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EFFECT OF PHYTOSTEROL EXTRACT FROM SESAME SEED ON EXPERIMENTALLY INDUCED HYPERLIPIDEMIC RATS: DOSE DEPENDENT STUDY

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ABSTRACT

Hyperlipidimia is a medical condition where there is an elevation of lipids, or fats, in the blood. The purpose of the study was to evaluate the hypolipidemic effect of sesame seed on experimentally induced hyperlipidemic albino rats. The study was carried out with thirty *Wistar* stain albino male rats and the groups were Group I was denoted as control, they were provided normal food, water with 1g multi-vitamin for 60 days. Group II was denoted as hyperlipidemic, from 1st to 14th day they were provided normal food, water and on the 15th to 60th day (45 days) they were provided normal food with 3.7 ml coconut oil with 1mg/kg cholesterol per rat, 1g multi-vitamin, and 5% sucrose for induction of hyperlipidemia. Group III, Group IV, Group V were denoted as treatment I, I, III respectively and they were treated as group II rats and fed orally sesame seed extract at the dose of 25, 50 and 75 mg/kg body weight/day/rat orally for 45 days respectively. The result of the present study showed that the toxicity level and total fat content of liver, intestine, and adipose tissue were significantly decreased and the antioxidant enzyme profiles were significantly increased in treatment I, II and III groups than the hyperlipidemic group. Sesame seed (*Sesamum indicum*) is the rich natural sources of phytostreol which have great reducing capability for lowering the blood cholesterol, triglyceride level and total fat content of the different tissues.

KEY WORDS: ***Phytosterol, sesame, hypercholesterolemia, hyperlipidimia.***

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INTRODUCTION

Hyperlipidemia and related cardio vascular diseases are now the first leading cause of death and World Health Organization (WHO) estimated that over 23 million people will die from cardiovascular diseases before 2030[1]**.** Prevention and management of this disease is now first priority of the research field among scientist in the world. NCEPP (National Cholesterol Education Prevention Programme) recommended 2 g plant sterol in the daily diet reduces the plasma cholesterol by 10 % in one month[2]. But normal daily Indian diet containing plantsterol not more than 200 mg/ day i.e. 425 tomatoes, 210 carrots, 150 apples, 83 oranges contain 2 g plant sterol, 100 g sunflower oil contain about 1.5 g plant sterol, which is impossible to consume daily. So, plant sterol extraction from some selected foods and supplements with diet or a combination of plant sterol rich food in daily menu is beneficial for hypercholesterolimic patients and also prevent progression of increasing plasma cholesterol. Phytosterols or plant sterols have a lipid lowering effect[3] and also reducing the low density lipoprotein-cholesterol[4]**. S**esame seed (*Sesamum indicum*)[5] is the rich natural sources of phytostreol. So, we selected sesame seed for its high plant sterol contents, *Sesamum indicum* L., is an ancient oil crop supplying seeds for confectionery purposes, edible oil, paste (tahini), cake and flour. Sesame is a broadleaf plant that grows about 5 to 6 feet tall, height dependent on the variety and growing conditions[6]. Sesame seed contains phytosterol, moisture, crude oil, crude proteins, carbohydrates, crude fiber, and ash. Sesame seeds also rich sources of mono unsaturated, poly unsaturated fatty acids[7] and tocopherols[8]**.** The present study is aimed to evaluate the potential effect of sesame seed on experimentally induced hyperlipidemic rats. Aim of the project work is to find out the anti-hyperlipidemic effect of sesame seed and the threshold level or dose of treatment on experimental induced hyperlipidemia in male albino rats.

Acute toxicity study of ESS

An acute toxicity of ESS was conducted using acute toxic class method as per Organization of Economic Co-operation and Development (OECD) guidelines 425 (OECD, 2001) where the limit dose of 2000 mg/kg body weight was used. Twelve healthy Wister strain rats (n=6) of either sex selected by random sampling technique were employed in this study. Wellness parameters of animals were made and recorded systematically 30 min, 4 hour, 24 hour and 48 hours after dose administration for skin and fur, eyes, mucus membrane, behavioral pattern changes, tremor, convulsions, salivations, diarrhea, lethargy, sleep and mortality.

