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EVALUATION OF HYPOLIPIDEMIC ACTIVITY OF SIDDHA FORMULATION VEPPAMPOO MAATHIRAI IN EXPERIMENTAL ANIMAL MODEL

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ABSTRACT

Dyslipidemia, a major contributor to cardiovascular disease, remains a global health challenge despite the availability of lipid-lowering drugs such as statins and fibrates, which are often limited by adverse effects. The present study evaluated the safety and hypolipidemic efficacy of Veppampoo Maathirai (VPM), a traditional Siddha polyherbal formulation, in high-fat diet-induced hyperlipidemic Wistar rats. Acute and sub-acute toxicity studies were conducted as per OECD guidelines, followed by assessment of lipid profiles in serum and liver after 27 days of treatment. Rats were divided into five groups: normal control, hyperlipidemic control, VPM low dose (200 mg/kg), VPM high dose (400 mg/kg), and atorvastatin (10 mg/kg) as standard. Acute toxicity results showed no mortality or behavioural changes up to 2000 mg/kg, indicating high safety. Sub-acute administration produced no significant alterations in hematological, biochemical, or histopathological parameters. VPM significantly reduced serum and hepatic total cholesterol, triglycerides, LDL, and VLDL while elevating HDL in a dose-dependent manner ($p < 0.01$–0.001), comparable to atorvastatin. Histopathological analysis revealed normalization of hepatic architecture and attenuation of fatty changes in treated groups. These findings suggest that Veppampoo Maathirai possesses potent hypolipidemic and hepatoprotective properties, with efficacy similar to standard drugs but with a superior safety profile. The study validates its traditional Siddha use and supports further pharmacological and molecular investigations to elucidate its mechanism of action.

KEYWORDS: Veppampoo Maathirai, Siddha medicine, dyslipidemia, hypolipidemic activity, high-fat diet, atorvastatin, hepatoprotection.

INTRODUCTION

Dyslipidemia, a major risk factor for atherosclerosis, cardiovascular disease (CVD), and stroke, represents a significant global health burden. Despite the efficacy of conventional lipid-lowering drugs (LLDs) such as statins, their long-term use is associated with various adverse effects including hepatobiliary disorders, renal disease, myalgia, myositis, rhabdomyolysis, and an increased risk of incipient diabetes and cognitive dysfunction. Fibrates, another class of LLDs, similarly present concerns regarding liver and renal toxicity and myopathy. These adverse reactions underscore the critical need for effective and safe alternative therapies, particularly for patients unable to tolerate the side effects of conventional LLDs. "Veppampoo Maathirai," a polyherbal Siddha formulation detailed in "The Pharmacopoeia of Siddha Research Medicines" (Pg. 5) by Dr. M. Shanmuga Velu (1st Edition, 1973), is specifically indicated for dyslipidemia. This formulation comprises 14 ingredients processed with lime fruit juice into 3-gram pills. Given the escalating prevalence of dyslipidemia and the limitations of current pharmacotherapies, scientific validation of traditional Siddha formulations like Veppampoo Maathirai is crucial. This study aims to evaluate the toxicity profile and hypolipidemic efficacy of Veppampoo Maathirai in experimental animal models, thereby providing evidence-based support for its traditional use and exploring its potential as a safer alternative in dyslipidemia management.

OBJECTIVES

This study was designed to assess the hypolipidemic activity of Veppampoo Maathirai in High Fat diet-induced hyperlipidemic rats.

MATERIALS AND METHODS

Drug Preparation and Authentication

The raw herbal ingredients for Veppampoo Maathirai were procured from a reputable herbal drug store in Thakkalay, Kanyakumari, Tamil Nadu. The identity and authenticity of all plant materials were verified by medicinal botanists and Gunapadam experts at Government Siddha Medical College and Hospital, Palayamkottai 672002.

ANTI CHOLESTROL REMEDY (SPECIAL) (S.R. Pharma.)

