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Original Article

Antidiabetic Effect of Selenium-Laden Garlic (*Allium sativum* L.) in Streptozotocin-Induced Diabetic Rats

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ABSTRACT

Background: Diabetes mellitus, being a prevalent and difficult-to-treat medical condition requires special concern from researchers and medicinal practitioners. Various research showed that Selenium (Se) and *Allium sativum* (garlic) have antidiabetic potential, indicating Se-laden garlic (SeG) to be an effective treatment strategy. This study investigated the effects of Se-laden garlic on the streptozotocin (STZ)-induced diabetic rats. **Materials and Methods:** 66 Wistar rats were divided into 11 groups. Diabetes was chemically induced using streptozotocin (STZ). The effects of SeG extract were tested on groups of normal (NR) and diabetic rats (DR) with three oral doses (125 mg/kg BW (SeG1), 250 mg/kg BW (SeG2), and 500 mg/kg BW (SeG3)) for 5 weeks. Two groups of each were administered parallelly with normal garlic extract (NG) and a standard antidiabetic drug, Glibenclamide (Gli) for comparisons. Each group underwent evaluation to examine hematological and serum biochemical characteristics. **Results:** The oral administration of Gli, NG, SeG1, and SeG2 showed effectively reduced serum glucose and close to restored levels of liver function parameters together with several other restorative indications. **Conclusion:** The study supports the anti-diabetic properties of garlic leaves supplemented with a defined concentration of Se. The data collected can be used as a guideline to determine the dose range to predict the MTD (maximum tolerated dose) of SeG plants.

Keywords: *Allium sativum*, Diabetes mellitus, Selenium, Garlic, Antidiabetic activity, Streptozotocin.

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INTRODUCTION

Diabetes Mellitus (DM), an important chronic syndrome is the fastest-growing metabolic disease of the 21st century resulting from a combination of hereditary and environmental variables. It has a substantial negative influence on a patient's quality of life, health, and life expectancy.[1] Both hereditary and acquired DM are essentially defined by increased blood glucose level, also known as hyperglycemia, and a progressive breakdown of multiple metabolic processes, if untreated. DM is widely treated throughout the world with a variety of medications as well as nutritional management methods that include herbal and traditional medicines. DM occurs due to the inability of the body to regulate the glucose. There are mainly two types of diabetes based on the reason for occurrence, Type I (autoimmune destruction of pancreatic beta cells) and Type 2 (insulin resistance). The pathophysiology of either type is mainly governed by the insulin resistance or deficiency. In Type I diabetes, the body metabolizes the fats at faster rates and makes the blood acidic causing diabetic ketoacidosis. Type II diabetes is characterized by the increased carbohydrate intake, decreased peripheral glucose uptake, increased hepatic glucose production, decreased insulin production, and increased peripheral insulin resistance.[2] DM shows islet paracrinopathy, where the normal interaction between the glucagon-producing alpha cells and the insulin-producing beta cells is disrupted, resulting in hyperglucagonemia and, consequently, hyperglycemia.[3] The development of type 2 diabetes is influenced by a combination of genetic and environmental factors.[2,4]

There are various effective strategies to cope up with the complications of diabetes. One of the traditional strategies is the consumption of garlic, *Allium sativum* L. Garlic, is a highly regarded anti-diabetic plant that has been extensively studied in both traditional medical systems and contemporary research.[5] This can be credited to its multi-target actions which affect more than one complication at a time.[6] The rich phytochemical profile of garlic, containing proteins, vitamins, macro- and micronutrients, alkaloids, phenols, essential oils, and most importantly the organo-sulfur compounds have gained it a repute as a miracle drug.[7] Studies suggest that garlic targets the potential proteins and decreases the negative impact of disease.