Selection of dose of ESS

Healthy *Wistar* strain rats were selected in this study and minimum dose of ESS fed was 25mg/ kg body weight (2000mg per 70 kg healthy adult person) [2]. Another two doses were chosen double (50 mg/kg body weight/day/rat) and triple (75 mg/kg body weight/day/rat) of the minimum dose according to the OECD 425.

Selection of animals and care (Experimental subjects)

The present experiment was conducted on 30 male *Wistar* strain adult pathogen free, healthy albino rats having weight of 100±15 g (Supplied from Ghosh animal, animal foods and animal cages Supplier, Kolkata 54). They were housed at laboratory condition for 2 weeks prior to experimentation. Animals were housed three rats/cage in a temperature-controlled room (22± 20C) with 12–12 h dark–light cycles (8.00–20.00 h light, 20.00– 8.00 h dark) at a humidity of 50 ±10%. They were provided with standard food and water *ad libitum*. Animal care was provided according to the Guiding Principle for the Care and Use of Animals (NIH, 1985)[9]**.** Our Institutional Animal Ethical Committee (IAEC) approved this study[10]**.**

MATERIALS AND METHODS

Plant Material (Preparation for sesame seed)

The sesame seed was purchased from nearest market (raja bazaar), paschim medinipur district of West Bengal. Seeds were dried at 40 ± 1°C in incubator. Then dried seeds were crushed in an electric grinder machine. Then the seeds were allowed to stand at room temperature for 24 hours. After that the fine dusts of sesame seeds were ready to prepare the extract as per requirement.

Preparation Hexane extracts

The dried sesame seed dust was dissolved in hexane and then placed it in shaker cum incubator at 37°C for 24 hours. Then it was filtered by whatman filter paper and evaporated by rotary evaporator and collect the hexane extract. Hexane extract was separated with hexane: ethanol-methanol (1:1) and hexane fraction was named as ESS (Sesame Seed Extract).

Experimental Models

We were tried to establish hyperlipidemia by dietary modification (high cholesterol diet). Grouping animal and experimental procedure**:** Total 30 rats were selected. Each group contains 6 rats and they were grouped as follows: Group I (control): six animals were subjected to control groups. They feed normal diet and water with multivitamin for 60 days. Group II (hyperlipidemic): Six animals were subjected to hyperlipidemic group. They were provided from 1st to 14th day normal food, water and on the 15th to 60th day (60 days) they were provided normal food with 3.7 ml coconut oil with 1mg/kg cholesterol per rat, 1g multi-vitamin, and 5% sucrose for 45 days to achieve hyperlipidemia. Group III or treatment I (sesame seed extract): Six animals were subjected to ESS I group. They were treated as group II rats and co administered with sesame seed extract (ESS) at 25mg/kg body weight/day for 45 days. Group IV or treatment II (sesame seed extract): Six animals were subjected to ESS II group. They were treated as group II rats and coadministered with sesame seed extract (ESS) at 50mg/kg body weight/day for 45 days. Group V or treatment III (sesame seed extract): Six animals were subjected to ESS III group. They were treated as group II rats and co administered with sesame seed extract (ESS) at 75mg/kg body weight/day for 45 days.

