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## Lipid lowering activity of the hydro-alcoholic extract of *Tecoma stans* L. Flowers in hyperlipidemic models of wistar albino rats

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### ABSTRACT

Lipid lowering effect of the hydro-alcoholic extract of the flower of *Tecoma stans* L. was evaluated in triton and diet induced hyperlipidemic models of wistar albino rats. The extract at 125 and 250mg/kg dose levels inhibited the elevation in serum cholesterol and triglyceride levels on Triton WR 1339 administration rats. The extract at the same dose level significantly attenuated the elevated serum total cholesterol and triglycerides with an increase in high-density lipoprotein cholesterol in high-fat diet-induced hyperlipidemic rats. The standard dose atorvastatin in the former and gemfibrozil in the later studies showed slightly better effects. The outcome of the study reveals the lipid lowering activity of hydro-alcoholic extract of *Tecoma stans* L. flowers in dyslipidaemic conditions by interfering with the biosynthesis of cholesterol and utilization of lipids.

**Keywords:** *Tecoma stans*, Antihyperlipidemic activity, High fat diet, Triton WR 1339, HDL-C, Triglycerides.

### INTRODUCTION

Hyperlipidemia (elevated levels of triglycerides or cholesterol) and reduced high- density lipoproteins (HDL-C) occur as a consequence of several interrelated factors that may be lifestyle, genetic, metabolic or other conditions that influence plasma lipoprotein metabolism (1). Elevated serum concentrations of total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C) appear to increase the risk of individual in developing coronary heart disease (CHD) (2). Lipid lowering therapy is indicated in primary and secondary prevention of cardiovascular diseases in addition to the management of all other risk factors including smoking, diabetes and obesity (3). The current antihyperlipidemic therapy includes principally statins and fibrates. The former corrects the altered blood lipid profile by inhibiting the biosynthesis of cholesterol and later acts by enhancing the clearance of triglyceride rich lipoproteins (1). The investigation of lipid lowering activity on nutraceutical will be useful strategy in the discovery of new lead molecules eliciting improved activity by regulating through different mechanism of action. The plant extracts maintaining the lipid metabolism and thus can be used in treating hyperlipidemia of varied etiology.

*Tecoma stans* (L.) from Bignoniaceae family is a semi-evergreen ornamental tropical shrub or small tree originally from Latin American which has been cultivated in Iran (particularly in west and southwest parts) recently. Also found in different parts of India. Its primary applications have been in treating diabetes and digestive problem. Extracts from *T. stans* leaves have been found to inhibit the growth of the yeast infection. Flower infusion can be taken orally for diabetes and stomach pains. A strong flower and root decoction is taken orally as a diuretic, to treat syphilis or for intestinal worms. The root is considered in the Satara District an effective remedy for snake and rats bites and for scorpion sting. The roots are used as a powerful diuretic, vermifuge and tonic. Flower and leaves have some medicinal value for the treatment of various cancer (5). Its leaves are used traditionally in Mexico in order to control diabetes (6, 7). The plant contains tecomonine, tannins, flavonoids, alkaloids, quinones and traces of

saponins (8). The leaves contain flavonoid, alkaloids such as tecomine and tecostidine. According to recent studies a new phenylethanoid, and a novel monoterpene alkaloid, along with eleven known compounds were isolated from the fruits and luteolin 7-O-beta-D-glucuronopyranoside, diosmetin 7-O-beta-D-glucuronopyranoside, diosmetin 7-O-beta-D-glucopyranoside, diosmetin 7-O-beta-D-glucuronopyranoside methyl ester and acetoside were isolated from the flowers (9). The tecostanine isolated from the leaves is suggested for antihyperglycemic effect (10). *T. stans* growing in Egypt has two alkaloids called tecomine-1 and tecostanine-2 with hypoglycemic effect in fasting rabbits which are inactive in the absence of pancreas (11, 12). Tecoma is not a toxic because this plant is used in Latin America as a remedy for diabetes and moreover for feeding cattle and goats in Mexico (13). The aim of the present study, therefore, was to investigate the hypolipidemic effect of hydro-alcoholic *Tecoma stans* (L.) flower extract (TFE) on albino rats.

## MATERIALS AND METHODS

### Plant material

*T. Stans* flowers were collected from the local area of Salipur, Cuttack, Orissa, during January-February and authenticated by Dr. P. Jayaraman, Director Plant Anatomy Research Center (PARC), Tambaram, Chennai, India, and a voucher specimen holding No. PARC/2007/83 was deposited in the same center. The air dried flowers were extracted with 50% ethanol by cold maceration. Dry extract was obtained by vacuum distillation and subsequent vacuum drying. The yield of the hydro alcoholic extract was 22% w/w and the extract was stored between 2°-8°C for further studies. Phytochemical analysis revealed the presence of alkaloid, flavonoids, tannins, terpenes and saponins. The total phenolic content of the extract was determined to be 1.2% as per Folin-Ciocalteu method (14).

