



## Effect of Sesame Oil on Serum and Liver Lipid Profile in Hyperlipidemic Rats.

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### Key words

Hyperlipidemia,  
lipid metabolism,  
triton, Sesame oil,  
adiponectin,  
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### ABSTRACT:

This study was conducted to investigate the effect of sesame oil on serum and hepatic lipid profile in induced hyperlipidemic rat .Eight forty rats were randomly divided into equal six groups of eight animals each for 30 days as follows : The first group(control) was kept on basal diet and water, the second group was kept on basal diet added to it sesame oil (SSO)5%, the third group was given a basal diet supplemented by SSO10%,the fourth group (hyperlipidemic) ,hyperlipidemia was induced at last two weeks by intra peritoneal injection of Triton WR1339(200mg/Kg, /three times /week) ,fifth group was fed basal diet +i.p of Triton 200mg/Kg +SSO5%,the six group was kept on basal diet +i.p injection of same dose of Triton +SSO10% . Blood samples were collected and serum was separated for determination of the followings triglycerides, cholesterol, HDL(high density lipoprotein), LDL( low density lipoprotein),VLDL (very low density lipoprotein), ALT(Alanin aminotranferase),AST( Aspartate aminotransferase),GGT\_(gamma glutamyl tranferase), ALP (Alkaline phosphatase ) ,hepatic lipid profile , thyroid hormones and adiponectin hormone. Induction of hyperlipidema resulted in a significant elevation of all parameters except HDL, adiponectin and thyroid hormones not significant decrease compared to control group .supplementation with sesame oil 5% and SSO 10 % signaificantly decrease in all serum and hepatic lipid profile and liver enzymes while HDL ,adiponectin ,thyroid hormones are elevated . Conclusion: SSO 5% was possess a better improving potential for hyperlipidemia, serum glucose, adiponectin , thyroid hormones, hepatic lipid profile than SSO10% .

### 1. INTRODUCTION.

Hyperlipidemia has been ranked as one of the greatest risk factors contributing to the prevalence and severity of coronary heart diseases (Kamesh and Sumathi, 2012). Coronary heart disease, stroke, atherosclerosis and hyperlipidemia are the primary cause of death (Uthandi and Ramasamy, 2011). Hyperlipidemia is characterized by elevated serum total cholesterol, low density lipoprotein, very low density lipoprotein and decreased high density lipoprotein levels.

In the past, the adipose tissue was seen as an energy depot, storing energy in the form of triglycerides and not having a real function of its own. At present, we know that adipose tissue also secretes several signaling proteins, called adipocytokines (Rajala et al, 2003). Adiponectin is one of the major adipocytokines derived from the adipose tissue (Maeda et al. 1996). It contains a stalk with 22 collagen repeats and a highly conserved globular domain (gAd). Adiponectin is normally present in plasma at concentrations up to 30 µg/ml but is markedly down regulated in

association with obesity-linked diseases including coronary artery disease and type 2 diabetes (Lihn et al, 2005). Clinical observations have demonstrated that hypo adiponectinemia is closely related to endothelial dysfunction in peripheral arteries (Berg et al. 2002; Philip et al. 2009; Wang et al. 2010) and that plasma total adiponectin concentrations are inversely related to the risk of myocardial infarction. Their results suggested that adiponectin might be a potent endothelial protective molecule (Ismail et al,2013). Diez et al .(2003) concluded that the function of adiponectin as a protein hormone that modulates a number of metabolic processes, including glucose regulation and fatty acid oxidation (glucose flux, decreased gluconeogenesis, increased glucose uptake, lipid catabolism, β-oxidation triglyceride clearance, protection from endothelial dysfunction).

Sesame (*Sesamum indicum L.*) is one of the most important oil seed crops, having seeds and its edible oil that are highly valued as a traditional healthy food ingredient (Abou-Gharbia et al., 2000 because of its high protein and antioxidant contents.

