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ORIGINAL ARTICLE

Assessing The Efficacy of *Delonix regia* Flowers in A High-Fat Diet for Hyperlipidemia

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ABSTRACT

Rats given a high-cholesterol diet are used in this study to assess the impact of the flavonoid-rich extract of the Delonix regia flower on hyperlipidemia. In this work, we employ established models of total cholesterol, triglyceride levels, LDL cholesterol, VLDL cholesterol, antioxidants, and histological observation to evaluate the efficacy of test drugs in treating hyperlipidemia in rats induced by a high-cholesterol diet. The total cholesterol levels in the blood serum were considerably and dramatically reduced when Delonix regia was taken orally. Furthermore, in a dose-dependent manner, Delonix regia dramatically decreases blood serum triglyceride levels. Intriguingly, however, a 200 mg/kg dose of Delonix regia results in a significant drop in blood serum HDL cholesterol levels. Furthermore, in a dose-dependent manner, Delonix regia dramatically decreases blood serum LDL cholesterol levels. Furthermore, in a dose-dependent manner, Delonix regia dramatically decreases blood serum levels of VLDL cholesterol. The interesting finding is that the atherogenic index dropped at 200 mg/kg. Test medications, however, exhibit a discernible drop in LPO levels. However, it was found that a test drug enhanced CAT and GSH. Based on the study's findings, oxidative metrics, and histological observations, Delonix regia was found to have passionate antihyperlipidemic effects.

Keyword- *Hyperlipidemia, TC, TG, LDL, HDL, VLDL*

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INTRODUCTION

A significant number of risk factors for the development of cardiovascular disease include hypercholesterolemia and hypertriglyceridemia. One effective way to treat atherosclerosis is to lower the plasma cholesterol level. High liquid-lowering speed and good efficacy are characteristics of many chemical medicines, including statins and fibrates. However, because there are a variety of hyperlipidemia patients, possible side effects, and high levels of medication dependence, they are unable to meet the needs for treatment [1]. In contrast, plant materials and their extracts have several targets for both preventing and treating hyperlipidemia, along with negligible side effects. Overweight and obesity affect over 2 billion people globally, making it one of the biggest public health problems of the twenty-first century. This is a dangerous long-term condition that raises the chance of death and concomitant conditions such as metabolic syndrome, cancer, and heart disease. This could indicate aging quickly. Atherosclerosis, obesity, arteriosclerosis, stroke, and other significant conditions are all included in the category of cardiovascular illnesses. Obesity has been linked to an increased risk of developing illnesses that affect the organ systems in the body. The main serious health concern linked to eating a high-fat diet (HFD) is dyslipidemia. A prevalent condition among those suffering from non-alcoholic fatty liver disease (NAFLD) is hyperlipidemia. The etiology of nonalcoholic fatty liver disease (NAFLD) includes triglyceride accumulation and the deleterious consequences of oxidative stress on hepatocytes, the cells that make up

the liver. Patients with hyperlidemia often struggle with oxidative stress. The development of oxidized low-density lipoprotein, or ox-LDL, raises the risk of atherosclerosis, type 2 diabetes, and coronary artery disease and is linked to hyperlipidemia [2]. Researchers can gain a better understanding of the genesis of hyperlipoproteinemias by looking at the documented routes of lipoprotein metabolism. Hyperlipidemia can refer to either hypertriglyceridemia, hypercholesterolemia, or both. To change hyperlipidemia into hyperlipoproteinemia, one must identify the high lipoprotein family or families. Hyperlipoproteinemia can result from reduced catabolism, excessive synthesis of a particular lipoprotein family or families, or from a mix of the two. Lipoprotein metabolism and structure Each lipoprotein family is made up of macromolecular complexes composed of proteins and lipids. The primary lipid components of the proteins, often referred to as apolipoproteins, are phospholipids, cholesterol, cholesteryl esters, and triglycerides. Since the 1960s, symptoms of metabolic syndrome have been associated with diets high in sugar. Most research on this topic has focused on the mechanism by which metabolites in the liver produced from fructose promote de novo lipogenesis. However, the exact mechanism by which excessive sucrose consumption results in metabolic syndrome is still unknown, as multiple studies have been unable to identify the origin of increased lipogenic enzyme activity or overt symptoms associated with excessive sucrose consumption. We must act right now since there is a public health concern about the growth in the morbidity rate linked to metabolic syndrome caused by significant dietary sugar consumption. Excess sucrose is known to create metabolic syndrome because fructose toxicity is involved. It is believed that the liver is the organ most important to fructose metabolism. A recent study did, however, also highlight the small intestine's vital role in reducing the quantity of fructose flow into the liver. Moreover, we have demonstrated that a high-sucrose diet (HSD) substantially alters the circadian rhythm of the small intestine, resulting in a decrease in the organ's ability to avert fructose poisoning. Therefore, more investigation is needed to understand the effects of fructose toxicity from an HSD on the distal intestine. In this study, we hypothesised that an excess of fructose damages the gastrointestinal tract, causing fatty liver and hyperlipidemia [3]. *Delonix regia* , a tropical and subtropical flowering plant well-known for its vivid ornamental blossoms, is often referred to as Flame Tree or Royal Poinciana. This plant's rich phytochemical makeup, which is the key to its possible therapeutic and medicinal qualities, has drawn scientific attention in addition to its aesthetic appeal. The phytochemical components of D. regia are thoroughly examined in this article, which also sheds light on the variety of bioactive substances it contains and their possible applications. Plants contain a type of polyphenolic chemicals called flavonoids, which are well-known for having anti-inflammatory and antioxidant characteristics. Flavonoids, including as rutin, kaempferol, and quercetin, are especially abundant in D. regia. These substances have astringent qualities. They have been found in the bark and leaves of D. regia, among other places. Tannins are responsible for the bitterness of the plant and may have antibacterial and wound-healing properties. By lowering oxidative stress and inflammation, the antioxidant and anti-inflammatory properties of D. regia constituents may improve cardiovascular health and may lessen the risk of heart disease [4].