Table 1
Preparation of food mix of different groups of animal

Food	Standard diet (n=6)	Hyperlipidemic diet (n=6)	Hyperlipidemic diet with sesame seed extract-I (n=6)	Hyperlipidemic diet with sesame seed extract-II (n=6)	Hyperlipidemic diet with sesame seed extract-III (n=6)
Atta (g)	66.5	45.43	45.43	45.43	45.43
Chatu (g)	19	12.98	12.98	12.98	12.98
Milk powder (g)	9.5	6.49	6.49	6.49	6.49
Sucrose (g)	-	5	5	5	5
Coconut oil (ml)	-	22.2	22.2	22.2	22.2
Cholesterol (g)	-	0.9	0.9	0.9	0.9
Salt (g)	6.0	6.0	6.0	6.0	6.0
Vitamin (g)	1.0	1.0	1.0	1.0	1.0
Total	100	100	100	100	100
Sesame seed extract (mg/kg body weight/rat)	-	-	25	50	75
Olive oil (ml)	-	-	0.5	0.5	0.5

Source of chemicals
Major biochemical parameters were measured by Semiautoanalyser (Mearck, Microlab 150), so, diagnostic kits like Urea, creatinine, Total cholesterol, HDL cholesterol, triglycerides, SGOT, SGPT, ALP supplied by Merck Ltd., Mumbai, India. Flavonoids like Querecetin and sodium galate were purchased from Sigma-Aldrich, India. All other chemicals were purchased from SRL, India and MERCK, India. s d FINE-CHEM LIMITED, India, HiMedia Laboratories Pvt. Ltd. Mumbai, India and Crest Biosystems Goa, India.

Preparations of sample for biochemical studies
This experimental design was continued for 60th days. On 61th day of the experiment, the animals were sacrificed and blood was collected from the aorta after which the liver was collected for different biochemical analysis.

Biochemical estimation of plasma lipid profile
Blood samples were collected from each rat. Prior to analysis, calibration of the analyzer for plasma triglyceride (Tg)[11], total cholesterol (TC) [12], high density lipoprotein cholesterol (HDL-c) [13], and low density lipoprotein cholesterol (LDL-c) [14] were done by semiautoanalyzer according to the standard method and the values are expressed as mg/dl.

Biochemical estimation of antioxidant enzyme profiles in plasma
The whole blood was centrifuged and plasma fraction was separated. The Superoxide dismutase (SOD) activity of plasma will be estimated by measuring the percentage of inhibition of the pyragallol auto-oxidation by SOD using spectrophotometer at 420 nm and values are expressed as nmol of H_2O_2 consumption/dl of plasma/min[15]. For the estimation of catalase (CAT) activity, in a spectrophotometric cuvette, 0.5 ml of hydrogen peroxide (H_2O_2) and 2.5 ml of distilled water will be mixed and reading of absorbance will be noted at 240 nm and plasma will be added at volume of 40µl separately the subsequent six reading will be noted at 30 sec. interval[16] and values are expressed as nmol of H_2O_2 consumption/dl of plasma/min.

Biochemical estimation of lipid peroxidation marker in plasma and liver tissue
The level of malondialdehyde (MDA) in plasma and liver tissue were measured by spectrophotometer at 535 nm and values are expressed as nM/ml of plasma or nM/mg of tissue[11].

Biochemical estimation of toxicity profile marker of liver tissue
Liver tissues were homogenized separately in 0.05 M Tris Hydrochloric acid (HCl) buffer solution (pH-7.0) at the tissue concentration of 50 mg/ml. These homogenate will be centrifuged separately at 10,000 g at 4^{o}C for 10 min and tissue supernatant was collected. Activities of serum liver enzymes Glutamic Oxalic Transaminase (GOT), Glutamate Pyruvate Transaminase (GPT) and alkaline phosphatase (ALP) were chemically determined[17] in liver tissue homogenate by spectrophotometer[18].

Histopathological analysis of liver tissue
Liver tissues from the experimental rats were fixed in 10% buffered formaline solution embedded in paraffin wax and five-micron sections were prepared with a rotary microtome. These thin sections were stained with hematoxylin and eosin (H and E), mounted on glass slides and observed for pathological changes under a

binocular microscope according to Mani, 2010[19, 20].

(i) *Statistical analysis*

Data are expressed as mean ± SE (n=6). ANOVA followed by Bonferroni t-test to detect inter group differences multiple two-tail t-test and bars differ from each other significantly ($p< 0.05$)[21].

