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Research Article

EVALUATION OF HYPOLIPIDEMIC POTENTIAL OF
TRICHOLOMA MATSUTAKE ON WISTAR ALBINO RATSChintana B N^{*1}, Suresha B S², T Balasubramanian³, Deepika J K⁴^{1,4}Department of Pharmacology, Bharathi College of Pharmacy, Bharathinagara, Karnataka, India.²Professor and Head, Department of Pharmacology, Bharathi College of Pharmacy, Bharathinagara, Karnataka, India.³Principal, Department of Pharmacology, Bharathi College of Pharmacy, Bharathinagara, Karnataka, India.**Abstract:**

Hyperlipidemia is an important risk factor for cardiovascular diseases and is characterized by abnormalities in serum lipid levels. *Tricholoma matsutake* is an edible medicinal mushroom containing various bioactive constituents with potential health benefits. The present study was undertaken to evaluate the hypolipidemic activity of the ethyl acetate extract of *T. matsutake* (EAETM) in high-fat diet (HFD)-induced hyperlipidemic Wistar albino rats. The ethyl acetate extract was prepared by Soxhlet extraction. Thirty male Wistar albino rats were divided into five groups (n=6), and hyperlipidemia was induced using a high-fat diet consisting of vanaspati ghee and coconut oil (3:1, v/v) for six weeks. EAETM was administered orally at doses of 200 and 400 mg/kg during the final three weeks, while atorvastatin (10 mg/kg) was used as the standard drug. Serum total cholesterol (TC), triglycerides (TG), HDL-C, LDL-C, VLDL-C and atherogenic index (AI) were evaluated. The HFD produced a significant alteration in the serum lipid profile, characterized by increased TC, TG, LDL-C, VLDL-C and AI and decreased HDL-C. Treatment with EAETM significantly improved these parameters compared with the HFD control group. At 200 and 400 mg/kg, EAETM reduced TC from 139.51 ± 6.12 to 113.32 ± 6.69 and 104.62 ± 4.89 mg/dL, respectively, while TG decreased from 141.67 ± 5.81 to 118.84 ± 5.96 and 110.39 ± 5.97 mg/dL. HDL-C increased from 23.31 ± 1.54 to 33.80 ± 2.13 and 36.52 ± 1.92 mg/dL, respectively. LDL-C and AI were also reduced following EAETM treatment, with the 400 mg/kg dose showing greater numerical improvement. These findings indicate that EAETM possesses hypolipidemic activity in HFD-induced hyperlipidemic rats and may have potential as a natural source for lipid management. Further studies are required to establish the underlying mechanisms and therapeutic potential.

Keywords: *Tricholoma matsutake*, hypolipidemic activity, high-fat diet, Wistar rats, serum lipids.

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INTRODUCTION:

Hyperlipidemia is a metabolic disorder characterized by an abnormal elevation of one or more plasma lipids, including triglycerides (TG), total cholesterol (TC), cholesterol esters and phospholipids, together with alterations in plasma lipoproteins such as very-low-density lipoprotein (VLDL) and low-density lipoprotein cholesterol (LDL-C), often accompanied by reduced levels of high-density lipoprotein cholesterol (HDL-C). Although lipids and lipoproteins are essential for maintaining normal physiological and microvascular functions, their persistent elevation can adversely affect vascular homeostasis. Elevated cholesterol levels, particularly hypercholesterolemia, have been associated with reduced coronary flow reserve, diminished capillary density and endothelial cell apoptosis, contributing to microvascular dysfunction. Persistent disturbances in lipid metabolism are closely associated with the development and progression of atherosclerosis and may subsequently impair cardiovascular function, thereby increasing the risk of cardiovascular diseases. Consequently, effective management of abnormal lipid levels is an important component of cardiovascular disease prevention [1,2].

Dietary factors, particularly excessive intake of dietary fat, can contribute to disturbances in lipid metabolism. High-fat diet (HFD)-induced dyslipidemia is widely used in experimental animals to investigate alterations in lipid metabolism and to evaluate potential hypolipidemic interventions. HFD feeding can produce elevations in circulating cholesterol and triglycerides and alterations in lipoprotein metabolism, thereby providing an experimental model for studying diet-associated dyslipidemia [3].

