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Original Research Article

Evaluation of hypolipidemic effect of *Pueraria tuberosa* extract on high-fat diet induced hyperlipidemia in Wistar rats

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ABSTRACT

Background: Low-density lipoproteins (LDL)-C plays a pivotal role in the development of atherosclerosis, and its reduction is a primary target in the management of dyslipidemia. *Pueraria tuberosa* contains Puerarin, increases LDL receptor (LDLr) mRNA and HMG-CoA reductase mRNA invitro. However, its role as an hypolipidemic agents needs further elucidation.
Methods: IAEC permission was sought (IAEC/GSMC/05/2022). Wistar rats of either sex around 150-200 grams were divided into 5 groups (n= 8). Group NC (Normal control) received a normal chow diet while Group DC (disease control), Group PC (Atorvastatin 10mg/kg/day), Group PTE LD (PTE 500mg/kg/day) and Group PTE HD (PTE 1000mg/kg/day) received high-fat diet for 45 days. Atorvastatin and *Pueraria tuberosa* aqueous extract (PTE) were given from the 15th day to the 45th day to the respective groups. Lipid profile, and HMG COA reductase enzyme levels were analysed and compared between groups using one-way ANOVA and Kruskal-Wallis test followed by post hoc analysis ($p<0.05$).
Results: PTE HD significantly decreased total cholesterol and LDL-C, compared to the disease control group.
Conclusions: According to our findings, *Pueraria tuberosa* aqueous extract exert a hypolipdemic effect at higher doses as evidenced by decrease in total cholesterol and LDL-C.

Keywords: Dyslipidemias, Low-density lipoproteins, HMG COA reductase inhibitors, Cholesterol, Plant extract

INTRODUCTION

Dyslipidaemias are a type of metabolic disorder that affects the lipid profile and can be characterized by a combination of elevated levels of TC (total cholesterol), LDL-C (low-density lipoprotein cholesterol), and TGs (triglycerides), as well as a lowered level of HDL-C (high-density lipoprotein cholesterol) in the blood plasma.[1] According to the Global Burden of Disease study conducted in 2019, a total of 3.78 million, which was about 44.3% of deaths from ischemic heart disease and 0.61 million which was around 22.4% of deaths from ischemic stroke were due to elevated LDL-C levels.[2] In India, 79% of people have at least one lipid abnormality in which around 11.8% were found to have elevated LDL-C, 72.3% individuals with lowered HDL-C, 29.5% with elevated triglycerides. High LDL-C forms a major risk factor for atherosclerotic cardiovascular events (ASCVD).[3] Access to healthcare is also not optimal in India, and the treatment for ASCVD remains expensive. The years of life lost because of ASCVD in India increased by 59%, from 23.2 million in 1990 to 37 million in 2010.[4] Hyperlipidaemia is a common predicament in society due to changes in lifestyle and food practices. Developing societies worldwide are shifting away from an agrarian existence to the current environment of high energy consumption, minimal physical activity and a lifestyle that includes stress and anxiety which are some of the several factors implicated in the development of CAD.[5] Recent studies conducted in India have unveiled alarming statistics: between 10% to 25% of myocardial infarctions (MIs), commonly known as heart attacks, occur in this younger demographic. Furthermore, over 50% of CAD-associated deaths in India transpire before individuals reach the age of 50. These findings underscore a significant and worrying trend of cardiovascular afflictions affecting younger populations, highlighting the urgent need for targeted interventions and preventive measures.[3] Lipid lowering therapies primarily aims to reduce the risk of atherosclerotic cardiovascular events (ASCVD). American College of Cardiology (ACC)/ American Heart Association (AHA) and European Society of Cardiology (ESC) are the most widely used guidelines so far. Statins are the first-line agent used to lower LDL-cholesterol and ezetimibe can be added if the desired LDL-C not achieved with maximally tolerated statins. If the LDL-C levels are still not under control and further reduction of ≤ 20% is needed bempedoic acid is added. PCSK9 inhibitors may be considered if > 20% LDL-C reduction required as they have shown to reduce LDL-C by 50-60% and also have shown CV risk reduction in large outcome trials.[6] Statin-related muscle symptoms have been variably reported by about 1-2% of patients.[7] In the real world, only 20-30% of patients attain recommended LDL-C levels with statin therapy.[8] The response to statins varies between 5 - 70% and this heterogeneity might be due to polymorphism in genes involved in cholesterol metabolism, and non-adherence to treatment, in that study only 47.5% (n=671) of patients had obtained less than 80% of the refills of their prescribed statins at 1 year.[9]

