ORIGINAL ARTICLE ASIAN JOURNAL OF MEDICAL SCIENCES

Effect of foxtail millet supplementation in comparison to atorvastatin on high-fat diet-induced hyperlipidemia in rats

Praneeth Reddy Onteddu[1], Suguna Sajja[2], Bhuvaneshwari Gajala[3], Shravya Edavaluru[4], Chaitanya Kandula[5]

[1,5]Final Year Postgraduate Resident, [2]Assistant Professor, [3]Professor and Head, Department of Pharmacology, Gandhi Medical College, Musheerabad, [4]Intern, Kamineni Academy of Medical Sciences and Research Center, Hyderabad, Telangana, India

Submission: 26-03-2023 Revision: 29-06-2023 Publication: 01-08-2023

ABSTRACT

Background: Hyperlipidemia can be defined as an increased concentration of lipids in the blood. Foxtail millet (FTM) is a nutrient-rich cereal containing several phytochemicals which have possible lipid-lowering and glucose-lowering abilities. Atorvastatin is an HMG Co-A Reductase Inhibitor commonly prescribed for Hyperlipidemia. **Aims and Objectives:** The aim of this study was to compare the hypolipidemic effect of FTM with that of Atorvastatin in Sprague–Dawley rats. **Materials and Methods:** Twelve 12 male Sprague–Dawley rats were segregated into Group-A and Group-B with six (6) rats in each group. The rats in both groups were fed high-fat diet for 21 days and for the next 21 days the 6 rats in Group-A were fed FTM in the form of pellets, the 6 rats in Group-B were administered Atorvastatin in a dose of 5 mg/kg. The body weight (BW) and lipid profiles of the rats were measured at three stages-day 0, day 21, and day 42. **Results:** FTM showed an 18.1% rise in high-density lipoprotein (HDL-C). It showed a fall in BW, total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL-C), and very LDL (VLDL-C) which were 15.5%, 14.0%, 12.8%, 18.3%, and 16.0%, respectively. With Atorvastatin the rise in HDL-C was 13.4%. The fall in BW, TC, TG, LDL-C, and VLDL-C was 30.2%, 30.5%, 24.9%, 27.5%, and 34.4%, respectively. **Conclusion:** The present study results showed that FTM had a noticeable positive effect on the lipid profile and BW in Dyslipidemia in comparison to Atorvastatin.

Key words: Hyperlipidemia; Foxtail millet; Atorvastatin; Sprague–Dawley rats; Dyslipidemia

Access this article online

Website: http://nepjol.info/index.php/AJMS

DOI: 10.3126/ajms.v14i8.53596

E-ISSN: 2091-0576

P-ISSN: 2467-9100

INTRODUCTION

The prevalence of dyslipidemia in the Indian Urban population was 31.7% in men and 32.8% in women while the prevalence in the rural population was 19.5% in men and 26.4% in women.[1] Lipids encompass molecules like Cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides (TG). The intestines are the site of absorption of TG and cholesterol. These lipids are then transported by lipoproteins and utilized for bile acid formation, steroid synthesis, and energy production. Organic and non-organic factors contribute to the disharmony in their levels which can ultimately result in dyslipidemia.[2]

Hyperlipidemia can be defined as total cholesterol (TC), TG, lipoproteins, or LDL >90th percentile when compared to the general population (or) HDL <10th percentile in comparison to the general population. Dyslipidemia is one of the crucial factors leading to cardiovascular diseases.[3] Till date, several studies have established that an elevated LDL-Cholesterol is strongly associated with atherosclerotic plaques and vascular diseases. On the other hand, sufficient HDL-cholesterol was found to be crucial for preventing atherosclerotic disease.[4]

Dyslipidemia is, fortunately, a modifiable risk factor.[5] Diets with lipid-lowering ability, like millets, and lifestyle

Address for Correspondence:
Dr. Suguna Sajja, Assistant Professor, Department of Pharmacology, Gandhi medical college, Musheerabad, Telangana, India.
Mobile: +91-9505014567. **E-mail:** suguna.pharmac@gmail.com

modifications like regular exercise have long been considered a reliable adjunct to pharmacotherapy of Dyslipidemias.