Pharmacological treatment of dyslipidemia commonly includes statins, which reduce cholesterol synthesis through inhibition of 3-hydroxy-3-methylglutaryl-coenzyme A reductase. Atorvastatin is an established lipid-lowering agent and has been used as a reference treatment in experimental models of HFD-induced lipid abnormalities. In a study using HFD-fed Wistar rats, atorvastatin treatment improved HFD-associated disturbances in lipid metabolism, supporting its use as a reference hypolipidemic agent in experimental investigations [4]. In recent years, increasing attention has been directed towards natural products and functional foods as potential sources of bioactive compounds with beneficial effects on lipid metabolism. Edible mushrooms contain a wide range of nutritionally and biologically active constituents, including polysaccharides, proteins, sterols, phenolic compounds, terpenoids, fatty acids, vitamins and minerals. These constituents have been associated with several biological activities,

including antioxidant, immunomodulatory and lipid-modulating effects [5].

A recent review specifically examining edible mushrooms reported evidence for their potential effects on lipid levels, obesity and related metabolic abnormalities [6]. Furthermore, a systematic review and meta-analysis of animal studies reported that *Ganoderma lucidum* supplementation was associated with reductions in TC, TG, LDL-C and VLDL, together with an increase in HDL-C, supporting continued investigation of mushrooms as potential sources of lipid-modulating agents [7].

Tricholoma matsutake, commonly known as matsutake or pine mushroom, is one of the highly valued wild edible mushrooms owing to its distinctive aroma, desirable taste, nutritional richness, and diverse medicinal properties [8]. It is considered a functional food as it contains high-quality proteins, essential amino acids, carbohydrates, dietary fibre, vitamins, and essential minerals, while being naturally low in fat. It is also a good source of minerals such as potassium, phosphorus, selenium, iron, and zinc, which are important for various physiological functions [9,10]. In addition to its nutritional constituents, *T. matsutake* contains several biologically active compounds, including polysaccharides, β -glucans, phenolic compounds, flavonoids, terpenoids, sterols, peptides, nucleosides, ergosterol, and volatile aromatic compounds, which contribute to its diverse biological activities. Among these, polysaccharides have received considerable scientific attention due to their reported antioxidant, immunomodulatory, antitumor, anti-inflammatory, antihypertensive, antidiabetic, and potential hypolipidemic activities [11].

Furthermore, pharmacological investigations have demonstrated that *T. matsutake* extracts exhibit free-radical scavenging activity, inhibit lipid peroxidation, enhance endogenous antioxidant defence mechanisms, and protect tissues against oxidative damage. These antioxidant and metabolic effects may be particularly relevant in the prevention and management of metabolic disorders such as hyperlipidemia and associated cardiovascular complications [10,11]. Although several biological activities of *T. matsutake* have been investigated, comparatively limited information is available regarding its potential effects on lipid metabolism, particularly in experimental models of diet-induced hyperlipidemia.

Therefore, further *in-vivo* investigations are required to determine whether *T. matsutake* extracts can influence the altered serum lipid profile associated with HFD-induced hyperlipidemia. The present study was designed to evaluate the hypolipidemic

activity of the ethyl acetate extract of *Tricholoma matsutake* (EAETM) in high-fat diet-induced hyperlipidemic Wistar albino rats. The effects of EAETM at doses of 200 and 400 mg/kg were evaluated by determining serum TC, TG, LDL-C, VLDL-C, HDL-C and atherogenic index, with atorvastatin (10 mg/kg) used as the reference treatment.

MATERIALS AND METHODS:

Collection and authentication of *Tricholoma matsutake*

Tricholoma matsutake mushroom powder was procured from a commercial supplier in the United States of America (USA) and imported to India. The mushroom material was authenticated by Dr. Thejesh Kumar M.P., M.Sc., Ph.D., Department of Botany (PG), Bharathi College, Bharathinagara. The authenticated mushroom powder was stored in an airtight container under suitable conditions until further use.

