

# The Saponin-Rich Fraction of a *Gymnema sylvestre* R. Br. Aqueous Leaf Extract Reduces Cafeteria and High-Fat Diet-Induced Obesity

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We examined the antiobesity effect of a saponin-rich fraction of a *Gymnema sylvestre* R. Br. aqueous leaf extract (SGE) using cafeteria and high-fat diet-induced obese rats for a period of eight weeks. SGE was orally administered at a dose of 100 mg/kg body weight once a day to the treatment group. It significantly decreased the body weight, food consumption, visceral organs weight, and the levels of triglycerides, total cholesterol, low-density lipoproteins, very low-density lipoproteins, atherogenic index, glucose, and increased the levels of high-density lipoproteins. There was no significant difference with respect to all parameters of the study in case of normal (N) diet and N diet + SGE rats. *In vitro*, SGE inhibited the pancreatic lipase activity. The present study gave clear evidence that the SGE has a significant antiobese action, supporting its use in traditional medicine, and can be used as a substitute for synthetic drugs.

**Key words:** *Gymnema sylvestre*, Cafeteria Diet, High-Fat Diet

## Introduction

The global prevalence of obesity is increasing rapidly among adults as well as among children and adolescents in places where high dietary fat intake is a major risk factor for the development of obesity (Canbakan *et al.*, 2008). Once considered a problem of developed countries, this global epidemic also affects developing countries. Coupled to this epidemic are obesity-related complications such as dyslipidemia (Fried *et al.*, 2008), type 2 diabetes mellitus (Pagotto *et al.*, 2008), fatty liver (Marovic, 2008), and cardiovascular diseases such as heart failure and coronary heart disease (Lavie *et al.*, 2008). Furthermore, it has been suggested that dietary fat promotes body fat storage more effectively than dietary carbohydrates. Consistent with these suggestions, high-fat diets can increase body weight and adiposity in humans and animals (Portillo *et al.*, 1999; Han *et al.*, 1999). Thus, inhibi-

tion of digestion and absorption of dietary fat is a key to treating obesity.

Nowadays there is an increased demand for using plants in therapy instead of using synthetic drugs which may have adverse effects. Traditional medicinal plants are often cheaper, locally available, and easily consumable. These simple medicinal preparations often mediate beneficial responses due to their active chemical constituents.

*Gymnema sylvestre* R. Br., belonging to the Asclepiadaceae family, is a native plant in the south-west of India, Australia and tropical Africa. From ancient times, *G. sylvestre* has been used in Indian traditional medicine, and is considered to be antiviral, diuretic, antiallergic, hypoglycemic, hypolipidemic, and to be effective in improving urination, digestion, and obesity (Anonymous, 2006). As for the active substances involved in *G. sylvestre*, triterpenoid saponins and their derivatives have been identified. These are glycosides; for example gymnemagenin is formed by the attachment of glucuronic acid to the triterpenoid structure as aglycone (Yoshikawa *et al.*, 1997). Other than these glycosides, conduritol-A with a tetrahydrox-

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**Abbreviations:** TG, triglycerides; TC, total cholesterol; LDL, low-density lipoproteins; VLDL, very low-density lipoproteins; HDL, high-density lipoproteins; AI, atherogenic index.

yxene structure has also been confirmed to be involved in glucose absorption. Moreover, the peptide grumarin, consisting of thirty-five amino acids, has been shown to be involved in suppression of sweetness (Ota *et al.*, 1998). It has been reported that the saponins of ginseng showed a strong inhibitory effect on pancreatic lipase activity *in vitro* and suppressed the increase in body weight (Yun *et al.*, 2004).

In this study, we examined the effect of saponin-rich *G. sylvestre* aqueous leaf extract on obesity induced in rats fed on cafeteria and high-fat diets, respectively.

## Material and Methods

### Collection of plant material

Leaves of *G. sylvestre* R. Br. were collected from Tirumala hills, A. P., India. The plant was botanically authenticated by a taxonomist of the Department of Botany, Sri Venkateswara University, Tirupati, A. P., India. A voucher specimen was deposited for future reference.

### Phytochemical study

The *G. sylvestre* leaves were subjected to a number of standard phytochemical screening tests for various phytoconstituents.