MATERIAL AND METHODS

Collection & Authentication of plant

The plant material was collected in the region of Maharashtra, India, from the native ecosystem. The entire plant, including the leaves, pods, and bloom, was collected at the time of collection, and the Botany Department of Jijamata Mahavidyalaya, authenticated it. For subsequent use as a reference, the voucher specimen (number 02/23-3-24) was assigned.

Extraction process

Two kilograms of *Delonix regia* flowers were dried in the shade and ground into a coarse powder. Plant's powdered stem was extracted using the maceration technique. Following the filtering of the solution, alcohol (ethanol) was added to obtain the polysaccharide precipitate. After filtering the solution, the filtrate was evaporated to make up one-fourth of the total volume. Ethyl acetate is used to extract the solution one step at a time after one-fourth of its volume has evaporated. After that, the ethyl acetate extract was evaporated to get the 1% w/w flavonoid-rich fraction of *Delonix regia* , which had a brownish orange color [5].

Animals

The study featured Wistar rats that were in good health and weighted between 150-200g. The rats were obtained from the animal house facility specifically from the Department of Pharmacology. Each animal was housed in a separate cage with an air-conditioned room that had a 12-hour light and dark cycle, a temperature of 22±20C, and a relative humidity of 50%±10%. The animals were kept in standard laboratory settings for the duration of the investigation. The animals were kept on a normal diet of rat pellets (Nutrivet Life science, Pune) and were given unlimited access to clean drinking water. Seven days

before the studies began; the animals were habituated to the laboratory environment. The Institutional Animal Ethical Committee (IAEC/1865/23-24/P-15) of (Registration No. 1865/PO/16/CPCSEA) authorized the experimental protocol.

Acute toxicity studies

The study used female Wister Albino rats weighing between 150 and 200 grams. The OECD-423 criteria were followed for performing acute oral toxicity. Prior to the experiment, the rats were fasted for an entire night, and their individual weights were noted. The animals were split into four groups at random, with two rats in each group. Following OECD-423 criteria, each group was given an oral dose of 1000, 2000, or 3000 mg/kg of the flavonoid-rich extract of the medicinal plant Flowers of *Delonix regia* [6]. After the extract was administered, the animals were closely monitored for four hours. After that, they were observed every day for seven days to look for any changes in their overall behavior or other physical activity.

Drugs

Atorcam-20 (Atorvastatin), flavonoid rich extract of *Delonix regia* . All other chemicals were of analytical grade procured from manufacturers.

Table: Experimental design Groups:

Group (n=6)	Drug	Dose	Route of Administration
Normal control	Saline	-	p.o
High fat control	Cholesterol diet	-	p.o
Positive control	Cholesterol diet+ Atorvastatin	10 mg/kg	p.o
Test 1	Cholesterol diet+ Test 1	100 mg/kg	p.o
Test 2	Cholesterol diet+ Test 2	200 mg/kg	p.o
Test 3	Cholesterol diet+ Test 3	300 mg/kg	p.o

There were six groups of animals (n=6). The regular group (Group 1) was fed an unlimited normal diet. Group II, the control group, was given a cholesterol-free diet. For four weeks, Group IV, V, and VI were given a cholesterol-lowering diet in addition to 100 mg/kg, 200 mg/kg, and 300 mg/kg of the plant extract *Delonix regia* , p.o. As the standard, Group III was given a cholesterol-lowering diet and atorvastatin at a dose of 10 mg/kg p.o. Following the conclusion of the experiment, the animal's blood was drawn by heart puncture, left to clot for 45 minutes, and the serum was separated by centrifugation and lipid profiling.

Preparation of hyperlipidemic diet

The diet was prepared by combining 1 mL of coconut oil, 1% cholic acid, and 2% cholesterol in precise quantities. Because of its high saturated fat content, which exacerbates the rats' atherogenic profile, Parachute coconut oil was selected [7].

Biochemical assays for lipids

The serum was used to assess the amounts of triacylglycerides, HDL, and cholesterol using the GPO-PAP and CHOD-PAP methods, respectively [8]. We estimated LDL and VLDL cholesterol using the method established by Johnson et al [9]. While the procedure outlined by Muruganandan et al [10]. was used to calculate the atherogenic index.

Preparation liver tissue homogenate

In order to extract the animals' livers, on day 30, intraperitoneal ketamine anesthesia (100 mg/kg) was used to make the mice unconscious. The liver was removed and then rinsed in an ice-cold isotonic tris-KCl solution. After the liver's weight was determined, it was homogenized in 7.4 pH phosphate buffer saline (PBS) at 20°C and centrifuged for 10 minutes at 2000 rpm. The supernatant was collected to estimate the results of the several tests [11].

Estimation of lipid peroxidase

A 4 milliliter assay mixture was mixed with 0.1 milliliter homogenate, 1.5 milliliters of 20% (v/v) acetic acid (pH 3.5), 1.5 milliliters of 0.8% (w/v) thiobarbituric acid, and 0.2 milliliters of SDS (8.1% w/v). The aforesaid mixture was blended, heated to 95 °C for an hour on a water bath, cooled under tap water, and then 1 milliliter of distilled water was added. Fill the tube with this liquid. Add 5ml of the 15:1 combination of n-butanol and pyridine. The mixture was centrifuged at 2200 rpm for 10 minutes. The results are expressed in terms of nmol MDA/mg of protein. The translucent, pink supernatant's absorbance was measured at 532 nm [12].

Estimation of glutathione level

Embark 0.1 mL of the supernatant. To lessen the strength of the supernatant, 0.9 milliliters of phosphate (PO4) water were added. Add 1 milliliter (20%) of TCA.I gave the mixture 20 minutes to sit. It was then

spun for ten minutes at 10,000 rpm. A quarter of the liquid was removed. To add, use 0.75 ml of phosphate water. Add 2 milliliters of DTNB, which has a strength of 0.0006M. I sat for ten minutes. By using a spectrophotometer, absorbance at 412 nm was measured [13].