Preparation of ethyl acetate extract

The finely powdered *T. matsutake* mushroom was subjected to hot continuous extraction using ethyl acetate in a Soxhlet extractor. The resulting extract was used for the subsequent experimental investigation.

Experimental animals

Healthy male Wistar albino rats of approximately 10–12 weeks of age and weighing 100–120 g were used for the evaluation of hypolipidemic activity. The animals were procured from Vertebrates, Magadi Road, Bengaluru, and acclimatized in the animal house facility of Bharathi College of Pharmacy, Bharathinagara. The animals were housed in polypropylene cages containing husk bedding under standard husbandry conditions with a 12-h light/12-h dark cycle. Commercial pelleted rat chow and water were provided *ad libitum*. The animals were maintained in accordance with the guidelines of the Committee for Control and Supervision of Experiments on Animals (CCSEA).

Ethical approval

The experimental protocol was approved by the Institutional Animal Ethics Committee (IAEC) of Bharathi College of Pharmacy, with approval reference number BCP/IAEC/2025/02, dated 12 July 2025. All experimental procedures were conducted in accordance with the applicable animal experimentation guidelines.

Preparation of extract and standard drug suspension

The ethyl acetate extract of *T. matsutake* and the standard drug atorvastatin were suspended in 2% acacia and administered orally to the experimental

animals. The vehicle was selected because the mushroom extract was insoluble in water.

Induction of high-fat diet-induced hyperlipidemia

Hyperlipidemia was induced by administering a high-fat diet consisting of a mixture of vanaspathi ghee and coconut oil in a 3:1 ratio (v/v). The high-fat mixture was administered orally at a dose of 3 mL/kg body weight daily, together with the normal laboratory diet.

Selection of extract doses

The ethyl acetate extract of *T. matsutake* was evaluated at doses of 200 and 400 mg/kg body weight. According to the methodology, these doses were selected based on previous *T. matsutake in-vivo* studies, in which oral administration of the extract at 200 and 400 mg/kg demonstrated biological activity. Accordingly, 200 mg/kg was selected as the lower dose and 400 mg/kg as the higher dose for the present investigation^[12].

Experimental design and grouping

A total of 30 male Wistar albino rats were randomly divided into five groups, with six animals in each group. The experimental groups were as follows:

Group I: Normal control.

Group II: Hyperlipidemic control.

Group III: Hyperlipidemic + Atorvastatin (10 mg/kg).

Group IV: Hyperlipidemic + Extract (200 mg/kg).

Group V: Hyperlipidemic + Extract (400 mg/kg).

The complete experimental period was 6 weeks. Group I received the standard pellet diet and purified water throughout the study and served as the normal control. Groups II–V received the standard pellet diet together with the high-fat diet throughout the experimental period. After 3 weeks of HFD administration, Group III received atorvastatin at 10 mg/kg for the subsequent 3 weeks. Groups IV and V received ethyl acetate extract of *T. matsutake* at doses of 200 and 400 mg/kg, respectively, during the final 3 weeks while HFD administration was continued^[13].

Blood collection and serum preparation

At the end of the experimental period, animals were fasted for 12 h and their body weights were recorded. The animals were then anesthetized, and blood samples were collected by cardiac puncture. The collected blood samples were centrifuged at 3000 rpm for 15 min, and the separated serum was used for estimation of serum lipid parameters.

Estimation of serum lipid parameters

Serum total cholesterol (TC), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) were estimated using commercially available diagnostic kits. Low-density lipoprotein cholesterol (LDL-C), very-low-density lipoprotein cholesterol (VLDL-C), and atherogenic index (AI) were calculated using the respective equations described below.