### Extraction of saponin fraction

The aqueous extraction was carried out as described earlier (Kurihara, 1969). Five hundred grams of shade-dried *G. sylvestre* leaves were powdered and extracted twice with 4 l of distilled water in a Soxhlet extractor at 60 °C for 5 h. After filtration, extracts were combined and acidified with 1 M sulfuric acid to pH 2.0. The precipitate was filtered, dried, and then extracted with ethanol and acetone. The insoluble matter was eliminated by filtration, and solvents were evaporated. The resulting dark green powder (7.1 g) was designated SGE and used in the feeding experiment. The fraction was tested for saponins using the Froth test and the Libermann-Burchard test (Evans, 1989).

### Acute toxicity studies

The acute toxicity of SGE was determined according to guideline No. 420 of the Organization for European Economic Cooperation (OECD) using male Wistar rats (110–130 g). Initial doses

of 100, 400, 800, 1200, 1600, and 2000 mg/kg body weight of SGE were administered to the respective six groups of four rats each and monitored for three weeks for mortality and general behaviour. Toxic symptoms or mortality were observed till the end of the study with doses of 800–2000 mg/kg body weight. The lethal dose (LD<sub>50</sub>) was determined as 400 mg/kg body weight. Hence, the experimental dose was selected as one-fourth (100 mg/kg body weight) of the LD<sub>50</sub>.

### Animal diets

The cafeteria diet consisted of three variants: (a) 10 g condensed milk + 10 g bread + 5 g pellet chow (4:4:2); (b) 3.75 g chocolate + 7.5 g biscuits + 7.5 g dried coconut + 6.25 g pellet chow (1.5:3:3:2.5); and (c) 10 g cheese + 12.5 g boiled potatoes + 2.5 g pellet chow (4:5:1). The three variants were presented to the individual rats on days one, two, and three, respectively, and then repeated for eight weeks in the same succession (Harris, 1993). High-fat diet (39% carbohydrate, 21.5% fat, 34.5% protein, 5% mineral and vitamin mixtures AIN 93) was obtained from the National Institute of Nutrition, Hyderabad, India.

### Experimental protocol

Male Wistar rats (110–130 g) were purchased from Sri Venkateswara Animal Agency at Bangalore, India. The rats were allowed free access to food and tap water under strictly controlled pathogen-free conditions at a room temperature of (26 ± 2) °C with relative humidity of 60% and a 12-h light/dark cycle. The rats were fed on standard rodent pellet chow and acclimatized to the environment for one week; the healthy animals were used for further study. The animals were divided into six groups (*n* = 6): (i) control for normal diet (N diet); (ii) normal diet + saponin-rich *G. sylvestre* aqueous leaf extract (N diet + SGE); (iii) control for cafeteria diet (CA diet); (iv) cafeteria diet + SGE (CA diet + SGE); (v) control for high-fat diet (HF diet); (vi) high-fat diet + SGE (HF diet + SGE). SGE at 100 mg/kg body weight was administered for 8 weeks once a day (between 8 a.m. and 10 a.m.) to the respective treatment group. The dose was suspended in distilled water and given orally using a gastric gavage. Since the study was carried out with antiobesigenic perspective, the animals received CA diet + SGE and HF diet + SGE from day one of the study. The food consumption rate

was calculated daily by subtracting the amount of food left over in each cage barrier per each rat from the measured amount of food provided on the previous day [g/(d rat)]. The mean of food consumption per each rat was calculated by dividing the amount of food eaten in a week by seven. The animals were weighed at the start of the experiment and then every week thereafter. At the end of the experimental period, blood samples were collected from the retro-orbital plexus in centrifuge tubes. The blood samples were allowed to clot for 30 min at room temperature and then centrifuged at 1200 x g for 15 min. Serum samples thus obtained were stored at -20 °C until biochemical assays were carried out. The animals were killed under anaesthesia using 85 mg/kg body weight ketamine and 95 mg/kg body weight xylazine (intraperitoneally), and different visceral organs (liver, kidney, spleen, heart, peritoneal and perirenal fat mass) were immediately removed and weighed. The animal experimentation was carried out according to Institutional Animal Ethical Committee Guidelines (CPCSEA), Sri Venkateswara University, Tirupati, A. P., India.