Estimation of Catalase level

We used Claiborne's (1985) method to measure the catalase (CAT) activity. 10% supernatant in 0.05 ml volume is the assay mixture.Incorporate one milliliter of H2O2. Add 1.95 ml of phosphate buffer (0.05 M, pH 7) to make a final volume of 3 ml. Absorbance at 240 nm was determined with a UV spectrophotometer [14].

Histopathological examination

The rats' livers were extracted and placed in a 10% formalin buffer solution. Regular paraffin embedding procedures were followed to prepare 4 µm slices of fixed tissues, which were then dyed with hematoxylin eosin. The stained areas were examined under an optical microscope set to 100 X and 200 X.

Statistical analysis

Values with *p<0.05 were deemed statistically significant. The results are shown as mean±SEM (standard error mean) and are submitted to one-way analysis of variance (ANOVA) and Dunnett's test.

RESULT

Acute toxicity studies

It was discovered from the data that there is no neurological or behavioral side effects, and no mortality at 3000 mg/kg. Therefore, using 100 mg/kg, 200 mg/kg, and 300 mg/kg for the current study's evaluation of the antihyperlipidemic action can be regarded as a safe dose.

Table 1: Acute toxicity study

Group	No. of animal	Dose	Result
1	3	1000mg/kg	No death
2	3	2000mg/kg	No death
3	3	3000mg/kg	No death

Effect of *Delonix regia* on Serum total cholesterol levels:

As depicted in **table 2 figure 1** Administration of Cholesterol diet in rats shown a significant **** (p<0.0001) increase in the cholesterol levels in blood serum compared to respective normal groups. Treatment with standard and test drug at two different doses (200mg/kg & 300mg/kg) had shown a significant **** (p<0.0001) reduction in the amount of total cholesterol when compared to the disease control group. Conversely *Delonix regia* administered orally was exhibited significant and dose dependent effect drastically reduction in serum total cholesterol levels dose dependently.

Effect of *Delonix regia* on Triglyceride levels:

As depicted in table 3 and figure 2 Administration of cholesterol diet in rats shown a significant **** (p<0.0001) increase in the triglyceride levels in blood serum compared to respective normal groups. Treatment with standard and test drug at three different doses (100mg/kg, 200mg/kg & 300mg/kg) had shown a significant *(p<0.05), **** (p<0.0001) reduction in the number of triglycerides when compared to the disease control group respectively. Conversely *Delonix regia* 100 mg/kg was failed to reduction in Serum Triglycerides was found to be non-significant. *Delonix regia* administered orally was exhibited significant and dose dependent effect drastically reduction in serum triglycerides levels dose dependently.

Effect of *Delonix regia* on HDL- cholesterol:

As depicted in table 4 and figure 3 There was a significant **** (p<0.0001) decrease in the HDL cholesterol levels in blood serum of cholesterol diet treated group compared to normal group. Treatment with standard and test drug at three different doses (100mg/kg, 200mg/kg & 300mg/kg) had shown a significant *** (p<0.001), **** (p<0.0001) increase in the amount of HDL-cholesterol level when compared to the disease control group. Whenever *Delonix regia* is administered, it can provide some noteworthy and remarkable effects. More precisely, it can selectively enhance the HDL levels in blood serum.

Effect of *Delonix regia* on LDL-cholesterol:

As depicted in table 5 and figure 4 Significant **** (p<0.0001) increase in the LDL cholesterol levels seen in the blood serum of cholesterol diet treated groups when compared to normal group. Treatment with standard and test drug at two different doses (200mg/kg & 300mg/kg) had shown a significant *(p<0.05), *** (p<0.001) reduction in the amount of LDL-cholesterol when compared to the disease control group. Conversely *Delonix regia* 100 mg/kg & 200 mg/kg was failed to reduction in LDL Cholesterol level in serum was found to be non-significant. *Delonix regia* 300mg/kg administered orally was exhibited

significant and dose dependent effect drastically reduction in serum LDL cholesterol levels dose dependently.

Effect *Delonix regia* on VLDL cholesterol levels:

As depicted in table 6 and figure 5 A Significant **** ($p<0.0001$) elevation in VLDL cholesterol levels in blood serum of cholesterol diet treated groups was observed when compared to normal group. Treatment with standard and test drug at three different doses (100mg/kg, 200mg/kg & 300mg/kg) had shown a significant *($p<0.05$), *** ($p<0.001$), **** ($p<0.0001$) reduction in the amount of VLDL-cholesterol when compared to the disease control group respectively. When administered *Delonix regia* it was shows that remarkable reduction in VLDL cholesterol levels in serum as compared to disease control group. It means test drug possesses the antihyperlipidemic activity against the level of VLDL cholesterol and drastically test drug shows the activity in dose dependent manner.

Effect of *Delonix regia* on Atherogenic index:

As depicted in table 7 and figure 6 During the course of study there was a significant *** ($p<0.001$) increase in the atherogenic index in cholesterol diet treated groups when compared to the normal groups. Treatment with standard and test drug at two different doses (200mg/kg & 300mg/kg) had shown a significant ** ($p<0.01$) reduction in the atherogenic index when compared to the disease control group. Conversely *Delonix regia* 100 mg/kg was failed to reduction in Atherogenic index was found to be non-significant. *Delonix regia* 200 mg /kg & 300mg/kg administered orally was exhibited significant and dose dependent effect drastically reduction in serum atherogenic index dose dependently.

Effect of *Delonix regia* on LPO level:

As depicted in table 8 and figure 7 the disease control group increase the level of LPO (**P< 0.1) as compared to normal group. The *Delonix regia* 100 mg/kg was not able to decrease the level of LPO, as compared to disease control group. Similarly The *Delonix regia* 200 mg/kg & 300 mg/kg was more significantly decrease the level of LPO, (**P < 0.01) as compared to disease control group. The standard group also more significantly decrease the level of LPO, (**P< 0.01) as compared to disease control group. The LPO level was significantly reduced by the *Delonix regia* when given at doses of 200 mg and 300 mg; in a similar vein, the LPO level was most significantly reduced by the 200 mg dose.

