# Hypolipidemic efficacy of Trigonella Foenum seeds in comparison with Rosuvastatin and Fenofibrate in hyperlipidemic rats

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## Abstract

Background: Trigonella foenum-graecum seeds (TGS) have been historically used for the treatment of various chronic human diseases and studies concerned with application of this plant in diabetes and dyslipidemia support this hypothesis. The present study was designed to evaluate and compare the effect of different doses of TGS powder with Rosuvastatin and Fenofibrate on lipid profile, liver function enzymes, body weight and malondialdehyde (MDA) in hyperlipidemic rats.

Method: Forty two rats were divided into two groups. The first group included 12 rats and received a standard diet throughout the experimental period and were subdivided into two subgroups of 6 rats each. The first subgroup served as a control group. The second subgroup received a standard diet containing TGS Powder at a concentration of 0.75% (w/w). The second group included 30 induced hyperlipidemic rats by feeding them with high cholesterol diet. They were subdivided into five subgroups each of 6 rats. First subgroup served as a positive control. The second subgroup received atherogenic diet containing TFSP at a concentration of 0.50% w/w, the third subgroup received the same diet containing TGS at a higher concentration of 0.75% w/w. The fourth and fifth subgroups received a daily dose of Rosuvastatin and Fenofibrate respectively. At the end of the treatment period (six weeks) all of these groups were subjected to various biochemical analyses of blood.

Results: After six weeks of therapy, TGS of both concentrations (0.50% and 0.75% w/w) significantly reduced serum low density lipoprotein cholesterol (LDL-C), total cholesterol (TC) when compared with hyperlipidemic rats. Both concentrations of TFS (0.50%, 0.75% w/w) increased serum high density lipoprotein cholesterol (HDL-C) significantly for both normal and hyperlipidemic rats. Daily administration of Rosuvastatin of (10mg/kg) for six weeks reduced serum TC, LDL-C and triglycerides (TG) level significantly when compared with hyperlipidemic rats. Administration of Fenofibrate for six weeks markedly and significantly reduced serum TG when compared with hyperlipidemic animals. Daily use of TFS (0.75% w/w) for six weeks increased both serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) significantly for both normal and hyperlipidemic rats.

Daily administration of TGS of both concentrations (0.50% and 0.75% w/w) significantly decreased serum MDA level of hyperlipidemic rats. There was a significant increase in body weight of normal rats taking diets containing TGS for six weeks.

Conclusion: TGS has similar efficacy of Rosuvastatin and Fenofibrate in reducing TC. whereas, TGS was non-significantly more effective than Rosuvastatin and Fenofibrate in changing serum HDL-C and LDL-C.

Key words: Hyperlipidemia, Trigonella Foenum, Rosuvastatin, Fenofibrate

## Introduction

Medicinal plant products have been highlighted as an alternative to current management of dyslipidemia to some extent (1). There are many types of medicinal herbals which are useful in treatment of hyperlipidemia such as Avena Sativa Which has been shown to decrease serum cholesterol (2). The lipid-lowering effects of Garlic (Allium sativum) may occur via inhibition of HMG-CoA reductase or other enzymes, possibly by diallyl di- and trisulphide components of garlic (3). The decoction of the (Coriandrum sativum) is effective in lowering blood lipid levels (4).

Trigonella foenum-graecum (TGSP), an annual medicinal plant of the Fabaceae family is extensively cultivated in most regions of the world for its medicinal value (5). TGSP seeds have been historically used for the treatment of various chronic human diseases and studies concerned with application of TGSP seeds in diabetes and dyslipidemia support this hypothesis (6, 7).

TGSP is full of 4-hydroxyisoleucine, which directly induces insulin secretion from pancreatic b cells (8). It's reported to have restorative and nutritive properties and to stimulate digestive processes, useful in healing of different ulcers in the digestive tract (9). TGSP has also been reported to exhibit pharmacological properties such as antitumor, anti-inflammatory, hypotensive and antioxidant (10, 11).