Estimation of total cholesterol

Serum total cholesterol was estimated by the enzymatic cholesterol oxidase-peroxidase (CHOD-POD) method. In this method, cholesterol esters are hydrolysed by cholesterol esterase to cholesterol and fatty acids. Cholesterol is subsequently oxidized by cholesterol oxidase, producing hydrogen peroxide, which reacts with 4-aminoantipyrine and phenol in the presence of peroxidase to form a colored quinoneimine product. The absorbance was measured at 505 nm. The cholesterol concentration was calculated using the standard supplied with the diagnostic kit [14].

Estimation of triglycerides

Serum triglycerides were estimated using the enzymatic glycerol phosphate oxidase-peroxidase (GPO-POD) method. Triglycerides are enzymatically hydrolysed to glycerol and fatty acids, followed by conversion of glycerol through a series of enzymatic reactions to generate hydrogen peroxide. The hydrogen peroxide participates in a color reaction to form a measurable quinoneimine dye. Absorbance was measured at 505 nm after incubation at 37°C [14,15]. Triglyceride concentration was calculated using the supplied standard according to the following equation:

$$\text{Triglycerides (mg/dL)} = (\text{Absorbance of test} / \text{Absorbance of standard}) \times 200$$

Estimation of HDL-C

Serum HDL-C was estimated using the PEGME/PVS direct HDL-C method. In this method, LDL, VLDL and chylomicrons are selectively masked by polyvinyl sulfonic acid (PVS) and polyethylene glycol methyl ether (PEGME), allowing selective measurement of HDL-associated cholesterol. The reaction was incubated at 37°C for 5 min, and absorbance was measured at 578 nm against the reagent blank. HDL-C concentration was

calculated using the calibrator supplied with the diagnostic kit [16].

Calculation of LDL-C

LDL-C was estimated using the Friedewald equation:

$$\text{LDL-C (mg/dL)} = \text{TC} - \text{HDL-C} - (\text{TG}/5)$$

where TC, HDL-C and TG are expressed in mg/dL.

Calculation of VLDL-C

VLDL-C was calculated from serum triglyceride concentration using the following equation:

$$\text{VLDL-C (mg/dL)} = \text{TG}/5$$

Calculation of atherogenic index

The atherogenic index was calculated using the following formula:

$$\text{AI} = \frac{\text{TC} - \text{HDL-C}}{\text{HDL-C}}$$

where: TC = Total cholesterol, HDL-C = High-density lipoprotein cholesterol

Statistical analysis

The experimental data were expressed as mean $\pm$ standard error of the mean (SEM) for six animals per group. Statistical analysis was performed using one-way analysis of variance (ANOVA), followed by Dunnett's multiple comparison test. A *P* value of <0.05 was considered statistically significant. The methodology classified $P<0.01$ as moderately significant, $P<0.001$ as highly significant, and $P<0.0001$ as very highly significant.

RESULTS AND DISCUSSION:

The effect of ethyl acetate extract of *Tricholoma matsutake* (EAETM) on the serum lipid profile was evaluated in high-fat diet (HFD)-induced hyperlipidemic Wistar albino rats. The results are expressed as mean $\pm$ SEM ($n = 6$). Statistical analysis was performed using one-way ANOVA followed by Dunnett's multiple comparison test. HFD administration produced marked alterations in the serum lipid profile compared with the normal control group, whereas treatment with EAETM at 200 and 400 mg/kg improved several of the altered lipid parameters. Atorvastatin (10 mg/kg) was used as the reference standard.

Table 1: Effects of *Tricholoma matsutake* mushroom extract on serum lipids at the end of 6th week of study.

Group	TC (mg/dL)	TG (mg/dL)	LDL-C (mg/dL)	VLDL-C (mg/dL)	HDL-C (mg/dL)	AI
Normal control	99.83 $\pm$ 7.10	95.00 $\pm$ 3.12	43.30 $\pm$ 7.98	19.00 $\pm$ 0.62	37.53 $\pm$ 2.97	1.73 $\pm$ 0.24
Hyperlipidemic control	139.51 $\pm$ 6.12####	141.67 $\pm$ 5.81####	87.86 $\pm$ 4.82####	28.33 $\pm$ 1.16#####	23.31 $\pm$ 1.54####	5.05 $\pm$ 0.26#####