1. Vembu flowers, dried in the shade and powdered - 1 part or 2 oz
2. Keezhkai Nelli Samoolam choornam - ½ part or 1 oz
3. Thuthuvelai choornam - ½ part or 1 oz
4. Potrilaikaiyan choornam - ½ part or 1 oz
5. Chukku
6. Milaku
7. Thippili
8. Kadukkaithole
9. Thandrikaithole
10. Nellivatral
11. Lavangam
12. Lavangapattai
13. Elarisi
14. Vettiver

Of each 1/8 th part or 2 grams fine choornam

All ingredients underwent purification processes as per Siddha literature protocols, under the supervision of guide/faculty members. The purified ingredients were then mixed in a kalvam (traditional grinding stone) and triturated with fresh lime fruit juice for three consecutive days. The resulting paste was rolled into 3-gram pills. The prepared pills, labelled "Veppampoo Maathirai," were stored in airtight containers.

Animal Study Design

Ethical Clearance: The experimental protocol was approved by the Institutional Animal Ethics Committee (IAEC), bearing registration number **[**AKCP/IAEC/12/2024-2025**]**. All animal experiments were conducted in accordance with national and international guidelines for animal care and use.

Animals: Wistar albino adult male rats weighing 150-250g were obtained from the animal housing facility of ARULMIGU KALASALINGAM COLLEGE OF PHARMACY. Animals were housed in polypropylene cages under controlled conditions (temperature 27°C ±1°C, 12-hour light/dark cycle) with free access to standard pellet diet (Sai Durga foods, Bangalore) and water *ad libitum*. A 7-day acclimatization period was provided before the commencement of the study.

High Fat Diet Induction: Experimental hyperlipidemia was induced using a High Fat diet composed of a well-pulverized mixture of cholesterol (400 mg/kg), cholic acid (50 mg/kg), and coconut oil. This mixture was formed into paste-like molds and fed to rats daily for 20 days.

Grouping and Treatment: Animals were randomly divided into five groups of six rats each (n=6).

- **Group I (Normal Control):** Administered 2% CMC only.
- **Group II (Hyperlipidemic Control):** Received High Fat diet and served as hyperlipidemic control.
- **Group III (VPM Low Dose):** Received High Fat diet and Veppampoo Maathirai (VPM) at a low dose of 200 mg/kg/day orally.
- **Group IV (VPM High Dose):** Received High Fat diet and Veppampoo Maathirai (VPM) at a high dose of 400 mg/kg/day orally.
- **Group V (Standard Control):** Received High Fat diet and Atorvastatin (10 mg/kg/day) orally, considered as the standard lipid-lowering drug.

After the 20-day induction period, treatments were continued for 7 days. All groups, except the normal control, received the High Fat diet throughout the treatment period. Animals also had access to standard pellet diet and water *ad libitum*.

Acute Toxicity Study: Acute oral toxicity was assessed as per OECD Test Guideline 423 (Acute Toxic Class Method) using a separate batch of animals. A starting dose of 2000 mg/kg of Veppampoo Maathirai was administered orally to a group of female rats. Animals were observed for 14 days for signs of toxicity and mortality. If no mortality was observed, the next higher dose was not administered, and the LD50 was considered to be above 2000 mg/kg.

Sub-acute Toxicity Study: A sub-acute toxicity study was conducted over a 28-day period using doses equivalent to the therapeutic doses in the hypolipidemic study (200 mg/kg and 400 mg/kg) in healthy Wistar rats. Animals were monitored daily for general health, body weight, food and water consumption, and behavioural changes. At the end of

the study, blood was collected for hematological and biochemical analysis, and major organs (liver, kidney, heart) were harvested for macroscopic and histopathological examination.

Biochemical Parameters

Collection of Blood and Organ Samples: On the day following the completion of the experimental study, all animals were fasted for 18 hours (with *ad libitum* water) before blood collection. Blood samples were collected from the retro-orbital sinus under mild anesthetic ether. The collected blood was centrifuged at 2500 rpm for 10 minutes to obtain serum. Serum samples were then used for various biochemical analyses. After blood collection, animals were sacrificed, and the liver, heart, and kidney were excised for gross visual examination, weight variation assessment, and subsequent histopathological study.