Effect of *Delonix regia* on CAT level:

As depicted in table 8 and figure 8 the disease control group significant decrease in the level of CAT (**P< 0.1) as compared to normal group. The *Delonix regia* 100 mg/kg was not able to increase the level of CAT, as compared to disease control group. Similarly The *Delonix regia* 200 mg/kg & 300 mg/kg was more significantly increases the level of CAT, (**P < 0.01) as compared to disease control group. The standard group also more significantly increases the level of CAT, (**P< 0.01) as compared to disease control group. The CAT level was significantly increased by the *Delonix regia* when given at doses of 200 mg and 300 mg; in a similar vein, the CAT level was most significantly increased by the 300 mg dose and it was exhibited significant and dose dependent effect.

Effect of *Delonix regia* on GSH level:

As depicted in table 8 and figure 9 the disease control group significant decreases the level of GSH (**P< 0.1) as compared to normal group. The *Delonix regia* 100 mg/kg was not able to increase the level of GSH, as compared to disease control group. Similarly The *Delonix regia* 200 mg/kg & 300 mg/kg was more significantly increases the level of GSH, (**P < 0.01) as compared to disease control group. The standard group also more significantly increases the level of GSH, (**P< 0.01) as compared to disease control group. The GSH level was significantly increased by the *Delonix regia* when given at doses of 200 mg and 300 mg; in a similar vein, the GSH level was most significantly increased by the 300 mg dose and it was exhibited significant and dose dependent effect.

Histopathological Examination

Figure 10(a) Normal group: Microscopic examination of liver tissue shows normal architecture of hepatocyte, sinusoidal space, central vein and portal tract (H&E, 200x and 100x). Figure 10(b): Disease Control: group- Microscopic examination of liver tissue increased sinusoidal space (black arrow) between hepatocyte and necrotic (red arrow) changes in hepatocyte (H&E, 200x and 100x) Figure 10(c): Standard: Microscopic examination of liver tissue shows minimal inflammatory cells (black arrow) in peri portal tract and normal architecture of hepatocyte and central vein (H&E, 200x and 100x). Figure 10(d) Test 1: Microscopic examination of liver tissue shows high lipid accumulation architecture of hepatocyte, sinusoidal space, central vein and portal tract (H&E, 200 x and 100 x). Figure 10(e) Test 2: Microscopic examination of liver tissue shows normal architecture of hepatocyte, sinusoidal space, central vein and portal tract (H&E, 200x and 100x) Figure 10(f) Test: 3 Microscopic examination of liver tissue shows normal architecture of hepatocyte, sinusoidal space, central vein and portal tract (H&E, 200x and 100x)

Under a microscope, the study's analysis of liver tissue revealed typical portal tract architecture, sinusoidal space, hepatocytes, and central veins. There were fewer inflammatory cells and more sinusoidal space between hepatocytes in the disease control group. The results of the tests revealed no appreciable alterations in the hepatocyte population, high lipid accumulation architecture, normal hepatocyte, sinusoidal space, central vein, and portal tract architecture.
Examining the liver areas revealed statistically significant differences between all groups based on the histological study results.

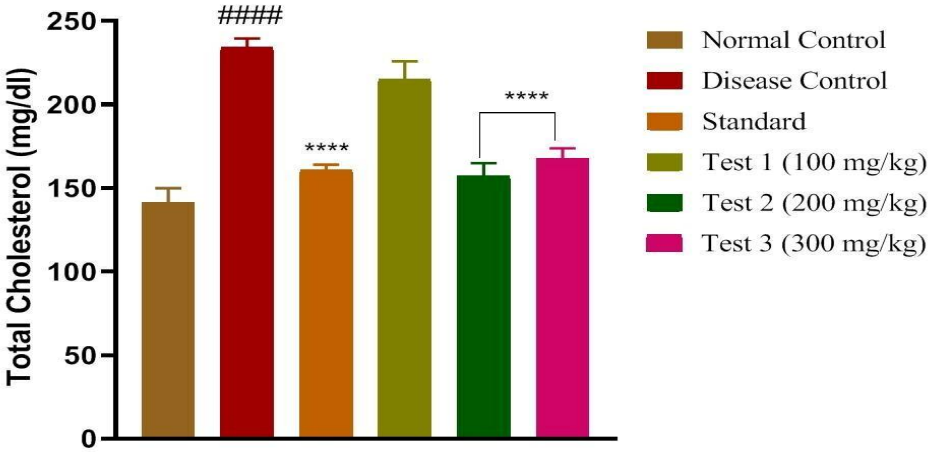

Figure 1 The effects of different doses (100,200 &300 mg/kg) of *Delonix regia* on serum total cholesterol levels. Data are represented as means± SEM (n=6). Ordinary one way ANOVA followed by Dennett's multiple comparison ****P<0.0001, **** P= 0.0005, ****P=0.0002 vs disease control.