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Some of the predicted targets of garlic are AMP-Activated Protein Kinase (AMPK),[8] C-reactive protein,[9] Glucose Transporter 4 (Glut-4),[10] Glucose Activated NO Synthase,[10] serum adenosine deaminase,[11,12,13] Glucokinase (GK),[14] Glycogen Synthase (GS),14 Phosphoenolpyruvate Carboxykinase (PEPCK),[14] Aldose Reductase (ALR2)[15] and Protein Tyrosine Phosphatase 1B (PTP-1B).[16,17] AMPK is an important regulator of energy metabolism which is found ubiquitously. Chronic inflammation leads to reduced AMPK activity. The activation of this protein has been found beneficial for several diseases by suppressing the inflammation. The garlic constituents, S-Allylcysteine (SAC), S-1-Propenylcysteine (S1PC) and S-Allylmercaptocysteine (SAMC), have been found to play a role in promoting the phosphorylation of AMPK by increasing the AMP/ATP ratio and/or the intracellular calcium concentration.[8] Another predicted target protein is CRP (C-Reactive Protein), which is an inflammatory factor associated with the diabetes complications and it impairs the Nitric Oxide (NO) production which is required for the synthesis of insulin in liver cells in the presence of sugar. Thus, it indirectly inhibits insulin production.[9] Also, glucose-induced NO synthesis in liver cells stimulates the production and translocation of GLUT-4, which further stimulates the insulin synthesis. It has been demonstrated that mannose binding lectin protein extracted from garlic can activate NO signaling by stimulating glucose-activated NO synthase, which in turn promotes the expression of insulin and GLUT-4, thereby reducing blood glucose levels.[10] An independent study suggests that garlic in combination with metformin reduces the CRP in diabetic rats which indicates its therapeutic potential. Another study reported that higher Adenosine Deaminase (ADA) activity in the tissues reduces sensitivity to insulin, leading to reduced glucose uptake.[11] Garlic has the potential to reduce ADA and ameliorate the diabetes complications.[12,13] Garlic polysaccharide may also regulate Glucokinase (GK), Glycogen Synthase (GS), and Phosphoenolpyruvate Carboxykinase (PEPCK) to alleviate the blood glucose level.[14] ALR is an important enzyme involved in the polyol pathway, which catalyzes the conversion of glucose to sorbitol and leads to substantial accumulation of Reactive Oxygen Species (ROS). A study on *Cucumis melo* demonstrated the selective inhibition of ALR2, which is the potential target compound of garlic, for diabetes.[15] Another important candidate target is PTP1B which alters cell signaling and plays an important role in etiology of diabetes. It plays a crucial role in insulin dephosphorylation and thus it's signaling. The computational analysis suggested that garlic possesses several compounds which can potentially inhibit the PTP1B.[16] A study found some motifs in PTP1B protein which can be used in the therapeutic formulation for diabetes.[17]

Additionally, studies have also suggested that Selenium-laden Garlic (SeG) could have modified phytochemical characteristics and exhibit therapeutic properties including anticancerous activity.[18] Several insightful experimentations have focused on the production, plant biochemistry,[19] bioavailability, and medical applications of Se-enriched crop plants,[20] but still, there exists a dearth of information on their safe consumption in relevance to various diseases, especially DM. Theoretically, both Se and garlic are antioxidants and that SeG is already considered as a Se supplement in a few Se-deficient countries like Finland.[21] This study investigated the effects of SeG on the Streptozotocin (STZ)-induced diabetic rats.

MATERIALS AND METHODS

Experimental animals

Healthy male Wistar rats (*Rattus norvegicus*), weighing around 200-250 g were procured from breeder DFHAS (LUVAS) Haryana, India. The rats were acclimatized and housed throughout the experimental duration following the standard CPCSEA guidelines in a well monitored, ventilated, air-conditioned room at 22°C, 60-70% relative humidity with the light/dark cycle of 12-hr, alternatively. The animals were fed with standardized pellets diet and hygienic RO drinking water ad libitum in the premises of the animal house facility, Sharda University.

Induction of diabetes- Streptozotocin (STZ) administration

Rats with good health with normoglycemia were selected for the experiment. Before diabetes induction the animals were randomized (± 20% mean difference in BW) and grouped followed by overnight starvation with free access to water. Rats of groups assigned for diabetes induction were individually weighed and their Fasting Blood Glucose (FBG) was measured from the blood withdrawn by tail nicking method (Day 0; D0) using a digital glucometer. The rats were then injected intra-peritoneally (i.p.)[22] using sterilized needles with freshly constituted STZ (Himedia) injection, in cold sodium citrate buffer solution (0.1 M, pH 4) in a dosage of 45 mg/ kg BW[23] and in a maximum volume of 0.5 mL.[24] Monitoring of the rats was continuously done. FBG was recorded to check for stable hyperglycemia at an interval of 3 days (Day 3; D3) and (Day 6; D6) in the overnight fasted rats. Rats with FBG ≥ 126 mg/dL were marked as hyper-glycemic and used in the study as diabetic rats. Animals showing ≤ 250 mg/dL FBG were examined as moderately diabetic and animals having ≥ 250 mg/dL were considered severely diabetic. These animals were, randomized again based on glycemic levels to maintain an averagely balanced cohort of FBG across the different diabetic groups.

Plant material and aqueous extract preparation

Garlic plants (Indian Var.YS G-282) were grown with and without Se supplementation using sodium selenate (2 mg/L Na_2SeO_4) in the experimental micromodel greenhouse at the University.[25] The leaves of garlic plants were collected before bulbing stage. Leaf aqueous extracts (stock concentration of 0.5 g/mL) were prepared

following the procedure of Nasim et al. 2009[26] by cold maceration method and stored at -20°C. They were labeled as NG-Normal garlic grown without Se supplementation and SeG-selenium garlic grown in Na_2SeO_4 supplementation.