#### *Biochemical analysis of serum*

The serum glucose level was determined using a glucometer (Accu Chek Sensor set; Roche Diagnostics, Mannheim, Germany). Serum lipids such as total cholesterol (TC), triglycerides (TG), and high-density lipoproteins (HDL) levels were measured by enzymatic colorimetric methods using biochemical kits purchased from Kamineni Life Sciences, Pvt (Hyderabad, India). Low-density lipoproteins (LDL) and very low-density lipoproteins (VLDL) levels were calculated using Friedewald's formula (Friedewald *et al.*, 1972). The atherogenic index (AI), a risk ratio for coronary heart disease, was calculated using the formula:  $AI = TC/HDL - \text{cholesterol}$ .

#### *In vitro pancreatic lipase activity*

The lipase activity was determined by measuring the rate of release of oleic acid from triolein. A suspension of triolein (80 mg), phosphatidylcholine (10 mg), and taurocholic acid (5 mg) in 9 ml of 0.1 M *N*-tris(hydroxymethyl)methyl-2-aminoethanesulfonic acid (TES), pH 7.0, containing 0.1 M NaCl was sonicated for 5 min. The sonicated substrate suspension (100  $\mu$ l) was incubated with 50  $\mu$ l (final content 10 units per tube) of pancreatic lipase and 100  $\mu$ l of various concentrations (0,

50, 100, 200, and 400 mg/ml) of SGE for 30 min at 37 °C in a final volume of 250  $\mu$ l, and the released oleic acid was measured by the method of Belfrage and Vaughan (1969). The lipase activity was expressed as mol of oleic acid released per ml of reaction mixture per h.

#### *Statistical analysis*

Data are expressed as the mean  $\pm$  SD. The statistical significance of differences between the mean values for the treatment groups was analysed by Student's *t*-tests using Instat (Graph Pad Software, Inc., Lajolla, CA, USA).

### **Result**

#### *Effect of SGE on body weight*

Body weights of rats that were fed on experimental diets with or without SGE are shown in Figs. 1–3. The body weights significantly increased with time, and percentage increase was higher for the cafeteria (15%) and high-fat diet (17%) fed rats than for the rats fed on normal diet. In contrast, non-significant differences were observed between N diet and N diet + SGE rats. The increase in body weight was lower in the rats treated with SGE (Figs. 1 and 2). In case of CA diet + SGE and HF diet + SGE rats, the body weights were lower by 9% and 13%, respectively, when compared with cafeteria and high-fat diet rats, respectively.

#### *Effect of SGE on food consumption*

Food consumption increased significantly in rats fed on cafeteria and high-fat diets, respectively, when compared with rats fed on normal diet. Treatment with SGE significantly decreased the food consumption during the treatment period (Table I).

#### *Effect of SGE on visceral organs weight*

There was a significant increase in the weight of visceral organs (liver, spleen, kidney, heart, peritoneal and perirenal fat mass) in rats fed on cafeteria and high-fat diets, respectively, compared with rats fed on normal diet, while treatment with SGE significantly suppressed the weight increase of visceral organs (Table II).

#### *Effect of SGE on serum biochemical parameters*

Serum TG, TC, LDL, VLDL, glucose levels, and AI were significantly elevated in rats fed on

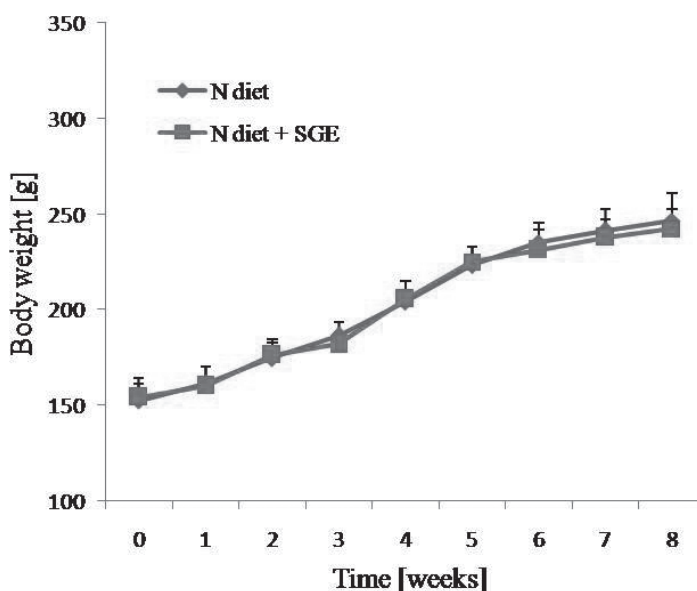


Fig. 1. Effect of saponin-rich *G. sylvestre* aqueous leaf extract on body weights of rats fed with normal diet during the treatment period.