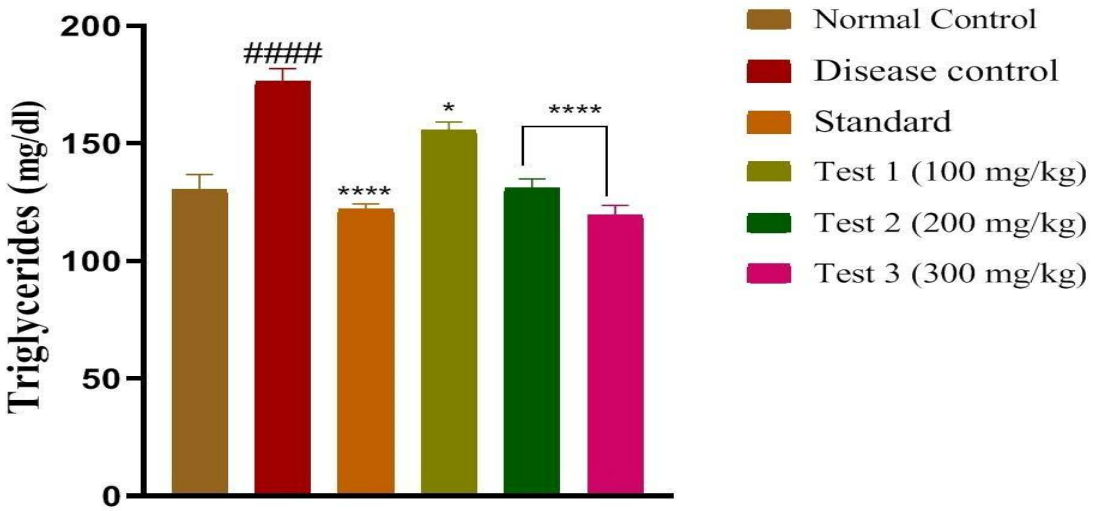

Figure 2 The effects of different doses (100,200 &300 mg/kg) of *Delonix regia* on triglycerides levels. Data are represented as means± SEM (n=6). Ordinary one way ANOVA followed by Dennett's multiple comparison ****P<0.0001, **** P= 0.0005, ****P=0.0002 vs disease control.

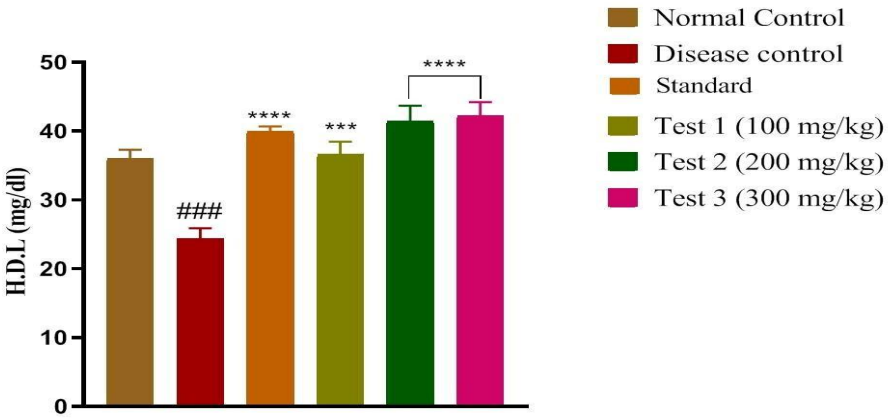

Figure 3 The effects of different doses (100,200 &300 mg/kg) of *Delonix regia* on HDL Cholesterol levels. Data are represented as means± SEM (n=6). Ordinary one way ANOVA followed by Dennett's multiple comparison ****P<0.0001, **** P= 0.0005, ****P=0.0002 vs disease control.

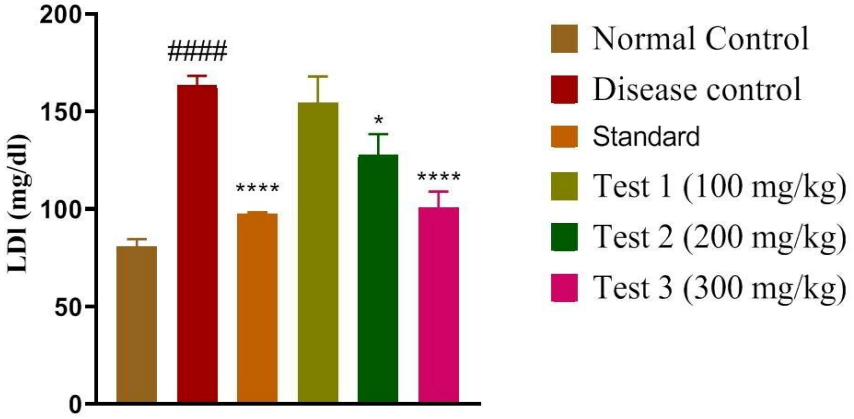

Figure 4 The effects of different doses (100,200 &300 mg/kg) of *Delonix regia* on LDL cholesterol levels. Data are represented as means± SEM (n=6). Ordinary one way ANOVA followed by Dunnetts multiple comparison ****P<0.0001, * P= 0.0005, ***P=0.0002 vs disease control.

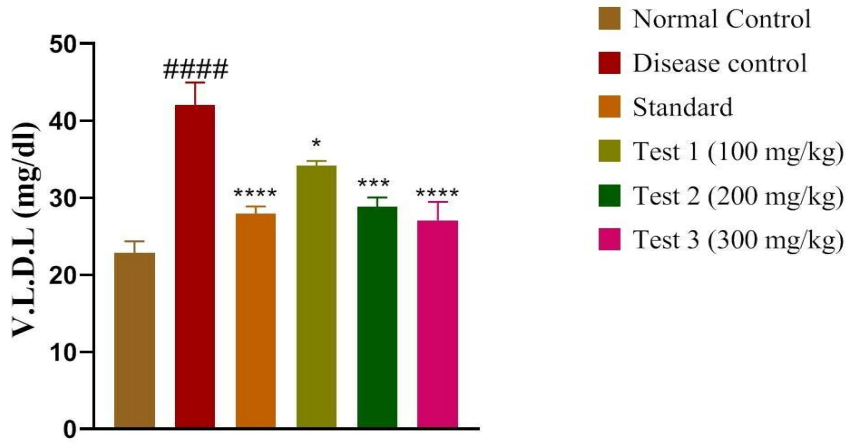

Figure 5 The effects of different doses (100,200 &300 mg/kg) of *Delonix regia* on VLDL cholesterol levels. Data are represented as means± SEM (n=6). Ordinary one way ANOVA followed by Dunnetts multiple comparison ****P<0.0001, *** P= 0.0005, ****P=0.0002 vs disease control.

