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Research Article

Assessing the Antioxidant and Hypolipidemic Potentials of Methanolic Extract of *Syzygium Cumini* Roots

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ABSTRACT

Hypercholesterolemia, or high blood cholesterol, increases the risk of atherosclerosis and coronary artery disease. Elevated cholesterol levels, especially high LDL and low HDL, are key risk factors. LDL contributes to plaque formation through oxidative modification, while HDL helps remove cholesterol and prevent LDL oxidation. Key contributors include high-fat diets, lack of exercise, and hypertension. Management involves dietary changes, exercise, and medications such as statins, fibrates, and cholesterol absorption inhibitors. Antioxidants can reduce oxidative damage. Traditional and alternative medicine, including Ayurveda and *Syzygium cumini* (L.), offer additional therapeutic options.

Keywords: Hypercholesterolemia, LDL, HDL, atherosclerosis, coronary artery disease, oxidative modification

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INTRODUCTION

Hypercholesterolemia

High cholesterol levels in blood are linked to increased risk of atherosclerosis and coronary artery disease. Elevated cholesterol levels, particularly above 230 mg/dL, raise the risk of coronary disease by 11%, with every 10 mg/dL increase in cholesterol corresponding to

a 10% increased risk. High levels of low-density lipoprotein (LDL) are a well-established risk factor for atherosclerosis. The oxidative modification hypothesis suggests that oxidized LDL in arterial walls leads to the formation of foam cells and atherosclerotic plaques through a process involving macrophages and increased oxidative stress. [1-3]

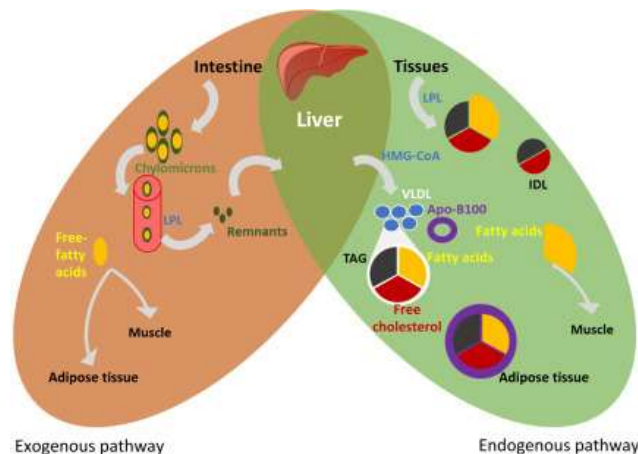


Fig.1 Production of cholesterol

High-Density Lipoprotein (HDL): HDL helps remove cholesterol from tissues and inhibits LDL oxidation, contributing to its antiatherogenic effects. Low HDL levels are linked to hypertension and diabetes, increasing atherosclerosis risk [4]

Types of Cholesterol: [5-7]

- **Triglycerides:** Controversial in atherosclerosis, but high serum levels are associated with coronary heart disease (CHD). Elevated triglycerides result from overproduction or defective metabolism of VLDL, and contribute to hypercoagulability.
- **Very Low-Density Lipoproteins (VLDL):** Transport endogenous triglycerides to tissues. VLDL remnants, converted to intermediate density lipoproteins (IDL) and then LDL, are involved in atherosclerosis.
- **Low-Density Lipoproteins (LDL):** Main carriers of cholesterol, elevated LDL increases atherosclerosis risk. LDLs are taken up by cells, where excess cholesterol can lead to atherosclerotic plaque formation.
- **High-Density Lipoproteins (HDL):** Synthesized in the liver and intestine, HDLs transport cholesterol back to

the liver. They also aid in reverse cholesterol transport and are crucial in reducing cardiovascular risk.

Etiology: [8-10]

- **High Fat Diet:** Increases cholesterol, malondialdehyde, and reactive oxygen species (ROS), leading to atherosclerosis.
- **Lack of Exercise:** Results in poor cholesterol metabolism and increased risk of cardiovascular diseases.
- **Hypertension:** Causes endothelial injury and promotes atherosclerosis through cytokine release.

Treatment [11-15]

Dietary management: Diet is the most important method in hyperlipidemia treatment. Many hyperlipidemia is caused by unscientific diet. Even those who are caused by heredity or other reasons, scientific diet treatment also achieve good result. Diet treatment has no side effect. Diet treatment can prevent other syndrome, helping to prevent coronary heart diseases, hypertension, peripheral vascular diseases and diabetes.

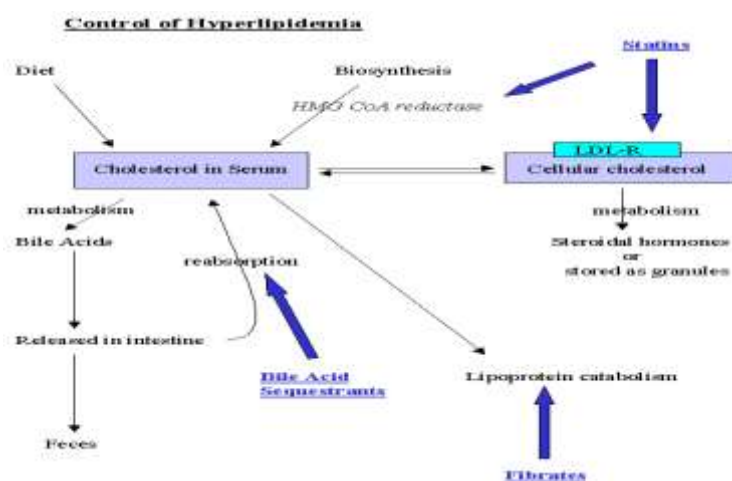


Figure-2 control of hyperlipidemia.

Exercise: Regular exercise, such as a brisk 30-minute walk 3-4 times a week, can lower LDL and increase HDL levels by burning cholesterol and fats. Patients with heart disease should consult their physician before starting an exercise program.

Drug Treatments: [16-19]

1. **Statins:** HMG-CoA reductase inhibitors (e.g., simvastatin, atorvastatin) reduce LDL-C by inhibiting cholesterol synthesis and increasing LDL receptor expression. Common side effects include mild muscle pain and digestive issues; severe side effects are rare.
2. **Fibrates:** (e.g., fenofibrate, gemfibrozil) lower VLDL and triglycerides, modestly increase HDL-C. They act as PPAR α agonists, enhancing lipoprotein lipase and reducing inflammation. Risks include myositis and gastrointestinal issues, especially in patients with renal impairment or those consuming alcohol.
3. **Cholesterol Absorption Inhibitors:** Ezetimibe reduces cholesterol absorption, increasing LDL receptor expression and lowering LDL-C. It is generally well-tolerated but can cause mild gastrointestinal symptoms.

4. **Nicotinic Acid Derivatives:** (e.g., niacin) lower triglycerides and LDL-C while increasing HDL-C. Side effects include flushing and gastrointestinal issues, but modified-release formulations offer improved tolerance.

5. **Fish Oil Derivatives:** Omega-3 fatty acids lower triglycerides but can increase cholesterol. They may reduce ischaemic heart disease risk and improve survival post-myocardial infarction, but are contraindicated in certain hyperlipoproteinaemias.

Antioxidants:

- **Definition:** Molecules that prevent or slow oxidation, thereby neutralizing free radicals and reducing cellular damage.

- **Types:**

Enzymatic Antioxidants: Include enzymes like superoxide dismutase and catalase.

Non-Enzymatic Antioxidants: Include vitamins, polyphenols, and thiols.