Biochemical Analysis: Serum total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) were estimated using standard enzymatic calorimetric methods.

Liver Lipid Extraction: Liver tissue was homogenized in cold 0.15 M KCl and subsequently extracted with CHCl3: CH3OH (2% v/v). This lipid extract was used for the estimation of hepatic lipid parameters (Total Cholesterol, Triglycerides, LDL, HDL, VLDL).

Histopathology

Liver, heart, and kidney tissues were fixed in 10% neutral formalin. The fixed tissues were then processed, embedded in paraffin, sectioned, and stained with hematoxylin and eosin (H&E). The stained sections were examined microscopically for any histopathological changes. A grading scheme was used for severity (Absent, Minimal, Mild, Moderate, Marked, Severe) and proportion of organ/tissue affected (Nil, very small amount, small amount, medium amount, large amount, very large amount), corresponding to grades 0-5 respectively.

Statistical Analysis

All data were expressed as mean ± standard error of the mean (SEM). Statistical analysis was performed using one-way ANOVA followed by Dunnett's t-test. A P-value < 0.05 was considered statistically significant.

RESULTS & DISCUSSION

Toxicity Study Results

Acute Toxicity: In the acute oral toxicity study, administration of Veppampoo Maathirai at a dose of 2000 mg/kg did not result in any mortality or observable signs of toxicity (e.g., changes in skin, fur, eyes, mucous membranes, respiratory, circulatory, autonomic and central nervous systems, somatomotor activity, and behaviour pattern) in female Wistar rats over a 14-day observation period. This suggests that the LD50 of Veppampoo Maathirai is greater than 2000 mg/kg, classifying it as practically non-toxic by the Globally Harmonized System (GHS) of classification.

Sub-acute Toxicity: The sub-acute toxicity study, conducted over 28 days with 200 mg/kg and 400 mg/kg doses of Veppampoo Maathirai, revealed no significant changes in body weight, food and water consumption, or general behaviour compared to the control group. Hematological and biochemical parameters were also found to be within normal limits, indicating no adverse effects on major organ systems. Macroscopic examination of vital organs (liver,

kidney, heart) showed no gross lesions. Histopathological examination of the liver and kidney in the sub-acute toxicity study further confirmed the safety of Veppampoo Maathirai.

- **Liver Histopathology:** Liver sections from the normal control rats showed normal hepatocytes (H), with intact central vein and portal tract. Similarly, the treatment groups administered with Veppampoo Maathirai (low and high dose) showed normal liver parenchyma with no significant pathological changes, or in some cases, minimal necrosis and inflammation, which were not considered dose-dependent or significant.
- **Kidney Histopathology:** Kidney sections from both control and Veppampoo Maathirai-treated groups consistently showed histomorphological observations within normal limits.

  These findings suggest that Veppampoo Maathirai is well-tolerated and exhibits a favourable safety profile at the tested doses, supporting its traditional use.

Hypolipidemic Activity Results

Body Weight Analysis: Table 1 and Figure 1 illustrate the effect of Veppampoo Maathirai on the body weight of High Fat diet-induced hyperlipidemic rats. The hyperlipidemia-induced control group exhibited a significant increase in body weight (181.88 ± 0.63 g) compared to the normal control group (163.52 ± 0.85 g), indicative of hyperlipidemia. Treatment with Veppampoo Maathirai at a low dose (VPM LOW) significantly reduced body weight to 176.15 ± 0.34 g, while the high dose (VPM HIGH) further reduced it to 168.40 ± 0.26 g. These reductions were statistically significant ($P < 0.001$ for VPM HIGH and $P < 0.01$ for VPM LOW) when compared to the hyperlipidemic control. Atorvastatin (standard drug) also significantly reduced body weight to 172.30 ± 0.19 g. The ability of Veppampoo Maathirai to mitigate the body weight gain associated with a High Fat diet suggests a positive effect on metabolic regulation in hyperlipidemic conditions.