Millet is a generic term for a series of small-seeded cereals, such as foxtail millet (FTM) (*Setaria* italica), proso millet (*Panicum* miliaceum), and finger millet (*Eleusine* coracana). FM, as a good source of whole grain, has been playing an important role in global food security, especially in arid and semiarid areas of Asia and Africa.[6]

Nutrition experts and scientists of late focusing on the possible health benefits of FM. This is probably due to the phytochemicals and nutrients present in FM. According to the currently available literature, FM has an established lipid-lowering ability and it was found to have low starch digestibility and its glycemic index was also moderate.[7-9]

Atorvastatin belongs to a class of drugs called Statins which is a 3-Hydroxy 3-methylglutaryl Co-enzyme-A Reductase inhibitor. Statins, apart from having strong lipid-lowering effects, produce pleiotropic effects, i.e., promoting the migration of Endothelial Progenitor cells, and lowering oxidation and inflammation.[10-12]

Among patients being prescribed Statins, 7%–29% of them experience adverse effects related to muscles. These could be myopathies, Myositis, rhabdomyolysis, or elevation of Creatine Kinase.[13] Hence, the current study was conducted to analyze and compare the effect of FTM supplementation and Atorvastatin on HFdiet-induced hyperlipidemia in rats. The intent was to aid the quest for safer alternatives to manage hyperlipidemias.

Aims and objectives

1. To evaluate the effect of Foxtail millet supplementation on high fat (HF) diet induced hyperlipidemia in rats.
2. To compare the effect of Foxtail millet supplementation and Atorvastatin on high fat (HF) diet induced hyperlipidemia in rats.

MATERIALS AND METHODS

Animals

After obtaining the approval of the Institutional Animal Ethics Committee (IAEC) (Approval number-27/GMC/IAEC), 12 Male Sprague–Dawley rats weighing 180–200 g were sourced. They were housed in the Animal House of the Department of Pharmacology at Gandhi Medical College, Musheerabad. The rats were housed at a temperature of 23°C±1°C and they had free access to water. Throughout the experiment, the rats were handled as per the guidelines laid down by CPCSEA under the supervision of IAEC, Gandhi Medical College. (Reg.no-428/GO/Re/S/2001/CPCSEA).

The 12 rats were divided into two equal groups - Group-A and Group-B. They were allowed to acclimatize for 1 week in the central animal house and the body weight (BW) and lipid profile of all the rats in both groups were measured at the start of the study (day 0). Each of the 12 rats was then fed 15 mL of HF diet every day in divided doses for 3 weeks.[14]

The high-fat diet (HFD) consisted of vanaspati ghee (clarified butter prepared from palm oil) and coconut oil in a ratio of 3:1 (v/v).[15] This was fed to the rats orally using the Gavage technique with the help of flexible catheters. After 3 weeks, the BW and lipid profile of all the rats were measured again. The rats in Group A (n=6) were fed FTM for the next 3 weeks orally, in the form of pellets. The rats in Group B (n=6) were given Atorvastatin at a dose of 5 mg/kg/d.[16] The BW and lipid profile of the rats in both groups were measured at the end of the study (day 42).

Blood sample collection and analysis

The saphenous vein was chosen as the site for blood sample collection.[15] Fasting blood samples were taken as lipid profiles were being measured. 4–5 mL of blood was collected from each rat on three instances on day 0, day 21, and day 42. TG, TC, HDL, and LDL were measured using a mid-volume Auto-analyzer Cobas c 501, as per the protocols laid out by the manufacturer, Roche Diagnostics.

Statistical analysis

The collected data were tabulated and analyzed by applying unpaired t-test with the help of Graph Pad Prism Version 9.

RESULTS

Twelve male Sprague–Dawley rats were fed HFD for 3 weeks. Later, these 12 rats were divided into two groups of 6 rats each Group-A and Group-B.

The 6 rats in Group-A were fed FTM in the form of pellets while the six (6) rats in Group-B were administered Atorvastatin in a dose of 5 mg/kg. The BW and lipid profiles of the rats were measured at three stages-day 0, day 21, and day 42.