TGSP has been used to treat peptic ulcers and inflamed conditions of the stomach and bowel; it absorbs toxic material and eliminate it. The healing and soothing action creates a protective coating, like a lubricant, over inflamed areas (12).

TGSP seed is widely used as a milk producing agent by nursing mothers to increase inadequate breast milk supply (13). Flavonoids of TGS extract have been observed to possess anti-oxidant activity (14).

The present study was designed to evaluate and compare the effectiveness of different doses of Fenugreek seeds, Rosuvastatin at 10mg/kg and Fenofibrate at 30mg/kg on the lipid profile, liver function tests, malondialdehyde level and weight in hyperlipidemic rats after six weeks of administration.

## Materials and Methods

**Animals:**
Adult female rats weighing between 100-250 g were used throughout the study. The rats were obtained from Mosul and Abu ghreb city. All animals were kept in the animal house at the College of Medicine under controlled conditions of 12 hours light and 12 hours dark cycles in a room temperature of 25 ºC. The rats were allowed to acclimatize to these conditions for one week.

**Experimental design:**
Forty two rats were divided into two groups. The first group included 12 rats which received standard diet throughout the experimental period. They were subdivided subsequently into two subgroups each of 6 rats. The first subgroup served as a control group. The second subgroup received a standard diet containing (TFSP) at a concentration of 0.75% (w/w).

The second group included 30 hyperlipidemic rats. Hyperlipidemia was induced by feeding the rats with high cholesterol diet; they received atherogenic diet (79% standard diet and 21% butter fat) for six weeks (15). The hyperlipidemic rats were subsequently subdivided into five subgroups of 6 rats. The first subgroup, served as a positive control (hyperlipidemic rats). The second subgroup, received atherogenic diet containing TGS Powder at a concentration of 0.50% (w/w) every day. The third subgroup, received atherogenic diet containing TGS powder at a concentration of 0.75% (w/w) every day. The fourth and fifth subgroups received a daily dose of Rosuvastatin (10mg/kg) and Fenofibrate (30mg/kg) respectively.

The solutions of two drugs Rosuvastatin (10mg/kg), and Fenofibrate (30mg/kg) were freshly prepared in normal saline and given to animals by oral gavage every day. (16, 17)

At the end of the treatment period (six weeks), the animals were subjected to various biochemical analysis of blood. They were fasted overnight and the following day blood samples were taken. The procedure started by anaesthetizing the rats by giving them a combination of ketamine in a dose of 35 mg/kg with xylazine in a dose of 5 mg/kg (18) which was followed by a cardiac puncture by a sterile disposable plastic syringe which was then put into a specified numerically labeled blood tube.

Blood samples were collected from rats for determination of serum lipid and lipoprotein profile (T.Ch, TGs, HDL-C and LDL-C), serum malondialdehyde (MDA) and liver function tests (serum alanine aminotransferase S.ALT, serum aspartate aminotransferase S.AST and serum alkaline phosphatase ALP).

During the experimental period, body weight was individually recorded for each rat before and after treatment.

**Statistical analysis:**
All data are expressed as means ± standard error of means (M ± SEM) and Statistical analysis was carried out using statistically available software (SPSS Version 19). Data analysis was made using one-way analysis of variables (ANOVA). Comparisons between groups were done using Duncan test and unpaired student t-test. P ? 0.05 was considered as statistically significant.

## Results

**Effects of Trigonella foenum graecum on lipid profile:**
Daily administration of TGS at a concentration of (0.75% w/w) had no significant effect on serum levels of total cholesterol, TG, LDL-C of normal rats (Table 1). The same dose of TGS has produced a statistically significant rise in serum HDL-C as seen in Table 1.