Atorvastatin 10 mg/kg	100.90 ± 3.73***	101.00 ± 4.72***	40.82 ± 3.26***	20.20 ± 0.94****	39.89 ± 2.95***	1.58 ± 0.15****
Extract 200 mg/kg	113.32 ± 6.69*	118.84 ± 5.96*	55.75 ± 6.78**	23.77 ± 1.19*	33.80 ± 2.13*	2.42 ± 0.28**
Extract 400 mg/kg	104.62 ± 4.89**	110.39 ± 5.97**	46.02 ± 4.15***	22.08 ± 1.19**	36.52 ± 1.92**	1.90 ± 0.18***

Data are presented as mean ± SEM values, n=6. Data were analyzed by one-way ANOVA followed by the Dunnett's multiple comparison test and the significance of results showed as ####p<0.0001, and ###p<0.001, when compared with normal control and ****p<0.0001, ***p<0.001, **p<0.01, *p<0.05 when compared with the Alzheimer's control.

The serum total cholesterol (TC) level in the normal control group was 99.83 ± 7.10 mg/dL, which increased to 139.51 ± 6.12 mg/dL in the hyperlipidemic control group, indicating a marked elevation following HFD administration. Atorvastatin treatment reduced TC to 100.90 ± 3.73 mg/dL. Treatment with EAETM at 200 and 400 mg/kg reduced TC to 113.32 ± 6.69 mg/dL and 104.62 ± 4.89 mg/dL, respectively. The reductions

produced by EAETM were statistically significant compared with the hyperlipidemic control group.

The increase in TC following HFD administration indicates disruption of normal lipid homeostasis. The reduction observed following EAETM treatment suggests that the extract may have a beneficial effect on HFD-associated cholesterol elevation. The effect was numerically greater at 400 mg/kg than at 200 mg/kg. Mushrooms contain several biologically active constituents, including polysaccharides, phenolic compounds, sterols, proteins and other secondary metabolites, which have been investigated for their potential effects on lipid metabolism. A review of edible mushrooms has reported lipid-modulating effects associated with mushroom consumption and mushroom-derived preparations.