Table 1: Effect of VPM on body weight of High Fat induced hyperlipidemic rats.

S.No	Groups	Body Weight (g)
1	Normal control	163.52 ± 0.85
2	Hyperlipidemic Control	181.88 ± 0.63
3	VPM (LOW)	176.15 ± 0.34
4	VPM (HIGH)	168.40 ± 0.26
5	Atorvastatin (10mg/kg/day)	172.30 ± 0.19

Table 1 *All values are represented as mean±SEM. All data were statistically analyzed by one-way ANOVA followed by Dunnett's test. Values p<0.05 were considered significant. *p<0.001; *p<0.01 vs control.

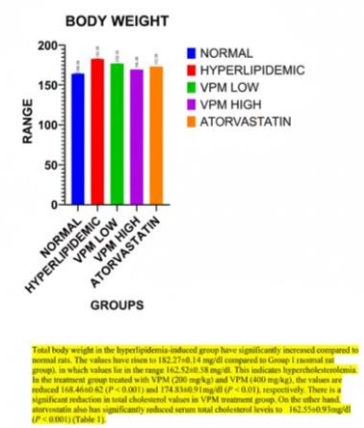


Figure 1: Body Weight of High Fat Induced Hyperlipidemic Rats.

Serum Lipid Profile Analysis: Table 2 present the impact of Veppampoo Maathirai on the blood lipid profile.

- **Total Cholesterol (TC):** The hyperlipidemic control group showed a marked increase in serum TC (201.05 ± 0.76 mg/dl) compared to the normal control (75.20 ± 0.75 mg/dl). Veppampoo Maathirai significantly reduced serum TC in a dose-dependent manner: VPM LOW to 166.73 ± 0.54 mg/dl and VPM HIGH to 141.07 ± 0.45 mg/dl. These reductions were highly significant ($P < 0.001$ for VPM HIGH, $P < 0.01$ for VPM LOW). Atorvastatin also effectively lowered TC to 122.98 ± 0.41 mg/dl.
- **Triglycerides (TG):** Similar to TC, serum TG levels were significantly elevated in the hyperlipidemic control (170.88 ± 0.48 mg/dl) compared to normal (79.88 ± 0.42 mg/dl). VPM treatment resulted in significant reductions: VPM LOW to 142.85 ± 0.37 mg/dl and VPM HIGH to 119.82 ± 0.29 mg/dl ($P < 0.001$ for VPM HIGH, $P < 0.01$ for VPM LOW). Atorvastatin reduced TG to 104.33 ± 0.24 mg/dl.
- **High-Density Lipoprotein (HDL):** HDL levels, often considered "good cholesterol," were significantly decreased in the hyperlipidemic control (29.10 ± 0.12 mg/dl) compared to normal (47.42 ± 0.23 mg/dl). Both doses of VPM treatment significantly increased HDL: VPM LOW to 35.10 ± 0.07 mg/dl and VPM HIGH to 40.57 ± 0.09 mg/dl ($P < 0.01$ for both). Atorvastatin also increased HDL to 44.90 ± 0.08 mg/dl.
- **Low-Density Lipoprotein (LDL):** Serum LDL levels were dramatically increased in the hyperlipidemic control (135.83 ± 0.25 mg/dl) versus normal (16.05 ± 0.11 mg/dl). VPM administration led to significant reductions: VPM LOW to 111.28 ± 0.23 mg/dl and VPM HIGH to 89.25 ± 0.22 mg/dl ($P < 0.001$ for both). Atorvastatin reduced LDL to 73.15 ± 0.18 mg/dl.
- **Very Low-Density Lipoprotein (VLDL):** The hyperlipidemic control showed elevated VLDL (34.18 ± 0.10 mg/dl) compared to normal (15.98 ± 0.08 mg/dl). VPM significantly reduced VLDL: VPM LOW to 28.57 ± 0.07 mg/dl and VPM HIGH to 23.96 ± 0.06 mg/dl ($P < 0.01$ for both). Atorvastatin lowered VLDL to 20.87 ± 0.04 mg/dl.