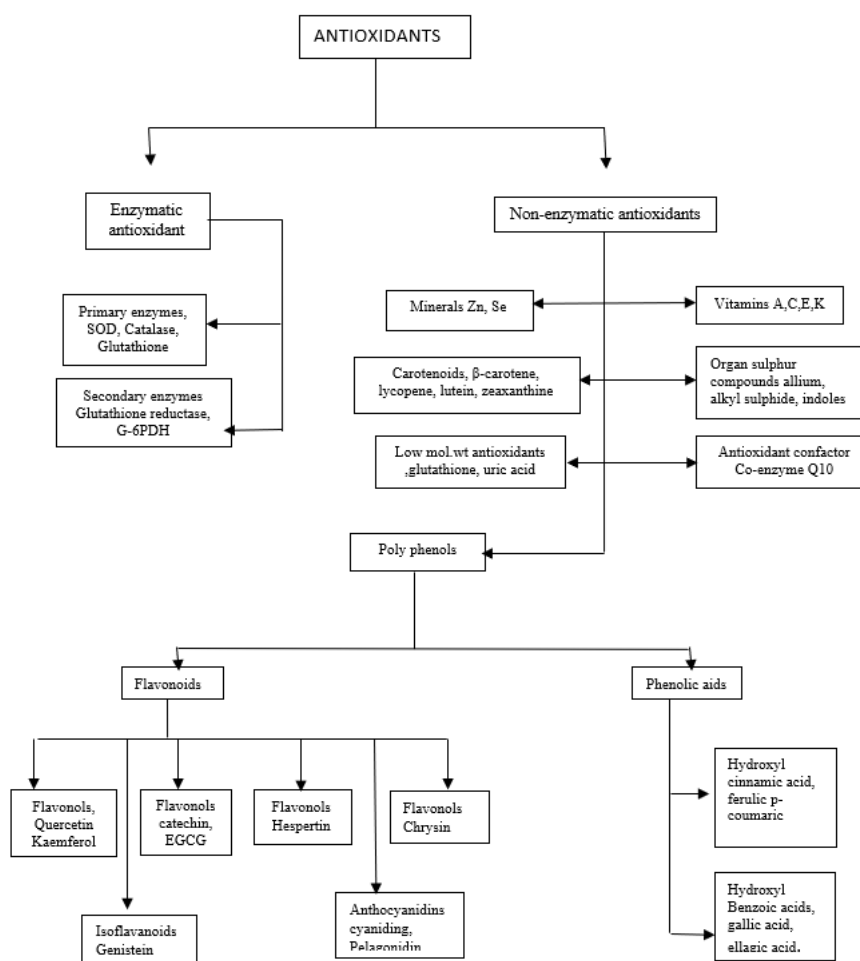


Fig- 3 Types of Antioxidants

Traditional Medicine:

Plants have been used as medicines for thousands of years in various forms such as tinctures, teas, and powders. Knowledge about medicinal plants was

traditionally passed down orally and later recorded in herbal pharmacopoeias. Modern medicine continues to draw from these ancient practices, with many remedies derived from traditional knowledge [20, 21].

Ayurveda:

Ayurveda, one of the oldest medicinal traditions, originated in India and emphasizes holistic balance and harmony. It predates traditional Chinese medicine and is rooted in ancient Hindu texts like the Vedas. Ayurveda focuses on maintaining balance through body humours (dosas) and the life force (prana). The practice aims to restore balance among the elements (earth, water, and fire, and air, space) to treat illness [22-24].

Homeopathy:

Developed in the 18th century by Samuel Hahnemann, homeopathy is based on the principle of "like cures like" (similia similibus curentur). It uses highly diluted substances to treat symptoms similar to those caused by the substance. Treatments are customized based on the patient's symptoms and are prepared through a process of dilution and succussion (vigorous shaking). Common homeopathic remedies include Nux vomica, thuja, and aconite [25, 26].

Plant Profile: *Syzygium cumini* (L.) Skeels [27-29]

- **Common Names:** Jambu, Java Plum, Jamun, Black Plum, Java Apple
- **Family:** Myrtaceae
- **Native Regions:** Indian subcontinent, Southeast Asia (Myanmar, Sri Lanka, Bangladesh, Andaman Islands)
- **Description:** Evergreen tropical tree, up to 30 m (98 ft) tall, living over 100 years. Introduced to many tropical and subtropical regions worldwide.
- **Uses:** Edible fruit, timber for railway sleepers, furniture, and village dwellings. Fruits are eaten by birds and small mammals.
- **Botanical Features:**
 - **Bark:** Rough, dark grey at the base, lighter and smoother higher up.
 - **Leaves:** Opposite, coriaceous, dark green, with a yellow midrib. Aromatic when young, used as livestock feed.
 - **Flowers:** Small, fragrant, white or pink, with a cup-like calyx and many stamens. Blooming from February to April.
 - **Fruit:** Ovoid-oblong berries, green when unripe, turning pink and then crimson to black. Juicy, with a combination of sweet, mildly sour, and astringent flavors.
- **Significance:** Cultivated for its edible fruits and medicinal uses. Seed powder is used to manage diabetes. Wood used for agricultural implements and construction.

MATERIAL AND MATHODOLOGY

Animals

Healthy, adult male rats of Wister strain, weighing 180 ± 5 gm was obtained from animal house. The animals were kept in a well-ventilated room and they were exposed to 12 hours day and 12 hours night cycle with a temperature between $20 \pm 2^\circ\text{C}$. The animals were housed in large spacious, hygienic polypropylene cages during the course of the experimental period. The animals were fed with water and mice pellet feed *ad libitum*, supplied by M/s. Hindustan Lever Ltd.,

Mumbai. All the experimental procedures and protocols used in this study were reviewed by institutional animal ethics committee and were in accordance with the guidelines of the CPCSEA.

Acute toxicity studies [30-33]

Acute toxicity was performed according to organization for economic cooperation and development guidelines.

1. Male swiss mice was selected by random sampling technique.
2. The animals were fasted for 4 hours with free study. *Syzygium cumini* roots extract was administered orally at a dose of 5mg/kg initially and mortality if any was observed for 3 days.
3. If mortality was observed in two out of three animals, then the dose administered was considered as toxic dose however, if the mortality was observed in only one animal out of three animals then the same dose was repeated again to conform the toxic effect.
4. If no mortality was observed, then only higher (50, 300, 2000 mg/kg) dose of *Syzygium cumini* roots extract were employed for further toxicity studies.

PHARMACOLOGICAL ACTIVITIES METHANOLIC EXTRACT OF SYZYGIUM CUMINI ROOTS.

Antioxidant Activity of Methanolic extract of *Syzygium cumini* roots.

Animal's treatment: Rats were randomly divided into five groups of six animals. Each group was kept in a separate cage.

Group I - Served as normal control and only treated (orally) with vehicle (0.5% Carboxyl methyl cellulose) for seven days.

Group II - Served as toxin control and treated (orally) with vehicle for seven days.

Group III - Served as standard and treated (orally) with Silymarin 25 mg/kg for seven days.

Group IV - Treated (orally) with 200 mg/kg SYZYGIUM CUMINI ROOTS methanolic extract for seven day by suspending in 0.5% Carboxyl methyl cellulose.

Group V - Treated (orally) with 400 mg/kg SYZYGIUM CUMINI ROOTS methanolic extract for seven days by suspending in 0.5% Carboxyl methyl cellulose.

On the 6th day, Rats of group II, III, IV and V were treated with a single dose of Thioacetamide (100 mg/kg s. c.) as 2 % w/v solution in double distilled water.