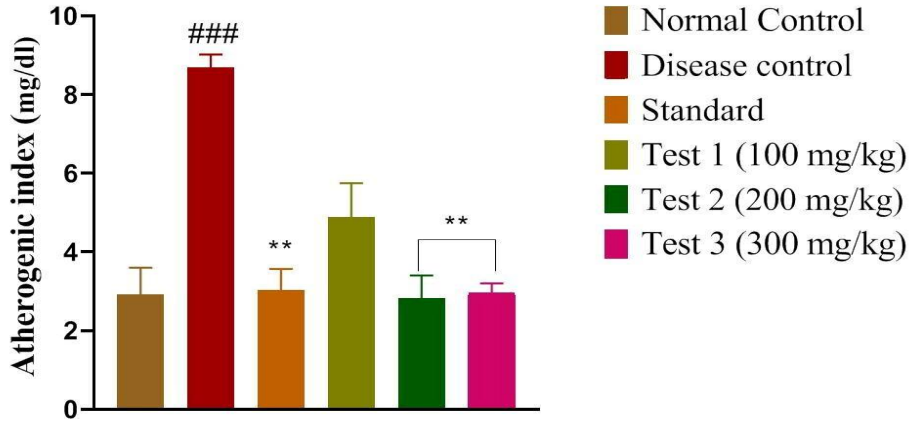

Figure 6 The effects of different doses (100,200 &300 mg/kg) of *Delonix regia* on atherogenic index. Data are represented as means± SEM (n=6). Ordinary one way ANOVA followed by Dunnetts multiple comparison **P<0.01, ** P= 0.01, **P=0.01 vs disease control.

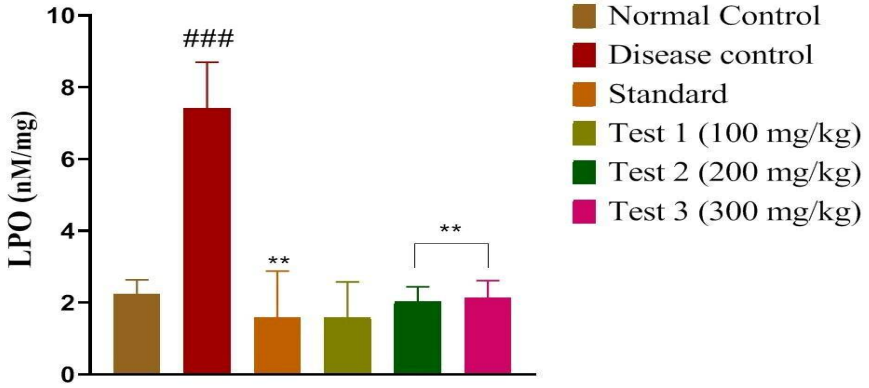

Figure 7 the effects of different doses (100, 200 &300 mg/kg) of *Delonix regia* on level of LPO. Data are represented as means± SEM (n=6). One-way ANOVA followed by Dunnett's multiple comparisons test.

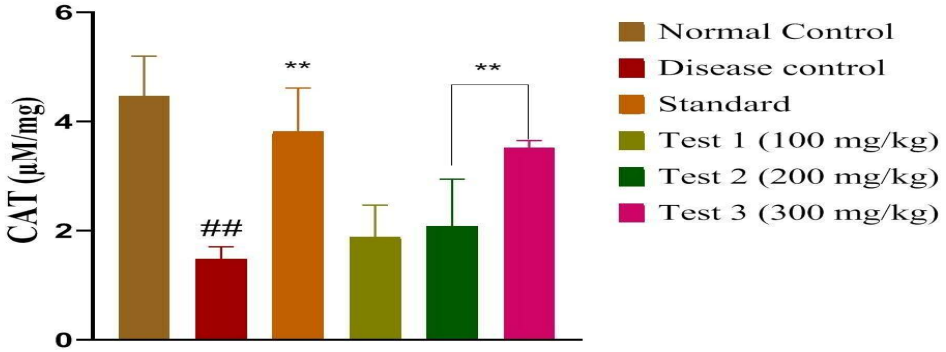

Figure 8 the effects of different doses (100, 200 &300 mg/kg) of *Delonix regia* on level of CAT. Data are represented as means± SEM (n=6). One-way ANOVA followed by Dunnett's multiple comparisons test.

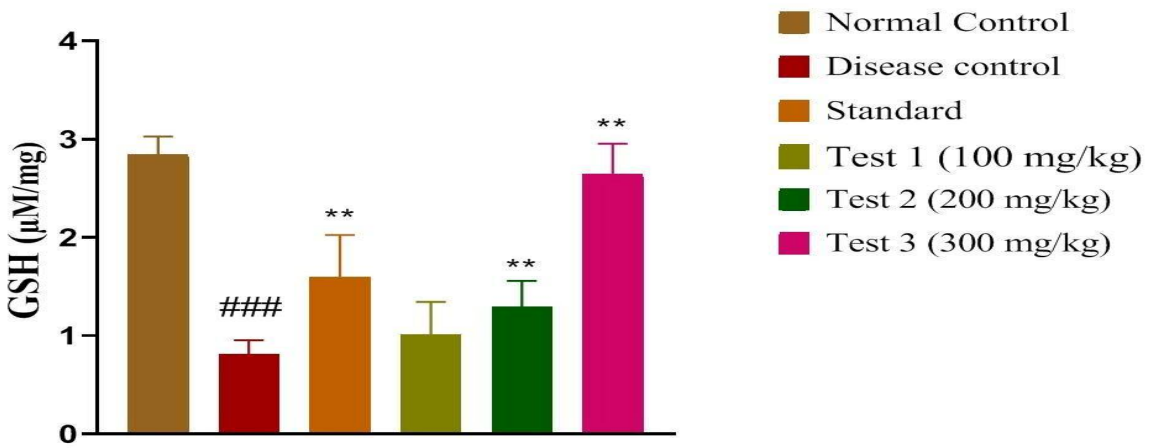

Figure 9 the effects of different doses (100, 200 &300 mg/kg) of *Delonix regia* on level of GSH. Data are represented as means± SEM (n=6). One-way ANOVA followed by Dunnett's multiple comparisons test.