RESULTS

Table 1

Represents the effect of ESS at three different doses (25, 50 and 75 mg/kg body weight/day) on changes of body weight and fat content of the liver, intestine, adipose tissue of experimentally induced hyperlipidemic albino male rats. The data were presented as mean ± SE and evaluated by One-way ANOVA followed by Bonferroni t-test to detect inter group differences. Differences were considered to be statistically significant if p<0.05.

Group	Body Weight (g)		Percentage of body weight (g) [Increase]	Fat Content (gm %)		
	Initial	Final		Liver	Intestine	Adipose tissue
Group I (Control)	98.72	134.18	35.92%	8.00±0.005 [a]	3.6±0.003 [a]	12±0.019 [a]
Group II (Hyperlipidemic)	109.62	162.2	47.24%	15.6±0.065 [b]	10±0.025 [b]	50±0.022 [b]
Group III or Treatment I (ESS)	103.57	141.07	36.55%	10.4±0.058 [c]	5.8±0.004 [c]	23±0.024 [c]
Group IV or Treatment-II (ESS)	104.72	139.45	33.16%	8.6±0.046 [a]	3.9±0.01 [a]	13.0±0.028 [a]
Group V or Treatment III (ESS)	108.17	147.2	36.08%	11.2±0.078 [c]	5.39±0.008 [c]	15±0.030 [a]

Data are expressed as Mean ± SE (n=6). ANOVA followed by multiple two-tail t-test and data with different superscripts (a, b and c) in a specific vertical column differ from each other significantly (*P*< 0.05). Group-I Untreated control; Group-II Hyperlipidemic (1mg cholesterol/rat/kg of body weight/day for 45 days); Group-III, IV, V ESS (Sesame seed extract) at three different doses (25, 50 and 75 mg/kg body weight/day respectively for 30 days).

Table 2

Represents the effect of ESS at three different doses (25, 50 and 75 mg/kg body weight/day) on lipid profile on cholesterol induced hyperlipidemic rats. The data were presented as mean ± SE and evaluated by One-way ANOVA followed by Bonferroni t-test to detect inter group differences. Differences were considered to be statistically significant if p<0.05.

Group	Triglyceride (mg/dl)	Low Density Lipoprotein -c (mg/dl)	High Density Lipoprotein -c (mg/dl)	Total Cholesterol (mg/dl)
Group I (Control)	114.29±2.03 [a]	14.30±1.10 [a]	36.70±2.10 [a]	123±0.19 [a]
Group-II (Hyperlipidemic)	202.00±2.12 [b]	46.90±2.98 [b]	12.20±1.30 [b]	177±0.22 [b]
Group III or Treatment I (ESS)	120.8±1.88 [c]	26.8±1.26 [c]	29.8±1.88 [c]	128±0.36 [a]
Group IV or Treatment II (ESS)	106.4±1.62 [d]	22.4±1.18 [c]	37.5±1.48 [a]	124±0.46 [a]
Group V or Treatment III (ESS)	114.2±2.44 [a]	23.7±2.48 [c]	28.4±1.32 [c]	127±0.49 [a]

Data are expressed as Mean ± SE (n=6). ANOVA followed by multiple two-tail t-test and data with different superscripts (a, b, c, and d) in a specific vertical column differ from each other significantly (*P*< 0.05). Group-I Untreated control; Group-II Hyperlipidemic (1mg cholesterol/rat/kg of body weight/day for 45 days); Group-III, IV, V Sesame seed extract at three different doses (25, 5. and 75 mg/kg body weight/day respectively for 30 days).

Table 3

Represents the effect of ESS at three different doses (25, 50 and 75 mg/kg body wt/day) on toxicity level on cholesterol induced hyperlipidemic rats. The data were presented as mean ± SE and evaluated by One-way ANOVA followed by Bonferroni t-test to detect inter group differences. Differences were considered to be statistically significant if p<0.05.