**Table 1: Effects of TGS 0.75% (w/w) on the lipid profile of normal rats (n=12).**

| Parameters | Control group | TGS |
|---|---|---|
| S. Total Cholesterol mg/100ml | 58.31 ± 9.703 | 66.83 ± 4.46 |
| S. Triglyceride mg/100ml | 57.25 ± 17.45 | 58.83 ± 8.33 |
| S.HDL mg/100ml | 35.33 ± 6 | 47.16 ± 0.79 * |
| S.LDL mg/100ml | 6.51 ± 1.71 | 7.16 ± 0.60 |

* P< 0.05

**Effects of Trigonella foenum graecum on lipid profiles of hyperlipidemic rats:**
There was a marked increase in the level of serum triglyceride, serum cholesterol and serum low density lipoprotein in the rats treated with atherogenic diet compared to the control group indicating the induction of hyperlipidemia as shown in Table 2.

**Table 2: Effects of different doses of TGS Rosuvastatin and Fenofibrate on lipid profile of hyperlipidemic rats (n=36)**

| Parameters | Control group | Hyperlipidemic group | TGS 0.50% | TGS 0.75% | Rosuvastatin 10 mg/kg | Fenofibrate 30 mg/kg |
|---|---|---|---|---|---|---|
| S. Cholesterol mg/100ml | 58.31±9.70 a | 104.33±23.91 b | 63.33±3.26 a | 57.33 ±4.17 a | 51.33 ± 7.85 a | 79 ± 5.23 ab |
| S. TG mg/100ml | 57.25±17.45 a | 174.83 ±53.8 b | 150.33±11.99 b | 148.50±9.76 b | 40.33 ±3.95 a | 42.83 ±3.97 a |
| S. HDL mg/100ml | 35.33 ± 6 ab | 32.66 ± 5.77 a | 44 ± 2.39 b | 44.66 ±3.16 b | 35.50 ± 3.64 ab | 37 ± 2.59 ab |
| S. LDL mg/100ml | 6.51 ± 1.71 a | 29 ± 11.51 b | 9.43 ± 1.08 a | 6 ± 0.47 a | 17.60 ± 3.60 a | 30.83 ±3.42 b |

The same letters mean that there is no significant difference
The different letters mean there is a significant difference at P < 0.05

There was a statistically significant reduction in both serum LDL and TC by both concentrations of TGS (0.50% and 0.75% w/w). While the same concentrations of TGS reduced serum TG when compared with hyperlipidemic rats, but the result was not statistically significant, Table 2.

Both concentrations of TGS (0.50%, 0.75% (w/w)) increased serum HDL-C significantly, when compared with hyperlipidemic rats.

Daily administration of Rosuvastatin of (10mg/kg) for six weeks reduced serum TC, LDL-C and TG level significantly when compared with hyperlipidemic rats (Table 2).

Administration of Fenofibrate at a dose (30 mg/kg) for six weeks significantly reduced serum TG when compared with hyperlipidemic animals. However the same dose had no significant effects on serum LDL-C and TC level in hyperlipidemic rats (Table 2).

Effects of TGS (0.75% w/w) on liver enzymes of normal rats (n= 12):
Daily use of TGS (0.75% w/w) for six weeks increased both serum AST and ALT significantly when compared with control group as seen in table 3. While the same dose of TGS (0.75% w/w) had no significant effect on serum ALP when compared with control group (Table 3).

**Table 3: Effects of TGS (0.75% w/w) on liver enzymes of normal rats (n= 12).**

| Parameter | Control group | TGS 0.75% | Statistical evaluation |
|---|---|---|---|
| S. AST | 106 ± 3.66 | 134.83 ± 7.58* | 0.007 |
| S. ALT | 37.66 ± 1.17 | 44.16 ± 2.18* | 0.025 |
| S. ALP | 25.26 ± 2.62 | 21.5 ± 1.54 | 0.244 |

* compared to the control.

**Effects of different doses of TGS (0.50% and 0.75% (w/w)), Rosuvastatin and Fenofibrate on liver enzymes of hyperlipidemic rats (n=36):**
Table 4 shows that the serum levels of AST, ALT and ALP did not change significantly in rats treated with atherogenic diet when compared with control group. As shown in Table 4 no significant change in liver enzymes (S. AST, S. ALT and S. ALP) was also observed in hyperlipidemic rats treated with TGS (0.50% w/w), while high concentration of TGS significantly affected both serum AST and ALP of hyperlipidemic animals.
Both Rosuvastatin (10mg/kg) and Fenofibrate (30mg/kg) significantly increased the serum level of ALP (alkaline phosphatase) of hyperlipidemic rats.