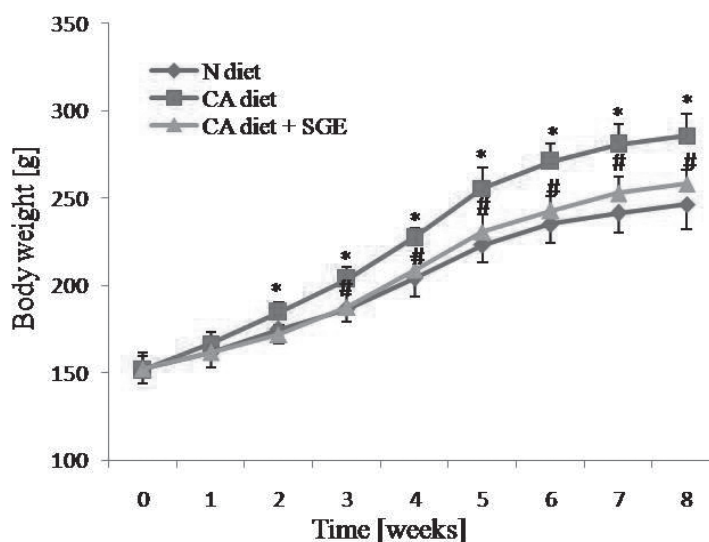


Fig. 2. Effect of saponin-rich *G. sylvestre* aqueous leaf extract on body weights of rats fed with cafeteria diet during the treatment period. Treatment with *G. sylvestre* aqueous leaf extract significantly prevented the elevations in body weight during the treatment period compared to the cafeteria diet group. Differences are significant as \* $P < 0.001$  when compared with the N diet group and # $P < 0.001$  when compared with the CA diet group.

cafeteria and high-fat diets, respectively, compared with rats fed on normal diet, while the HDL level was significantly decreased compared to the rats fed on normal diet. Additional administration of SGE significantly prevented these changes (Table III).

#### Effect of SGE on pancreatic lipase activity

SGE produced a dose-dependent inhibition of pancreatic lipase activity at concentrations of 0, 50, 100, 200, and 400 mg/ml, as indicated by the reduction in the amount of free fatty acids released

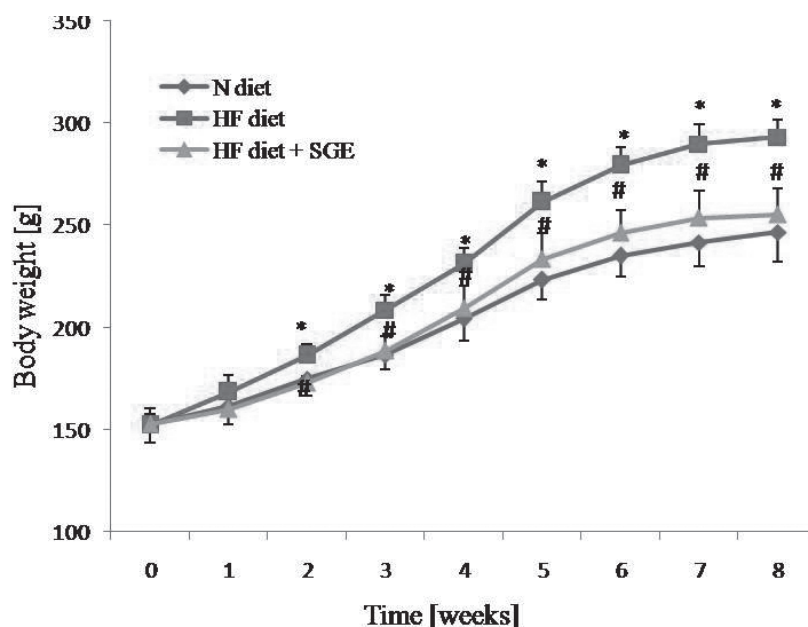


Fig. 3. Effect of saponin-rich *G. sylvestre* aqueous leaf extract on body weights of rats fed with high-fat diet during the treatment period. Treatment with *G. sylvestre* aqueous leaf extract significantly prevented the elevation in the body weight during the treatment period compared to the high-fat diet group. Differences are significant as  $*P < 0.001$  vs. the N diet and  $^{\#}P < 0.001$  vs. the HF diet.

