116.28±0.08**</td>
</tr>
<tr>
<td>MOHE (200 mg/kg) plus DMBA (15 mg/kg)</td>
<td>10</td>
<td>61.43±0.05△△</td>
<td>37.33±0.06△△</td>
<td>81.44±0.13△△</td>
<td>4.98±0.13△△</td>
<td>92.87±0.05△△</td>
</tr>
<tr>
<td>MOHE (400 mg/kg) plus DMBA (15 mg/kg)</td>
<td>10</td>
<td>72.54±0.04△△</td>
<td>44.67±0.07△△</td>
<td>92.44±0.05△△</td>
<td>5.25±0.07△△</td>
<td>81.65±0.03△△</td>
</tr>
<tr>
<td>BHA (0.5%) plus DMBA (15 mg/kg)</td>
<td>10</td>
<td>43.58±0.17△△</td>
<td>26.41±0.09△△</td>
<td>63.77±0.09△△</td>
<td>4.32±0.09△△</td>
<td>109.65±0.10△△</td>
</tr>
<tr>
<td>BHA (1%) plus DMBA (15 mg/kg)</td>
<td>10</td>
<td>55.67±0.14△△</td>
<td>32.42±0.02△△</td>
<td>76.42±0.06△△</td>
<td>4.56±0.02△△</td>
<td>101.23±0.12△△</td>
</tr>
</tbody>
</table>

$* *$ $P<0.01$, vs control group; $△△ P<0.01$, vs DMBA group. DMBA: 12-dimethylbenz[a]anthracene; MOHE: hydroethanolic extract of Moringa oleifera; BHA: butylated hydroxyanisole; PNP: p-nitrophenol; AST: aspartate aminotransaminase; ALT: alanine aminotransaminase; ALP: alkaline phosphatase.

skin and the mammary gland[21] and to some extent liver and kidney[10,11]. DMBA-induced experimental carcinogenesis is preceded by a sequence of hyperplasia, dysplasia, and carcinoma. The cancer-chemopreventive efficacy is assessed by its ability to modulate the activities of enzymes associated with drug metabolism and bifunctional modulators that reduce the availability of ultimate carcinogen metabolites in the epithelial stage[22]. The activities of Cyt P450 and Cyt b5 in hepatic phase I were decreased and GST in hepatic phase II, and the GSH level were significantly restored when mice were fed by oral gavage with 200 and 400 mg/kg of MOHE for 14 d. This leads to the supposition that the inhibition of tumorgenesis by the plant extract might have been executed either by preventing the formation of active carcinogens from their precursors or by augmenting detoxification process, preventing promotional events in the mice kidney through free radical-scavenging mechanism.

Cyt P450 isoenzymes are necessary to initiate the conversion of metabolizing the lipophilic carcinogens compounds to more water-soluble metabolites, which are then acted upon by phase I enzymes to promote their polarity and assisting in their excretion[27]. Similarly, the production of phase II enzymes is measured as a cancer risk factor because of the starting carcinogens to ultimate carcinogens[28].

One of the most important antioxidant systems is the GSH redox cycle. GSH is one of the essential compounds for maintaining cell integrity. The depletion in the renal GSH level has been observed in mice in the response to oxidative stress caused by DMBA treatment. It has been suggested that DMBA is able to generate reactive oxygen species (ROS), and we found that it also inhibits the activity of antioxidant enzymes in renal tissue such as catalase and superoxide dismutase[10,11].

GST and DT-diaphorase are two major phase II enzymes. GSTs are a family of enzymes that catalyze the conjugation of reactive chemicals with GSH and play a major role in protecting cells[29]. GST catalyzes the conjugation of a variety of endogenous and exogenous compounds with the non-protein thiol, GSH. This reaction inhibits the reactive electrophiles from reaching cellular targets, and results in the production of a thioether-linked glutathionyl conjugate that is less cytotoxic.

Various approaches to cancer chemoprevention exist. For example, the inducer of GSTs has received much attention as a potential chemopreventive agent, because the ability to induce GST is a property found in many chemopreventive agents, ameliorating toxicity and carcinogenicity[2,20]. Another mechanism of chemoprevention is the elevation of antioxidant defense that can combat the oxidative stress produced by ROS, which often leads to mutation and cancer.