On the 8th day, the rats were anesthetized with light ether anesthesia and blood samplings was performed by cardiac puncher. The collected blood samples were allowed to clot for 45min at room temperature. Serum for separated by centrifugation at 4000 rpm for 20 min. serum enzymes like SGOT (MOD.IFCC method); SGPT (Modified IFCC method) and ALP (PNPP Kinetic Method) values were evaluated by using Biochemical kits.

After collection of blood samples, animals were sacrificed by cervical decapitation under excessive anaesthesia on the 8th day. Immediately after sacrifice, the liver was dissected out, washed in the ice-cold saline, and the homogenate was prepared in 0.1M Tris-HCl buffer (pH 7.4). The homogenate was centrifuged

and the supernatant liquid was used for the assay of marker enzymes, namely Glutathione peroxidase [34-38, 52, 53].

Estimation of Antioxidant enzyme of Methanolic extract of *Syzygium cumini* roots on thioactamide intoxicated rats [39-46, 51]

Determination of Lipid Peroxidation (LPO) activity
LPO were estimated by the method of Jiang *et al.*, (1992). 1.8 ml of the Fox reagent was mixed with 0.2 ml of the tissue homogenate/plasma and incubated for 30 min at room temperature. The colour developed was read at 560 nm.

Values were expressed as $\times 10^{-5}$ mM/dL plasma and mmol/100g wet tissue.

Determination of glutathione Reductase (GRD) activity

Reduced glutathione (GSH) was estimated by the method of Ellman, (1959). A known weight of tissue was homogenized in phosphate buffer. From above, 0.5 ml was pipetted out and precipitated with 2.0 ml of 5% TCA. 1.0 ml of the supernatant was taken after centrifugation/0.5 ml of plasma and was added to it 0.5 ml of Ellman's reagent and 3.0 ml of phosphate buffer. The yellow colour developed was read at 412 nm. A series of standards were treated in a similar manner along with a blank containing 3.5 ml of buffer.

The amount of glutathione was expressed as mg/dL plasma or mmol/g wet tissue.

Determination of glutathione peroxidase (GPX) activity

Glutathione peroxidase in the heart was assayed by the method of Rotruck *et al.*, (1973). To 0.2 ml of tris buffer, 0.2 ml of EDTA, 0.1 ml of sodium azide and 0.5 ml of tissue homogenate was added. To the mixture, 0.2 ml of glutathione followed by 0.1 ml of H_2O_2 was also added. The contents were mixed well and were incubated at 37°C for 10 min along with a tube containing all the reagents except the sample. After 10 min, the reaction was arrested by the addition of 0.5 ml of 10% TCA, centrifuged and the supernatant was used for the estimation of glutathione by the method of Ellman (1973).

The activity of GPx was expressed as μ g of GSH consumed/min/mg protein.

Determination of glutathione S-transferase

Glutathione S-transferase activity in the heart was assayed by the method of Habig and Jackoby (1981). The reaction mixture containing 1.0 ml of buffer, 0.1 ml of CDNB and 0.1 ml of enzyme was made up to 3.0 ml with water. The reaction mixture was pre incubated at 37°C for 5 min. 0.1 ml of GSH was added and change in the absorbance was measured at 340 nm for 3 min at 30 sec intervals in a Systronics UV-visible spectrophotometer.

The activity of GST was expressed as nmoles of CDNB conjugated/min/mg protein

Determination of superoxide dismutase (SOD) activity

The activity of SOD in the heart was determined according to the procedure of Kakkar *et al.*, (1973). 0.5 ml of tissue homogenate was diluted to 1.0 ml with water. Then 2.5 ml of ethanol and 1.5 ml chloroform (all reagents chilled) was added. This mixture was shaken for one minute at 4°C and then centrifuged. The enzyme activity in the supernatant was determined.

The assay mixture contained 1.2 ml of sodium pyrophosphate buffer (0.025 M, pH 8.3), 0.01 ml of 186 μ M phenazine methosulphate, 0.3 ml of 30 μ M nitroblue tetrazolium, 0.2 ml of 780 μ M NADH, appropriately diluted enzyme preparation and distilled water in a total volume of 3.0 ml. Reaction was initiated by the addition of NADH. After incubation at 30°C for 90 sec., the reaction was stopped by the addition of 1.0 ml glacial acetic acid. The reaction mixture was stirred vigorously and shaken with 4.0 ml of n-butanol. The intensity of the chromogen in the butanol layer was measured at 560 nm against butanol blank. A system devoid of enzyme served as control.

One unit of the enzyme activity is defined as the enzyme concentration required to inhibit the optical density at 560 nm of chromogen production by 50% in 1 min.

Determination of catalase (CAT) activity

The activity of catalase in the heart was assayed by the method of Sinha, (1972). To 0.9 ml of phosphate buffer, 0.1 ml of tissue homogenate and 0.4 ml of hydrogen peroxide were added. After 60 s, 2.0 ml of dichromate acetic acid mixture was added. The tubes were kept in a boiling water bath for 10 min and the colour developed was read at 620 nm. Standards in the range of 2-10 μ M were taken and preceded as test with a blank containing reagent alone.

The activity of catalase was expressed as μ moles of H_2O_2 consumed/min/mg protein.

Serum Glutamate oxaloacetate transaminase (SGOT)

SGOT in serum was estimated by MOD-IFCC method using an Asritha Diagnostic Kit.

Pipetted out the serum samples into a clean dry test tube labeled as test. The test sample tube contains working reagent and serum sample. Mixed well and the initial absorbance after 1 min. at 340nm and repeated the absorbance reading after 1, 2 & 3 min. calculate the mean absorbance changed per min. ($\Delta A/\text{min}$).

Calculation:

SGOT activity in U/L 37°C = $\Delta A/\text{min} \times 1746 \times \text{Tf}$

Serum Glutamate pyruvate transaminase (SGPT)

SGPT in serum was estimated by MOD-IFCC method using an Asritha Diagnostic Kit.

Pipetted out the serum sample into a clean dry test tube labeled as test. The test sample tube containing working reagent and incubated at the assay temperature for 1 min. at 340 nm then added sample (serum). Mix well and read the initial absorbance after 1 min. and repeated the absorbance changed per min. ($\Delta A/\text{min}$).

Calculation:

SGPT activity in U/L 37°C = $\Delta A/\text{min} \times 1746$.

Alkaline phosphatase (ALP)

ALP in serum was estimated by PNPP kinetic method using an Asritha Diagnostic Kit.

Pipetted out the serum sample into a clean dry test tube labeled as test. The test sample tube containing working reagent and incubate at the assay temperature for 1 min. at 405 nm then add sample (serum). Mixed well and read the initial absorbance after 1 min. and repeated the absorbance changed per min. ($\Delta A/\text{min}$).

Calculation:

ALP activity in U/L 37°C = $\Delta A/\text{min} \times 2754 \times \text{tf}$.

Statistical analysis

The collected data were subjected to appropriate statistical test like one-way ANOVA (Analysis of variance), followed by an appropriate turkey test. P values of less than 0.01 were considered as significant. The analysis was carried out using Graph pad prism software of version 4.

RESULT AND DISCUSSION

Table -1 Result of phytochemical analysis of methanolic extract of *Syzygium cumini* roots.

Component	Present (+) or absent (-)
Alkaloids	+
Carbohydrates	+
Flavonoids	+
Tannins	+
Amino acids	+
Saponins	-
Triterpens	-

On the base of above results, chromatography was performed for the detection of active compound in the extract.

Thin layer chromatography (TLC) has been performed with the above solvent system and we found that solvent system {Chloroform +Ethyl acetate (10:1)} show single spot on the TLC plate.