**Table 4: Effects of different concentration of TGS (0.50%, 0.75%w/w), Rosuvastatin and Fenofibrate on liver enzymes of hyperlipidemic rats (n=36)**

| Parameter | Control group | Hyperlipidemic group | TGS 0.5% | TGS 0.75% | Rosuvastatin (10mg/kg) | Fenofibrate (30mg/kg) |
|---|---|---|---|---|---|---|
| AST | 106± 3.66 ab | 94 ± 2.55 a | 107.5±5.69 ab | 126.33±20.46 b | 114.50±4.07 ab | 114.8±3.94 ab |
| ALT | 37.66±1.17 a | 41.66±2.13 a | 41 ± 3.10 a | 43.33±1.20 ab | 45.16±4.36 ab | 51.50±3.37 b |
| ALP | 25.26±2.62 ab | 34.50±2.68 b | 23.4±3.37 ab | 19.93 ± 1.33 a | 38.5±5.55 c | 41.66±6.76 c |

**Effects of TGS on MDA of normal rats (n=12).**
No significant changes in the serum of malondialdehyde were observed between the normal rats taking TGS (0.75% w/w) and control group as shown in Table 5.

**Table 5: Effects of TGSP on MDA in normal rats (n=12)**

| Groups | Serum MDA µmol/L |
|---|---|
| Control | 3.35 ± 0.16 |
| TGS 0 (0.75% w/w) | 3.36 ± 0.22 |

**Effects of TGS on MDA of hyperlipidemic rats (n=24):**
Serum MDA level of rats fed with atherogenic diets increased significantly when compared with control group as shown in (Table 6).

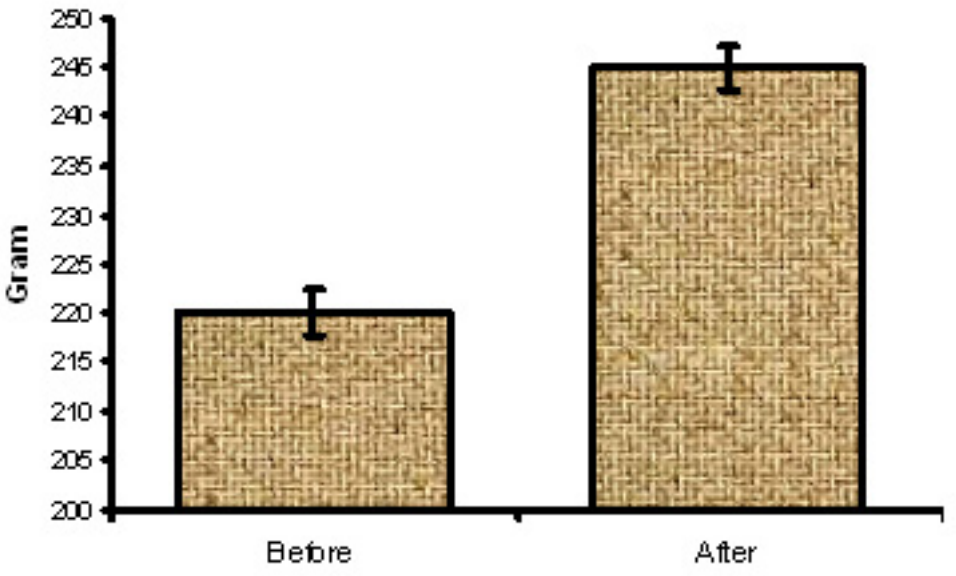
Daily administration of TGS of both concentrations (0.50% and 0.75% w/w) significantly decreased serum MDA level of hyperlipidemic rats.