Group	SGOT (U/L)	SGPT (U/L)	ALP (U/L of plasma)
Group I (Control)	44.425±0.89[a]	48.52±1.45 [a]	116.46±3.46 [a]
Group II (Hyperlipidemic)	83.41±1.71[b]	109.88±4.83 [b]	303.1±19.46 [b]
Group III or Treatment I (ESS)	62.88±0.66 [c]	69.64±1.08 [c]	138.2±5.59 [c]
Group IV or Treatment II (ESS)	54.76±1.16 [d]	53.01±2.74 [d]	121.46±2.46 [d]
Group V or Treatment III (ESS)	57.8±0.69[d]	59.04±1.51 [e]	124.01±7.61 [d]

Data are expressed as Mean ± SE (n=6). ANOVA followed by multiple two-tail t-test and data with different superscripts (a, b, c, d and e) in a specific vertical column differ from each other significantly (*P*< 0.05). Group-I Untreated control; Group-II Hyperlipidemic (1mg cholesterol/rat/kg of body weight/day for 45 days); Group-III, IV, V Sesame seed extract at three different doses (25, 50 and 75 mg/kg body weight/day respectively for 30 days).

Table 4

Represents the effect of ESS at three different doses (25, 50 and 75 mg/kg body wt/day) on antioxidant profile on cholesterol induced hyperlipidemic rats. The data were presented as mean ± SE and evaluated by One-way ANOVA followed by Bonferroni t-test to detect inter group differences. Differences were considered to be statistically significant if p<0.05.

Group	Plasma		Liver	
	Catalase (m mol of H2O2 consumption /dL of Plasma/min)	SOD (m mol of H2O2 consumption /dL of Plasma/min)	Catalase (m mol of H2O2 consumption/mg of tissue/min)	SOD (m mol of H2O2 consumption/mg of tissue/min)
Group I (Control)	0.16±0.05 [a]	0.83±0.05 [a]	0.45±0.03 [a]	1.33±0.08 [a]
Group II (Hyperlipidemic)	0.02±0.04 [b]	0.49±0.04 [b]	0.22±0.07 [b]	0.49±0.08 [b]
Group III or Treatment I (ESS)	0.14±0.06 [a]	0.81±0.06 [a]	0.42±0.06 [a]	1.31±0.05 [a]
Group IV or Treatment II (ESS)	0.18±0.04 [a]	0.90±0.06 [c]	0.54±0.02 [c]	1.46±0.05 [c]
Group V or Treatment III (ESS)	0.15±0.06 [a]	0.78±0.06 [a]	0.44±0.06 [a]	1.28±0.05 [a]

Data are expressed as Mean ± SE (n=6). ANOVA followed by multiple two-tail t-test and data with different superscripts (a, b and c) in a specific vertical column differ from each other significantly (*P*< 0.05). Group-I Untreated control; Group-II Hyperlipidemic (1mg cholesterol/rat/kg of body weight/day for 45 days); Group-III, IV, V Sesame seed extract at three different doses (25, 50 and 75 mg/kg body weight/day respectively for 30 days).

Table 5

Represents the effect of ESS at three different doses (25, 50 and 75 mg/kg body wt/day) on lipid peroxidation level on cholesterol induced hyperlipidemic rats. The data were presented as mean ± SE and evaluated by One-way ANOVA followed by Bonferroni t-test to detect inter group differences. Differences were considered to be statistically significant if p<0.05.

Group	Liver	Plasma
	MDA (n mol/ mg of tissue)	MDA (n mol/ dL of plasma)
Group I (Control)	76.96±0.01 [a]	33.66±0.15 [a]
Group II (Hyperlipidemic)	121.14±0.34 [b]	84.25±0.54 [b]
Group III or Treatment I (ESS)	89.44±0.01 [c]	42.56±0.26 [c]
Group IV or Treatment II (ESS)	71.24±0.04 [a]	32.66±0.23 [a]
Group V or Treatment III (ESS)	79.04±o.o6 [a]	36.56±0.26 [a]

Data are expressed as Mean ± SE (n=6). ANOVA followed by multiple two-tail t-test and data with different superscripts (a, b and c) in a specific vertical column differ from each other significantly (*P*< 0.05). Group-I Untreated control; Group-II Hyperlipidemic (1mg cholesterol/rat/kg of body weight/day for 45 days); Group-III, IV, V Sesame seed extract at three different doses (25, 50 and 75 mg/kg body weight/day respectively for 30 days).