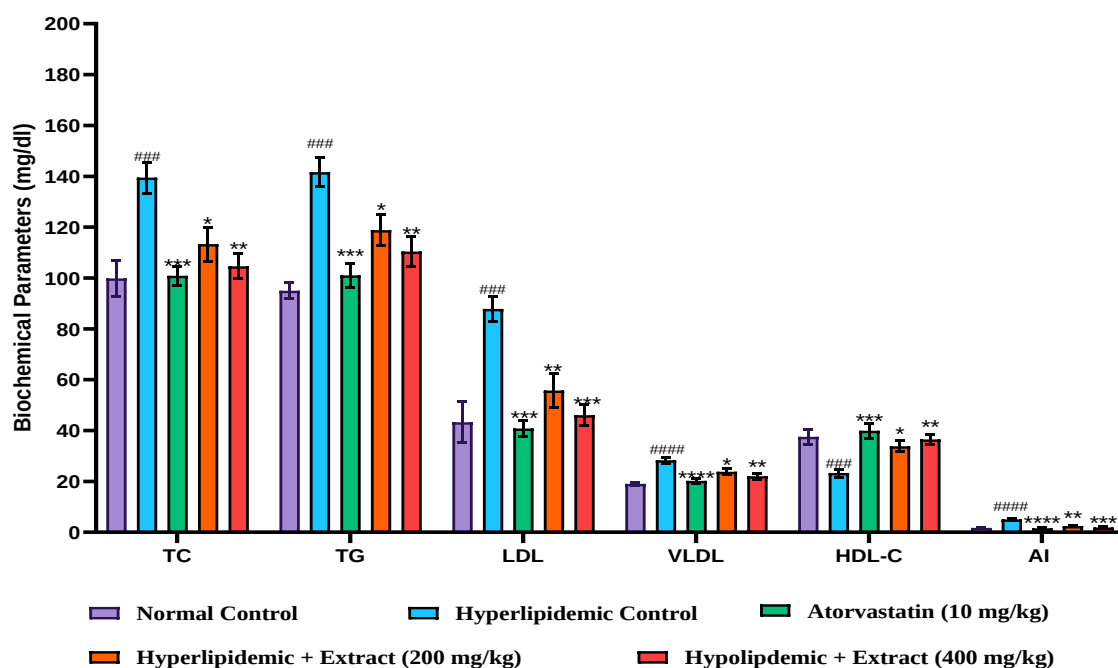


Figure 1: Graph showing mean serum lipid parameters in 5 groups at the end of 6th week.

The triglyceride (TG) level increased from 95.00 ± 3.12 mg/dL in the normal control group to 141.67 ± 5.81 mg/dL in the hyperlipidemic control group. Atorvastatin reduced the TG level to 101.00 ± 4.72 mg/dL. EAETM treatment at 200 and 400 mg/kg reduced TG levels to 118.84 ± 5.96 mg/dL and 110.39 ± 5.97 mg/dL, respectively, compared with the hyperlipidemic control group.

The increase in TG observed after HFD administration is consistent with the development of

diet-associated dyslipidemia. The reduction in TG following EAETM administration indicates an improvement in the triglyceride component of the altered lipid profile. The greater numerical reduction observed at 400 mg/kg compared with 200 mg/kg suggests that the higher dose produced a greater effect under the present experimental conditions. Since triglyceride-rich lipoproteins are closely associated with VLDL metabolism, the accompanying reduction in VLDL-C further

supports an improvement in the HFD-associated lipid disturbance.

The LDL-C concentration was 43.30 ± 7.98 mg/dL in the normal control group and increased to 87.86 ± 4.82 mg/dL in the hyperlipidemic control group. Atorvastatin reduced LDL-C to 40.82 ± 3.26 mg/dL, while EAETM at 200 and 400 mg/kg reduced LDL-C to 55.75 ± 6.78 mg/dL and 46.02 ± 4.15 mg/dL, respectively. Both extract-treated groups showed significant reductions compared with the hyperlipidemic control group.

LDL-C is an important component of the atherogenic lipid profile. The marked increase in LDL-C in HFD-fed animals indicates an adverse alteration in lipid metabolism. The reduction in LDL-C following EAETM treatment, together with the reductions in TC and TG, indicates an overall improvement in the dyslipidemic profile. The effect was numerically more pronounced at 400 mg/kg. However, the present study did not directly investigate hepatic cholesterol synthesis, LDL receptor expression or other molecular pathways; therefore, the precise mechanism responsible for the LDL-C-lowering effect cannot be established from the present findings.

VLDL-C increased from 19.00 ± 0.62 mg/dL in the normal control group to 28.33 ± 1.16 mg/dL in the hyperlipidemic control group. Atorvastatin treatment reduced VLDL-C to 20.20 ± 0.94 mg/dL, whereas EAETM treatment at 200 and 400 mg/kg reduced VLDL-C to 23.77 ± 1.19 mg/dL and 22.08 ± 1.19 mg/dL, respectively. The extract-treated groups showed significant reductions compared with the hyperlipidemic control group.

The increase in VLDL-C following HFD administration is consistent with the elevation of TG observed in the same group. The reduction in VLDL-C following EAETM treatment may therefore reflect an improvement in the altered triglyceride-rich lipoprotein profile produced by the HFD. The concurrent reductions in TG and VLDL-C provide further support for the lipid-modulating activity of the extract.

Unlike the other lipid parameters, HDL-C showed a reduction following HFD administration. The HDL-C level decreased from 37.53 ± 2.97 mg/dL in the normal control group to 23.31 ± 1.54 mg/dL in the hyperlipidemic control group. Atorvastatin increased HDL-C to 39.89 ± 2.95 mg/dL, while EAETM treatment at 200 and 400 mg/kg increased HDL-C to 33.80 ± 2.13 mg/dL and 36.52 ± 1.92 mg/dL, respectively. Both extract doses produced significant increases compared with the hyperlipidemic control group.