Experimental treatment regimen

66 animals were divided into eleven groups (n=6). The animals in each group were orally fed once daily with different treatments solutions, as shown in Table 1. The feeding was given for a period of 35 days (5 weeks). The dose selection was based on the dose standardization experiment (data not shown) and literature survey of other similar experiments.[27,28]

NG plant extract was given in dose of 500 mg/kg BW and SeG extract was given in a dose of 125, 250 and 500 mg/kg BW. The stock concentrate of NG and SeG leaf extract (0.5 g/mL) was freshly thawed and diluted with cold, sterile 0.9% saline solution just before feed administration to the animals. The two feeding solutions of SeG corresponding to 0.1 g/mL and 0.25 g/mL were freshly prepared everyday just before the feed. Glibenclamide (Daonil tablet- Sanofi India Ltd.,) was used as the reference standard antidiabetic drug and was orally fed as a solution of 500 µg/mL in sterile distilled water at an oral dose of 600 µg/kg animal BW. The volume of the feeding solutions (0.1, 0.25 and 0.5 g/mL) of NG, SeG extracts and Gli solution (600 µg/mL) as per their body weight were calculated by the following formula:

$$\text{volume of feeding solution (ml)} = \frac{\text{weight of animal (kg)} \times \text{required dose (g kg}^{-1}\text{) or (µg kg}^{-1}\text{)}}{\text{concentration of feeding solution (g mL}^{-1}\text{) or (µg mL}^{-1}\text{)}}$$

Each animal was fed with the selected solutions in a volume strictly ranging between ≥0.3 mL to ≤1 mL. Regular and scheduled observations were made on the mortality and general clinical symptoms for the expression of any adverse effects.

Euthanasia and collection of blood and tissue

Blood was sampled during treatment period by the tail vein nicking method during the treatment period on the 0th, 7th, 14th, 21st, 28th, and 35th days for monitoring of FBG by glucometer. After end of the treatments (40th day), all the animals were euthanized by Xylazine/Thiopentone sodium injection method and the collection of blood was done from the heart. Blood was drawn out in oxalate fluoride mixture tubes for glucose determination and plain sterile centrifuge tubes for serum separation for determination of other tests. The non-haemolyzed serum was then analyzed after storage at 0^0 – 4^0C or -20^0C for different parameters and for enzymes contents instantly within 24 hours of storage. Tissues of liver and soleus muscle from the hind legs were removed, washed with cold sterile distilled water, and stored at -70°C for glycogen assay.[29]

Biochemical assay

Analysis of FBG was done in several instances starting before diabetes induction and thereafter. It was performed by a portable glucometer (Accu-check, Roche Diabetes Care, Inc.). Serum Glucose was quantified by using the method of GOD-POD (glucose oxidase- peroxidase) method by Trinder, 1969 and Lott, 1975.[30,31] Serum total Triglycerides (TAG) were quantified by using the GPO-Trinder Wako method and modifications proposed by McGowan *et al.*, 1983, and Fossati and Prencipe, 1982.[32,33] Serum total Cholesterol (CHO) was quantified by using Roeschlau's method.[34] Serum High-Density Lipoproteins (HDL) were quantified by using Polyethylene-Glycol-Methyl Ether (PEGME) and Polyvinyl Sulfonic acid (PVS) and coupled classical precipitation method with improvements by Pisani T *et al.*, 1995.[35] The Very Low-Density Lipoproteins (VLDL) were calculated by dividing the TAG content by 5. The Low-Density Lipoproteins (LDL) were calculated by applying the traditional Friedewald's equation.[36] Serum Alkaline Phosphatase (ALP) was quantified by using the recommended method of the International Federation of Clinical Chemistry.[37] Biuret reaction methodology was used

Table 1: Division of treatment groups according to treatment material.

Type of rats	Group no.	Material of treatment (1 mL/dose/day)
Non diabetic	1	Sterile normal saline
	2	NG at 500 mg/kg animal BW
	3	SeG1 at 125 mg/kg animal BW
	4	SeG2 at 250 mg/kg animal BW
	5	SeG3 at 500 mg/kg animal BW
Diabetic	6	Sterile normal saline
	7	Glibenclamide at 600 µg/kg animal BW
	8	NG at 500 mg/kg animal BW
	9	SeG1 at 125 mg/kg animal BW
	10	SeG2 at 250 mg/kg animal BW
	11	SeG3 at 500 mg/kg animal BW

• NR- Non Diabetic Rat, DR - Diabetic Rat, NG - Normal garlic leaf extract (control plants), SeG - Se treated garlic leaf extract (treated garlic with 2 mg/L Se), Gli - Glibenclamide, BW - Body weight. • Each bar and value represent mean±*sem* (n=6). Means are represented with alphabets highlighting one-way or two-way ANOVA. Means with similar alphabets in each column (in tables) are not significantly different at $p \leq 0.05$ (level of significance) calculated by Dunnett's or Tukey's post hoc test for multiple comparisons of means after ANOVA analysis as applicable. • Group 6 (diabetic control) was compared with group 1 (non-diabetic control), represented by the asterisk symbol '*'. Groups 3, 4, and 5 (non-diabetic-Se-garlic) were compared with groups 1 (normal control) and 2 (non-diabetic- normal garlic) represented by asterisk symbols '*' and alphabet 'e', respectively. • Groups 7 (diabetic- std. drug), 8 (diabetic- untreated; control plant group), 9, 10, and 11 (diabetic- Se-garlic) were compared with group 6 (diabetic control) represented by octothorpe symbol '**#**'. • Group 8 (diabetic- non-Se-treated garlic), 9, 10, and 11 (diabetic- Se-garlic) were compared with group 7 (diabetic- std. drug) represented by alphabet 'a'. • Groups 9, 10, and 11 (diabetic- Se-garlic) were also compared with group 8 (diabetic- non-Se-treated garlic) represented by the alphabet 'b'. • Groups 9, 10, and 11 (Se-garlic) were also compared among each other and were represented by alphabets 'c, d' for groups 9 and 10 respectively, wherever necessary. The criteria for statistical significance were $p < 0.05$ and is represented as a single symbol "*, #, a, b, c, d, e" = $p < 0.05$ and as a double- or upper-case symbol "**, ##, A, B, C, D, E" = $p < 0.02$.