Due to increase in cost and adverse effects of the lipid lowering therapy, use of natural substances has been increasing nowadays. As per WHO, 75-80% of the world population in developing countries seek herbal medicines for the chronic condition.[10] Medicinal properties of only about 12.5% of the total 4,22,000 plant species were documented.[11] This indicates that there is an unmet need for lipid lowering therapy and to explore herbal medicines. *Pueraria tuberosa* (Roxb. ex Willd.) DC. (Fabaceae), commonly known as Indian Kudzu or Vidari kand, contains various flavonoids and isoflavones, including puerarin (8.31%), daidzein (1.70%), genistein (1.37%), tuberosin, 4-methoxy puerarin, quercetin, hydroxytuberosone, biochanin A, biochanin B, irisolidone, C-glycoside (4′,6-diacetyl), puerarone, tectoridin, and robinin.[12,13] Puerarin, has been found to upregulate LDL receptor (LDLr) mRNA and suppression of HMG-CoA reductase mRNA.[14] Additionally, *Pueraria tuberosa* shows significant antioxidant, anti-inflammatory, and anti-diabetic properties, which are important in preventing atherosclerosis.[15] Though there are studies which explored the total cholesterol and triglycerides lowering properties of *Pueraria tuberosa,* our study is the first of its kind to explore its effect on LDL-C, HDL-C and HMG CoA reductase enzyme.

The high-fat diet model (HFD) is a well-established method for inducing hypercholesterolemia, mimicking the dietary patterns observed in humans consuming western diet. Chemical models which using Triton X100, results in acute hypercholesterolemia whereas the HFD establishes chronic hypercholesterolemia allowing to study the long-term lipid metabolism and the progressive nature of cholesterol buildup.[16] The HFD model's tendency to elevate low density lipoproteins (LDL-C) and TC makes it a valuable model to study the effects of the test drug in lipid profile and atherosclerosis.

In this study we used Wistar rats due to their ease of handling, low cost and the extensive availability of genomic data.[17] Although rats are resistant to atherosclerosis, the use of a HFD containing additives like cholic acid can induce a significant increase in LDL-C which makes them a useful model for diet-induced hypercholesterolemia.[16]

Our study aimed to evaluate the hypolipidemic effect and anti-atherosclerotic effect of *Pueraria tuberosa* extract in HFD induced hyperlipidemia in Wistar rats.

METHODS

The institutional animal ethics committee permission for taken (IAEC/GSMC/05/2022). The Committee for Control and Supervision of Experiments on Animals (CCSEA) guidelines were followed during the entire study.

Experimental animals

56 Wistar Rats of either sex (16 for standardization and 40 for study groups), weighing around 150-200 grams and aged 4-6 weeks, randomly bred in the central animal house of the institute were used for the study. The rats were housed in the experimental area 7 days prior to the initiation of the experiment to allow for acclimatization.

Husbandry conditions

Animals were housed in polypropylene cages with stainless steel top grill having facilities for providing food and water. Paddy husk was used as the bedding. Animals were housed in an air-conditioned room with 12 – 15 filtered fresh air changes and regulated conditions were maintained with temperature 23°C±4°C, relative humidity 30-70% and a 12-hour light-dark cycle. Animals were housed under standard laboratory conditions with free access to filtered water and commercial animal feed in the form of pellets.