Table 1 shows the baseline mean BW and the baseline mean lipid profile values of rats at the start of the study. The least BW recorded in both Group-A and Group-B was 180 g. The highest BW in both groups was 210 g. The TC in Group-A ranged from 87 mg/dL to 96 mg/dL. In Group-B, it ranged from 88 mg/dL to 95 mg/dL. TG in Group-A ranged from 71 to 76 while in group-B ranged from 69 mg/dL to 78 mg/dL. The LDL-C in group-A ranged from 41 mg/dL to 47 mg/dL. In Group-B, the LDL-C ranged from 41 mg/dL to 48 mg/dL. The very

LDL (VLDL-C) in Group-A ranged from 18 mg/dL to 21 mg/dL, while in Group-B, it ranged from 18 mg/dL to 20 mg/dL. HDL-C in Group-A ranged from 24 mg/dL to 27 mg/dL. The same parameter in Group-B ranged from 24 mg/dL to 29 mg/dL.

Table 1 The mean BW of Group-B was found to be marginally higher than that of Group-A. The baseline mean lipid profile parameters were found to be similar in both Group-A and Group-B while there was a slight difference in the standard deviations.

It can be observed from Table 2 that, on day–21, there is not much difference between the mean BW and the mean lipid values of rats in both groups. However, the mean LDL-C and VLDL-C in Group-B were marginally higher than those in Group-B.

Table 2 shows that in Group A, the mean BW was found to be 280±10.80, the mean TC was 138.2±6.72, the mean TG was 129.8±4.76, the mean LDL-C was 72.8±2.67, the mean VLDL-C was 28.8±1.77, the mean HDL-C was 36.0±1.29.

In Group B, the mean BW was found to be 281.7±6.24, the mean TC was 138.8±5.40, the mean TG was 129.8±4.78, the mean LDL-C was 75.2±1.07, the mean VLDL-C was 30.5±1.71, the mean HDL-C was 36.2±2.03.

From Table 3, it is evident that the FTM could lower the BW and lipid levels but it could not bring them down to baseline. On the other hand, with atorvastatin these values came close to the baseline values (day–0).

Table 3 shows that in Group A, after feeding them with FTM, the mean BW was found to be 236.7±8.00, the mean TC was 118.8±4.06, the mean TG was 113.2±7.73, the mean LDL-C was 59.5±4.57, the mean VLDL-C was 24.2±1.07, the mean HDL-C was 42.5±2.06.

In Group B, after administering 5 mg/kg atorvastatin, the mean BW was found to be 196.7±7.99, the mean TC was 96.5±4.79, the mean TG was 97.5±3.5, the mean LDL-C was 54.5±2.00, the mean VLDL-C was 20.0±1.15, the mean HDL-C was 41.81.46.

Table 1: Mean body weight and mean lipid profile values of rats in Group-A on day–0 (baseline)

S. No.	Body weight (in grams)	TC (in mg/dL)	TG (in mg/dL)	LDL-C (in mg/dL)	VLDL-C (in mg/dL)	HDL-C (in mg/dL)
MEAN±SD =	195±11	91.3±3.14	72.7±1.86	43.5±2.26	19±1.26	25.8±1.17
Mean body weight and mean lipid profile values of rats in Group-B on day 0 (baseline)						
S.no	**Body Weight (in grams)**	**TC (in mg/dL)**	**TG (in mg/dL)**	**LDL-C (in mg/dL)**	**VLDL-C (in mg/dL)**	**HDL-C (in mg/dL)**
Mean±SD =	198.3±1.33	91.2±2.93	72.7±3.88	44.7±3.01	19.2±0.75	26±1.90

TC: Total cholesterol, TG: Triglycerides, LDL-C: Low-density lipoprotein cholesterol, VLDL-C: Very low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein Cholesterol

Table 2: Mean body weight and mean lipid profile values categorized as Group-A on day–21

Mean body weight	Mean TC	Mean TG	Mean LDL-C	Mean VLDL-C	Mean HDL-C
280±10.80	138.2±6.72	129.8±4.76	72.8±2.67	28.8±1.77	36.0±1.29
Mean body weight and mean lipid profile values were categorized as Group-B on day-21					
Mean body weight	**Mean TC**	**Mean TG**	**Mean LDL-C**	**Mean VLDL-C**	**Mean HDL-C**
281.7±6.24	138.8±5.40	129.8±4.78	75.2±1.07	30.5±1.71	36.2±2.03

TC: Total cholesterol, TG: Triglycerides, LDL-C: Low-density lipoprotein cholesterol, VLDL-C: Very low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol

Table 3: Mean body weights and mean lipid profile of Group-A after feeding with FTM on day-42