for the quantification of serum proteins.[38] Serum albumin was quantified by using the Bromocresol Green (BCG) Dye method by Doumas and Peters, 2009.[39] The total globulin concentration in the serum was calculated by subtracting total serum albumin from total serum protein. Serum bilirubin was quantified by using diazo method by Pearlman and Lee, 1974.[40] Estimation of glycogen in the liver and skeletal muscle tissue was done following the method of Good *et al.*, 1993[41] and Carroll *et al.*, 1996.[42]

Statistical analysis

Statistical analyses were made using the software SPSS version 22 and GraphPad Prism 8 for windows. One way-ANOVA and Two way-ANOVA (as required) were performed to compare the means as required and multiple comparisons among the different groups were made by the Post hoc Tukey's or Dunnett's test. All results were expressed as Mean ± SEM. The values were considered statistically significant when $p<0.05$.

RESULTS

FBG and serum glucose level

The diabetic group 6 (DR-C) showed a continuous increase in the FBG every week which was the maximum increase among the diabetic groups (6-11) and raised up to 18.45% from the start. In the third week, sparing group 11 (SeG 3) all other treatment groups (7-10) showed a significant decrease in FBG compared to the control group; DR-C, which were 22.16%, 19.88%, 19.58%, and 24.26%, respectively. Furthermore, a significant decrease in NG and SeG groups 8 to 10 was observed from the third week when compared to the Gli group 7, which showed a final decrease of 24.26%. Group 10 fed with SeG2 showed a maximum reduction in FBG compared to all the diabetic groups (Figure 1). There was no significant reduction observed in the highest dose SeG3 which showed a constant increase of up to 12.05% from the starting week.

The diabetic groups animals showed significantly high serum glucose levels than the NR-C Group (Table 2). The diabetic

