RESEARCH ARTICLE

STUDIES ON ANTIOXIDANT AND HEPATOPROTECTIVE ACTIVITY OF ETHANOLIC EXTRACT OF MOREL MUSHROOM MYCELIUM

R.RAVIKUMAR¹ AND A.KALIDOSS²

¹Department of Biotechnology, J.J.College of Arts and Science, Sivapuram, Pudukkottai Tamil Nadu, India.
²Department of Microbiology, Mohamad Sathak college of Arts and Science, Chennai, Tamil Nadu, India.
Corresponding Author: ravimicrobiotech@gmail.com

Abstract

Species of Morchella are known as morels. Morchella is a brain like fungus, it tastes so good. It is often found under oak, maple, likory and other hard wood especially in May. It is an economically important mushroom. Attempts to grow Morchella species have not been successful. Hence their mycelia are produced commercially by submerged fermentation. Morel mycelia can be used as a good source of protein supplement to vegetarian diets. Proteins from the mycelia are comparable to vegetable protein used by several sections of the population in our country. Morchella esculenta is an important morel mushroom belonging to the family Morchellaceae of Ascomycotina Division and is locally known as Guchhi. Morchella grows naturally on the forest floor rich in humus they are difficult to grow commercially. In India this mushrooms is found growing in the forest of Jammu and Kashmir and Himachal Pradesh. In 1982 Morchella esculenta was reported to be grown under laboratory condition. Morchella esculenta is an expensive product because of its rich nutritional value and unique flavour. It is cooked as food and used in medicinal and health care systems by the traditional societies and also considered as important for clinical use.

Key words: Morchella, Morchellaceae, Ascomycotina, medicinal, health care systems.

Introduction

It is interesting to note that the oxygen, which is most essential for the very existence of higher organism can become a monostroously dangerous molecule when combined with free electron. In the living cell several electrons can be leaked out from the electron transports chain and get attached to the acceptor molecule in tissue. A free radical in any species is capable of independent existence, that contain one or more electrons with in its outer orbital. The unpaired electron alter chemical reactivity of an atom or molecule making it more reactive than the corresponding nonradical atom or molecule.

Reactive oxygen species is a collective name which is used by biologist which includes not only the oxygen radicals (superoxide-O₂ and hydroxyl radicals-OH) but also some derivatives of oxygen that do not contain unpaired electrons such as hydrogen peroxide (H₂O₂), singlet oxygen (O₂) and peroxynitrate (ONOO). Most of the reactive oxygen species come from the endogenous source as a by-product of
normal essential reaction such as generation of energy from mitochondria or the detoxification reactions involving the liver cytochrome P-450 system. The exogenous sources include bacterial, viral or fungal infections. Reactive oxygen species (ROS) are considered to be important factors in etiology of several pathological conditions (Hemanani and Prihar, 1998). Recent studies on free radical and biochemical research in infectious diseases show involvement of free radical generation in several diseased conditions.

Human liver is the main organ that contain complex parenchymal cells that perform multiple and diverse functions essential for life. Hepatocytes have a unique regeneration capacity as well as a marked capacity to respond to increased metabolic demands of the organisms.

Liver directly receives, process, and stores the materials absorbed from the digestive tracts such as aminoacids, carbohydrates, fatty acids and cholesterol as well as vitamins and is capable of releasing metabolites of these compounds on demands. Many plasma proteins including 1,2 and 3 globulin, clotting factors and transport proteins are synthesized by the liver.

The liver is the main organ of detoxification and is the site of metabolic conversion of endogenous and exogenous compounds. Another major functions of liver is to synthesize bile acids from cholesterol and to secrete these compounds from the hepatocytes into the intestine, there by generating bile flow and facilitating dietary fats, encalcification and absorption. Tetracycline, ethanol, carbontetrachloride, bromobenzene, paracetamol sultonamide, erythromycin etc are the chemicals that induces hepatotoxicity.

Carbon tetrachloride is a pungent volatile fluid used as a drycleaner because of its ability to easily dissolve greasy stains. Its lipid solubility allows it to cross cell membrane rapidly and any tetrachloromethylene taken into the body is quickly distributed to all organs. When inhaled or swallowed it may severely damage the heart, liver and kidney, causing cirrhosis and necrosis and it can also affect the optic nerves. Treatment is by administration of the oxygen.