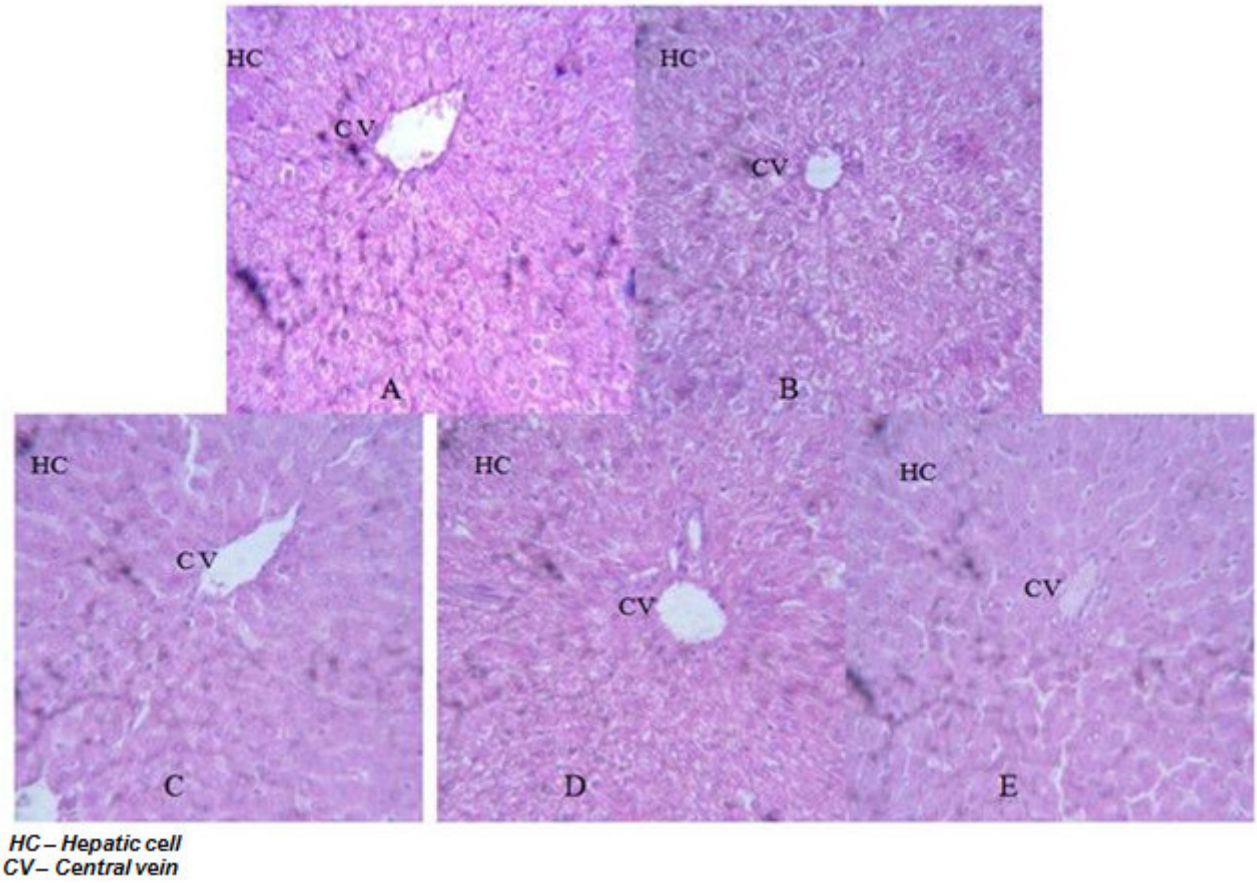

Figure 1

In Figure 1-B, clearly showed the disorganization of hepatic cells and the central vein of liver cell and after treatment by different doses of ESS the cells were well organized like normal liver cells. Histology slides showing liver tissue section (haematoxylin and eosin stained X 400). A: photography of histoarchitecture of liver tissue of control rats as group-I, B: photography of histoarchitecture of liver tissue of hyperlipidemic rats as group-II, C: photography of histoarchitecture of liver tissue of treatment I (ESS) rats as group-III, D: photography of histoarchitecture of liver tissue of treatment II (ESS) rats as group-IV, E: photography of histoarchitecture of liver tissue of treatment III (ESS) rats as group-V.

Changes in body weight and fat content in liver, adipose and intestine

Body weight (g) and liver somatic and adipose tissue decreased significantly ($p< 0.05$) was increased at the time of experiment in group II (hyperlipidemic) compared to the other groups from their initial body weight (Table: 1). In group IV or treatment II (ESS) dose at 50mg/kg body weight/day showed drastically less body weight (g) than the other two groups (group III or treatment-I dose at 25mg/kg body weight/day and group V or treatment III dose at 75 mg/kg body weight/day). The percentage of elevation in body growth was drastically less than the other groups. Liver somatic and adipose tissue decreased significantly ($p< 0.05$) in group IV or treatment II dose at 50mg/kg body weight/day, in comparison to other groups and after administration of the sesame seed extract in group III, IV and V these indices were resettled towards the control level (Table: 1).

Changes in the level of lipid profile

Lipids being insoluble in water need a transport system made up of lipoproteins such as LDL-C, HDL-C etc.Estimation of these lipoproteins was used as an index to measure the levels of lipids present in the plasma. Table 2 shows that the effect of sesame seed extracts (ESS) on lipoprotein (LDL, triglyceride and HDL) of control and experimental animals. Circulating levels of LDL-c was significantly ($p< 0.05$) increased followed by a parallel decrease in HDL-c in different doses of ESS treated rats compared to the control groups. Nevertheless the levels were statistically similar in control and treated with ESS groups. In control rats the level of cholesterol, triglyceride, HDL, LDL was normal and the treated groups of ESS these levels near to the control rat when compared with the control group (Table: 2).