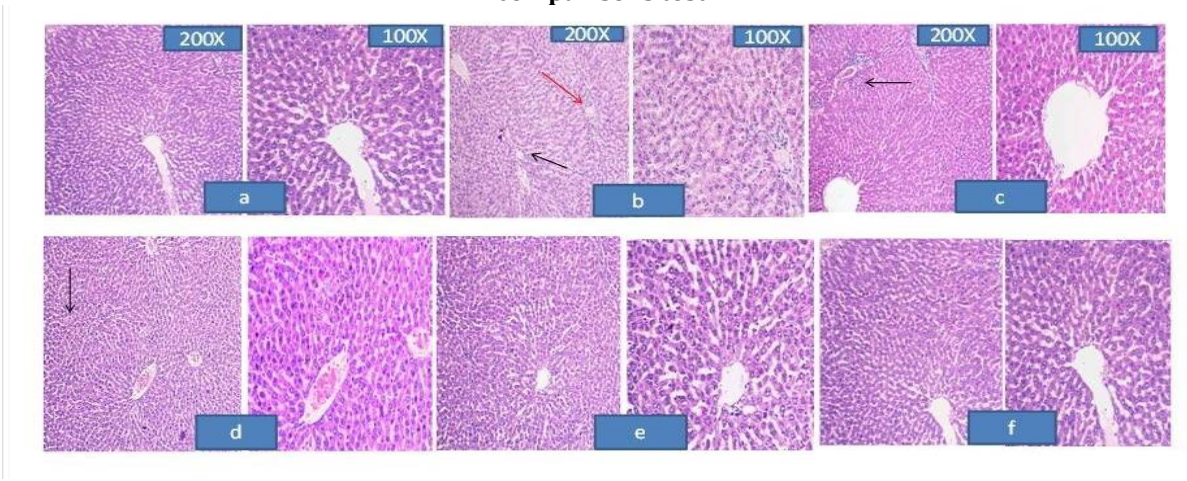

Figure1: Histopathological examination of rat liver.

Table 2: Effect of *Delonix regia* on Serum Total cholesterol levels (mg/dl).

Sr. No	Groups	Total cholesterol (mg/dl)
I	Normal	141.364±8.56
II	Disease control	234.21±5.39
III	Standard	161.10±2.88
IV	Test 100 mg/kg	215.33±10.51
V	Test 200mg/kg	157.22±7.80
VI	Test 300mg/kg	167.63±6.14

Table 3: Effect of *Delonix regia* on Serum Triglycerides (mg/dl).

Sr. No	Groups	Triglycerides(mg/dl)
I	Normal	130±6.43
II	Disease control	176.46±5.85
III	Standard	122±2.30
IV	Test 100mg/kg	155.80±3.29
V	Test 200mg/kg	131±3.97
VI	Test 300mg/kg	119.50±4.11

Table 4: Effect of *Delonix regia* on Serum HDL-cholesterol (mg/dl)

Sr. No	Groups	HDL-cholesterol (mg/dl)
I	Normal	36.04±1.22
II	Disease control	24.40±1.47
III	Standard	39.966±0.69
IV	Test 100mg/kg	36.60±1.86
V	Test 200mg/kg	41±4.04
VI	Test 300mg/kg	42.22±1.98

Table 5: Effect of *Delonix regia* on Serum LDL-cholesterol (mg/dl)

Sr. No	Groups	LDL-cholesterol(mg/dl)
I	Normal	80.65±3.89
II	Disease control	163±4.87
III	Standard	97.46±0.77
IV	Test 100mg/kg	154.40±3.57
V	Test 200mg/kg	127±5.28
VI	Test 400mg/kg	102.73±8.24

Table 6: Effect of *Delonix regia* on Serum VLDL-cholesterol (mg/dl)

Sr. No	Groups	VLDL-cholesterol(mg/dl)
I	Normal	22.87±1.48
II	Disease control	42.00±2.96
III	Standard	27.95±0.96
IV	Test 100mg/kg	34.13±0.65
V	Test 200mg/kg	28.86±1.17
VI	Test 400mg/kg	27.07±2.39

Table 7: Effect of *Delonix regia* on Atherogenic index

Sr. No	Groups	Atherogenic index
I	Normal	2.92±0.68
II	Disease control	8.69±0.63
III	Standard	3.03±0.54
IV	Test 100mg/kg	4.88±0.87
V	Test 200mg/kg	2.83±0.57
VI	Test 400mg/kg	2.97±0.23

Table 8: Effect of *Delonix regia* in liver biochemical parameters (CAT, GSH and LPOValues)

Sr. No.	Groups	PRO- OXIDANT LEVELS		
		Catalase (μM_2H_2OConsumed/ mg protein)	GSH (µM of GSH/mg protein)	LPO (nM of MDA/mg protein)
I	Normal	4.466±0.734	2.843±0.183	2.236±0.401
II	Disease control	1.487±0.219	0.813±0.140	7.417±1.28
III	Positive control	3.819±0.795	1.597±0.626	1.597±0.250
IV	Test 100mg/kg	1.89±0.58	1.012±0.73	2.89±0.98
V	Test 200mg/kg	2.086±0.86	1.296±0.26	2.032±0.413
VI	Test 300mg/kg	3.521±0.1	2.643±0.81	2.130±0.486