S.No	Body weight (in grams)	TC (in mg/dL)	TG (in mg/dL)	LDL-C (in mg/dL)	VLDL-C (in mg/dL)	HDL-C (in mg/dL)
Mean ±SD =	236.7±8.00	118.8±4.06	113.2±7.73	59.5±4.57	24.2±1.07	42.5±2.06
Mean body weights and mean lipid profile of Group-B after administering 5 mg/kg Atorvastatin on day-42						
S.No	**Body weight (in grams)**	**TC (in mg/dL)**	**TG (in mg/dL)**	**LDL-C (in mg/dL)**	**VLDL-C (in mg/dL)**	**HDL-C (in mg/dL)**
Mean ±SD =	196.7±7.99	96.5±4.79	97.5±3.5	54.5±2.00	20.0±1.15	41.8±1.46

TC: Total cholesterol, TG: Triglycerides, LDL-C: Low-density lipoprotein cholesterol, VLDL-C: Very low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein Cholesterol

Table 4 shows that after feeding the rats in Group A with FTM a decrease in mean BW by 43.3 g, a decrease in mean TC by 19.4 mg/dL, a fall in mean TG by 16.6 mg/dL, a drop in mean LDL-C by 13.3 mg/dL, a decrease in mean VLDL-C by 4.6 mg/dL and a rise in mean HDL-C by 6.5 mg/dL was observed.

Table 4 shows that after feeding the rats in Group A with FTM a decrease in mean BW by 15.5%, a decrease in mean TC by 14.0%, a fall in mean TG by 12.8%, a drop in mean LDL-C by 18.3%, a decrease in mean VLDL-C by 16.0% and a rise in mean HDL-C by 18.1% was observed.

Table 5 shows that after administering atorvastatin for the rats in Group B, a decrease in mean BW by 85 g, a decrease in mean TC by 42.3 mg/dL, a fall in mean TG by 32.2 mg/dL, a drop in mean LDL-C by 20.7 mg/dL, a decrease in mean VLDL-C by 10.5 mg/dL and a rise in mean HDL-C by 5.6 mg/dL was observed.

Table 5 shows that after administering Atorvastatin for the rats in Group B, a decrease in mean BW by 30.2%, a decrease in mean TC by 30.5%, a fall in mean TG by 24.9%, a drop in mean LDL-C by 27.5%, a decrease in mean VLDL-C by 34.4% and a rise in mean HDL-C by 13.4% was observed.

Table 6 shows that the difference between Group-A and Group-B in terms of a drop in BW, the drop in TC, TG, LDL-C and VLDL-C is statistically significant. However, the difference between the two groups in terms of rise in HDL-C is statistically not significant. An unpaired t-test was used.

Table 6 shows that the difference between Group-A and Group-B in terms of drop in BW, the drop in TC, TG, LDL-C, and VLDL-C is statistically significant. However, the difference between the two groups in terms of rise in HDL-C is statistically not significant. An unpaired t-test was used.

DISCUSSION

Dyslipidemia, i.e., abnormal elevations in the lipid profile parameters such as TC, TG, LDL-C, VLDL-C, and HDL-C were found to be established contributors to coronary heart Disease.[17] The literature available at present shows that the dietary fiber, the phenolics, the lipid profile, and aminoacid profile of FTM confer lipid-lowering and glucose-lowering qualities on it.[18] Atorvastatin has well-established capabilities of elevating HDL-C while lowering the remaining lipid profile parameters. Hence, it is of extensive use in managing dyslipidemia.[19]

As evident from Table 2, there was a noticeable rise in BW and lipid profile values after 3 weeks of HFD. These results were similar to those in a study by Jia et al.[20] It was observed in their study that When 3-week-old male Sprague–Dawley rats weighing 50±1 g were fed HFD for 4 weeks, their BW increased to 391±13.09 g. Their TC increased by 30.55 mg/dL, their TG increased by 98.32 mg/dL, their LDL-cholesterol increased by 5.41 mg/dL while their HDL cholesterol increased by 13.92 mg/dL. The findings in the present study were similar to these results.