### Chemicals and reagents

Triton WR 1339 (Sigma USA) and Folin-Ciocalteu reagent (Sd. fine) were from commercial sources. Serum Cholesterol, triglycerides and high-density lipoprotein cholesterol (HDL-C) were determined using kits of Qualigens fine chemicals. Atrovastatin and gemfibrozil were obtained from Dr. Reddy's Lab, Hyderabad and Sun pharmaceuticals, India respectively.

### Animals

Male wistar strain albino rats (150-250g) were obtained from Central Animal House of Institute of Pharmacy and Technology Salipur, Cuttack, Orissa India. The animals were housed under standardized environmental conditions (at normal room temp, with a 12 hour light and dark cycle) and fed with standard pellet chow feed and water *ad libitum*. The animal protocol was approved by Institutional Animal Ethical Committee of Institute of Pharmacy and Technology, Salipur, Cuttack, Orissa, India with registration number 1053/ac/07/CPCSEA. All the experiments were performed as per the CPCSEA guidelines.

### Triton induced hyperlipidemic study (15)

The use of Triton WR 1339 induced hyperlipidemia through accelerated hepatic cholesterol synthesis was suggested as an important approach to screen the action of hypolipidemic drugs (16). Male wistar rats weighing 200-250g were divided into 4 groups of 6 animals each. Group 1 (Vehicle control) received 0.3% w/v carboxy methyl cellulose (CMC) orally for one week. Group 2 and 3 were treated with the extract of *T. Stans* at the dose 125 and 250mg/kg (p.o) respectively. The Group 4 received atrovastatin 1mg/kg body weight once daily for one week. On seventh day, 200mg/kg Triton WR 1339 (isooctyl polyoxyethylene phenol) was injected (i.p.), to all the four groups of rats immediately after drug administration. Serum total cholesterol and triglycerides were estimated for individual animals in autoanalyser (Microlab 100) on seventh day previous to drug treatment and after 24 hr of Triton administration. Blood was withdrawn from retro-orbital sinus using glass capillary in EDTA coated tubes and serum was separated in cooling centrifuge (Remi, C24) by centrifuging at 2500 rpm for 10 min. The observations made were recorded in Table-2.

### High-fat diet-induced hyperlipidaemic study (17)

Hyperlipidemia was induced in male wistar rats weighing 150-180g by feeding them with a high fat diet, (Table 1) for 4 weeks. High-fat diet increased the serum cholesterol and triglycerides to about 75-80% of the normal levels and reduced the HDL-C levels significantly (Table-3). The rats with significantly higher values of serum cholesterol and triglyceride values compared to that of normal animals were considered to be hyperlipidaemic and six hyperlipidaemic animals were grouped for one treatment. Group 1 received 0.3% w/v CMC and served as vehicle control, while group 2 and group 3 hyperlipidemic rats were orally treated with the extract of *T. Stans* at the dose 125 and 250mg/kg (b/w) respectively, once a day for one week. Animals of fourth group of hyperlipidaemic animals were administered the standard drug Gemfibrozil 50mg/kg body weight for one week. All the four groups were kept on the same high fat diet throughout drug treatment. Serum total cholesterol, triglyceride and HDL-C of the non-

fasted animals were estimated on seventh day after 1hr of dosing. Atherogenic index was calculated using the formula:

$$\text{Atherogenic Index} = (\text{TC} - \text{HDL})/\text{HDL}$$

### Statistical analysis

Data are represented as mean  $\pm$ SEM (Standard error of mean). The group means were compared for significant difference ( $p < 0.01$ ) by Student's t test in triton model and paired t test in diet model.

## RESULTS AND DISCUSSION

The systemic administration of the surfactant Triton to rats resulted in an enormous elevation of serum cholesterol and triglycerides at 24 hour (Table 2). The extract of *T. Stans* inhibited highly significant elevation in cholesterol by 19.28 and 31.98% at 125 and 250 mg/kg (b/w) dose levels, respectively as compared to that of untreated vehicle control group. Triglyceride level was lowered in *T. Stans* treated rats by 17.06 and 41.46 % at 125 and 250 mg/kg (b/w) doses, respectively in comparison to that vehicle control rats (Table 2). Atrovastatin, the lipid controlling mechanism of which is inhibition of synthesis of cholesterol in the liver, was employed as the standard drug in Triton induced model. The treatment with atrovastatin resulted in a slightly better effect than *T. Stans*. These results indicate that the extract of *T. Stans* may interfere with cholesterol biosynthesis as Triton accelerates the hepatic synthesis of cholesterol (18).