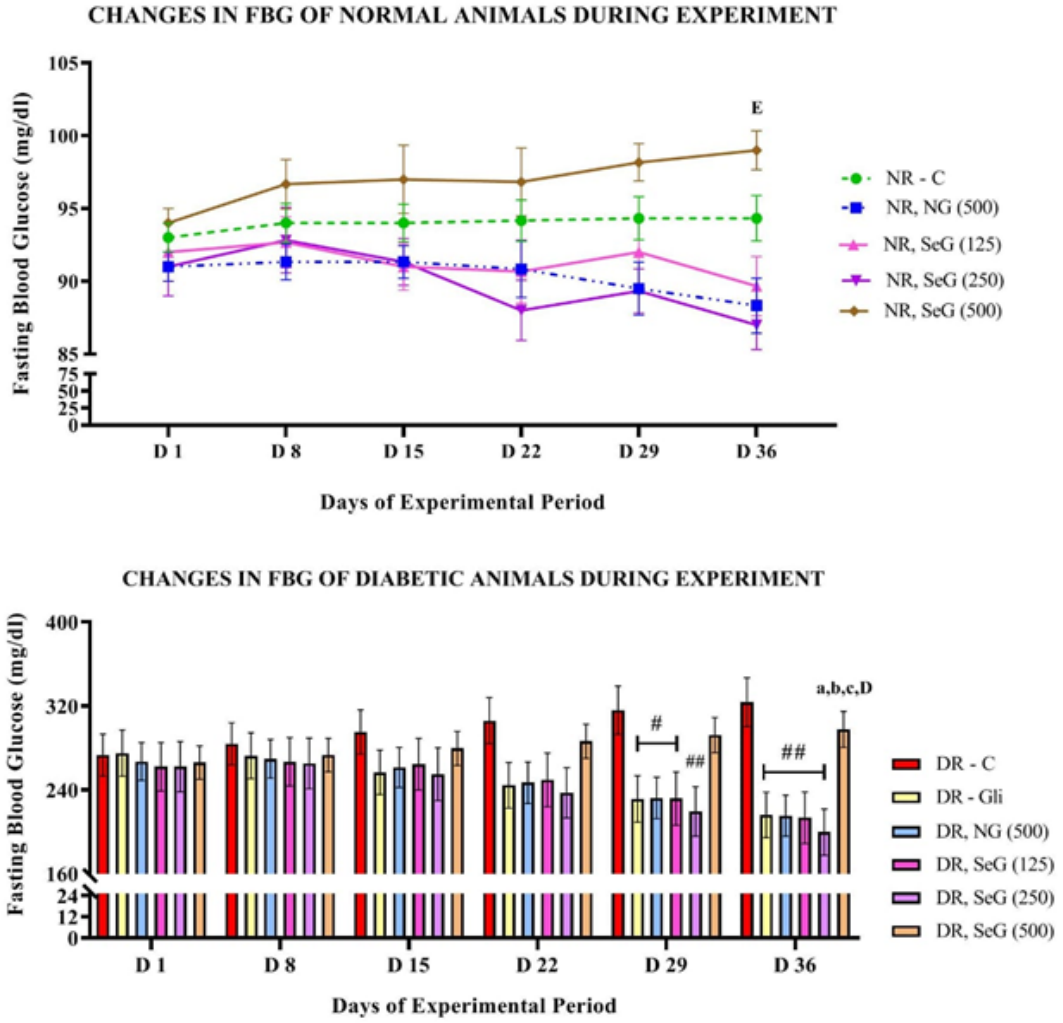

Figure 1: Effect of normal and Se-treated garlic extract on fasting blood glucose (FBG) levels of normal animals (upper panel) and diabetic animals (lower panel) during 5 weeks of treatment period. DR, SeG (125); DR, SeG (250); DR, SeG (500) represent SeG1, SeG2 and SeG3 groups, respectively.

Table 2: Effect of normal and Se-treated garlic extract on the serum glucose, Triglycerides (TAG), Total Cholesterol (Tot CHO), HDL (High-Density Lipoprotein), LDL (Low-Density Lipoprotein) and VLDL (Very Low-Density Lipoprotein) content of animals after 35 days of treatment.

Grp. No.	Type of Animal, Material and Dose of treatment	Serum glucose (mg/dL)	Triglycerides (TAG) (mg/dL)	Total Cholesterol (mg/dL)	HDL-Cholesterol (mg/dL)	LDL (mg/dL)	VLDL (mg/dL)
1	NR-C (NaCl)	100.8±1.11	101.5±2.97	98.33±1.38	31.54±1.08	46.5±0.79	20.29±0.59
2	NR-NG (500 mg/kg BW)	98.83±1.8	97.26±1.67	95.86±0.51	31.25±0.49	45.15±0.61	19.45±0.33
3	NR-SeG1 (125 mg/kg BW)	99±2	95.4±2	96.04±0.25	33.7±1.07	43.26±1.13	19.08±0.4
4	NR-SeG2 (250 mg/kg BW)	96.83±1.94	95.73±1.42	95.56±0.43	31.48±0.46	44.94±1.02	19.15±0.28
5	NR-SeG3 (500 mg/kg BW)	107.5±2.43	106.7±1.95^{e}	98.03±0.71	28.91±0.39	47.78±0.62	21.33±0.39^{e}
6	DR-C (NaCl)	354.8±27.15**	142.5±2.02**	123±1.17**	22.09±0.37**	72.47±1.13**	28.49±0.4**
7	DR-Gli (600 µg/kg BW)	226.7±24.84**##	120.3±1.89**##	112±1.95**##	25.12±0.39**#	62.82±1.77**##	24.06±0.38**##
8	DR-NG (500 mg/kg BW)	228.7±22.74**##	121.5±1.52**##	114.9±2.06**##	26.99±0.4**##	63.66±2.11**##	24.3±0.3**##
9	DR-SeG1 (125 mg/kg BW)	225.4±28.31**##	119.8±1.6**##	114.3±1.17**##	25.9±0.65**##	64.4±1.46**##	23.96±0.32**##
10	DR-SeG2 (250 mg/kg BW)	209.3±25.05**##	115.9±1.17**##	108.8±1.3**##	26.67±0.44**##	58.93±1.37**##	23.17±0.23**##
11	DR-SeG3 (500 mg/kg BW)	324.8±19.82**abcD	130.7±1.58**##AbCD	118.5±1.05**abD	22.73±0.63**BcD	69.67±1.34**Ad	26.15±0.32**##AbCD

control animals (DR-C) showed highest serum glucose which was, averagely 354.8 mg/dl ±27.15. Compared to DR-C group the other DR groups 7 to 10 showed a significant decrease of 36.10%, 35.54%, 36.47 %, and 41.0 %, respectively. Group 11 fed with SeG3 showed no significant difference in serum glucose with the DR-C control group and was significantly higher than that of groups 7 to 10. Group 10 animals fed with SeG2 showed a maximum reduction in serum glucose with a final average of 209.3 mg/dl ±25.05 among the entire diabetic group (Table 2).

Effects on Lipid Profile-Triglycerides (TAG), Total Cholesterol (Tot CHO), Low-Density Lipoprotein (LDL), Very Low-Density Lipoprotein (VLDL) and High-Density Lipoprotein (HDL)

Compared to the DR-C (group 6), the diabetic group 10 fed with SeG2 showed the highest significant decrease (P≤0.02) in TAG, Tot CHO, LDL, and VLDL (18.7%, 11.5%, 18.7%, and 18.5 %, respectively). The lowest significant increase (P≤0.02) in HDL levels (2.8%) was seen in SeG3 group (group 11). Groups 7 to 9 showed comparable results to group 10 and there were no significant differences in all parameters among them, however SeG2 (group 10) showed the highest control over STZ-induced hyperlipidemic conditions in the diabetic animals in comparison to the DR-Gli, DR-NG and DR-SeG1 which was approximately uniform for most of the lipid parameters (Table 2).