The decrease in HDL-C produced by the HFD contributed to the adverse lipid profile observed in the hyperlipidemic control group. The increase in HDL-C following EAETM administration indicates improvement in this component of the lipid profile. HDL is involved in reverse cholesterol transport and therefore represents an important component of lipid homeostasis. The numerical increase observed at 400 mg/kg compared with 200 mg/kg was accompanied by reductions in TC, TG, LDL-C and VLDL-C, suggesting an overall improvement in the serum lipid profile.

The atherogenic index (AI), calculated as $(TC - HDL-C)/HDL-C$, was 1.73 ± 0.24 in the normal control group and increased markedly to 5.05 ± 0.26 in the hyperlipidemic control group. Atorvastatin reduced AI to 1.58 ± 0.15 , whereas EAETM treatment at 200 and 400 mg/kg reduced AI to 2.42 ± 0.28 and 1.90 ± 0.18 , respectively. The reductions in AI produced by the extract were statistically significant compared with the hyperlipidemic control group.

The marked increase in AI following HFD administration reflects the combined increase in TC and reduction in HDL-C. The reduction in AI following EAETM treatment is therefore consistent with the simultaneous improvement in these two lipid parameters. AI provides an integrated representation of the relationship between cholesterol associated with potentially atherogenic fractions and HDL-C, and the reduction observed following EAETM treatment further supports the improvement in the overall lipid profile.

Taken together, HFD administration produced a dyslipidemic pattern characterized by increased TC, TG, LDL-C, VLDL-C and AI, accompanied by decreased HDL-C. EAETM treatment improved all of these measured parameters at both tested doses. The 400 mg/kg dose showed numerically greater improvement than the 200 mg/kg dose for TC, TG, LDL-C, VLDL-C, HDL-C and AI. These findings indicate that EAETM exerted a broad lipid-modulating effect in the present HFD-induced hyperlipidemic rat model.

The observed activity may be related to the diverse chemical composition of *T. matsutake*. Previous literature has described *T. matsutake* as a source of polysaccharides, proteins and amino acids, fatty acids, minerals, phenolic compounds, steroids and volatile constituents, and has reported several biological activities associated with its constituents. However, the present experiment did not identify the specific constituent responsible for the observed lipid-modulating activity. Therefore, the mechanism of action of EAETM cannot be conclusively established from

the present study.

Atorvastatin, used as the reference drug, produced marked improvement in the measured lipid parameters, consistent with its established lipid-lowering activity and with experimental observations in HFD-fed rats. EAETM also produced significant improvements in the lipid profile, although the magnitude of response varied among individual parameters. The atorvastatin group therefore provides a reference for evaluating the experimental response of EAETM rather than establishing equivalence between the extract and the standard drug.

Overall, the present findings provide experimental evidence that EAETM has hypolipidemic activity in HFD-induced hyperlipidemic Wistar albino rats. The extract reduced TC, TG, LDL-C, VLDL-C and AI and increased HDL-C. Further studies investigating hepatic lipid metabolism, cholesterol-related gene and protein expression, oxidative stress markers, histopathological changes and characterization of the active constituents would help clarify the mechanisms responsible for the observed effects.

CONCLUSION:

The present study demonstrated that the ethyl acetate extract of *Tricholoma matsutake* exhibited hypolipidemic activity in high-fat diet-induced hyperlipidemic Wistar albino rats. Administration of EAETM at 200 and 400 mg/kg improved the altered serum lipid profile by reducing total cholesterol, triglycerides, LDL-C, VLDL-C and atherogenic index, while increasing HDL-C levels. The 400 mg/kg dose showed greater numerical improvement across the measured lipid parameters than the 200 mg/kg dose. These findings suggest that *T. matsutake* may have potential as a natural source of lipid-modulating agents. However, further studies are required to identify the active constituents and elucidate the molecular mechanisms responsible for the observed hypolipidemic effects.

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