Figure 5. TLC of *Syzygium cumini* roots extract.

Result of the chemically intoxicated and its associated Antioxidant Activity of Methanolic extract of *Syzygium cumini* roots on rats are shown in Table and some figures.

LIPID PEROXIDATION (LPO)

LPO activity was significantly ($P < 0.001$) increased in Thioacetamide treatment when compared to control. The SCRE (200 mg/kg and 400 mg/kg dose) significantly decreased ($P < 0.001$) LPO in liver homogenate when compared to Thioacetamide treated animals. Silymarin treated group also showed significant ($P < 0.001$) decreased of LPO when compared to Thioacetamide treated animals.

GLUTATHIONE PEROXIDASE (GPX)

GPX activity in liver homogenates was significantly ($P < 0.001$) reduced in Thioacetamide treated animals when compared to control. The SCRE treatment (200 mg/kg and 400 mg/kg dose level) significantly increased ($P < 0.001$) the GPX level when compared to Thioacetamide treated animals. Silymarin treated animals also showed significant ($P < 0.001$) increases of GPX level in the liver homogenate compared with Thioacetamide treated animals.

GLUTATHIONE S-TRANSFERASE (GST)

GST level in liver was significantly reduced ($P < 0.001$) in Thioacetamide treated animals when compared with normal animals. Treatment with SCRE at 200 mg/kg and 400 mg/kg dose showed significant increase (P

<0.001) the GST level when compared to Thioacetamide treated group. Silymarin treated animals also showed significant ($P < 0.001$) increases of GST level in the liver homogenate compared with Thioacetamide treated animals.

GLUTATHIONE REDUCTASE (GRD)

GRD activity was significantly decreased ($P < 0.001$) in Thioacetamide treated animals when compared to control. A significant increase ($P < 0.001$) in the level of GRD was observed in SCRE (200 mg/kg and 400 mg/kg) treated rats when compared with Thioacetamide treated animals. Silymarin treated group also showed significant ($P < 0.001$) increase in the level of GRD when compared to Thioacetamide treated animals.

SUPEROXIDE DISMUTASE (SOD)

SOD level was significantly reduced ($P < 0.001$) in Thioacetamide treated animals when compared with normal animal. The SCRE (200 mg/kg and 400 mg/kg) showed significant increase ($P < 0.001$) in SOD when compared to Thioacetamide treated animals. Silymarin treated animals also showed significant ($P < 0.001$) increase in SOD when compared to Thioacetamide treated animals.

CATALASE (CAT)

CAT activity was significantly ($P < 0.001$) reduced in Thioacetamide treatment when compared to control. The SCRE (200 mg/kg and 400 mg/kg dose) significantly increased ($P < 0.001$) CAT in liver homogenate when compared to Thioacetamide treated animals. Silymarin treated group also showed significant ($P < 0.001$) increase of CAT when compared to Thioacetamide treated animals.

HYPOLIPIDEMIC ACTIVITY OF METHANOLIC EXTRACT OF SYZYGIUM CUMINI ROOTS ON NORMAL RATS

Hyperlipidemia is a major risk factor for coronary artery disease and is leading cause of death. Major component of total cholesterol is LDL-cholesterol, which leads to atherosclerosis. Dietary wheat bran has been known to modulate hypolipidemic effect of fish oils in rats.

Glutamate oxaloacetate transaminase (SGOT) activity of *Syzygium cumini* roots extract on thioacetamide-intoxicated rats

SGOT activity was significantly ($P < 0.001$) increased in Thioacetamide treatment when compared to control. The SCRE (200 mg/kg and 400 mg/kg dose)

significantly decrease ($P < 0.001$) SGOT in blood serum when compared to Thioacetamide treated animals. Silymarin treated group also showed significant ($P < 0.001$) decrease of SGOT when compared to Thioacetamide treated animals.

Glutamate pyruvate transaminase (SGPT) activity of *Syzygium cumini* roots extract on thioacetamide-intoxicated rats:

SGPT activity was significantly ($P < 0.001$) increased in Thioacetamide treatment when compared to control. The SCRE (200 mg/kg and 400 mg/kg dose) significantly decrease ($P < 0.001$) SGPT in blood serum when compared to Thioacetamide treated animals. Silymarin treated group also showed significant ($P < 0.001$) decrease of SGPT when compared to Thioacetamide treated animals.

Alkaline phosphatase (ALP) activity of *Syzygium cumini* roots extract on thioacetamide-intoxicated rats

ALP activity was significantly ($P < 0.001$) increased in Thioacetamide treatment when compared to control. The SCRE (200 mg/kg and 400 mg/kg dose) significantly decrease ($P < 0.001$) ALP in blood serum when compared to Thioacetamide treated animals. Silymarin-treated group also showed significant ($P < 0.001$) decrease of ALP when compared to Thioacetamide treated animals.

In the present study pretreatment with methanolic extract was found to significantly reverse the Thioacetamide rise in the biochemical parameter like SGOT, SGPT and ALT level, there by demonstrating the membrane stabilizing activity of the extract. The activities of SGPT and SGOT were almost brought down to normal suggesting the membrane stabilizing effect of the extract. The difference between group II (Thioacetamide alone) and group V (extract pretreated followed by Thioacetamide) was found to be statistically significant. The level of ALP, which was elevated it was also brought down in the rats pretreated with the extract followed by Thioacetamide.

Organ collection and histopathological studies

At the ends of 7th day study. The animals were sacrificed with excess dose of anesthetic ether. Animals were dissected; liver was collected and weighed. Then the organs were stored in 10% buffered neutral formalin. The tissues were embedded in liquid paraffin, cut in to 5-6 μ m and stained with haemotoxylin and eosin stain for histopathological findings.

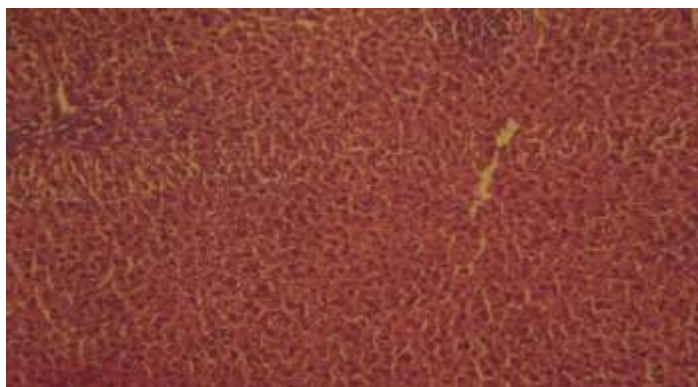


Figure 6. Histopathology of Control group: treated with CMC only.

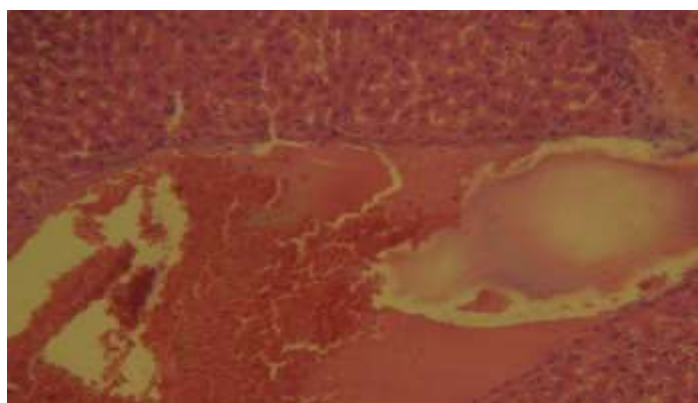


Figure 7. Histopathology of Toxic Control: treated with Thioacetamide.