Study drugs

Test drugs

Aqueous extract of pueraria tuberosa: Standardised aqueous extracts were procured from the Shree

Dhootapapeshwar Limited, Ayurvedic Research Foundation, Panvel, Navi Mumbai (Extractive Value-23.57%). The extract was prepared by using 50 grams of coarse powder of *Pueraria tuberosa* was boiled with 5 volumes of water. The volume was later reduced to quarter and filtered. The filtered extract was then washed with hexane in separating funnel and then aqueous part was collected and concentrated by a rotatory evaporator and lyophilized. The doses used in the study were 500 mg/kg/day (low-dose) and 1000 mg/Kg/day (high-dose) which were derived from the previous study of aqueous extract of *Pueraria tuberosa* extract done to evaluate its anti-oxidant effect on Wistar rats.[13]

Positive control

Atorvastatin: The drug was procured from a standard drug e-market for research purposes (Pharmacupboard.com). The dose used in the study was 10 mg/kg and was taken from previous study.[18]

Diet

Normal diet: Normal chow diet in the form of pellet.

High-fat diet: The composition of the diet used was powdered NPD – 235gm/kg , DL-methionine -3 gm/kg, sucrose- 200gm/kg, lactose – 200gm/kg, Vanaspati oil (dalda) (hydrogenated vegetable oil) – 200gm/kg, cholesterol 50 gm/kg, Cholic acid – 20gm/kg, choline chloride- 4 gm/kg, Thiouracil – 1.5 gm/kg, Mineral mix- 35gm/kg, vitamin mix – 10gm/kg, cellulose – 41.50 gm/kg.[16]

Experimental procedure

40 Wistar rats were divided into 5 different groups. All the groups received high-fat diet except normal control group which received normal diet. The procedure was followed for 6 weeks and variables were assessed as per study protocol. The overview of the experimental procedure is given in Table 1.

Table 1: Overview of the study groups.

No.	Group (n=8)	Diet received	Drug (oral gavage)
1.	Normal control (NC)	Normal diet	Normal saline (1 ml)
2.	Disease control (DC)		Normal saline (1 ml)
3.	Positive control (PC)		Atorvastatin 10mg/kg/day
4.	*Pueraria tuberosa* aqueous extract low dose (PTE LD)	High-fat diet	PTE 500mg/kg/day
5.	*Pueraria tuberosa* aqueous extract high dose (PTE HD)		PTE 1000mg/kg/day

Drug administration

The aqueous extracts of *Pueraria tuberosa* 500mg/Kg/day and 1000mg/kg/day were suspended in normal saline. Drugs were administered orally using feeding cannula. The appropriate doses were given to their respective groups from 15th day till 45th day for a duration of 1 month.

Variables assessed

At the end of the 45th day (6 week), total body weight, total cholesterol, LDL-C, HDL-C, triglycerides, were assessed from serum. The animals were sacrificed by cervical dislocation method. The dissection was done on ice pack platform to maintain the temperature to prevent degradation of the enzymes. Liver tissue obtained and made into homogenate for measuring HMG COA Reductase levels by ELSIA. Aorta identified and dissected for histopathology (HPE) analysis. The ELISA kit was purchased from Allianz Bioinnovation (Fine Test), Mumbai. Lipid profile and HPE analysis were done in unique Biodiagnostics Vet Path lab in Mumbai by veterinary pathologist.

Statistical analysis

The results were expressed in mean±SD. The level of significance (p-value) for each comparison in the analysis was calculated at 0.05. Data were analysed using GraphPad Instat V3.06 (free version). Normality was checked by Kolmogorov and Smirnov Test.

The parametric data between five groups were compared using one-way-ANOVA followed by post hoc Tukey's test while the non-parametric data between five groups were compared using Kruskal-Wallis Test followed by post hoc analysis.

RESULTS

Total body weight

At the end of 45th day, the groups which consumed HFD (DC, PC, PTE LD, PTE HD) showed decreased body weight than the group which consumed the normal diet (NC) and the weights were comparable to each other. (Figure 1).