Effects on Liver Functions

Liver enzymes and bilirubin level

Among the groups 6-11, the least increased levels of Alkaline phosphatase (ALP), Aspartate aminotransferase (AST), and Alanine aminotransferase/transaminase (ALT) (27.2%, 21.8%, and 27.2 % respectively) was seen in the group 10 which was highly significant (P≤0.02) compared to the DR-C group. Groups 7–9, also showed comparable results as that of group 10 and there was no significant difference in all parameters among them (Table 3). However, group 11 failed to show significant decrease in the

serum enzymes as compared to DR-C group. Compared to the NR-C group, the diabetic groups (6-11), except group 10, showed a significant increase ($p \leq 0.02$ and 0.05) of 57.6% (highest), 27.3%, 27.3%, 27.3%, 15.15% (lowest, not significant) and 42.4%, respectively, in the Direct (D)-bilirubin levels. Similarly, compared to the NR-C group, diabetic groups (6-11) showed an increase of 64%, 22.2%, 33.3%, 22.2% (lowest, not significant), 22.2% (lowest, not significant) and 38.9% respectively, in the Indirect (I)-bilirubin levels. The maximum reduction in the levels of D- and I-bilirubin (26.9% and 25.4%) was seen in SeG2 group (group 10) which was highly significant ($p \leq 0.02$) when compared to the DR-C group. The other test groups (groups 7-9) also showed comparable results as group 10 and there were no significant differences in D- and I-bilirubin levels among them (Table 3).

Total protein, albumin, globulin content

Compared to the NR-C group the animals of DR groups 6-11 showed statistically significant ($p \leq 0.02$) decrease in the levels of total protein and DR groups 7-9 showed statistically significant decrease ($p \leq 0.02$) in albumin content. However, compared to the NR-C group, no significant change was observed in the globulin content and A/G ratio of the DR groups 7-10. When compared to the DR-C group, the DR groups 7-10 showed statistically significant ($p \leq 0.02$) increase in total protein, albumin and globulin content but the highest comparable increase of 39%, 70% and 19.2%, respectively, was observed in group 10 rats fed with SeG2 extract. Except group 11, all other DR groups 7-10 showed no statistically significant difference among each other. Hence, SeG2 showed slightly better control over STZ-induced hypo-proteinemia and hypo-albuminemia content in diabetic animals compared to DR-NG. DR-Gli, DR-SeG1 extracts. Group 11 however, showed a significant decrease in all the parameters compared to normal rats but with least improvements (Table 4).

Liver and muscle glycogen content

Compared to the NR-C animals, all the diabetic groups 6-11 showed reduced levels of glycogen content in liver and muscle. In the liver, group 6 to11 showed highly significant ($P \leq 0.02$) decrease in the total glycogen content by 56.5% (highest), 34.6%, 30.8%, 30.4%, 20.7 (least), and 46.4%, respectively. And in muscle, groups 6 to11, showed significant decrease by 65.5% (highest), 32.6%, 25.5%, 31.6%, 16.4% (least, insignificant) and 44.6%, respectively, when compared with the NR-C group. Compared to the DR-C group, a statistically significant rise was observed in the groups 7 to 10. The maximum increase in liver glycogen (82.23%) and in muscle glycogen (142%) was observed in group 10 (SeG 2). Groups 7 to 9 did not show any significant difference compared to the group 10. Group 11 animals showed the least increase of 23.25% and 60% in liver and muscle, respectively, compared to the DR-C group (Table 4).

DISCUSSION

Modern research has also advocated anti-diabetic, anti-hyperlipidemic, hepato-protective, reno-protective, and anti-oxidative properties of almost all parts of garlic. In the

Table 3: Effect of normal and SeG extract on the liver enzymes (ALP, AST, ALT) and bilirubin content of animals after 35 days of treatment.

Grp. No.	Type of Animal, Material and Dose of treatment	ALP (IU/L)	AST (IU/L)	ALT (IU/L)	Direct Bilirubin (mg/dL)	Indirect Bilirubin (mg/dL)
1	NR-C (NaCl)	96.57±1.93	102.7±2.33	85.92±1.43	0.33±0	0.36±0.01
2	NR-NG (500 mg/kg BW)	94.5±2.54	100.4±1.04	83.83±2.79	0.32±0.01	0.37±0.01
3	NR-SeG1 (125 mg/kg BW)	93.67±3.16	101±2.98	81.33±4.36	0.31±0.01	0.39±0.01
4	NR-SeG2 (250 mg/kg BW)	96.9±3.26	104±2.63	83±2.62	0.32±0.01	0.38±0.02
5	NR-SeG3 (500 mg/kg BW)	105.3±1.33	112±2.73	94.5±2.41	0.34±0.01	0.39±0.01
6	DR-C (NaCl)	161.6±4.64**	173±4.45**	159.5±2.49**	0.52±0.01**	0.59±0.04**
7	DR- Gli (600µg/kg BW)	122.7±1.94**##	136.2±1.71**##	121.2±2.11**##	0.42±0.01**##	0.44±0.01##
8	DR-NG (500 mg/kg BW)	118.8±2.34**##	135.7±5.84**##	117.2±4.77**##	0.42±0.01**##	0.48±0.04**##
9	DR-SeG1 (125 mg/kg BW)	120.7±3.12**##	139.8±4.74**##	121.1±2.92**##	0.42±0**##	0.44±0.01##
10	DR-SeG2 (250 mg/kg BW)	117.6±2.29**##	135.3±1.74**##	116±3.18**##	0.38±0.02##	0.44±0.01##
11	DR-SeG3 (500 mg/kg BW)	143.8±4.22**##ABCD	159.3±4.14**ABCD	135 ±4**##BD	0.47±0.02**abD	0.5±0.02**#