These results unequivocally demonstrate that Veppampoo Maathirai possesses significant hypolipidemic activity by reducing total cholesterol, triglycerides, LDL, and VLDL, while simultaneously increasing beneficial HDL cholesterol. The dose-dependent nature of these effects highlights the formulation's potential as a therapeutic agent for dyslipidemia.

Table 2: Effect of VPM on Blood lipid profile of High Fat induced hyperlipidemic rats.

Group	Treatment	T.C. (mg/dl)	T.G. (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
I	Normal Control	75.20 ± 0.75	79.88 ± 0.42	47.42 ± 0.23	16.05 ± 0.11	15.98 ± 0.08
II	Hyperlipidemic Control	201.05 ± 0.76	170.88 ± 0.48	29.10 ± 0.12	135.83 ± 0.25	34.18 ± 0.10
III	VPM (LOW)	166.73 ± 0.54	142.85 ± 0.37	35.10 ± 0.07	111.28 ± 0.23	28.57 ± 0.07
IV	VPM (HIGH)	141.07 ± 0.45	119.82 ± 0.29	40.57 ± 0.09	89.25 ± 0.22	23.96 ± 0.06
V	Atorvastatin (10mg/kg)	122.98 ± 0.41	104.33 ± 0.24	44.90 ± 0.08	73.15 ± 0.18	20.87 ± 0.04

Table 1 *All values are represented as mean±SEM. All data were statistically analyzed by one-way ANOVA followed by Dunnett's test. Values $p<0.05$ were considered significant. *$p<0.001$; *$p<0.01$ vs control.

Liver Lipid Profile Analysis: Table 3 and Figure 3 provide insight into the effect of VPM on the liver lipid profile, which is critical given the liver's central role in lipid metabolism.

- **Hepatic Total Cholesterol:** The hyperlipidemic control exhibited significantly increased hepatic TC (8.25 ± 0.04 mg/g) compared to the normal control (4.12 ± 0.03 mg/g). Both VPM LOW (6.72 ± 0.03 mg/g) and VPM HIGH

(5.18 ± 0.02 mg/g) significantly reduced hepatic TC (P < 0.01 for VPM LOW, P < 0.001 for VPM HIGH). Atorvastatin also showed a reduction to 4.36 ± 0.02 mg/g.

- **Hepatic Triglycerides:** Hepatic TG was elevated in the hyperlipidemic control (11.02 ± 0.04 mg/g) compared to normal (4.95 ± 0.03 mg/g). VPM LOW (9.21 ± 0.03 mg/g) and VPM HIGH (7.36 ± 0.02 mg/g) significantly reduced hepatic TG (P < 0.01 for VPM LOW, P < 0.001 for VPM HIGH). Atorvastatin reduced TG to 6.08 ± 0.02 mg/g.
- **Hepatic LDL:** Hepatic LDL levels were markedly increased in the hyperlipidemic control (49.29 ± 0.03 mg/g) compared to normal (1.19 ± 0.01 mg/g). VPM LOW (4.36 ± 0.02 mg/g) and VPM HIGH (3.24 ± 0.01 mg/g) significantly lowered hepatic LDL (P < 0.01 for VPM LOW, P < 0.001 for VPM HIGH). Atorvastatin showed a reduction to 2.56 ± 0.01 mg/g.
- **Hepatic HDL:** Hepatic HDL was decreased in the hyperlipidemic control (0.92 ± 0.01 mg/g) compared to normal (1.63 ± 0.02 mg/g). VPM LOW (1.21 ± 0.01 mg/g) and VPM HIGH (1.36 ± 0.01 mg/g) significantly increased hepatic HDL (P < 0.01 for VPM LOW, P < 0.001 for VPM HIGH). Atorvastatin increased HDL to 1.45 ± 0.01 mg/g.
- **Hepatic VLDL:** Hepatic VLDL was elevated in the hyperlipidemic control (2.20 ± 0.01 mg/g) compared to normal (0.99 ± 0.01 mg/g). VPM LOW (1.84 ± 0.01 mg/g) and VPM HIGH (1.47 ± 0.01 mg/g) significantly reduced hepatic VLDL (P < 0.01 for VPM LOW, P < 0.001 for VPM HIGH). Atorvastatin reduced VLDL to 1.22 ± 0.01 mg/g.