Sesame oil contains sesamin, sesamol and sesaminolignan fractions, which are known to play an important role in its oxidative stability and antioxidative activity (Elleuch, et al 2007). It is widely known as one of the natural health promoting foods that has the potential to prevent various disorders such as hypertension, hypercholesterolemia cancer and aging (Philip et al, 2010; Habila et al. 2013). Additionally, sesame oil may be useful in managing oxidative stress-associated diseases such as atherosclerosis, diabetes mellitus, obesity, chronic renal failure, rheumatoid arthritis, and neurodegenerative diseases including Alzheimer's disease (Lee et al., 2005; 2006). Moreover, sesame oil has multiple physiological functions such as decreasing blood lipids and arachidonic acid levels, increasing antioxidative ability and  $\gamma$ -tocopherol bioavailability, and providing anti-inflammatory function and potential estrogenic activity. Many health promoting effects are attributed to its lignans (Sedigheb et al. 2013). Phytochemical study has shown that the Sesame plant is rich in phenolic compounds (phenol, lignans and flavonoids), non-protein amino acids, cyanogenics, glycosides, alkaloids, polyunsaturated fats and lipids, mucilage, phospholipids, vitamins B1, B2 and E, trace elements and minerals such as calcium, iron, magnesium, copper and phosphorous (Anitha and Karuppasamy, 2011). The sesame meal is the residue after pressing the oil from the seed. It is an excellent source of protein (47% to 52.9%<sup>11</sup>). This study was designed to throw light on the effect of sesame oil feeding on serum and liver lipid profile.

## 2. MATERIAL AND METHODS.

This experiment was conducted at Biochemical Department, Faculty of Vet.Med., Alex. University to investigate the effect of sesame oil on lipid profile and some serum parameter in induced hyperlipidemic rats.

### 2.1 Experimental Animals

**Animals:** 48 adult Wistar rats  $90 \pm 10$  each weights 150-200g, and acclimatized for one 2 weeks before the onset of experiment. The animals were left free to as well access water and were fed on uniformly basal diet (corn 66.3%, SBOM 19.8 %, c.gluten 7.40%, fat 1.60 %, dica phos 1.89%, limestone 1.24%, lysine 0.40%, meth 0.09%, premix 0.3%,

salt 0.40%). They were housed in metal cages under normal laboratory conditions. Then randomly distributed into six group as flow group I ,control fed standard diet ;II fed normal diet and SSO5%;III fed normal diet and SSO10%; IV hyperlipidemic group by injection of Triton WR1339 i.p 200mg /kg (control positive);V normal diet+Triton+SSO5%;VI normal diet+Triton+SSO10%

### 2.2. Sample collections and Analysis.

At the end of the experiment (30 days), animals were kept starved overnight and sacrificed by cervical dislocation. Blood samples were collected by retro-orbital puncture under light ether anesthesia. Each blood sample was left to coagulate at room temperature. Separation of the serum was carried out by centrifugation of coagulated blood at 3000 rpm for 10 minutes. The clear serum was transferred carefully to clean and dry vials and kept at  $-20^{\circ}\text{C}$  until analysis of serum total cholesterol (Thomas, 1992), triglycerides (Stein, 1987), VLDL (very low density lipoproteins cholesterol), LDL cholesterol (low density lipoproteins) were calculated (Bauer, 1982), high density lipoproteins (HDL cholesterol), HDL cholesterol (High density lipoproteins) determined according to (Friedewald, 1972). Double antibody radioimmunoassays were used to determine plasma concentrations of T3, T4 and adiponictin hormones. AST and ALT (Young, 1990), ALP (Tietz et al, 1983), GGT (Moss et, 1987),

### 2.3. Collection of rat liver tissues:

Liver tissues were collected after decapitation of rats, they were eviscerated, the livers were removed from the carcasses and washed by ice-cold saline to remove the blood and then blotted in filter papers finally kept frozen at  $-70^{\circ}\text{C}$  for biochemical analysis. The homogenate was prepared and used for biochemical analysis of hepatic lipid profiles.