Changes in the toxicity level

The present study showed that the activity of ALP, GOT and GPT level were increased with hepatotoxicity due to oxidative stress by hyperlipidemia. ALP, GOT and GPT level were decreased significantly ($p< 0.05$) which was increased at the time of experiment in group-II (hyperlipidemia) compared to the treatment groups. In Group IV dose at 50mg/kg body weight/day showed drastically less ALP in comparison to the other two ESS groups (group III dose at 25 mg/kg body weight/day and group V dose at 75 mg/kg body weight/day) and after administration of the sesame seed extract these indices were resettled towards the control level (Table: 3).

Changes in the level of antioxidant enzymes

The present study showed that the activity of antioxidative enzyme levels was decreased due to hyperlipidemic condition. SOD and CAT level were increased significantly ($p< 0.05$) in Treatment groups, which were decreased at the time of experiment in group II (hyperlipidemia) compared to the treatment groups. In Group IV dose at 50mg/kg body weight/day showed drastically less SOD and CAT level in comparison to the other two ESS groups (group III dose at 25 mg/kg body weight/day and group V dose at 75 mg/kg body weight/day) and after administration of the sesame seed extract these indices were resettled towards the control level (Table: 4).

Changes in the component of Lipid Peroxidation Assay

The effect of total cholesterol on the lipid peroxidation (LPO) was drastically iecreased than control group. The effect of different doses of sesame seed extracts showed significantly decreased the lipid peroxidation level near about the normal when compared to the control group. Oral administration of ESS prevented the increased LPO (MDA) to almost normal level. Between 3 doses of ESS, group IV (50mg/kg body weight/day) showed better result than other treated groups was observed (Table: 5).