The observed reductions in hepatic total cholesterol, triglycerides, LDL, and VLDL, along with an increase in HDL, signify that Veppampoo Maathirai effectively modulates lipid metabolism within the liver. This direct effect on liver lipid content is a crucial mechanism contributing to its overall hypolipidemic activity.

Table 3: Effect of VPM on liver lipid profile of High Fat -induced hyperlipidemic rats.

Group	Treatment	T.C (mg/g)	T.G. (mg/g)	LDL (mg/g)	HDL (mg/g)	VLDL (mg/g)
I	Normal Control	4.12 ± 0.03	4.95 ± 0.03	1.19 ± 0.01	1.63 ± 0.02	0.99 ± 0.01
II	Hypolipidemic Control	8.25 ± 0.04	11.02 ± 0.04	49.29 ± 0.03	0.92 ± 0.01	2.20 ± 0.01
III	VPM (LOW)	6.72 ± 0.03**	9.21 ± 0.03**	4.36 ± 0.02**	1.21 ± 0.01**	1.84 ±0.01**
IV	VPM (HIGH)	5.18 ±0.02***	7.36 ± 0.02***	3.24 ±0.01***	1.36 ± 0.01***	1.47 ± 0.01**
V	Atorvastatin (10mg/kg/day)	4.36 ± 0.02***	6.08 ± 0.02***	2.56 ± 0.01***	1.45 ± 0.01***	1.22 ± 0.01**

Table 2 *All values are represented as mean±SEM. All data were statistically analyzed by one-way ANOVA followed by Dunnett's test. Values p<0.05 were considered significant. *p<0.001;*p<0.01 vs control.

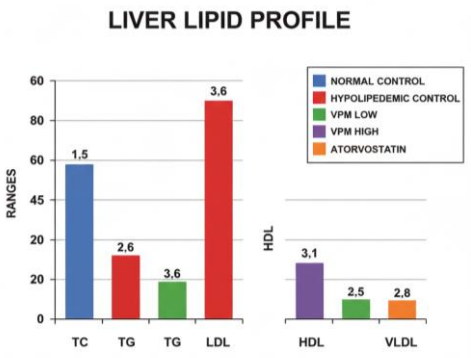
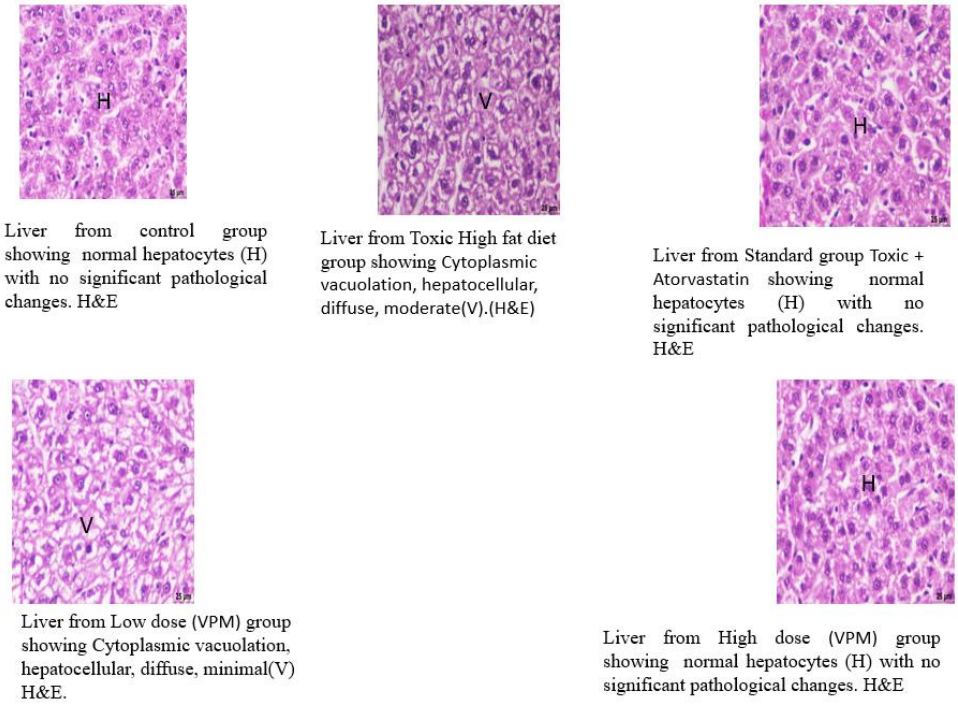