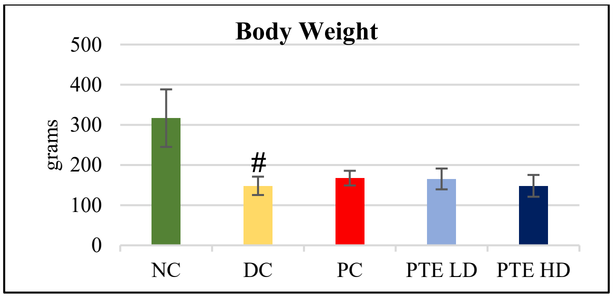

Figure 1: Comparison of total body weight of wistar rats (n=8).

$p<0.01$ vs NC, Kruskal-Wallis test followed by post hoc Dunn's test.

Total cholesterol

The Normal Control (NC) group exhibited a mean total cholesterol level of 65.88±32.38 mg/dL. In the Disease Control (DC) group, the mean total cholesterol level significantly increased to 300.75±38.52 mg/dl. The Positive Control (PC) group, which received Atorvastatin, showed a significant reduction in total cholesterol levels compared to the DC group, with a mean of 190.75±18.81 mg/dl (**$p<0.01$ vs. DC). For the groups treated with *Pueraria tuberosa*, the low dose (PTE LD) resulted in a mean total cholesterol level of 294.13±20.63 mg/dL, which was not significantly different from the DC group but was significantly higher than the PC group ($$$$p<0.001$). The high dose (PTE HD) group showed a further reduction in total cholesterol levels to 239.88±32.59 mg/dL, which was significantly lower than the DC group (*$p<0.05$) (Figure 2).

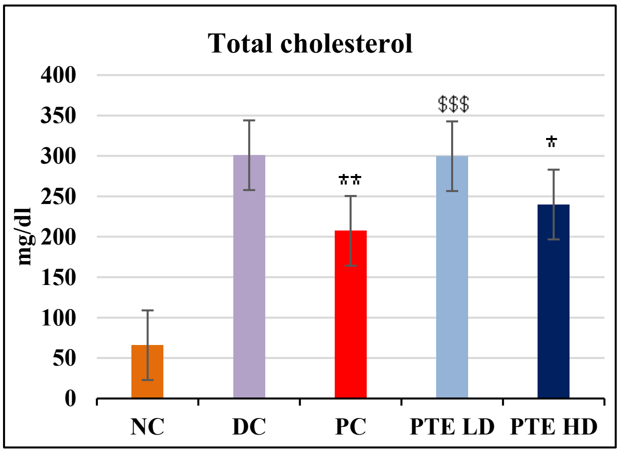

Figure 2: Comparison of total cholesterol (n=8).

*$p<0.05$, **$p<0.01$ vs DC, $$$$p<0.001$ vs PC; ANOVA followed by post hoc Tukey's test.

Low-density lipoprotein cholesterol

The Normal Control (NC) group exhibited a mean LDL-C level of 23.85±17.73 mg/dL. In contrast, the Disease Control (DC) group, showed a significantly elevated mean LDL-C level of 243.21±39.62 mg/dl. The Positive Control (PC) group, demonstrated a significant reduction in LDL-C levels, with a mean of 117.46±24.74 mg/dl (***$p<0.001$ vs. DC). The low dose (PTE LD) resulted in a mean LDL-C level of 224.66±19.55 mg/dL which did not show significant difference from the DC group. However, the high dose (PTE HD) group exhibited a mean LDL-C level of 159.25±31.84 mg/dl, which was significantly lower than the DC group (***$p<0.001$), but significantly higher than the PC group ($$p<0.05$). (Figure 3).

High-density lipoprotein cholesterol

The groups which consumed HFD (DC, PC, PTE LD, PTE HD) showed elevated HDL levels compared to the group which consumed normal diet (NC). PTE HD group showed significantly elevated HDL-C compared to DC group (Figure 4).