Tetrachloromethane is metabolized by the p450 system to give the trichloromethyl radical (CCl₃)

\[
\text{CCl}_4 \rightarrow \text{CCl}_3 + \text{Cl}
\]

This CCl₃ is brought about by the cytochrome p450 molecule itself. The trichloromethyl radical then to attracts a hydrogen atom from the membrane lipid, setting of the chain reaction lipid peroxidation. Products of peroxidation are known to inhibit protein synthesis and the activity of the certain enzymes.

Cancer is the second largest single cause of death in both men and women worldwide. Cancer arises from the abnormal and uncontrolled division of cells, then invade and destroy the surrounding tissue. Spread of cancer cells may occur via the blood streams or the lymphatic channels, thus setting up secondary tumor. Hepatocellular carcinoma is the one of the most common malignances with limited effective options. Chronic hepatitis B virus (HBV) are the etiological factor’s. Chemotherapy, radiotherapy and surgery are more effective treatment of cancer. But these treatments are found to damage the normal cells.

Mushrooms are widely distributed in nature. They are the largest reproductive structure of edible fungi. Humanities use of mushrooms extends to ancient days. About 10,000 species of mushrooms are known all over the world. Mushrooms are rich source of proteins vitamins and minerals.
A large number of mushrooms have been reported to possess medicinal properties. Many of them are found useful to treat number of disease conditions. Some important medicinal properties of mushrooms are antibiotic, immunoregulatory, cardiotonic, hypcholesteremic, antiviral and antitumor. Extract and powders of mushroom’s (mycelium, sporocarp) in the form of sugar coated tablets are being marketed for the treatment of diseases such as diabetes, cancer etc. Attempts have been made in many parts of the world to explore the use of mushroom metabolites for the treatment of human sufferings (Janardhanan, 1999). However, information on the medicinal properties of this valuable mushroom is fragmentary. Since, commercial cultivation of morel mushroom have not been successful, the cultured mycelium of morel are extensively used as food and flavour. Thus it was considered desirable to investigate the medicinal properties of the cultured mycelium. The present investigations where undertaken to evaluate the antioxidant and hepatoprotectice activity of the ethanolic extract of the mycelium of Morchella esculenta.

Materials and methods

Experimental materials

Mycelia of *Morchella esculenta*

Animals

Wistar rats purchased from Small Animal Breeding Center, Kerala Agriculture University, Mannuthy, Thrissur and were used. The animals were kept for a week under environmentally controlled conditions with free access to standard food (Lipton, India) and water.

Methods

All the ingredients were dissolved in 1000ml distilled water. The medium was poured into Roux bottles and sterilized by autoclaving at 121°C for 30 minutes. The medium was inoculated with 10 days old culture of *M.esculenta* and incubated at 30°C for 20 days as stationary culture.

Preparation of extract

After 20 days growth the liquid medium was filtered and the mycelium was separated. The mycelium was washed and dried at 40°-50°C for 48 hours. Powdered mycelium was extracted by boiling with 50% ethanol repeatedly for a period of 10 hours. The extracts were pooled and evaporated at low temperature. The alcoholic extracts thus obtained was named as the ethanol extract and was employed in the experiments.

Determination of in vitro antioxidant activity

Superoxide scavenging activity

Procedure

Superoxide radicals (O₂⁻) are generated by the photoreduction of riboflavin was detected by NBT reduction method of McCord and Fridovich (1968).

The reaction mixture contained EDTA (6mM) 3µg NaCN, riboflavin (2mM), NBT(50mM), KH₂PO₄-Na₂HPO₄ (67mM, pH 7.8) and various concentration of the ethanolic extract in a final volume of 3ml. The tubes were illuminated under incandescent lamp for 15 min. The optical density (OD) at 530 nm was measured before and after illumination. The inhibition of superoxide radicals was determined by comparing the absorbance value of the control with that of the treatments.