Triton induced hypercholesterolaemia, though simple and rapid for evaluating hyperlipidemic compounds, is rather artificial. Hence the lipid controlling potential of *T. Stans* flower was further validated in diet-induced hyperlipidemic rat model. When male wistar albino rats were kept on high-fat diet supplemented with 1% cholesterol for 4 weeks, there was elevated serum cholesterol levels and triglyceride levels were almost doubled whereas, HDL-C levels were reduced significantly as indicated by low value of atherogenic index (Table 3). Elevated circulating lipid levels may be the outcome of inhibitory effect of high dietary fat intake on lipogenesis (19). The treatment of hyperlipidemic rats with *T. Stans* for one week brought down the elevated serum total cholesterol and triglycerides improving the HDL-C levels as shown by reduced atherogenic index (Table 3). Similar to gemfibrozil (50mg/kg) the standard fibrate drug used, the extract may have enhanced the breakdown of lipids, thus modifying the altered lipid metabolism induced by high fat-diet. Increase in HDL levels and reduction in LDL shows the intensive conversion of LDL to HDL and clearance of circulating lipids. Total cholesterol- HDL-C/HDL-C ratio of  $> 4.5$  is associated with increased coronary heart disease (CHD) risk and the ideal ratio is  $\leq 3.5$  (20). A significant reduction in the atherogenic index by *T. Stans* flower extract treatment demonstrates the protective efficacy of the extract against atherogenesis. Consequently the lipid regulating efficacy of the hydro-alcoholic extract of *T. Stans* flower would be beneficial in the prevention of plaque formation leading to atherosclerosis and CHD accelerated by high fat diets.

The lipid lowering activity of *T. Stans* flower extract may be attributed to the phytoconstituents present, such as  $\beta$ -carotenes, tannins, saponin & polyphenols ingredient present in it as reported for other plant extracts (21, 22, 23). Saponin derived from *Medicago sativa* were reported to reduce blood cholesterol by competing with cholesterol at binding sites or interfering with cholesterol biosynthesis in the liver (23). Phenolic active principle present in *Anethum graveolens* were observed to be responsible for lowering TC and LDL-C and elevating HDL-C in hypercholesterolaemic rats (24).

The findings of the study reveals that hydro-alcoholic extract of *Tecoma stans* L. flowers can effectively control the blood lipid levels in dyslipidaemic conditions by interfering with the biosynthesis of cholesterol and utilization of lipids.

**Table 1- Constituents of high fat diet**

| Ingredients  | Quantity(g/100g) |
|--------------|------------------|
| Corn flour   | 25               |
| Milk powder  | 15               |
| Sucrose      | 15               |
| Casein       | 5                |
| Egg yolk     | 3                |
| Lard         | 35               |
| Salt mixture | 1                |
| Cholesterol  | 1                |

Table 2: Effects of the hydro-alcoholic extract of *T. Stans* flower on lipid profile in triton induced study.

| Group | Total cholesterol(mg/dl) |            | Triglycerides(mg/dl) |                |
|-------|--------------------------|------------|----------------------|----------------|
|       | 0 hr                     | 24 hr      | 0hr                  | 24 hr          |
| 1     | 47.0±3.2                 | 182.3±4.2  | 73.32 ± 5.57         | 356.2 ± 24.75  |
| 2     | 52.5±2.7                 | 158.4±3.4* | 68.30±2.4            | 295.4 ± 28.66* |
| 3     | 45.3±2.9                 | 124.0±2.6* | 72.2±3.1             | 208.5 ± 26.34* |
| 4     | 48.7±1.3                 | 118±4.3*   | 66.5±7.9             | 196.3±21.42*   |

Values are mean±SE of 6 rats in each group  
\**P*<0.001 compared with vehicle (untreated) control

Table 3: Effects of the hydro-alcoholic extract of *T. Stans* flower on lipid profile of hyperlipidemic wistar rats in diet-induced hyperlipidemia.

| Group | Total cholesterol(mg/dl) |                                |                     | Triglycerides(mg/dl) |                                |                     | Atherogenic index<br>(total cholesterol- HDL/HDL) |                                |                     |
|-------|--------------------------|--------------------------------|---------------------|----------------------|--------------------------------|---------------------|---|--------------------------------|---------------------|
|       | Normal value             | On induction of hyperlipidemia |                     | Normal value         | On induction of hyperlipidemia |                     | Normal value                                      | On induction of hyperlipidemia |                     |
|       |                          | 0 <sup>th</sup> day            | 7 <sup>th</sup> day |                      | 0 <sup>th</sup> day            | 7 <sup>th</sup> day |   | 0 <sup>th</sup> day            | 7 <sup>th</sup> day |
| 1     | 51.6±2.6                 | 72.1±3.1                       | 78.6±2.9*           | 69.8±2.9             | 132.6±2.1                      | 173.6±4.3*          | 3.65±0.23   | 3.87±0.12                      | 5.28±0.27*          |
| 2     | 49.8±2.3                 | 75.6±1.8                       | 63.4±4.3*           | 71.7±2.3             | 134.8±2.6                      | 111.3±3.1*          | 3.37±0.20   | 4.13±0.08                      | 3.02±0.07*          |
| 3     | 49.3±2.1                 | 76.3±1.5                       | 55.2±1.2*           | 68.8±3.7             | 136.2±2.3                      | 95.32±2.1*          | 3.17±0.32   | 4.12±0.17                      | 2.46±0.08*          |
| 4     | 48.3±2.7                 | 75.2±2.6                       | 48.6±1.8*           | 66.3±3.1             | 135.4±2.7                      | 76.8±2.7*           | 3.23±0.26   | 4.36±0.17                      | 2.75±0.28*          |

Values are mean ±SE of 6 rats in each group  
\*- Represent values significantly different in paired t test as compared to 0 day values (*P*<0.01)

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