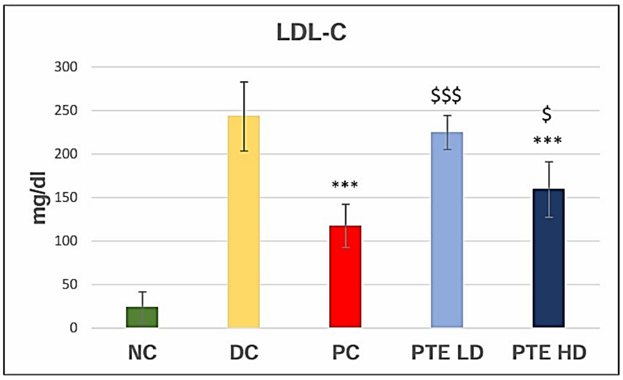

Figure 3: Comparison of LDL-C (n=8).

***$p<0.001$ vs DC, $$p<0.05$ $$$$p<0.001$ vs PC; one-way ANOVA followed by post hoc Tukey's test.

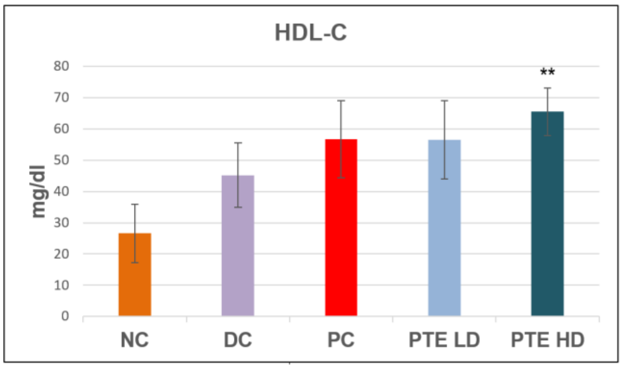

Figure 4: Comparison of HDL-C (n=8).

**$p<0.01$ vs DC, One-way ANOVA followed by post hoc Tukey's test.

Triglycerides

The groups which consumed NC showed elevated triglyceride levels compared to the groups which fed HFD (DC, PC, PTE LD, PTE HD). They showed significantly decreased triglycerides levels which were comparable among each other.

HMG-COA reductase

The Normal Control (NC) group exhibited a mean HMG-CoA reductase level of 15.58±1.05 ng/ml. In the Disease Control (DC) group, there was a significant increase in HMG-CoA reductase levels to 26.40±1.62 ng/ml (###p<0.001 vs. NC). The Positive Control (PC) group showed a significant reduction in HMG-CoA reductase levels compared to the DC group, with a mean of 18.88±1.39 ng/ml (*p<0.05 vs. DC).

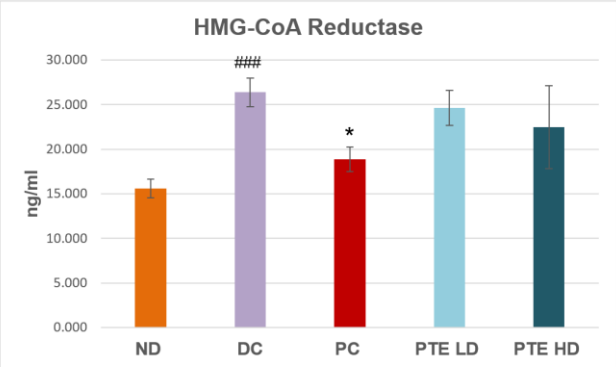

Figure 5: Comparison of HMG-COA reductase enzyme (n=8).

###p<0.001 vs NC; *p<0.05 vs DC; Kruskal-Wallis test followed by post hoc analysis.

For the groups treated with *Pueraria tuberosa*, the low dose (PTE LD) had a mean HMG-CoA reductase level of 24.67±1.96 ng/mL, while the high dose (PTE HD) showed a level of 22.49±4.63 ng/ml. Neither the PTE LD nor the PTE HD group showed a statistically significant reduction in HMG-CoA reductase levels compared to the DC group and PC group.