Inhibition of lipid peroxidation

Procedure

The reaction mixture contained the rat liver homogenate of 0.1ml (25% W/V) in Tris-Hcl
buffer (20mM, pH 7.0); KCl (30mM); FeSO₄ (NH₄)₂; SO₄ 6H₂O (0.16mM); ascorbate (0.06mM) and various concentration of mycelium extract to a final volume of 5.0ml. The reaction mixture was incubated at 37°C for 1 hour. After the incubation period, 4ml was taken and treated with 0.2ml sodium dodecyl sulphate (SDS) 8.1%; 1.5ml thiobarbituric acid (0.8%) and 1.5ml acetic acid (20%, pH 3.5). The total volume was made up to 4ml with distilled water and kept in a water bath at 95°C to 100°C for 1 hour. After cooling, 1.0 ml of the distilled water and 5.0ml of the n-butanol pyridine mixture (15:1) was added to the reaction mixture, shaken vigorously and centrifuged at 4000 rpm for 10 min. The butanol-pyridine layer was removed and its absorbance at 532 nm was read. Inhibition of the lipid peroxidation was determined by comparing the OD of treatment with that of the control.

**Hydroxyl radical scavenging activity**

**Procedure**

The reaction mixture contained deoxyribose (2.8mM); FeCl₃ (0.1mM); KH₂PO₄-KOH buffer (20mM, pH 7.4); EDTA (0.1mM); H₂O₂ (1.0mM); ascorbic acid (0.1mM) and various concentrations of the extract to a final volume of 1ml. The reaction mixture was incubated at 37°C for 1 hr. The TBARS formed was estimated by the above method of Ohkawa and Oshishi (1979). The activity was determined by comparing the absorbance of the control with that of the treatment.

**Nitric oxide scavenging activity**

**Procedure**

Nitric oxide generated from sodium nitroprusside was measured by the Griess reagent by the method of Marcocci et al (1994). Immediately before the experiment, 10mM stock solution of sodium nitroprusside was prepared in PBS (pH 7.4), Various concentrations of the extract and sodium nitroprusside (1mm) in PBS to a final volume of 3ml were incubated at 25°C for 150 minutes. After incubation, 0.5ml of the solution was taken and diluted with 0.5ml of the Griess reagent (1% sulphanilamide, 2% orthophosphoric acid and 0.1% naphthylethylene diamene). The absorbance of the chromophore formed during the diazotation of nitrite with sulphanilamide and subsequent coupling with the naphthylethylene diamenedihydrochloride was read immediately at 546nm.

**Statistical analysis**

All the data are expressed as mean ± SD.

**Determination of the hepatoprotective activity**

Hepatoprotective activity was determined using Carbon tetrachloride (CCl₄) induced acute hepatotoxicity in rats.

**Experimental Procedure**

Hepatoprotective activity was determined by the method of Lin et al (1995) with some modifications (Ajith and Janardhanan, 2002).

Animals were divided into four groups of six animals each. Group 1 was administrated with normal saline and kept as normal. Group 2 was given CCl₄ paraffin oil (1:IV/V, 1.5m/kg ), intraperitonially and kept as control. Group 3 and 4 were administrated orally with the ethanolic extract (500, 250mg/kg body weight). The administration of the drug continued for three consecutive days. One hour before the administration of last dose of the extract, the CCl₄ was given intrapertontially to group 2, 3, 4. There after the experimental animal were sacrificed after 24 hours.
Collection of Liver and Serum

Blood was collected from the animals by puncturing of the heart. Serum from the control and the experimental rats were separated by centrifugation of the blood. The liver was washed, dried with a filter paper, weighed and processed immediately for biochemical analysis.

Preparation of liver homogenate

The liver was homogenized in 50mM phosphate buffer (pH 7.0) to give a 10% homogenate. The homogenate was centrifuged at 10,000 rpm for 10 minute in a cold centrifuge at 4° C and the supernatant were used for enzyme assays and protein determination.

Serum sample was used for the estimation of Glutamate oxaloacetate transaminase (GOT) and Glutamate pyruvate transaminase (GPT) by the method of Reitman and Frankel and Alkaline phosphatase (ALP) by the method of Kind and King. Tissue homogenate was used for the assay of superoxide dismutase (SOD) by the method McCord and Fridovich; Malondialdehyde (MDA) by the method of Ohkawa et al using 1,1,3,3-tetramethoxy propane as the standard. Protein was estimated by the method of Lowry et al.,(1951) using Bovine serum albumin as standard and catalase by the method of Aebi et al.,(1974).

Assay of enzymes

Alkaline phosphatase (ALP)

Procedure

Alkaline phosphatase was assayed by the method of Kind and King,(1980).

500 l of the working buffered substrate (Phenyl Phosphate) was made up to 2ml with purified water. It was incubated for 3 minutes at 37°C. To this 50 l of the serum was added and again incubated for 15 minute at 37°C. To this 1ml of the chromogen reagent was added. The optical density was read at 510nm. 10mg% phenol was used as a standard.