From Table 3, a drop in the mean BW and an alteration of the lipid profile can be observed following FTM supplementation. This finding is congruent with that of a study conducted by Sireesha et al.[21] They observed that following FTM supplementation there was a significant hypolipidemic effect which is evident from lower levels of TG, total, LDL and VLDL cholesterol and increase in the

Table 4: Difference in mean body weight and lipid profile in Group-A

Study Indices	Before foxtail millet	After foxtail millet	Difference in means	Percentage change (%)
Mean body weight	280	236.7	43.3	15.5
Mean TC	138.2	118.8	19.4	14.0
Mean TG	129.8	113.2	16.6	12.8
Mean LDL-C	72.8	59.5	13.3	18.3
Mean VLDL-C	28.8	24.2	4.6	16.0
Mean HDL-C	36.0	42.5	6.5	18.1

TC: Total cholesterol, TG: Triglycerides, LDL-C: Low-density lipoprotein cholesterol, VLDL-C: Very low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein Cholesterol

Table 5: Difference in mean body weight and lipid profile in Group-B

Study Indices	Before atorvastatin	After atorvastatin	Difference in means	Percentage change (%)
Mean body weight	281.7	196.7	85	30.2
Mean TC	138.8	96.5	42.3	30.5
Mean TG	129.8	97.5	32.3	24.9
Mean LDL-C	75.2	54.5	20.7	27.5
Mean VLDL-C	30.5	20.0	10.5	34.4
Mean HDL-C	36.2	41.8	5.6	13.4

Table 6: Statistical significance of the difference between body weight changes and lipid profile changes in Group-A and Group-B

Body weight (BW)							**P-Value**
Group-A (S.no)	**1**	**2**	**3**	**4**	**5**	**6**	
Drop in BW after FTM	30 g	55 g	25 g	30 g	50 g	70 g	
Group-B (S.no)	7	8	9	10	11	12	0.0005
Drop in BW after atorvastatin	90 g	90 g	90 g	85 g	90 g	65 g	
Total cholesterol (TC)							**P-Value**
Group-A (S.no)	**1**	**2**	**3**	**4**	**5**	**6**	
Drop in TC after FTM	16 mg/dL	19 mg/dL	22 mg/dL	23 mg/dL	27 mg/dL	9 mg/dL	0.0001
Group-B (S.no)	7	8	9	10	11	12	
Drop in TC after atorvastatin	40 mg/dL	35 mg/dL	50 mg/dL	49 mg/dL	36 mg/dL	44 mg/dL	
Triglycerides (TG)							**P-Value**
Group-A (S.no)	**1**	**2**	**3**	**4**	**5**	**6**	
Drop in TG after FTM	13 mg/dL	2 mg/dL	14 mg/dL	29 mg/dL	16 mg/dL	26 mg/dL	0.0098
Group-B (S.no)	7	8	9	10	11	12	
Drop in TG after Atorvastatin	26 mg/dL	37 mg/dL	35 mg/dL	26 mg/dL	27 mg/dL	43 mg/dL	
Low-density Lipoprotein (LDL-C)							**P-value**
Group-A (S.no)	**1**	**2**	**3**	**4**	**5**	**6**	
Drop in LDL-C after FTM	16 mg/dL	17 mg/dL	20 mg/dL	5 mg/dL	11 mg/dL	11 mg/dL	0.0110
Group-B (S. No)	7	8	9	10	11	12	
Drop in LDL-C after Atorvastatin	23 mg/dL	23 mg/dL	18 mg/dL	21 mg/dL	20 mg/dL	19 mg/dL	
Very low-density lipoprotein (VLDL-C)							**P-value**
Group-A (S.no)	**1**	**2**	**3**	**4**	**5**	**6**	
Drop in VLDL-C after FTM	7 mg/dL	1 mg/dL	3 mg/dL	3 mg/dL	9 mg/dL	5 mg/dL	0.0027
Group-B (S.no)	7	8	9	10	11	12	
Drop in VLDL-C after Atorvastatin	11 mg/dL	10 mg/dL	11 mg/dL	14 mg/dL	9 mg/dL	8 mg/dL	
High-density lipoprotein (HDL-C)							**P-value**
Group-A (S.no)	**1**	**2**	**3**	**4**	**5**	**6**	
Rise in HDL-C after FTM	6 mg/dL	7 mg/dL	8 mg/s	5 mg/dL	9 mg/dL	4 mg/dL	0.4807
Group-B (S.no)	7	8	9	10	11	12	
Rise in HDL-C after atorvastatin	9 mg/dL	3 mg/dL	5 mg/dL	5 mg/dL	5 mg/dL	7 mg/dL	

levels of HDL cholesterol. The findings were similar in the present study with a noticeable rise in HDL-C.