DISCUSSION

LDL-C plays a vital role in the development of atherosclerosis, and its reduction is the primary target in the management of dyslipidaemia. Experimental models such as LDL receptor deficient mice have shown to have elevated LDL-C which directly contribute to atherosclerosis, emphasizing the importance of targeting LDL-C to reduce cardiovascular risk.[19,20]

In our study high dose of aqueous extract of PTE treated group demonstrated a significant decrease in low-density lipoprotein (LDL-C) and total cholesterol level (TC) compared to the disease control group (DC). Although the Positive Control (Atorvastatin) group showed an even greater reduction in total cholesterol and LDL-C, the effect of PTE HD was significant, confirming its ability to lower the harmful cholesterol fraction. The lower dose of PTE showed less significant effect than PTE HD suggesting that aqueous extract of PTE shows dose-dependent response.

In a study conducted by Aditi and Tripathi et al, (2019), where male Charles Foster strain rats fed on a high fat diet for 100 days to induce NAFLD both low dose and high dose of PTE resulted in reductions of total cholesterol and triglycerides compared to the disease control group. Although their study did not asses LDL and HDL, the results for total cholesterol reduction is consistent with our findings.[21] Another study done by Satpathy et al, (2021) evaluated the hypolipdemic effect of ethyl acetate fraction of *Pueraria tuberosa* which is rich in phenolic content and antioxidant activity, in an ovariectomized rat model. This model, which simulates menopause-induced osteoporosis, also leads to increased cholesterol, triglycerides, and body weight due to reduced estrogen levels. While their study used the ethyl acetate extract, and did not measure HDL and LDL, our study utilized the aqueous extract, yet both extracts demonstrated significant reductions in total cholesterol. This suggests that *Pueraria tuberosa* possesses hypolipidemic potential across different types of extracts, highlighting the plant's versatile therapeutic properties in managing hyperlipidemia.[22]

Our study provides new evidence supporting the in vitro findings by Chung et al (2008), demonstrating that, particularly at high doses, can effectively reduce LDL-C levels in vivo. This aligns with Chung et al's observations of puerarin glycosides enhancing LDL receptor promoter activity in a dose-dependent manner in HepG2 cells, suggesting that 's LDL-C-lowering effect is consistent across different experimental conditions. Puerarin also reduced HMG-CoA reductase mRNA in vitro whereas in our study PTE HD manage to reduce the enzyme levels which was not statistically significant and not as pronounced in the Positive Control group (Atorvastatin) suggesting that it may be influenced by other in vivo factors which needs further investigations to understand the mechanism.[14]

In our study, the high-fat diet consumed groups (DC, PC, PTE LD, PTE HD) showed an elevated HDL-C levels and the administration of PTE at high dose have showed significant elevation than DC group. This contrast with the general expectation that the high-fat diets typically lower HDL-C levels and similar findings were previously reported by Yurina et al, where Wistar rats fed high -fat diet initially showed increased HDL-C levels followed by a decline with prolonged feeding.[17] It may be attributed to the activation of reverse cholesterol transport (RCT), a process which facilitates the removal of cholesterol from peripheral tissues and its transport back to liver for excretion. Also, the low or absent expression of cholesterol ester transfer protein (CETP) in Wistar rats, which is responsible for the conversion of HDL to LDL and VLDL contribute to elevated HDL-C.

In our study the DC, PC, PTE LD and PTE HD groups which consumed high-fat diet showed reduction in TG levels compared to the NC group, which was an unexpected outcome considering the high-fat diets usually increase TG levels. These findings suggest that the presence of cholate which is known to enhance the absorption of dietary cholesterol and compensatory decrease in the cholesterol synthesis in liver. Additionally, it has been shown to downregulate Acyl-CoA synthase mRNA, reducing the conversion of fatty acids into triglycerides which explains the reduced TG levels in our study. These findings align with a previous study done by Ikemoto et al.[23]