Figure 2: Liver Lipid Profile of High Fat Induced Hyperlipidemic Rats.

Histopathological Observations



Liver from control group showing normal hepatocytes (H) with no significant pathological changes. H&E

Liver from Toxic High fat diet group showing Cytoplasmic vacuolation, hepatocellular, diffuse, moderate(V).(H&E)

Liver from Standard group Toxic + Atorvastatin showing normal hepatocytes (H) with no significant pathological changes. H&E

Liver from Low dose (VPM) group showing Cytoplasmic vacuolation, hepatocellular, diffuse, minimal(V) H&E.

Liver from High dose (VPM) group showing normal hepatocytes (H) with no significant pathological changes. H&E

Figure 3: Liver Histopathology Report - Control and Treatment Groups.

- **Liver Histopathology:** The liver sections from the hyperlipidemic control group (Figure 3) showed cytoplasmic vacuolation, hepatocellular, diffuse, and moderate changes, indicative of fatty liver and scattered focal areas of necrosis. In contrast, the group treated with Veppampoo Maathirai (VPM LOW) showed cytoplasmic vacuolation, diffuse, and minimal changes, indicating a protective effect. The VPM HIGH group showed liver parenchyma with minimal necrosis and inflammation, otherwise normal. The Atorvastatin-treated group showed normal hepatocytes with normal appearance of central vein and portal tract, demonstrating the formulation's ability to normalize liver architecture affected by hyperlipidemia.
- **Kidney and Heart Histopathology:** Histopathological examinations of the kidney and heart from the treatment groups did not reveal any significant adverse changes, supporting the safety profile observed in biochemical and gross examinations.

The amelioration of cytoplasmic vacuolation and necrosis in liver cells by Veppampoo Maathirai highlights its hepatoprotective effects against High Fat diet-induced damage, further strengthening its therapeutic potential. The normalization of lipid profiles in both serum and liver, coupled with the absence of significant toxicity, aligns with the traditional Siddha understanding of Veppampoo Maathirai as a beneficial agent for dyslipidemia. The polyherbal nature of the formulation likely contributes to its multifaceted action, potentially targeting various pathways involved in lipid synthesis, absorption, and clearance.

CONCLUSION

This comprehensive study demonstrates that the Siddha formulation Veppampoo Maathirai is safe and possesses significant hypolipidemic activity in High Fat diet-induced hyperlipidemic rats. The acute toxicity study revealed no signs of toxicity up to 2000 mg/kg, indicating a high safety margin. In the sub-acute study, Veppampoo Maathirai

showed no adverse effects on body weight, biochemical parameters, or organ histopathology. Furthermore, Veppampoo Maathirai effectively attenuated the dyslipidemic state by significantly reducing elevated serum and hepatic total cholesterol, triglycerides, LDL, and VLDL, while increasing beneficial HDL levels. These effects were dose-dependent and comparable to the standard lipid-lowering drug, Atorvastatin. The histopathological findings corroborated the biochemical data, showing a reversal of hyperlipidemia-induced fatty changes in the liver. Based on these findings, Veppampoo Maathirai emerges as a promising natural alternative for the management of dyslipidemia, offering both efficacy and a favourable safety profile. Further research, including isolation of active compounds and elucidation of molecular mechanisms, is warranted to fully explore its therapeutic potential.

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