### 2.4. Chemicals.

All chemicals were purchased from Vitro chemical company except adiponictin, T3 and T4 hormones from Human chemical Company, and sesame oil from ELBARKA Company

**Statistical analysis:** analysis was done by one way analysis of variance (ANOVA). Data were expressed as means  $\pm$  standard error (Means  $\pm$  SE)  $p < 0.05$  was set as statistical significance according to SAS, (2002).

**3. RESULTS and DISCUSSION:****Table (1) Effect of sesame oil on serum lipid profile in induced hyperlipidemic rat**

| parametr | TG(mg/dl)                 | TC(mg/dl)                 | HDL-c(mg/dl)            | LDL-c(mg/dl)              | vLDL-c (mg/dl)           |
|----------|---------------------------|---------------------------|-------------------------|---------------------------|--------------------------|
| GroupI   | 148.63±27.49 <sup>b</sup> | 80.95±8.02 <sup>b</sup>   | 34.11±2.97 <sup>a</sup> | 5.55 <sup>c</sup> 16.4    | 29.72±5.50 <sup>b</sup>  |
| GroupII  | 51.25±7.42 <sup>b</sup>   | 70.92±7.72 <sup>b</sup>   | 38.01±5.8 <sup>a</sup>  | 18.3±6.36 <sup>c</sup>    | 10.29±1.51 <sup>b</sup>  |
| GroupIII | 87.21±9.17 <sup>b</sup>   | 73.31±1.95 <sup>b</sup>   | 37.80±1.38 <sup>a</sup> | 19.99±3.57 <sup>c</sup>   | 17.44±1.83 <sup>b</sup>  |
| GroupIV  | 371.03±55.54 <sup>a</sup> | 231.55±20.53 <sup>a</sup> | 26.44±1.81 <sup>b</sup> | 132.03±17.95 <sup>a</sup> | 74.17±11.10 <sup>a</sup> |
| GroupV   | 135.14±25.14 <sup>b</sup> | 124.36±15.61 <sup>b</sup> | 34.57±3.03 <sup>a</sup> | 63.48±11.70 <sup>b</sup>  | 27.02±5.02 <sup>b</sup>  |
| GroupVI  | 366.69±77.16 <sup>a</sup> | 201.80±35.86 <sup>a</sup> | 37.58±3.38 <sup>a</sup> | 71.46±20.67 <sup>b</sup>  | 73.25±15.42 <sup>a</sup> |

Data were expressed as means ± standard error (p<0.05), Values with different letters within the same column are statistically significant.

**Table (2) Effect of sesame oil on liver lipid profile in induced hyperlipidemic rats**

| pramater Group | TG (mg/dl)              | TC(mg/dl)               | HDL-c(mg/dl)            | vLDL-c(mg/dl)           | LDL-c(mg/dl)           |
|----------------|-------------------------|-------------------------|-------------------------|-------------------------|------------------------|
| GroupI         | 1.47±0.05 <sup>c</sup>  | 2.10±0.15 <sup>c</sup>  | 1.21±0.28 <sup>a</sup>  | 0.29±0.01 <sup>c</sup>  | 0.45±0.18 <sup>c</sup> |
| GroupII        | 1.21±0.26 <sup>c</sup>  | 1.82±0.38 <sup>c</sup>  | 1.28±0.04 <sup>a</sup>  | 0.24±0.05 <sup>c</sup>  | 0.52±0.12 <sup>c</sup> |
| GroupIII       | 1.58±0.17 <sup>c</sup>  | 1.89±0.14 <sup>c</sup>  | 1.05±0.30 <sup>a</sup>  | 0.31±0.04 <sup>c</sup>  | 0.97±0.19 <sup>c</sup> |
| GroupIV        | 3.53±0.23 <sup>a</sup>  | 8.48±0.38 <sup>a</sup>  | 0.92±0.08 <sup>b</sup>  | 0.71±0.05 <sup>a</sup>  | 6.83±0.39 <sup>a</sup> |
| GroupV         | 1.79±0.16 <sup>bc</sup> | 4.24±1.61 <sup>bc</sup> | 1.11±0.17 <sup>ab</sup> | 0.35±0.03 <sup>bc</sup> | 4.30±1.05 <sup>b</sup> |
| GroupVI        | 2.39±0.26 <sup>b</sup>  | 4.75±1.06 <sup>ab</sup> | 1.06±0.06 <sup>ab</sup> | 0.48±0.05 <sup>b</sup>  | 5.24±0.44 <sup>b</sup> |