1. Changes in the structure of liver cells

Morphological observations showing normal histology of liver of control group of rats with radiating chords of hepatocytes around central vein indicate well organized histoarchitecture (Figure 1- A). Figure 1- B, showed severe disorganization of liver cells (CV and HC) in cholesterol induced hyperlipidemic rats and damaged hepatocytes and blood vessels were seen prominently (Figure 1- B). By the treatment of ESS at the dose 25mg/kg body weight/day/rat orally there was evidence of progress in blood vessel and liver tissue (Figure 1- C) but also co-administration of ESS at the dose 50mg/kg body weight/day/rat and 75mg/kg body weight/day/rat orally were seen well organized liver cells like normal CV and normal hepatocytes (Figure 1- D, Figure 1- E) as well as in control group rats.

DISCUSSION

Plantsterols or phytosterols are cholesterol homologous compete with cholesterol in the absorption by intestine into blood circulation [22]. In the literature study, phytosterol reduces the plasma cholesterol concentration by competitively inhibit the cholesterol absorption by displacing them from bile salt micelles [23]. They may modify the LDL-c to bind with cholesterol and lowers the LDL-c concentration in plasma [24]. Sesame seed contains about 50% of oil, 25% of protein, 15% of carbohydrate and 10% of other nutrients and secondary metabolites [25]. The sesame seed oil is rich in phytosterol, which reported the lipid lowering effect [26]. The present study revealed that consumption of high cholesterol and fructose diet causes hyperlipidemia evidenced by high fat content of the different tissues in group II untreated hyperlipidemic rats, but ESS fed group III, IV and V animals showed significantly reduced fat content of the different tissues like- adipose, intestine, liver etc [27]. Cholesterol rich diet also can increased the level of TC, Tg, LDL-c in group II untreated hyperlipidemic rats, but ESS fed group III, IV and V animals showed significantly lower TC, Tg, LDL-c than group II. ESS inhibits the cholesterol absorption by intestine and lowers the LDL-c and enhances the HDL-c concentration in plasma, ultimately control the TC [24]. In this study the GOT, GPT and ALP levels which are the toxicity measurement, increased in group II untreated hyperlipidemic condition than the control group. The biologically active different doses of ESS were resettled the level of GOT, GPT and ALP level [28]. Where the treatment II group (50mg/kg body weight/day) showed more reduced level of GPT, GOT and ALP level than the other groups. The antioxidant enzymes are the protective substances, which plays as crucial role against the oxidants [29, 30]. The level of antioxidative enzymes (SOD and CAT) was drastically low in hyperlipidemic group. After administration of ESS, the level of the antioxidative enzymes significantly increased. In which the treatment II (50mg/kg body weight/day) showed the good result than the other two treatment groups. The study showed that the lipid peroxidation level (MDA) was drastically high in hyperlipidemic group. After administration of different doses of ESS the level of the MDA was significantly decreased. In which the treatment II (50mg/kg body weight/day) showed the good result than the other two treatment groups. The histopathological analysis showed disarrangement of HC and CV of liver cell in untreated group II hyperlipidemic rats. After oral co-administration of ESS at the doses of 50mg/kg body weight/day/rat and 75mg/kg body weight/day/rat showed the better protective action with well arranged HC and normal CV than the untreated groups.

CONCLUSION

In conclusion, the results of this present study revealed that sesame seed extract at the concentration of 50mg/kg body weight/day lower the level of cholesterol in hyperlipidemic condition. The protective effect of sesame seed extract is performed through multiple ways as it scavenges free radicals generated by hyperlipidemic condition, and also increases the activity of antioxidant defense system by elevating antioxidant enzymes like SOD, catalase and protect the liver through anticipating oxidative stress. The corrective effect of sesame seed extract also control the blood lipid profile along with the fat content of the tissues. Therefore it may be conferred that sesame seed extract possesses strong anti oxidative properties and ameliorates hyperlipidemia caused by cholesterol.

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