Figure 8. Histopathology of Standard: treated with Thioacetamide along with Silymarin.

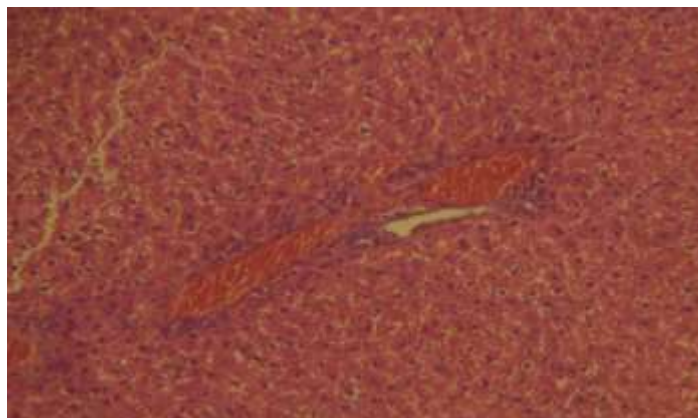


Figure 9. Histopathology of 200 mg/kg: treated with Thioacetamide along with *Syzygium cumini* roots extract.

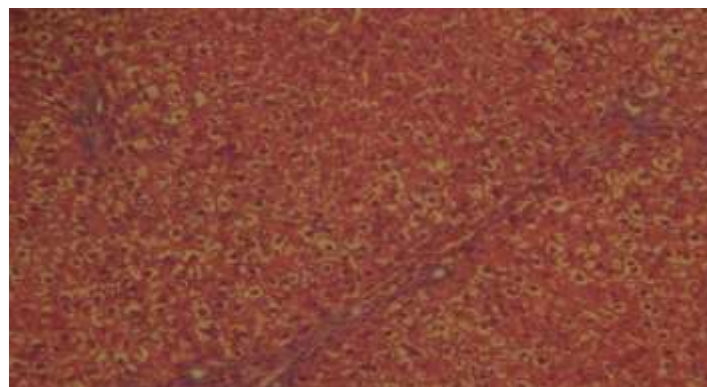
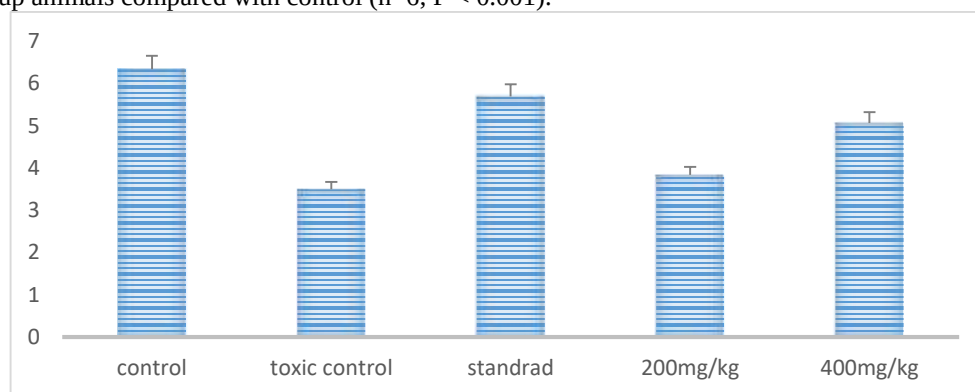


Figure 10. Histopathology of 400mg/kg: treated with Thioacetamide along with *Syzygium cumini* roots extract.

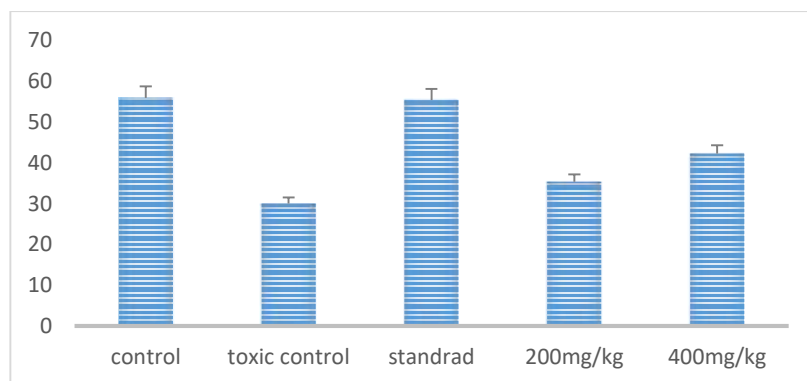
Results are expressed as mean $\pm$ SEM of 6 animals.

A treated group animals compared with control (n=6, $P < 0.001$).



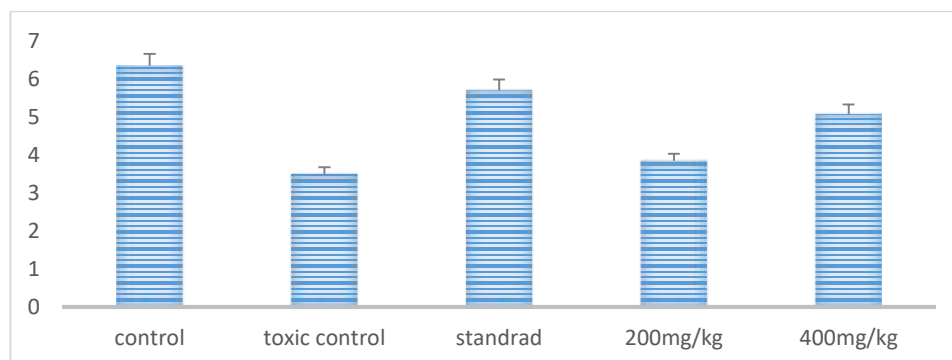
Toxic control (Thioacetamide 100mg/kg s.c), standard (silymarin 25mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Fig -11. Superoxide dismutase (SOD) activity of *Syzygium cumini* roots extract on Thioacetamide-intoxicated rats:



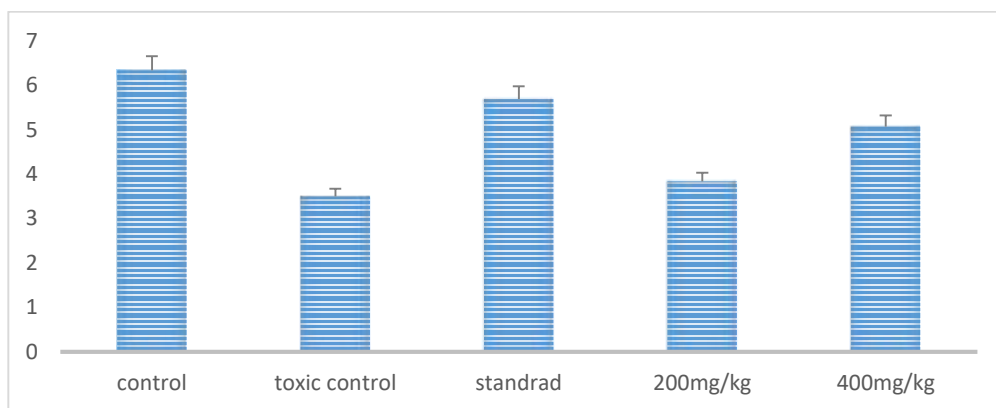
Toxic control (Thioacetamide 100mg/kg s.c), standard (silymarin 25mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 12. Catalase (CAT) activity of *Syzygium cumini* roots extract on Thioacetamide-intoxicated rats:



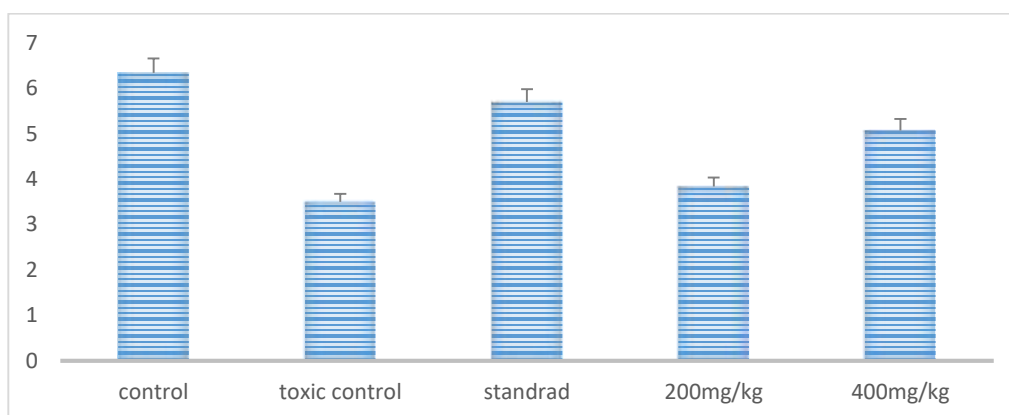
Toxic control (Thioacetamide 100mg/kg s.c), standard (silymarin 25mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 13. Glutathione reductase (GRD) activity of *Syzygium cumini* roots extract on Thioacetamide-intoxicated rats:



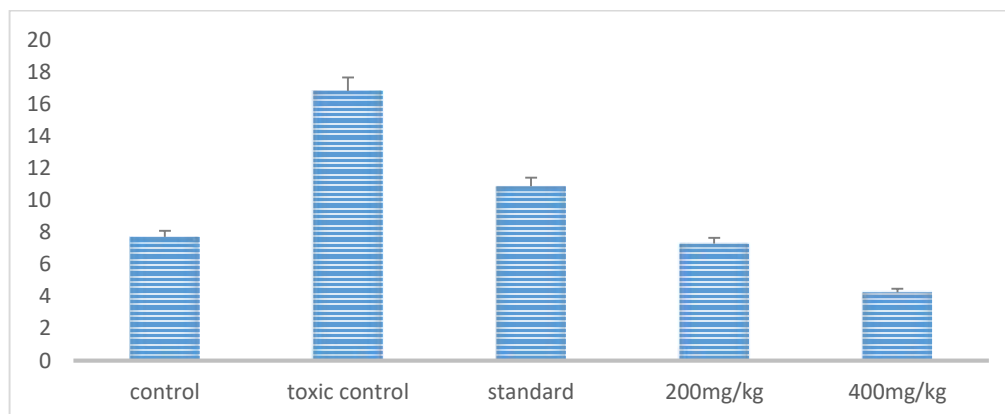
Toxic control (Thioacetamide 100mg/kg s.c), standard (silymarin 25mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 14. Glutathione peroxidase (GPX) activity of *Syzygium cumini* roots extract on Thioacetamide-intoxicated rats:



Toxic control (Thioacetamide 100mg/kg s.c), standard (silymarin 25mg/kg), 200mg/kg (SCRE), 400 mg/kg (SCRE)

Figure 15. Glutathione S-transferase (GST) activity of *Syzygium cumini* roots extract on Thioacetamide-intoxicated rats:

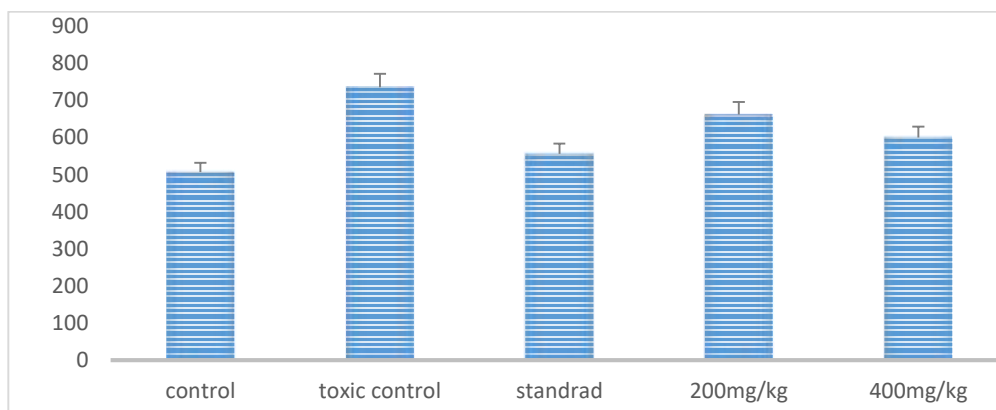


Toxic control (Thioacetamide 100mg/kg s.c), standard (silymarin 25mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 16. Lipid peroxidation (LPO) activity of *Syzygium cumini* roots extract on Thioacetamide-intoxicated rats:

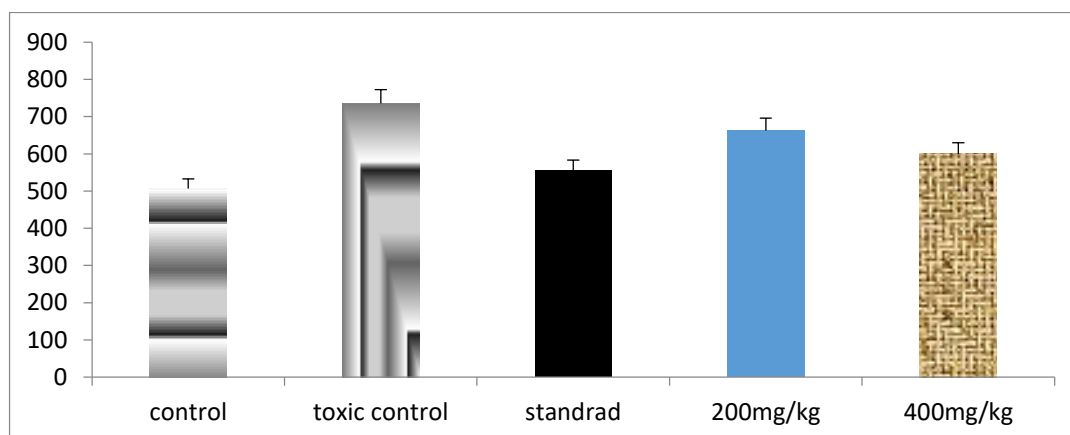
Results are expressed as mean $\pm$ SEM.

A treated group animals compared with control (n=6, P < 0.001).