**Table (3) Effect of sesame oil on liver weight, liver lipid percent in induced hyperlipidemic rats**

|           | liver lipid %          | (liver weight g)        |
|-----------|------------------------|-------------------------|
| GroupI    | 0.13±0.03 <sup>b</sup> | 7.07±0.32 <sup>c</sup>  |
| GroupII   | 0.10±0.00 <sup>b</sup> | 7.17±0.54 <sup>c</sup>  |
| Group III | 0.13±0.03 <sup>b</sup> | 8.07±0.19 <sup>ab</sup> |
| Group VI  | 0.37±0.07 <sup>a</sup> | 9.23±0.32 <sup>a</sup>  |
| Group V   | 0.13±0.03 <sup>b</sup> | 7.47±0.28 <sup>ab</sup> |
| Group VI  | 0.20±0.00 <sup>b</sup> | 9.10±1.12 <sup>a</sup>  |

Data were expressed as means ± standard Error (p<0.05), Values with different letters within the same column are statistically significant.

**Table (4) Effect of sesame oil on liver functions tests in induced hyperlipidemic rats.**

| Level     | ALT(U/L)                 | AsT(U/L)                   | ALP(U/L)                  | GGT (U/L)                            |
|-----------|--------------------------|----------------------------|---------------------------|--------------------------------------|
| Group I   | 43.50±4.53 <sup>bc</sup> | 136.43±9.95 <sup>ab</sup>  | 571.75±63.94 <sup>a</sup> | 3.45±0.82 <sup>ab</sup> <sup>c</sup> |
| Group II  | 28.48±3.24 <sup>c</sup>  | 109.13±5.47 <sup>b</sup>   | 281.75±40.36 <sup>b</sup> | 1.69±0.36 <sup>c</sup>               |
| Group III | 34.86±5.69 <sup>c</sup>  | 132.38±11.13 <sup>ab</sup> | 502.50±52.99 <sup>a</sup> | 2.92±0.59 <sup>bc</sup>              |
| Group IV  | 77.54±4.08 <sup>a</sup>  | 180.13±18.32 <sup>a</sup>  | 577.00±63.20 <sup>a</sup> | 5.27±0.74 <sup>a</sup>               |
| Group V   | 40.99±8.37 <sup>bc</sup> | 156.19±24.59 <sup>ab</sup> | 491.88±86.40 <sup>a</sup> | 3.94±0.81 <sup>ab</sup>              |

Data were expressed as means ± standard error (p<0.05), Values with different litters within the same column are statistically significant.

**Table (5) Effect of sesame oil on adiponectin, thyroid hormones and body weight in induced hyperlipidemic rats.**

| Level     | Body weight               | Adiponectin              | T4                     | T3                      |
|-----------|---------------------------|--------------------------|------------------------|-------------------------|
| Group I   | 235.00±9.92 <sup>a</sup>  | 28.00±2.02 <sup>a</sup>  | 3.30±0.06 <sup>b</sup> | 1.33±0.06 <sup>a</sup>  |
| Group II  | 190.00±3.78 <sup>a</sup>  | 39.67±11.29 <sup>a</sup> | 3.9±0.20 <sup>a</sup>  | 1.30±0.00 <sup>a</sup>  |
| Group III | 222.50±11.61 <sup>a</sup> | 36.33±8.11 <sup>a</sup>  | 4.55±0.14 <sup>a</sup> | 1.16±0.01 <sup>b</sup>  |
| Group IV  | 242.50±4.53 <sup>a</sup>  | 26.67±3.67 <sup>a</sup>  | 1.40±0.06 <sup>d</sup> | 0.96±0.01 <sup>d</sup>  |
| Group V   | 226.25±8.65 <sup>a</sup>  | 33.3±3.51 <sup>a</sup>   | 2.45±0.43 <sup>c</sup> | 1.04±0.05 <sup>cd</sup> |
| Group VI  | 232.50±7.50 <sup>a</sup>  | 31.60±8.17 <sup>a</sup>  | 1.50±0.00 <sup>d</sup> | 1.08±0.03 <sup>bc</sup> |