From Table 3, a drop in the mean BW and an alteration of the lipid profile can be observed following atorvastatin administration. This finding is in similar lines with that of a study conducted by Roglans et al.[22] They concluded that Atorvastatin 5 mg/kg for 2 weeks markedly reduced plasma triglyceride, hepatic triglyceride content (45%), and plasma non-esterified fatty acids (49%). The findings in the present study were similar to the aforementioned study results.

Limitations of the study

1. The current study being diet-based should have had more number of follow-ups with a longer study duration.
2. The sample size is another limitation.

CONCLUSION

The present study concludes that FTM can produce a noticeable reduction in the levels of LDL-C, VLDL-C, TG, TC, and BW. This reduction was found to be statistically significant. FTM and Atorvastatin could show a considerable positive influence on HDL-C, the effect of FTM being more than that of Atorvastatin but the difference was not statistically significant.

The importance of drugs like statins cannot be ignored. However, combining a diet rich in FTM with adequate exercise could probably evolve as an effective alternative to pharmacotherapy, at least during the initial stages of dyslipidemia. This may offer a chance for patients to avoid the adverse effects of pharmacotherapy when hyperlipidemia is in the early stages.

ACKNOWLEDGMENT

We would like to thank our institution, Gandhi Medical College, Secunderabad, for supporting us throughout our study.

REFERENCES

1. Shah B and Mathur P. Surveillance of cardiovascular disease risk factors in India: The need & scope. Indian J Med Res. 2010;132(5):634-642.