**Table 6. Effects of TGSP on MDA of hyperlipidemic rats (n=24)**

| Groups | Serum MDA µmol/L |
|---|---|
| Control | 3.35 ± 0.16 |
| Hyperlipidemia | 6.39 ± 0.87 * |
| TGS 0.50% (w/w) | 2.3 ± 0.1 |
| TGS 0.75% (w/w) | 2.07 ± 0.12 |

**Effects of TGS (0.75% w/w) on body weight of normal rats (n=12) after six weeks.**
There was a significant increase in body weight of rats taking diets containing TGS as shown in Figure 1.

**Figure 1: The effects of TGS (0.75 %w/w) on the body weight of normal rats**




**Comparison of hypolipidemic efficacy of TGS with Rosuvastatin and Fenofibrate of hyperlipidemic rats:**
As shown in Table 2 no significant differences in hypolipidemic efficacy were found in serum TC between rats taking both concentrations of TGS (0.50% and 0.75% (w/w)) when compared with each of Rosuvastatin (10mg/kg) and Fenofibrate (30mg/kg) group.

Unlike Rosuvastatin and Fenofibrate, both concentrations of TGS (0.50% and 0.75% (w/w)) did not significantly decrease serum TG of hyperlipidemic rats.

Effect of TGS of both concentrations (0.50% and 0.75% (w/w)) on serum HDL-C was non-significantly higher than that of Rosuvastatin (10mg/kg) and Fenofibrate (30mg/kg), Table 2.

As shown in Table 2. TGS (0.75% w/w) non-significantly was more effective in reducing serum LDL-C than that of Rosuvastatin.

**Comparison of TGS effects on liver enzymes with Rosuvastatin and Fenofibrate of hyperlipidemic rats:** Unlike both Rosuvastatin and Fenofibrate, which caused a significant rise in ALP level, Trigonella foenum graceum seeds (0.75% w/w) significantly deceased serum ALP of hyperlipidemic rats.

Table 4 shows no significant difference in the effect of the TGS at (0.75% w/w) with Rosuvastatin (10mg/kg) and Fenofibrate (30mg/kg) on each of serum AST and ALT of hyperlipidemic rats.

## Discussion

Dyslipidemia is a major cause of atherosclerosis and atherosclerosis associated with conditions such as ischemic cerebrovascular disease, coronary heart disease and peripheral vascular disease (19, 20). Animal and human studies have established the role of cholesterol in the development and progression of atherosclerosis. Low density lipoprotein cholesterol (LDL) constitutes approximately 60-70% of serum total cholesterol (TC). Epidemiological studies directly implicated LDL to the development of atherosclerosis and coronary heart disease (21).

The result of the present study, showed that both concentrations of TGS (0.50% and 0.75% w/w) had reduced serum TC and LDL significantly and reduced TG level of hyperlipidemic rats. This result was similar to the findings of Kumar and Bhandari, (2013) who reported that giving aqueous extract of Trigonella foenum graecum (0.5 and 1g/kg orally) to hyperlipidemic rats for 28 days produced a significant reduction in serum TC and TGs (22). In another study Saxena and Saxena (2009) observed that administration of aqueous seed extract of Trigonella foenum graecum (120mg/kg, p.o.) for seven weeks to high fat diet and triton induced hyperlipidemic models of albino rats showed a 20.42% reduction in plasma cholesterol level and significantly attenuated the elevated plasma triglycerides and LDL level (23). Similar findings were reported by Patel et al., (2011) who showed that administration of Trigonella foenum graecum ethanol extract at a dose of 250 mg/kg for one week induced a significant reduction in serum LDL, TC and TG and significantly increased HDL level of hyperlipidemic rats (24).

In this study, TGS (0.75% w/w) significantly increased serum HDL level of normal and hyperlipidemic rats. These result were in agreement with the results of another study by Xue et al., (2007) who found that rats treated with Trigonella foenum graecum extracts of different doses (0.44 g/kg/day, 0.87g/kg/day and 1.74g/kg/day for 6 weeks) had lower blood glucose, serum TG and TC and higher serum HDL in a dose dependent way of diabetic rats (25).