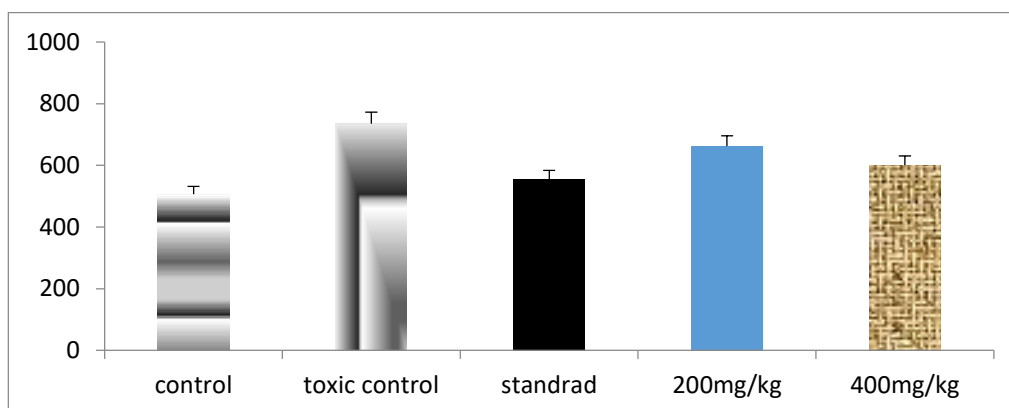
Toxic control (Thioacetamide 100mg/kg s.c), standard (silymarin 25mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 17. Serum Glutamate pyruvate transaminase (SGPT) activity of *Syzygium cumini* roots extract on Thioacetamide-intoxicated rats:



Toxic control (Thioacetamide 100mg/kg s.c), standard (silymarin 25mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 18. Serum Glutamate oxaloacetate transaminase (SGOT) activity of *Syzygium cumini* roots extract on Thioacetamide-intoxicated rats:



Toxic control (Thioacetamide 100mg/kg s.c), standard (silymarin 25mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 19. Alkaline phosphatase (ALP) activity of *Syzygium cumini* roots extract on Thioacetamide-intoxicated rats:

The results of Hypolipidemic activity of *Syzygium cumini* roots extract (SCRE) on Normal rats are shown in Table 4 and some figure.

Estimation of Triglycerides

In the triglyceride concentration from the serum in the normal animal, which received only the solvent, had the value of 284.59 ± 15.49 . The animals treated with standard showed the value of 178.03 ($P < 0.001$), whereas animals treated with *Syzygium cumini* roots extract at a dose of 200 mg/kg and 400 mg/kg had showed the value of 222.08 ± 6.14 ($P < 0.05$) and 189.61

± 7.79 ($P < 0.01$) correspondingly. The above values showed that *Syzygium cumini* roots extract treatment lowered the normal triglyceride level in experimental rats.

Estimation of Total Cholesterol

In the total cholesterol concentration from the serum in the normal animal, which received only the solvent, had

the value of 316.40 ± 7.98 . The animals treated with standard showed the value of 137.75 ± 13.83 ($P < 0.001$). whereas animals treated with *Syzygium cumini* roots extract at a dose of 200 mg/kg and 400 mg/ kg had showed the value of 253.12 ± 6.50 ($P < 0.01$) and 191.66 ± 10.42 ($P < 0.001$) correspondingly. The above values showed that *Syzygium cumini* roots extract treatment lowered the normal total cholesterol level in experimental rats.

Estimation of HDL cholesterol

In the HDL cholesterol concentration from the serum in the normal animal, which received only the solvent, had the value of 254.79 ± 16.80 . The animals treated with standard showed the value of 66.82 ± 5.86 ($P < 0.001$). whereas animals treated with *Syzygium cumini* roots extract at a dose of 200 mg/kg and 400 mg/ kg had showed the value of 186.19 ± 15.40 ($P < 0.05$) and 106.24 ± 17.77 ($P < 0.001$) correspondingly. The above values showed that *Syzygium cumini* roots extract treatment lowered the normal HDL cholesterol level in experimental rats.

Estimation of VLDL cholesterol

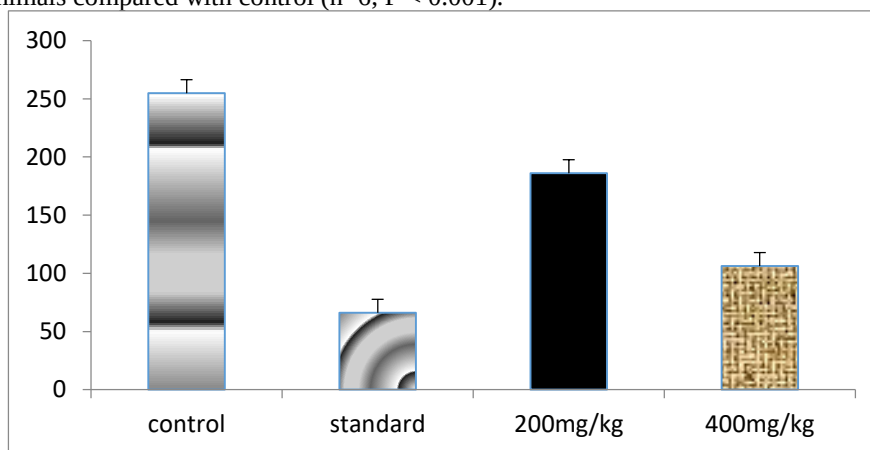
In the VLDL cholesterol concentration from the serum in the normal animal, which received only the solvent, had the value of 56.85 ± 3.16 . The animals treated with standard showed the value of 35.60 ± 2.62 ($P < 0.001$). whereas animals treated with *Syzygium cumini* roots extract at a dose of 200 mg/kg and 400 mg/ kg had showed the value of 44.41 ± 1.22 ($P < 0.05$) and 37.92 ± 0.90 ($P < 0.001$) correspondingly. The above values showed that *Syzygium cumini* roots extract treatment lowered the normal VLDL cholesterol level in experimental rats.

Estimation of LDL cholesterol

In the LDL cholesterol concentration from the serum in the normal animal, which received only the solvent, had the value of 118.46 ± 15.96 . The animals treated with standard showed the value of 108.15 ± 11.59 . Whereas animals treated with *Syzygium cumini* roots extract at a dose of 200 mg/kg and 400 mg/ kg had showed the value of 111.35 ± 16.18 and 123.30 ± 10.96 correspondingly. The above values showed that *Syzygium cumini* roots extract treatment did not produce any significant activity in LDL cholesterol level in experimental rats.

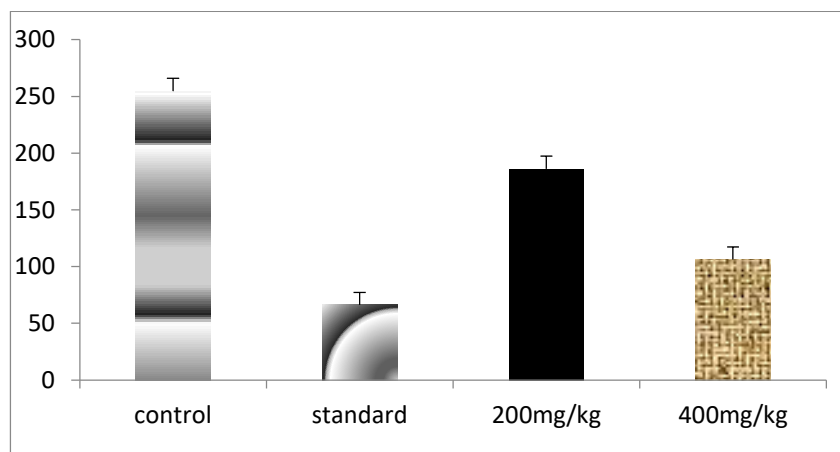
Results are expressed as mean $\pm$ SEM.

A treaded group animals compared with control (n=6, $P < 0.001$).