https://doi.org/10.4103/0971-5916.73420

2. Rader DJ, Hoeg JM and Brewer HB Jr. Quantitation of plasma apolipoproteins in the primary and secondary prevention of coronary artery disease. Ann Intern Med. 1994;120(12): 1012-1025.
   https://doi.org/10.7326/0003-4819-120-12-199406150-00008
3. Grundy SM. Hypertriglyceridemia, atherogenic dyslipidemia, and the metabolic syndrome. Am J Cardiol. 1998;81(4):18B-25B.
   https://doi.org/10.1016/s0002-9149(98)00033-2
4. Fredrickson DS. An international classification of hyperlipidemias and hyperlipoproteinemias. Ann Intern Med. 1971;75(3): 471-472.
   https://doi.org/10.7326/0003-4819-75-3-471
5. Haffner SM. Diabetes, hyperlipidemia, and coronary artery disease. Am J Cardiol. 1999;83(9):17F-21F.
   https://doi.org/10.1016/s0002-9149(99)00213-1
6. Saleh AS, Zhang Q, Chen J and Shen Q. Millet grains: nutritional quality, processing, and potential health benefits. Compr Rev Food Sci Food Saf. 2013;12(3):281-295.
7. Choi YY, Osada K, Ito Y, Nagasawa T, Choi MR and Nishizawa N. Effects of dietary protein of Korean foxtail millet on plasma adiponectin, HDL-cholesterol, and insulin levels in genetically Type 2 diabetic mice. Biosci Biotechnol Biochem. 2005;69(1): 31-37.
   https://doi.org/10.1271/bbb.69.31
8. Ren X, Chen J, Molla MM, Wang C, Diao X and Shen Q. *In vitro* starch digestibility and *in vivo* glycemic response of foxtail millet and its products. Food Funct. 2016;7(1):372-379.
   https://doi.org/10.1039/c5fo01074h
9. Ren X, Chen J, Wang C, Molla MM, Diao X and Shen Q. *In vitro* starch digestibility degree of gelatinization and estimated glycemic index of foxtail millet-derived products: Effect of freezing and frozen storage. J Cer Sci. 2016;69:166-173.
10. Tziomalos K, Athyros VG, Karagiannis A and Mikhailidis DP. Lipid lowering agents and the endothelium: An update after 4 years. Curr Vasc Pharmacol. 2012;10(1):33-41.
    https://doi.org/10.2174/157016112798829742
11. Balakumar P, Kathuria S, Taneja G, Kalra S and Mahadevan N. Is targeting eNOS a key mechanistic insight of cardiovascular defensive potentials of statins? J Mol Cell Cardiol. 2012;52(1): 83-92.
    https://doi.org/10.1016/j.yjmcc.2011.09.014
12. Antonopoulos AS, Margaritis M, Lee R, Channon K and Antoniades C. Statins as anti-inflammatory agents in atherogenesis: Molecular mechanisms and lessons from the recent clinical trials. Curr Pharm Des. 2012;18(11):1519-1530.
    https://doi.org/10.2174/138161212799504803
13. Stroes ES, Thompson PD, Corsini A, Vladutiu GD, Raal FJ, Ray KK, et al. Statin-associated muscle symptoms: Impact on statin therapy-European Atherosclerosis Society consensus panel statement on assessment, aetiology and management. Eur Heart J. 2015;36(17):1012-1022.
14. McConnell EL, Basit AW and Murdan S. Measurements of rat and mouse gastrointestinal pH, fluid and lymphoid tissue, and implications for *in-vivo* experiments. J Pharm Pharmacol. 2008;60(1):63-70.
    https://doi.org/10.1211/jpp.60.1.0008
15. Marques C, Meireles M, Norberto S, Leite J, Freitas J, Pestana D, et al. High-fat diet-induced obesity Rat model: A comparison between Wistar and Sprague-Dawley Rat. Adipocyte. 2015;5(1):11-21.
    https://doi.org/10.1080/21623945.2015.1061723
16. Priyadharsini KS, Ramanna MK, Somu L and Prasad TK. A comparative study on efficacy of lutein and atorvastatin on lipid profile and lipoprotein(A) in hypercholesterolemic male wistar rats. Biomed Pharmacol J. 2020;14(1):503-511.
17. Gupta R, Rao RS, Misra A and Sharma SK. Recent trends in epidemiology of dyslipidemias in India. Indian Heart J. 2017;69(3):382-392.
    https://doi.org/10.1016/j.ihj.2017.02.020
18. Singhal P and Kaushik G. Therapeutic effect of cereal grains: A review: Crit Rev Food Sci Nutr. 2016;56(5):748-759.
    https://doi.org/10.1080/10408398.2012.714417
19. Malhotra HS and Goa KL. Atorvastatin: An updated review of its pharmacological properties and use in dyslipidaemia. Drugs. 2001;61(12):1835-1881.
    https://doi.org/10.2165/00003495-200161120-00012
20. Jia YJ, Liu J, Guo YL, Xu RX, Sun J and Li JJ. Dyslipidemia in rat fed with high-fat diet is not associated with PCSK9-LDL-receptor pathway but ageing. J Geriatr Cardiol. 2013;10(4):361-368.
    https://doi.org/10.3969/j.issn.1671-5411.2013.04.007
21. Sireesha Y, Kasetti RB, Nabi SA, Swapna S and Apparao C. Antihyperglycemic and hypolipidemic activities of *Setaria italica* seeds in STZ diabetic rats. Pathophysiology. 2011;18(2): 159-164.
    https://doi.org/10.1016/j.pathophys.2010.08.003
22. Roglans N, Sanguino E, Peris C, Alegret M, Vázquez M, Adzet T, et al. Atorvastatin treatment induced peroxisome proliferator-activated receptor alpha expression and decreased plasma nonesterified fatty acids and liver triglyceride in fructose-fed rats. J Pharmacol Exp Ther. 2002;302(1):232-239.
    https://doi.org/10.1124/jpet.302.1.232

Authors Contribution:
PRO- Definition of intellectual content, literature survey, implementation of the study protocol, data collection, statistical analysis, first draft of manuscript preparation; **SS**- Concept, design, pre clinical protocol, manuscript preparation, editing, manuscript revision, submission of article; **BG**- Design of study, data analysis, and interpretation; **SE**- Literature survey and preparation of Figures; **CK**- Co-ordination and manuscript revision.

Work attributed to:
Gandhi Medical College, Musheerabad, Secunderabad - 500 003, Telangana, India.

Orcid ID:
Praneeth Reddy Onteddu - https://orcid.org/0009-0004-6620-6332
Suguna Sajja - https://orcid.org/0000-0001-8874-3076
Bhuvaneshwari Gajala - https://orcid.org/0000-0002-2052-5301
Shravya Edavaluru - https://orcid.org/0009-0009-2118-2513
Chaitanya Kandula - https://orcid.org/0009-0008-3713-2249

Source of Funding: None, **Conflicts of Interest:** None.