Assay of serum glutamate pyruvate transaminase (GPT)

Procedure

GPT activity was determined according to the method of Reitman and Frankel (1957). 0.25ml of the buffered substrate (2-ketoglutarate and L-Alanine, pH 7.4) was incubated for 5 minutes at 37°C. 50 l of the serum was added, mixed well and incubated at 37°C for 30 minute. 0.25ml DNPH colour reagent was added and allowed to stand at room temperature for 20 minute. 2.5ml of 4N sodium hydroxide was added to this and allowed to stand at room temperature for 10 minute. The optical density was read against purified water at 505nm. The enzyme activity was calculated from the standard (Sodium pyruvate, 2mM) calibration curve.

Assay of serum glutamate oxaloacetate transaminase (GOT)

Procedure

GOT activity was determined according to the method of Reitman & Frankal (1957). 0.25ml buffered substrate (Aspartate and α-Ketoglutarate pH 7.4) was incubated for 5 minutes at 37°C. 50 l of serum was added to this and again incubated at 37°C for 60 minute. 0.25ml of DNPH colour reagent was added, mixed well and allowed to stand at room temperature for 20 minutes. 2.5ml of 4N Sodium hydroxide was added and allowed to stand at room temperature for 10 minutes. The optical density was read against purified water at 505nm. The enzyme activity was calculated from the
standard (Sodium pyruvate 2mM) calibration curve.

**Assay of superoxide dismutase (SOD)**

Superoxide dismutase activity was determined by NBT reduction method of McCord and Fridovich (1969). The reaction mixture contained, EDTA (6mM) containing 3 g, NaCN; riboflavin (2mM); NBT (50 M); KH$_2$PO$_4$-Na$_2$HP buffer (67mM, pH 7.8) and tissue homogenate to a final volume of 3.0 ml, the tubes were illuminated under incandescent lamp for 15 minutes. the optical density (O.D) at 530nm was measured before and after illumination. The superoxide dismutase (SOD) was determined by comparing the absorbance values of the control.

**Inhibition of tissue lipid peroxidation**

The amount of malondialdehyde (MDA) formed was quantitated by reaction with thiobarbituric acid (TBA) and used as an index of lipid peroxidation. 0.4 ml of tissue homogenate was treated with 0.2 ml SDS (8.1%); 1.5 ml TBA (0.8%); and 1.5 ml acetic acid (20%, pH 3.5). The total volume was made up to 4.0 ml with distilled water and then kept in a water bath at 95°-100°C for 1 hour. After cooling 1.0 ml distilled water and 5.0 ml n-butanol and pyridine mixture (15:1) were added to the reaction mixture, shaken vigorously and centrifuged at 4000 rpm for 10 min. The organic layer was removed and its absorbance at 532 nm was measured. Inhibition of lipid peroxidation was determined by comparing the O.D of the treatment with that of control.

**Catalase activity**

catalase activity in mice liver homogenate was determined by the method of Aebi et al. The reaction mixture contained liver homogenate 0.1 ml in 1.9 ml phosphate buffer (0.2 mM : pH 7.0) mixed H$_2$O$_2$ instantly with the reaction mixture and measured hydrogen absorbance at 240 nm for 1 minutes interval at 4 times. Catalase activity was determined by comparing the optical density of treatment with that of control.

**Assay of protein estimation**

Procedure described by Lowry et al.,(1951) was used.

**Procedure**

10 l of tissue homogenate (10%) was made up to 1.0 ml with distilled water. Mixed with 5.0ml of alkaline copper reagent, incubated at room temperature for 10 minutes in a final volume of 6.0 ml. After the incubation, 0.5ml of Follins reagent (1:1 with distilled water) was added and kept in room temperature for 30 minutes. The optical density of the coloured solution was read at 660 nm using the reagent blank. A standard curve was prepared similarly using various concentration of BSA (10-200 g/ml).

**Statiscal analysis**

All experimental data were as mean ± SD.
Results

Antioxidant activity

Super oxide radical scavenging activity

Ethanol extract of the mycelim of *Morchella esculenta* was found to be a scavenger of superoxide generated by photoreduction of riboflavin. (Table 1). The extract showed superoxide inhibiting activity. The extract inhibited 47.13% superoxide generated at a concentration of 1000 µg/ml.