In our study the reduction in total body weight in the groups which consumed high-fat diet (DC, PC, PTE LD, PTE HD) compared to the NC group contrasts the expected outcome of increase in weight when high fat diet is consumed. These results align with the study done by Ble-Castillo et al (2012)[24], where rats fed high-fat diet showed reduced weight gain compared to those which fed normal chow diet. The differences in food consistency between the high-fat and normal chow diets could have influenced feeding patterns, potentially leading to reduced overall food intake in the high-fat diet group. Additionally, the high-fat diet's slower digestion may have had a higher satiating effect, contributing to the observed weight reduction. The presence of cholate in the diet, which prevents obesity by influencing bile acid composition and fat absorption, further supports this unexpected outcome, as previously reported by Ikemoto et al.[23]

While the DC, PC, and PTE LD, PTE HD groups exhibited similar body weights, it is worth noting that *Pueraria tuberosa* did not significantly influence weight compared to the DC group. This observation contrasts with findings from Tripathi et al and Satpathy et al, where *Pueraria tuberosa* and its ethyl acetate fraction significantly reduced body weight in rats fed a high-fat diet.[21,22] The discrepancy could be attributed to external factors, duration, or the type of extract used. Although the exact mechanism remains unclear, these studies suggest that *Pueraria tuberosa* may exert weight-modulating effects under certain conditions, warranting further exploration into its underlying mechanisms.

When compared to the study conducted by Chung et al, 2008[14] where puerarin was shown to reduce HMG-CoA reductase mRNA and protein levels in vitro, our findings align in showing the enzyme-inhibitory properties of PTE. The in vitro study demonstrated a more pronounced reduction, which might be attributed to the controlled conditions and direct application of puerarin. In contrast, our in vivo model, while showing a similar trend, suggests that the effectiveness of PTE in reducing HMG-CoA reductase may be less potent or influenced by other in vivo factors. This comparison highlights the need for further investigation to fully understand the mechanisms and optimize the use of for cholesterol reduction.

Also, the histopathology of aorta did not reveal any atherosclerotic plaques which can be explained by the high HDL-C levels in Wistar rats which could have counteracted the development of atherosclerosis. HDL-C facilitates reverse cholesterol transport (RCT), promoting efflux of cholesterol from peripheral tissues back to the liver for excretion. Additionally, rats possess inherent physiological differences compared to humans, including variations in vessel size, surface area, blood flow, pulse rate, blood pressure, and shear stress, all of which influence the susceptibility to atherosclerosis. These hemodynamic factors create a vascular environment in rats that is less conducive to plaque formation compared to humans, who are more prone to atherosclerotic changes under similar lipid conditions.[25] The absence of plaque in our study could also be related to the specific sections of the aorta analysed, as the precise location within the vasculature plays a crucial role in plaque formation and progression. Identifying and quantifying atherosclerotic lesions accurately is often challenging due to these location-specific differences. The absence of lesions in the examined sections does not necessarily indicate a complete lack of atherosclerotic changes but rather highlights the complexities associated with plaque localization and detection in animal models.

Our study is one of the first study to explore the effects of *Pueraria tuberosa* on LDL-C, TC, HDL-C, TG, HMG - CoA Reductase and compare it with the standard treatment atorvastatin. Testing low and high doses of extract provides valuable data in dose-dependent effects aiding in optimal dosing strategy in future research.

The limitation of this study was its short duration limited to a period of six weeks in Wistar rats due to budgetary constraints. This may have limited the ability to assess the long-term effects of PTE on lipid levels and the development of atherosclerotic plaques.

Evaluation in diverse models such as transgenic mice (apoE-/-,LDLr-/-), rabbits, with extended study duration and inclusion of broader biomarkers like lipoprotein(a), apolipoprotein levels in the plasma, S-100 proteins in the atherosclerotic plaques are warranted to confirm its translatability and understand the molecular mechanism of *Pueraria tuberosa*.

CONCLUSION

According to our findings, high dose of Pueraria tuberosa exhibited a hypolipidemic effect as evidenced by reduction in total cholesterol and LDL-C. Further studies in transgenic models are needed to confirm its efficacy and translational relevance.

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