Table I. Effect of saponin-rich *G. sylvestre* aqueous leaf extract on food consumption [g/(d rat))] of rats fed with cafeteria and high-fat diets, respectively.

Time	N diet	N diet + SGE	CA diet	CA diet + SGE	HF diet	HF diet + SGE
1 <sup>st</sup> week	12.21 ± 1.47	11.02 ± 1.59 <sup>ns</sup>	14.66 ± 1.0*	10.84 ± 1.88 <sup>##</sup>	13.99 ± 1.44***	10.93 ± 0.68 <sup>#</sup>
2 <sup>nd</sup> week	13.04 ± 0.42	13.96 ± 1.02 <sup>ns</sup>	15.13 ± 0.60*	12.74 ± 0.95 <sup>#</sup>	14.44 ± 1.39***	11.30 ± 0.53 <sup>#</sup>
3 <sup>rd</sup> week	13.88 ± 1.36	14.36 ± 1.01 <sup>ns</sup>	16.21 ± 1.14*	13.94 ± 2.31 <sup>###</sup>	16.16 ± 2.39***	13.05 ± 1.92 <sup>###</sup>
4 <sup>th</sup> week	16.10 ± 1.31	15.63 ± 2.03 <sup>ns</sup>	18.85 ± 1.04**	14.83 ± 2.57 <sup>###</sup>	18.10 ± 1.10**	14.94 ± 2.27 <sup>###</sup>
5 <sup>th</sup> week	17.38 ± 1.32	16.32 ± 2.31 <sup>ns</sup>	20.84 ± 2.49***	16.71 ± 2.05 <sup>###</sup>	19.88 ± 2.05***	16.44 ± 1.50 <sup>#</sup>
6 <sup>th</sup> week	17.83 ± 1.88	16.35 ± 1.09 <sup>ns</sup>	21.38 ± 2.21**	17.88 ± 1.32 <sup>#</sup>	21.94 ± 2.84***	17.83 ± 1.77 <sup>##</sup>
7 <sup>th</sup> week	18.99 ± 2.40	17.89 ± 1.82 <sup>ns</sup>	24.88 ± 2.23***	19.09 ± 1.13 <sup>#</sup>	23.5 ± 2.73**	18.16 ± 1.98 <sup>#</sup>
8 <sup>th</sup> week	19.44 ± 2.50	18.02 ± 2.04 <sup>ns</sup>	25.15 ± 1.74*	19.55 ± 1.37 <sup>#</sup>	23.75 ± 2.92***	18.49 ± 2.46 <sup>#</sup>

Values are expressed as means ± SD ( $n = 6$ ). Values are significantly different. ns, not significant; \*\*\* $P < 0.05$ , \*\* $P < 0.01$ , \* $P < 0.001$  vs. N diet; ### $P < 0.05$ , ## $P < 0.01$ , # $P < 0.001$  vs. CA diet and HF diet.

Table II. Effect of saponin-rich *G. sylvestre* aqueous leaf extract on visceral organs weights of rats fed with cafeteria and high-fat diets, respectively.

Parameter	N diet	N diet + SGE	CA diet	CA diet + SGE	HF diet	HF diet + SGE
Liver (g)	9.91 ± 0.44	9.28 ± 0.69 <sup>ns</sup>	13.96 ± 1.15*	10.48 ± 0.81 <sup>#</sup>	15.09 ± 1.62*	11.08 ± 0.62 <sup>#</sup>
Spleen (g)	1.7 ± 0.26	1.61 ± 0.86 <sup>ns</sup>	2.24 ± 0.42***	1.77 ± 0.26 <sup>##</sup>	2.27 ± 0.41**	1.88 ± 0.62 <sup>###</sup>
Heart (g)	1.69 ± 0.04	1.52 ± 0.13 <sup>ns</sup>	2.02 ± 0.04*	1.76 ± 0.10 <sup>#</sup>	2.07 ± 0.10*	1.77 ± 0.09 <sup>#</sup>
Kidney (g)	2.25 ± 0.25	2.28 ± 0.36 <sup>ns</sup>	2.98 ± 0.07*	2.3 ± 0.22 <sup>#</sup>	3.05 ± 0.10*	2.31 ± 0.20 <sup>#</sup>
Peritoneal fat (g)	4.19 ± 0.66	3.52 ± 1.03 <sup>ns</sup>	12.97 ± 0.67*	6.26 ± 0.74 <sup>#</sup>	13.84 ± 0.64*	7.01 ± 0.62 <sup>#</sup>
Perirenal fat (g)	3.54 ± 0.36	2.86 ± 0.78 <sup>ns</sup>	11.10 ± 0.94*	5.09 ± 0.70 <sup>#</sup>	11.84 ± 0.73*	6.27 ± 0.69 <sup>#</sup>