Inhibition of lipid peroxidation

Ethanol extract of the mycelim of *Morchella esculenta* was effective in inhibiting the lipid peroxidation induced by Fe°₂ ascorbate system in rat liver homogenate (Table-1). The generation of malondialdehyde (MDA) and related substance that react with thiobarbituric acid (TBARS) was found to be inhibited by the extract. This indicated the significant lipid peroxidation inhibition activity of the extract. The extract inhibited 56.2% lipid peroxidation at a concentration of 1000µg/ml.

Hydroxyl radical scavenging activity

The degradation of deoxyribose TBARS by hydroxyl radical generated from Fe°₂ – ascorbate – EDTA – H₂O₂ system was markedly decreased by ethanolic extract of the mycelim of *Morchella esculenta* (Table-1). The extract inhibited 64.98% of hydroxyl radical at a concentration of 1000µg/ml. This indicated the significant hydroxyl radical scavenging activity of the extract.

Nitric oxide radical scavenging activity

Incubation of solution of sodium nitroperoxide in phosphate buffered saline at 25°C for 150 min. resulted in generation of nitric oxide. The ethanolic extract of the mycelim of *Morchella esculenta* effectively reduced the generation of nitric oxide radicals. The extract inhibited 54.07% nitric oxide radical generation at a concentration of 1000µg/ml. This showed marked nitric oxide scavenging activity of the extract (Table-1).

Hepatoprotective activity

In living system livers is considered to be highly sensitive to toxic agents. Hepatic dysfunction due to ingestion (or) inhalation of hepatotoxins are increasing world wide. Carbontetrachloride (CCl₄) is a well known dry cleaning agent, that produces acute hepatotoxicity.

In modern medicine, there is hardly any drug that stimulates liver function, offer protection to liver from damage and help regeneration of hepatic cells.

In the present study, a dose of CCl₄ was used to induce liver damage in the rats CCl₄ caused significant changes in the liver function enzymes. A significant elevation of serum marker enzymes namely GOT, GPT and ALP was observed.

Serum ALP levels registered drastic increase in CCl₄ treated rat as compared to normal. Administration of ethanolic extract of the mycelim of *Morchella esculenta* (250 and 500 mg/kg body wt.) also significantly reduced the increase in serum ALP levels (Table 2) Administration of (500 mg/kg body weight) extract reduced the increase in serum ALP levels to almost normal level.

Serum GOT and GPT levels registered a significant increase in CCl₄ treated rat as compared to normal. Administration of ethanolic extract of *Morchella esculenta* (250 and 500 mg/kg, body weight) significantly reduced the increase in serum GOT and GPT levels (Table 2).

Super oxide dismutase activities in the liver significantly decreased in CCl₄ treated rat as compared to the normal. Administration of ethanol extract of the mycelium of
Table .1 Invitro antioxidant activity of ethanol extract of mycelium of *morchella esculenta* (% inhibition)

<table>
<thead>
<tr>
<th>Activities</th>
<th>Ethanol Extract</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1000 µg/ml</td>
</tr>
<tr>
<td>Super oxide scavenging activity</td>
<td>47.13</td>
</tr>
<tr>
<td>Hydroxyl radical scavenging activity</td>
<td>64.98</td>
</tr>
<tr>
<td>Lipid Peroxidation inhibiting activity</td>
<td>56.25</td>
</tr>
<tr>
<td>Nitric oxide scavenging activity</td>
<td>54.07</td>
</tr>
</tbody>
</table>

Table.2 effect of ethanol extract of *Morchella esculenta* mycelium on hepatic enzymes consequent to the challenge with CCl₄ activities of GPT, GOT, ALP levels in serum (values are mean ± SD, for animals)

<table>
<thead>
<tr>
<th>GROUP</th>
<th>TREATMENT (mg/kg)</th>
<th>SGPT</th>
<th>SGOT</th>
<th>ALP (KA/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>73±11.08</td>
<td>128±5.53</td>
<td>139.5±11.05</td>
</tr>
<tr>
<td>Control</td>
<td>CCl₄+paraffin oil(1:1)</td>
<td>428±60.3</td>
<td>281±15.9</td>
<td>373±62.14</td>
</tr>
<tr>
<td>CCl₄ + ME</td>
<td>500</td>
<td>181.33±24.7</td>
<td>140.3±8.9</td>
<td>181±7.52</td>
</tr>
<tr>
<td>CCl₄ + ME</td>
<td>250</td>
<td>284.33±22.7</td>
<td>190±12.05</td>
<td>252.5±25</td>
</tr>
</tbody>
</table>