In this study, the renoprotective effect of M. oleifera is evident by the restoration of ALT, AST and ALP along with total protein and cholesterol content. The preservation of kidney function was significantly increased in the groups pretreated with MOHE. The recovery towards to normalization of the enzymes suggested that the plant extract has some poten role in preserving structural integrity of renocellular membrane, thus preventing the enzymes leakage into blood circulation. In the current investigation, it was noticed that DMBA altered the level of enzyme activities that may be due to discharge of the cell content into the blood stream, and therefore enzyme activities decreased in tissue and increased in blood during carcinogen toxicity. It is well known that free radicals generate a cascade, producing lipid peroxidation, protein oxidation, DNA damage and cell death, and contribute to the occurrence of pathological conditions[20,21]. In the present study, the data indicate the protein damage in kidney may be due to the oxidative stress generated by DMBA. Free radicals generated by DMBA decreased the total protein content in kidney that was elevated by the administration of MOHE in experimental groups. Saponins also form complexes with proteins and could decrease protein degradability[40]. DMBA intake increased the mean values of cholesterol in tissues. DMBA-mediated development of hypercholesterolemia
entails the activation of cholesterol biosynthetic enzymes and the simultaneous suppression of cholesterol catabolic enzymes. In general, the beneficial effects of medicinal plants may be attributable to one or more *M. oleifera* phytochemicals including antioxidants, flavonoids, saponins and other substances. Thus, compounds exhibiting antioxidant and anti-inflammatory activities are expected to be effective antitumor promoting agents. It can be postulated that the cancer-chemopreventive effect of the active bioconstituents of *M. oleifera* in the present study could be due to one or more of the following mechanisms: by blocking the carcinogen so that it cannot pass through the plasma membrane, by induction of enzymes that detoxify carcinogens, by inhibiting competitively DNA adduct formation, and by scavenging ROS. Therefore, the result of the present study suggests that MOHE has potential anticarcinogenic and antioxidative properties, and may act as a potent chemopreventive agent.

4 Conclusion

In conclusion, the present study demonstrates that *M. oleifera* mediates its chemopreventive effects by enhancing the antioxidant status and quenching of ROS. Their constituents scavenge the free radicals and exert a protective effect against free radicals and the oxidative damage to cellular macromolecules. Previous *in vitro* studies performed by us in our laboratory have proved that *M. oleifera* acts as a free radical scavenger and has wide variety of antioxidants and phyto-constituents. Traditional use of natural plant parts as food ingredients may confer some protection from cancer. The results of present study indicate that *M. oleifera* may emerge as putative chemopreventive agent against carcinogen toxicity.

5 Acknowledgements

The authors are grateful to University Grants Commission for providing financial assistance. The authors are thankful to the authorities of Banasthali University for providing support to the study.

6 Competing interests

The authors declare that they have no competing interests.

REFERENCES


analysis and evaluation of antioxidant activities of hydro-

油椒木豆荚提取物对二甲基基苯并芘诱导小鼠肾组织损伤的保护作用

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目的：通过二甲基苯并(a)anthracene（DMBA）诱导雄性白化小鼠肾组织损伤来研究油橄榄木豆荚的水乙醇提取物对肾脏的保护作用。

方法：予实验小鼠口服 15 mg/kg DMBA 两周前，分别予 200 和 400 mg/kg 的油橄榄木豆荚的水乙醇提取物以及浓度为 0.5%和 1%的叔丁基对羟基苯甲酸灌胃。测定各组小鼠细胞色素（cytochrome，Cyt）P450 及 Cyt b5、还原型谷胱甘肽（reduced glutathione，GSH）、谷胱甘肽转移酶（glutathione-S-transferase，GST）以及肾组织天冬氨酸氨基转移酶（aspartate transaminase，AST）、丙氨酸氨基转移酶（alanine transaminase，ALT）、碱性磷酸酶（alkaline phosphatase，ALP）活性及总胆固醇和蛋白质含量。

结果：口服 15 mg/kg 的 DMBA 可显著增加小鼠 Cyt P450 和 Cyt b5 的浓度（P<0.01），小鼠肾组织 GSH、GST 活性显著降低（P<0.01，P<0.05）。实验小鼠在口服 DMBA 后，肾组织 AST, ALT, ALP 活性和蛋白质含量也显著降低，总胆固醇含量则显著增加（P<0.01）。但是经 200 和 400 mg/kg 油橄榄木豆荚提取物预处理 14 d 的实验小鼠，其由 DMBA 诱发的各项组织生化指标变化发生显著逆转（P<0.01）。

结论：油橄榄木豆荚的提取物可通过提高细胞抗氧化活性、减少自由基的生成来减少 DMBA 的毒副作用，起到保护肾组织的作用。

关键词：油橄榄；植物提取物；9,10-二甲基-1,2-苯并蒽；致癌物；细胞毒性；免疫；小鼠