Elmnan et al., (2012) also observed that TGS added to experimental diets at concentrations of 0.25%, 0.50% and 0.75%w/w have significantly decreased plasma total lipid and TG (26).

Other studies reported that the seeds of Trigonella foenum-graecum contain an unusual amino acid, which significantly decreased the plasma TG levels and TC, and free fatty acids, accompanied by an increase in HDL /TC ratio in the dyslipidemic hamster model ( 27, 28).

The reduction in TG and TC level in hyperlipidemic rats induced by TGS could be due to presence of bioactive fibers which act to decrease the rate of gastric emptying thereby delaying the absorption of lipid from the small intestine (29) or it directly inhibits the absorption of cholesterol by enterocytes of small intestine as ezetimibe (30), or it binds to bile acids and increases excretion of bile acids and neutral sterols in faeces (31). This action prevents the enterohepatic cycling of acids and obligates the liver to synthesize replacement of bile acids from cholesterol (32).

Rosuvastatin (10mg/kg) caused a significant decrease in serum TC, TG and LDL of hyperlipidemic rats. This result was in accordance with the observations of Ansari et al., (2012) who found that oral administration of Rosuvastatin (10mg/kg/day) for 21 days along with high fat diet had reduced serum TC,TG and LDL significantly when compared with hyperlipidemic rats (17).

This reduction of serum TC of Rosuvastatin is due to inhibition of HMG-CoA reductase which catalyzes the conversion of HMG-CoA to mevalonate which decreases the cholesterol synthesis (33, 34).

The results of the present study showed that Fenofibrate (30mg/kg/day) significantly reduced serum TG when compared with hyperlipidemic rats and this is similar to the findings of Santiago et al., (2013) who found that administration of 10mg/kg of Fenofibrate in hyperlipidemic mice significantly decreased TG by 54.87% (35). The remarkable decrease in TG levels by Fenofibrate supports the literature stating that it increases the expression of genes for lipoprotein lipase, and decreases the expression of apolipoprotein CIII. Apoliproprotein CIII is a known potent inhibitor of lipoprotein lipase while apolipoprotein CII activates the same enzyme. An imbalance in apo CIII/CII ratio due to increase in plasma apolipoprotein CIII may cause inactivation of lipoprotein lipase (36 Moberly et al., 1999).

In this study Fenofibrate did not reduce serum LDL of rats fed with atherogenic diet and this is in contrast with the study of Li et al, (2010) who found that hyperlipidemic rats treated with a high dose of Fenofibrate of 80mg/kg/day for 12 weeks reduced serum TG ,TC and LDL significantly. This indicates that the beneficial effects of Fenofibrate on

serum LDL probably needs more time and higher doses would have been more informative in our study (37).

Serum transaminases (ALT, AST) in this study, increased significantly in hyperlipidemic rats treated with TGS of both concentrations (0.50% and 0.75% w/w). This result was incompatible with another study reported by Kumar and Bhandari (2013) who observe that aqueous extract of TGS of (0.5 and 1g/kg, orally) for 28 days caused a significant reduction in serum transaminases (22).This discrepancy with our findings could probably be due to the higher doses of TGS and longer duration of the study. Conversly, Toppo et al., (2009) reported that TGS powder did not alter ALT, AST and alkaline phosphatase (ALP) levels maintained on (1%, 5%, 10%) up to 90 days (38). In another study Haeri et al., (2009) found that unusual amino acid (4-hydroxyisoleucine) isolated from the plant did not affect liver damage markers but it significantly improved HDL cholesterol levels (31% increase) in diabetic rats (30).

Serum alkaline phosphatase (ALP) of hyperlipidemic rats was significantly Increased by daily administration of Rosuvastatin (10mg/kg), this result was similar to a study reported by Dodiya et al., (2013) who found that orally administration of Rosuvastatin (40mg, 80mg/kg) for 21 days to rats significantly increased AST, ALP and total bilirubin levels. The serum ALP elevation might be in response to direct irritant effect of Rosuvastatin on hepatic cells (40).