Table 4: Effect of normal and SeG extract on serum total protein, albumin, globulin, and tissue glycogen contents of animals after 35 days of treatment.

Grp. No.	Type of Animal, Material and Dose of treatment	Total Protein (g/dL)	Albumin (g/dL)	Globulin (g/dL)	Albumin/Globulin (A/G) ratio	Glycogen (mg/g WW)	
						Liver tissue	Muscle tissue
1	NR-C (NaCl)	7.02±0.02	3.38±0.04	3.65±0.04	0.93±0.02	59.13±1.9	4.75±0.25
2	NR-NG (500 mg/kg BW)	7.03±0.05	3.39±0.03	3.64±0.05	0.93±0.02	63.68±2.57	4.73±0.32
3	NR-SeG1 (125 mg/kg BW)	7.02±0.04	3.45±0.07	3.58±0.07	0.97±0.04	61.25±3.42	4.68±0.36
4	NR-SeG2 (250 mg/kg BW)	7.1±0.03	3.5±0.04	3.6±0.06	0.97±0.03	64.95±2.65	4.68±0.24
5	NR-SeG3 (500 mg/kg BW)	6.46±0.02^{**E}	2.98±0.08^{*e}	3.49±0.09	0.86±0.05	54.45±3.38	4.5±0.16
6	DR-C (NaCl)	4.68±0.21**	1.81±0.14**	2.87±0.11**	0.63±0.04**	25.72±0.83 **	1.64±0.08 **
7	DR-Gli (600 µg/kg BW)	6.3±0.1$^{**\#\#}$	2.96±0.09$^{**\#\#}$	3.61±0.16$^{\#\#}$	0.9±0.06$^{\#\#}$	38.62±1.86 $^{**\#\#}$	3.2±0.08 $^{**\#\#}$
8	DR-NG (500 mg/kg BW)	6.39±0.04$^{**\#\#}$	3.01±0.08$^{*\#\#}$	3.38±0.08$^{\#\#}$	0.9±0.04$^{\#\#}$	40.93±1.34 $^{**\#\#}$	3.54±0.18 $^{*\#\#}$
9	DR-SeG1 (125 mg/kg BW)	6.46±0.05$^{**\#\#}$	3±0.05$^{**\#\#}$	3.46±0.04$^{\#\#}$	0.87±0.02$^{\#\#}$	41.13±2.18 $^{**\#\#}$	3.25±0.22 $^{**\#\#}$
10	DR-SeG2 (250 mg/kg BW)	6.51±0.05$^{**\#\#}$	3.09±0.06$^{\#\#}$	3.42±0.03$^{\#\#}$	0.91±0.02$^{\#\#}$	46.87±1.77 $^{*\#\#}$	3.97±0.15 $^{\#\#}$
11	DR-SeG3 (500 mg/kg BW)	5.78±014$^{**\#\#ABCD}$	2.33±0.08^{**ABCD}	3.45±0.11$^{\#\#}$	0.68±0.03^{**ABcD}	31.72±1.74 ^{**D}	2.63±0.25 ^{**D}

presented study, it was seen that there was a reduction of FBG by ~3% in the garlic extracts fed normal rats. This observation was in accordance with the reports of Eidi *et al*., 2006.[26] When compared to DR-C, the Gli and NG, SeG1 and SeG2 groups showed a tremendous decrease in the FBG and serum glucose, indicating that the leaf extracts of SeG extract in a dose of 250 mg/kg BW produces a maximum reduction in blood glucose and hence, best control over the diseased condition.

DM is characterized by disrupted lipid metabolism, various hyperlipidemic conditions are characterized by increased concentrations of triglycerides, LDL, VLDL and low HDL.[43] The diabetic groups showed increased lipid parameters and reduced HDL content in serum when compared to the NR-C animals but in comparison to the DR-C animals, the Gli and NG, SeG1, and SeG2 fed rats showed a tremendous decrease in all of the lipid parameters and an increase in HDL. Although the difference between the Gli and NG treatments was not statistically significant, this decrease was clinically significant because it showed better results than the standard medication and normal garlic extract, which is a sign of a rapid recovery, suggestive of the fact that SeG1 and SeG2 extract when administered for an extended period of time, can produce better control over glycemic and hyperlipidemic situations at partial doses than that of the regular garlic extract.