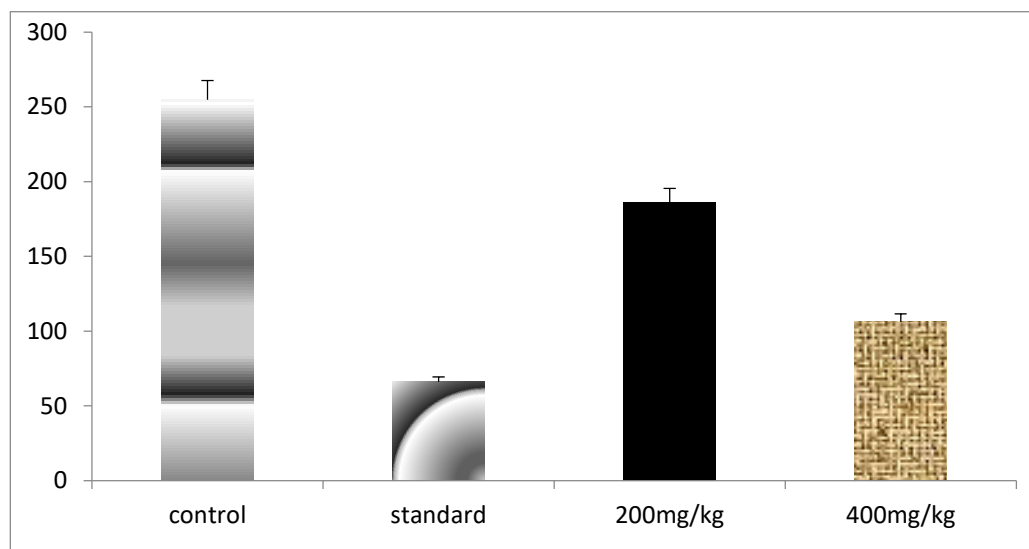
Standard (Lovastatin 7.2 mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 20. Estimation of total cholesterol from the blood serum of the normal rats:



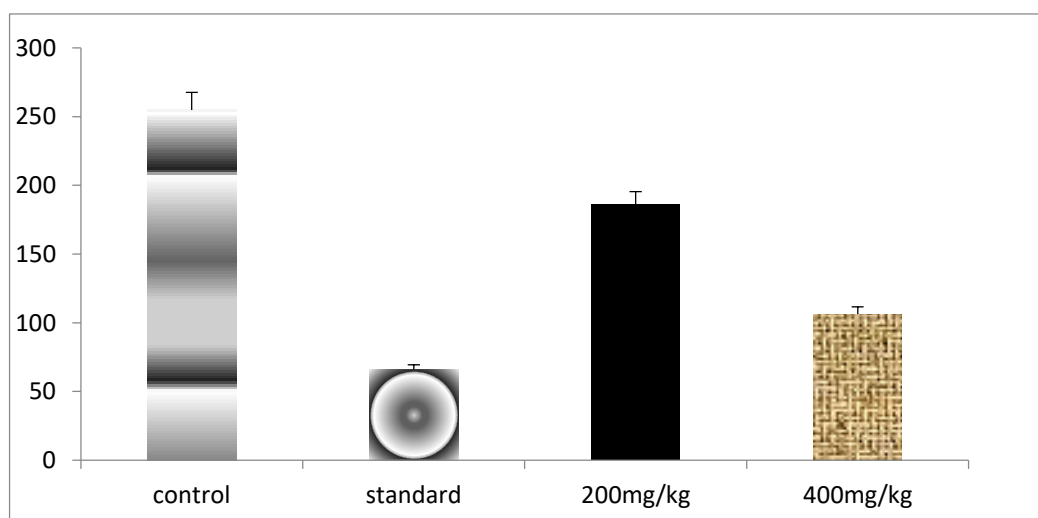
Standard (Lovastatin 7.2 mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 21. Estimation of HDL cholesterol from the blood serum of the normal rats:



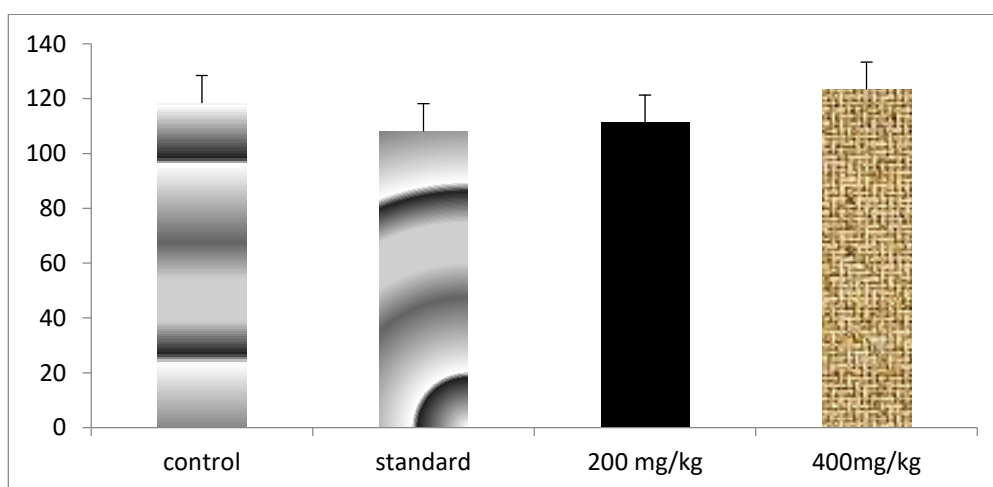
Standard (Lovastatin 7.2 mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 22. Estimation of triglyceride from the blood serum of the normal rats:



Standard (Lovastatin 7.2 mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 23. Estimation of VLDL cholesterol from the blood serum of the normal rats:



Standard (Lovastatin 7.2 mg/kg), 200mg/kg (SCRE), 400mg/kg (SCRE)

Figure 24. Estimation of LDL cholesterol from the blood serum of the normal rats:

SUMMARY AND CONCLUSION

Plant for the present studies was selected on the bases of literature review. The collected Roots were powdered in

hand grinder and the methanolic extract were prepared. This extract was used for further studies. Phytochemical studies had been performed on the extract and it was

found to have alkaloids, carbohydrates, flavonoids, tannins, triterpens and amino acids. TLC was performed by using chloroform + ethyl acetate (10:1 v/v) [47].

Dose of the extract was selected on the bases of previous work done on the *Syzygium cumini* roots. Dose was calculated on the bases of acute toxicity studies. Antioxidant activity of the methanolic extract of *Syzygium cumini* roots against Thioacetamide induced liver damaged rats were done after 7 days oral administration drug. On the next day blood was collected and animals were sacrificed and livers were removed immediately. The serum was separated from blood and serum enzyme like SGOT, SGPT and ALP had been estimated and livers were homogenate with tris- HCl buffer and centrifuged it. Supernatant was used for the estimation of liver enzyme like Glutathione peroxidase, glutathione S-transferase, Glutathione reductase, superoxide dismutase, catalase and LPO [48]. After getting data statistical analysis was performed and calculated significant value and I found that higher dose of extract was showing higher significant as compared lower dose.

Hypolipidemic activity also was performed with *Syzygium cumini* roots extract on the normal rats. Animals were treated with extract for 7 days and after seven days blood samples was collected and serum was separated. Lipid profile like Total cholesterol, triglyceride, HDL, LDL and VLDL was estimated by using biochemical kits [49].

After getting data, statistical analysis was performed and significant values was calculated and it was found that extract treated groups was showing significant decrease the level of total cholesterol, triglyceride, HDL and VLDL. Higher dose was showing more significant as compared lower dose [50].

In conclusion, the plasma level triglyseride (TG), total cholesterol (TC), LDL-cholesterol decreased after 7 days supplementation of *Syzygium cumini* roots extract in normal rats . decreased total cholesterol and TG levels in blood indicate the production of oxygen free radical was effectively inhibited by *Syzygium cumini* roots extract .so that I could say that methanolic extract of *Syzygium cumini* roots shows the antioxidant and hypolipidemic activity

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