Values are expressed as means ± SD ( $n = 6$ ). Values are significantly different. ns, not significant; \*\*\* $P < 0.05$ , \*\* $P < 0.01$ , \* $P < 0.001$  vs. N diet; ### $P < 0.05$ , ## $P < 0.01$ , # $P < 0.001$  vs. CA diet and HF diet.

Table III. Effect of saponin-rich *G. sylvestre* aqueous leaf extract on serum lipid profile and glucose (mg/100 ml) of rats fed with cafeteria and high-fat diets, respectively.

Parameter	N diet	N diet + SGE	CA diet	CA diet + SGE	HF diet	HF diet + SGE
TG	97.99 ± 6.71	90.78 ± 7.86 <sup>ns</sup>	153.89 ± 12.1*	112.39 ± 6.48 <sup>#</sup>	164.35 ± 10.14*	116.78 ± 4.98 <sup>#</sup>
TC	73.33 ± 13.98	71.01 ± 9.36 <sup>ns</sup>	140.11 ± 18.03*	104.44 ± 10.03 <sup>#</sup>	161.1 ± 14.83*	111.10 ± 10.88 <sup>#</sup>
HDL	45.55 ± 1.71	46.98 ± 2.09 <sup>ns</sup>	27.21 ± 2.51*	37.77 ± 5.71 <sup>#</sup>	24.99 ± 4.08*	38.33 ± 4.59 <sup>#</sup>
LDL	43.37 ± 2.68	41.29 ± 3.01 <sup>ns</sup>	62.88 ± 5.05*	50.37 ± 2.5 <sup>#</sup>	70.02 ± 5.07*	55.13 ± 3.86 <sup>#</sup>
VLDL	19.59 ± 1.33	17.95 ± 2.01 <sup>ns</sup>	30.77 ± 2.42*	22.46 ± 1.31 <sup>#</sup>	32.87 ± 2.03*	23.35 ± 1.24 <sup>#</sup>
AI	1.67 ± 0.46	1.57 ± 0.82 <sup>ns</sup>	5.17 ± 0.80*	2.77 ± 0.41 <sup>#</sup>	6.55 ± 0.96*	2.91 ± 0.38 <sup>#</sup>
Glucose	85.66 ± 3.93	81.96 ± 4.35 <sup>ns</sup>	146.66 ± 5.31*	109.16 ± 7.6 <sup>#</sup>	132.66 ± 4.08*	106.33 ± 6.02 <sup>#</sup>

Values are expressed as means ± SD ( $n = 6$ ). Values are significantly different. ns, not significant; \* $P < 0.001$  vs. N diet; <sup>#</sup> $P < 0.001$  vs. CA diet and HF diet.

from triolein per hour. The amounts of free fatty acids from triolein were found to be 0.58, 0.39, 0.28, 0.26, and 0.20  $\mu\text{mol}/(\text{ml h})$  by 0, 50, 100, 200, and 400 mg/ml of SGE, respectively.

## Discussion

The present study demonstrated that the saponin-rich fraction of *G. sylvestre* aqueous leaf extract reduces the diet-induced obesity in rats fed on either cafeteria diet or high-fat diet. The effects on body weight, food consumption, weight of visceral organs, serum biochemical parameters, and inhibition of pancreatic lipase were investigated.

Rats fed on a variety of highly palatable, energy-rich, high-carbohydrate cafeteria foods elicited a significant increase in body weight and fat pad mass. Cafeteria diets have been previously reported to increase energy intake and cause obesity in humans (Bull, 1988) as well as animals (Rothwell *et al.*, 1983). Obesity is considered to be a disorder of energy balance, occurring when energy expenditure is no longer in equilibrium with daily energy intake, so as to ensure body weight homeostasis (Van Herpen and Schrauwen-Hinderling, 2008). Although the etiology of obesity is complex, dietary factors, particularly the consumption of a cafeteria diet (Srinivasan *et al.*, 2008) and high-fat diet (Kim *et al.*, 2000) are considered risk factors for its development. The current study revealed that the body weight increased significantly in the cafeteria diet and high-fat diet group compared with the normal diet group (Figs. 1 and 2), a result in accordance with Xu *et al.* (2008). A gain in body weight is a common index of obesity (Toplak *et al.*, 2000). The increased body weight in rats fed on cafeteria and high-fat diets, respec-

tively, when compared to animals fed on normal diet, might be due to hyperphagia (Soundararajan *et al.*, 2010). The gain in body weight is largely due to increased fat mass as a result of preadipocyte proliferation by differentiation and, to some extent, accumulation of lipids in the liver (Llado *et al.*, 2000).