ME = Mycelium Extract

Table.3 effect of ethanol extract of *Morchella esculenta* mycelium on hepatic antioxidant enzyme activities and lipid peroxide (MDA) levels consequent to CCl₄ challenge (values are mean± SD, for 6 animals)

<table>
<thead>
<tr>
<th>GROUP</th>
<th>Treatment mg/kg</th>
<th>SOD (g/mg protein)</th>
<th>CAT (g/mg protein)</th>
<th>MDA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Tissue (nmole/mg protein)</td>
</tr>
<tr>
<td>Normal</td>
<td>--</td>
<td>18.25±1.57</td>
<td>63.47±5.7</td>
<td>1.30±0.32</td>
</tr>
<tr>
<td>Control</td>
<td>CCl₄+paraffin oil(1:1)</td>
<td>1.39±3.31</td>
<td>16.47±3.41</td>
<td>2.78±4.44</td>
</tr>
<tr>
<td>CCl₄ + ME</td>
<td>500</td>
<td>10.91±4.2</td>
<td>58.80±7.2</td>
<td>1.38±3.35</td>
</tr>
<tr>
<td>CCl₄ + ME</td>
<td>250</td>
<td>8.4±1.79</td>
<td>24.20±5</td>
<td>2.07±1.13</td>
</tr>
</tbody>
</table>

ME = Mycelium Extract
Morchella esculenta to CCl₄ treated animals significantly increased the SOD activities as compared to control group (Table 3). The catalase activity was significantly decreased in CCl₄ treated group as compared to normal. Administration of the extract of caused significant increase in catalase activity (Table 3). The concentration of malondialdehyde in liver and serum significantly increased in CCl₄ treated group as compared to normal. Ethanol extract of Morchella esculenta treatment significantly decreased malondialdehyde levels as compared to control group (Table 3) indicating the in vivo inhibition of lipid peroxidation.

Discussion

Reactive oxygen species (ROS) and free radicals are involved in a variety of pathological events. In addition to ROS, nitric oxide is also implicated in inflammation, cancer and other pathological conditions (Sreejayan and Rao, 1997). A potential mechanism of oxidative damage is the nitration of tyrosine residues of proteins, peroxidation of lipids, degradation of DNA and oligonucleosomal fragments (Hemanani and Prihar, 1998). Nitric oxide or reactive nitrogen species, formed during its reaction with oxygen or with superoxide, such as NO₂⁻, N₂O₄, N₃O₄ and nitrite are very reactive. These compounds alter the structure and function of many cellular components. Any compound natural or synthetic, with antioxidant properties might contribute towards the partial or total alleviation of this damage (Lin et al., 1995).

Mycelial extract of Morchella esculenta (50% ethanol) shows potent scavenging activity of free radical such superoxide, hydroxyl and nitric oxide and inhibition of lipid peroxidation activity. The significant antioxidant activity of the extract, thus suggests the possible therapeutic potentials of this mushroom.

Studies on the hepatoprotective activity in experimental model indicate that CCl₄ is first metabolized by cytochrome P₄₅₀ in the liver endoplasmic reticulum to the highly reactive CCl₃ radical. The main cause of CCl₄ induced liver injury is lipid peroxidation by free radical derivatives of CCl₄ (Recknagel, 1967).

The results of the present study reveal that the crude ethanolic extract of M. esculenta mycelium (250,500 mg/kg) have preventive action of CCl₄ induced hepatotoxicity in a dose dependent manner. The amelioration of liver toxicity by the extract is evident from its significant effect on the serum transaminases (SGOT and SGPT) and Alkaline Phosphatase (ALP) levels. The protective effect of the extract of M. esculenta on CCl₄ induced hepatotoxicity was evident from the increase in the generation of antioxidant enzymes like superoxide dismutase and catalase. The lipid peroxidation was also lowered by the administration of the extract. This indicates that antioxidant activity of the extract plays a significant role in the amelioration of hepatotoxicity induced by CCl₄. The marked in vitro antioxidant activity of the extract also support this conclusion. The experiment results reveal that ethanolic extract of the mycelium of Morchella esculenta possess profound antioxidant and hepatoprotective effects. The findings thus suggest the potential therapeutic use of moral mushroom mycelium.

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