Commonly investigated enzymes in Liver Function Test (LFT) are Serum Aspartate Aminotransferases/ Serum Glutamic-Oxaloacetic Transaminase and Alanine Aminotransferases/Serum Glutamic Pyruvic Transaminase (AST/SGOT and ALT/SGPT) enzymes. ALT and AST determine the concentration of hepatic enzymes having a low threshold in blood, increased concentrations of these enzymes serve as markers of hepatic cellular damage. Bilirubin and ALP act as the major markers of cholestasis and biliary functions. In our study, the diabetic rats treated with garlic extract showed a restored effect in the enzymes similar to that of the drug indicating the potential of garlic to reduce the ill effects of diabetes on the liver.

The oral administration of Gli, and garlic extracts (NG, SeG1 and SeG2) showed close to restored effects in the serum proteins, albumin levels, and Albumin/Globulin (A/G) ratio. Data obtained for the liver indices demonstrated the absence of significant damage to liver attributed to the SeG1 and SeG2 extract treatment of diabetic rats. Total albumin and protein concentrations and A/G ratio were found to be nearly restored in the serum of diabetic rats fed by three garlic extracts and Gli which may be due to the inhibition of proteolytic degradation because of increased secretion of insulin and efficient blood glucose utilization. Similar results have been put forward by previous studies as well.[44,45] The STZ induces diabetes by targeting pancreatic beta cells producing

insulin. The bulk loss and destruction of these cells results in the oxidative stress and reduced antioxidant defense which further leads to the reduction in insulin and more damage of beta cells. This results in hyperglycemia which causes complications in other organs.[46] One of the complications caused by hyperglycemia is the non-enzymatic glycation of proteins and albumin. The glycated albumin elicits the immunological response which may further reduce the level of albumin.[47] Independent studies have suggested that garlic possesses anti-glycation properties that may contribute to the reduced glycation and increased albumin and protein levels in diabetic rats.[48,49] The glycation is followed by the ROS production that results in the diabetes complications. The ROS affects almost all of the components of a cell; they affect the protein via different ways which makes them more susceptible to proteolysis, resulting in a reduced protein level in diabetic patients. In the case of garlic-treated diabetic rats, the antioxidant compounds of garlic- quercetin, allicin and sulphur-containing compound must have counterbalanced the ROS and consequently restored level of albumin and other proteins. Apart from this study, various clinical studies suggest that the exogenous supply of antioxidants to the diabetic organisms can attenuate the ROS induced-damage.[50] It has also been suggested that garlic exerts hypoglycemic effect as a result of allyl propyl disulfide or diallyl disulfide action, and affects purine metabolism.[51] Another reason for decreased albumin in diabetic rats is kidney damage which leads to the leakage and causes albuminuria. A study showed that garlic reduces kidney damage and helps rats to retain more albumin.[52] One study suggested that garlic has the ability to stimulate the regeneration of hepatic tissue, which increases protein synthesis in damaged liver, improves the functional status of the liver cells and prevents protein oxidation.[53] In addition, it is believed that garlic affects whole body protein metabolism through hormonal regulation by stimulating adrenaline and nor-adrenaline hormone secretion. Treatment with garlic normalized the total proteins by stimulating protein synthesis, contributing to an improved hepatoprotective mechanism, and accelerating the regeneration process of liver cells.[54]

Our study also demonstrated the significant reduction in serum uric acid, BUN, and creatinine in the diabetic rats treated with Gli and garlic extracts (NG and SeG1 and SeG2) compared to the DR-C group (data not shown). Liver and skeletal muscle glycogen content has been observed to fall remarkably in diabetic rats to ~3/4th of their basic levels.[55] Garlic and its formulations have been shown to promote glucose utilization by significantly enhancing insulin sensitivity and secretion, and glucose tolerance in skeletal muscles. In our study, we observed a significant decrease ($p \leq 0.05$) in the glycogen content of liver and muscle tissues in the diabetic control, DR-C rats. The treatment with the garlic extracts (NG and SeG1 andSeG2) including Gli, showed a significant decrease in glycogen content compared to the NR-C but increased compared to the DR-C group. This indicates their restricted degradation effects on glycogen in the rats of those groups.

CONCLUSION

Various researches showed that Selenium and *Allium sativum* have antidiabetic potential, indicating Se-laden garlic to be an effective treatment. This research was undertaken to compare the effects of control (NG) and SeG plant extracts on the biochemical parameters in the chemically induced diabetic animal models, hence comprehending the anti-diabetic activity. The results in this pre-clinical study are suggestive of better antidiabetic potential of leaf aqueous extracts of SeG. Future elaborations on SeG at the molecular levels may give clearer insights about the molecular mechanisms underlying such pre-clinical observations. Furthermore, the use of commercially acknowledged parts of the garlic plants i.e. bulbs also need to be researched upon.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ETHICAL STATEMENT

The protocol was approved by the CPCSEA and Institutional Ethics Committee for Animal Research of the University (1173/PO/Re/S/08/CPCSEA).

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