Consumption of a high-fat diet leads to obesity because it facilitates the development of a positive energy balance leading to an increase in fat deposition which leads to abdominal obesity in particular. In the current study, rats fed on cafeteria and high-fat diets consumed considerably more food than the control rats fed on normal diet throughout the experiment (Table I). As a result their caloric intake was increased and they showed a large increase in perirenal and peritoneal fat mass (Table II), suggesting that the excess energy led to an increase in adiposity. Rats consuming the cafeteria and high-fat ration, respectively, actually received more calories, and had more weight and a larger fat mass than rats fed on normal diet. The SGE-treated group showed a significant decrease in body weight, food consumption, and visceral organs weight. This is in accordance with the findings of Shigematsu *et al.* (2001).

The cafeteria and high-fat diets, respectively, produced a significant increase in serum glucose level (Table III), which parallels the results obtained by Leibowitz *et al.* (1998). Diminished hepatic and muscular uptake of glucose produces hyperlipidemia due to increased fat mobilization from adipose tissue and resistance to the antilipolytic actions of insulin. Impaired insulin action is associated with an oversupply of lipids. This increased availability leads to either elevated lipid storage in insulin target tissues (*e.g.* muscle, liver

adipose) or increased plasma-free fatty acids or triglycerides (Frayn, 2002). A significant decrease of serum glucose level in rats fed on high-calory diets along with SGE was observed (Table III) in agreement with Gholap and Kar (2003).

Cafeteria diet and high-fat diet resulted in dyslipidemic changes as illustrated by increasing TG, TC, LDL, and VLDL levels and a decrease in HDL levels (Table III), a finding in accordance with that of Soundararajan *et al.* (2010) and Woo *et al.* (2008). SGE treatment produced a significant decrease in serum TG, TC, LDL, and VLDL levels while there was a significant increase in HDL cholesterol in high-fat diet-fed rats. These results are in agreement with those of Bishayee and Chatterjee (1994). The possible explanation for the hypocholesterolemic activity of SGE is an increased excretion of cholesterol, neutral steroids, bile acids in the feces, and lipid-lowering effects, resulting in depression of lipid accumulation. It consequently has antiatherosclerotic properties (Nakamura *et al.*, 1999).

Cafeteria and high-fat diets induce atherosclerosis which is the major cause of morbidity from coronary heart disease (CHD). An increased atherogenic index is attributed to enhanced intestinal absorption and secretion and catabolism of cholesterol (Safur Rehman *et al.*, 2010). SGE caused a significant reduction in the atherogenic

index, which is considered a better indicator of coronary heart disease risk than the individual lipoprotein concentration. SGE would be beneficial in the prevention of plaque formation leading to atherosclerosis and CHD accelerated by cafeteria and high-fat diets.

The dietary lipid is not directly absorbed from the intestine unless it has been hydrolyzed by pancreatic lipase enzyme. The products formed are fatty acids and 2-monoacylglycerides, which are absorbed (Verger, 1984). Thus the inhibition of this enzyme is beneficial in the treatment of obesity. Orlistat, an approved antiobese drug is clinically reported to prevent obesity and hyperlipidemia through inhibition of the pancreatic lipase and increased fat excretion into the feces (Drent *et al.*, 1995). The saponins of *G. sylvestre* showed a strong inhibitory effect on pancreatic lipase activity *in vitro* which might have suppressed the increase in body weight induced by cafeteria and high-fat diets *in vivo*. The results are in accordance with those of Yun *et al.* (2004) and Han *et al.* (2005) for ginseng.

SGE might exert its antiobesity action, intestinal absorption of dietary fat, through the inhibition of pancreatic lipase activity. Such obesity and its associated problems could be suppressed by the saponin-rich fraction of a *G. sylvestre* aqueous leaf extract.

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