In the present study Fenofibrate (30mg/kg) increased serum ALP and ALT significantly when compared with hyperlipidemic rats. It has been reported that Fenofibrate activates the aminotransferase gene expression, thus leading to a mild and transient elevation of aminotransferase via PPAR? through mechanisms involving increased levels of reactive oxygen species and intracellular glutathion depletion, thus leading to mitochondrial dysfunction and a perturbation of intracellular Ca++ homeostasis and also cell death (41); (37).

In this study, Malondialdehyde an end product of polyunsaturated fatty acid peroxidation, was increased significantly in the hyperlipidemic group (taking atherogenic diet 21% w/w) when compared with control group.

Hypertriglyceridemia and hypercholesterolemia were associated with oxidative modification of LDL, protein glycation, glucose-auto oxidation, thus leading to excess production of lipid peroxidation products which may cause elevation of oxidative stress in higher lipid and hyperlipidemic subjects. Clinical and epidemiological studies have proven that individuals with elevated LDL showed an increased risk for cardiovascular diseases (42). HDL may be protective by reversing cholesterol transport, inhibiting the oxidation of LDL and by neutralizing the atherogenic effects of oxidized LDL (43).

Increased lipid peroxidation is thought to be a consequence of oxidative stress which occurs when the dynamic balance between pro oxidant and antioxidant mechanism is impaired (44). It is known that hyperlipidemic states are associated with altered physical properties of cellular membranes (45), which may facilitate the escape of free radicals from the mitochondrial electron transport chain or the activation of NADPH oxidase (46).

The present study demonstrated that TGS significantly reduced malondialdehyde level of hyperlipidemic rats. However the same dose of the plant did not show a significant reduction in malondialdehyde level of normal rats. These results are in agreement with the study of Kumar and Bhandari, (2013) who detected that aqueous extract of TGS (0.5 and 1g/kg, orally) for 28 days produced a significant reduction in serum malondialdehyde of hyperlipidemic rats (22). This could be suggestive of an antioxidative effect during oxidative stress induced by hyperlipidemia and increased lipid peroxidation. Myhrstad et al (2002) found the antioxidant activity of flavonoids isolated from the seed of Trigonella foenum graecum, as it exhibited scavenging of hydroxyl radicals (OH) and inhibition of hydrogen peroxide-induced lipid peroxidation in rat liver mitochondria (47).

In this study, TGS (0.75% w/w) increased the body weight of normal rats significantly. This outcome is in agreement with the results of Elmnan et al., (2012) who observed that TGS added to experimental diets at concentrations of 0.25%, 0.50% and 0.75%w/w have significantly increased the body weight of normal rats (26). This effect on body weight could be attributed to the appetizing activity of steroidal saponins (diosgenin, yamogenin, tigogenin and neotigogenin) which are the major constituents of TGS (44, 45). Therefore, it is possible that the presence of saponins in the plant is responsible for the antioxidant and appetizing activity.

According to the above results on the effects of TGS, Rosuvastatin and Fenofibrate on the lipid profile and liver enzymes of hyperlipidemic rats, it can be observed that TGS has similar efficacy of Rosuvastatin and Fenofibrate in reducing TC. Whereas the seed powder was non-significantly more efficient than Rosuvastatin and Fenofibrate in changing serum HDL and LDL. Moreover, unlike to Rosuvastatin and Fenofibrate it significantly decreased serum ALP of hyperlipidemic rats.

## Conclusion

TGS has similar efficacy of Rosuvastatin and Fenofibrate in reducing TC. Whereas, TGS was non-significantly more effective than Rosuvastatin and Fenofibrate in changing serum HDL-C and LDL-C.

Fenofibrate and Rosuvastatin significantly increased serum alkaline phosphatase (ALP), while Trigonella foenum graecum seeds significantly decreased ALP for hyperlipidemic rats.

Rats that received TGS exhibited a significant decrease in malondialdehyde level when compared to hyperlipidemic rats.

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