DISCUSSION

Since the beginning of human history, people have used plants for medicinal purposes. All countries have historically used herbs, but when it comes to the richest, oldest, and most diverse cultural practices around the use of medicinal plants, India leads the pack. There is rising recognition on a global scale of the significant contribution that medicinal plants make to human health. The World Health Organization (WHO) estimates that traditional medicine, which mostly involves plant extracts or active

substances, provides primary medical treatment to 80% of the world's population (6 billion people). Big pharmaceutical companies are currently doing extensive research on plant materials for potential medical use. Hyperlipidemia is a common and widespread health problem these days. Hyperlipidemia is the primary cause of atherosclerosis and conditions associated with it, such as ischemic cerebrovascular disease, peripheral vascular disease, and coronary heart disease (CHD). Examining the anti-hyperlipidaemic qualities of a few therapeutic plants and the phytoconstituents they contain is the aim of this review [15]. Even in the modern era, medicinal plants and herbal treatments are still frequently employed in therapy. The World Health Organization estimates that 80% of individuals in developing countries use these types of therapy. Although herbal medicine products are generally regarded as low risk, it is nevertheless vital to carefully assess any potential health risks. Products made from herbal medicine may be dangerous for a number of causes, including as adulteration, environmental contamination with heavy metals or pesticides, potentially toxic plant components or metabolites, or microbial (toxic fungal) contamination. Accurate assessment is necessary for the patient's safety. Both in vitro and in vivo procedures are used in the assessment of the toxicity of herbal medicine; however, in addition to the classic ways, several new approaches have been created in recent decades. Even though adulteration and pollution can be detected using DNA sequencing, omics has become a valuable research tool for toxicity assessment and prediction. Invertebrate models, like Galleria mellonella or Danio renio, became more popular because employing vertebrate models posed ethical concerns. The purpose of this article is to provide a general overview of the methods and strategies being used to investigate the possible negative effects of herbal medicines as well as the challenges this field of research faces [16]. MetS therapy modalities include pharmacologic and non-pharmacologic interventions, with varying degrees of effectiveness. For those with borderline values and those who cannot take medication, the second strategy is the most economical preventive measure. The first technique is advised for those with a high risk of cardiovascular issues. Nutraceuticals, mainly phytochemicals produced from plants, can be used as non-pharmacological treatments for Metabolic Syndrome (MetS) when combined with lifestyle modifications. This chapter will review the research that has been done on soluble fibers that come from psyllium and other sources, as well as phytochemicals like cinnamaldehyde and cinnamic acid that are present in cinnamon. Berberine, charantin from bitter gourd, corosolic acid from banaba, and flavonols and catechins from chocolate and green tea will also be covered. Vegetable omega-3 polyunsaturated fatty acids, soy peptides, garlic-derived alliin, and curcumin from curcuma longa are the ingredients listed [17]. Flavonoids are a broad class of naturally occurring phenolic chemicals that can be found in many different plant-based foods and beverages, including fruits, vegetables, cereals, bark, roots, stems, flowers, tea, and wine. These natural products are well known for their beneficial effects on health, and efforts are currently underway to separate out the flavonoid components. Nowadays, it is acknowledged that flavonoids are crucial for a variety of pharmacological, therapeutic, cosmetic, and nutraceutical applications. These characteristics have to do with their capacity to stop oxidation, lessen inflammation, stop mutations, and stop the growth of cancer. They can also control the activity of significant biological enzymes. The discovery of the low risk of cardiovascular death and the ability to prevent coronary heart disease has greatly advanced the field of flavonoid research. The exact ways in which flavonoids work are still not entirely understood. It has been widely acknowledged for millennia that plant derivatives possess a diverse array of biological activity [18]. The plant often known as the royal Poinciana is *Delonix regia* a member of the Fabaceae subfamily and family Leguminosae. Flamboyant, another name for the May flower plant, is a deciduous tree with many branches and a broad, spreading, flat crown. It is well known for its vivid red-orange flowers, which cover the tree entirely in May and June. Planting the *Delonix regia* in a spot that receives direct sunshine will result in best flowering and growth. According to the literature review, the bark of *Delonix regia* includes carotene, hydrocarbons, flavonoids, alkaloids, saponins, phytotoxins, and β-sitosterol. Carotenoids, tannins, saponins, flavonoids, steroids, alkaloids, and β-sitosterol are all present in *Delonix regia* flowers. Galactomannon and saponins make up the seeds. The leaves of the plant are rich in β sitosterol and lupeol. To yet, the overall amount of flavonoids and phenolic chemicals has not been quantified [19]. Dietary modifications influence normal metabolic function and provide significant risks for cardiovascular disease and oxidative stress. The goal of the current study was to determine whether extracts of *Delonix regia* may help wistar rats fed an experimental high-fat, high-cholesterol diet, as well as any potential mechanisms of action. We created an experimental design to look into how *Delonix regia* affects conditions similar to hyperlipidemia in test animals based on the available data. The effects of the formulation of *Delonix regia* on circumstances resembling hyperlipidemia caused by a high-cholesterol diet are evaluated in this study using animal models. The study's goal is to organize multiple stages, such as the choice and acquisition of medications and animals, as well as the evaluation of biochemical parameters using different assays,

including atherogenic index, total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides. Here, we noticed and assessed changes in biochemical indicators, such as the test groups considerably lower dose-dependent triglyceride level. The test drug's impact on HDL cholesterol was considerably amplified by *Delonix regia* . On the other hand, the test medication administration resulted in a considerable and abrupt reduction in the levels of total cholesterol, VLDL cholesterol, LDL cholesterol, and atherogenic index. In order to confirm this, we investigated the effects of lipid peroxidation (LPO), catalase (CAT), and glutathione levels (GSH) in *Delonix regia* . The findings indicated that long- term administration of the test medicine dramatically decreased LPO and raised GSH and Catalase levels.

CONCLUSION

The current investigation found that *Delonix regia* has an antihyperlipidemic effect. The antihyperlipidemic impact of a diet high in cholesterol was lessened by delonix regimen. The primary goal of the current study was to critically assess the test drug's effectiveness in treating experimental animals' hyperlipidemia, which was brought on by a high-cholesterol diet model. Following the creation of hyperlipidemia in rats through the use of a high-cholesterol diet, various biochemical parameters were measured in the animals, including total cholesterol, LDL cholesterol, HDL cholesterol, VLDL cholesterol, and the atherogenic index. antioxidants in addition to evaluation of the histopathology. Based on the findings of the behavioral investigation, oxidative parameters, and histological examination, *Delonix regia* (300 mg/kg p.o.) was found to have more notable effects in the animal model of hyperlipidemia brought on by a diet high in cholesterol. Further research is required to determine the mechanism and evolving factors responsible for the test drug's antihyperlipidemic activity in experimental animals.

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