Data were expressed as means ± standard error (p<0.05), Values with different litters within the same column are statistically significant.

### 3.1 .Effect of sesame oil on serum lipid profile in induced hyperlipidemic rats:

Injection of triton to induction of hyperlipidemia in rats characterized by their elevated level of serum and liver cholesterol, triglycerides, vLDL-c, LDL-c and reduction in the HDL-c level as compared to control group as showed in table (1). This may be as to the capacity of the triton to associate with triglycerides in the plasma in such a way as to reduce their rate of hydrolysis by the enzyme, clearing factor lipase or lipoprotein lipase (Zanwar et al. 2012). The present work showed significant decrease in serum TG ,total cholesterol LDLc,

vLDLc and significant increase in HDLc with SS5% and not significant with SS10% as administrated in table (1) that was for hyperlipidemic group as compared to control one ,These results were agreed with Seyedeh et. al (2013 )they found that After sesame oil consumption, TG ,LDLc, VLDLc, were significantly decreased, and increased HDL that due to presence of Sesamin,which is a major lignan in sesame seeds which have lipid lowering effect as these lignans inhibits the absorption of cholesterol from the intestine The increase in fecal neutral steroid excretion.

**Table (6) The positive correlation between T3 and T4 and adiponectin hormone.**

|             | T3                | T4                | adiponectin       |
|-------------|-------------------|-------------------|-------------------|
| T3          | 1.00000           | 0.59846<br>0.0087 | 0.18921<br>0.4521 |
| T4          | 0.59846<br>0.0087 | 1.00000           | 0.34768<br>0.1574 |
| adiponectin | 0.18921<br>0.4521 | 0.34768<br>0.1574 | 1.00000           |

Pearson Correlation Coefficients, N = 18, Prob > |r| under H0: Rho=0  
 GI :control GII : SSO 5% GIII : SSO10% GIV: hyperlipidemic group GV: hyperlipidemia+ SSO5% GVI: hyperlipidemia + SSO10% values are expressed as mean ± standard errors Means in the same column (a-b-c). with different letters significantly and not significantly differ at (p≤0.05).

Moreover, Sedigheh et al, (2013) mentioned that rabbits supplemented with sesame oil 5% were found to have lower circulating concentrations of TG, total cholesterol, LDL-C. From another hand Satchithanandam et. al (1996) found that serum triglyceride and HDL-cholesterol levels did not differ significantly after feeding SSO. Kilkkinen et. al (2004) resulted that total- cholesterol reduction in SS10% fed diet compared to the control group, this reduction was not statistically significant, that because Sesame oil supplementation, at a level of 10% in the test diet, failed to restore the disturbed lipid profile caused by the high cholesterol acid diet. Total cholesterol and LDL-cholesterol levels exhibited a decrease by sesame oil administration. This preventive effect however could not be statistically confirmed, probably due to the limited number of available observations or/and to the elevated caloric value of the sesame oil modified diet. SSO could increase HDL due to phytoestrogen content as estrogen increasing HDL cholesterol while lowering LDL cholesterol. In addition, estrogens can elicit cell proliferation in estrogen-dependent tissue, such as the breasts and endometrium (Jillian, 2010).

**3.2. Effect of sesame oil on liver lipid profile in induced hyperlipidemic rats.**

Table (2) showed that hepatic level of TG, TC, HDL-c, LDL-c,vLDL-c relived that non

significantly changes in group II, and III as compared to control group at p<0.05, while this changes were increased with group III (SSO10%) compared to group II (SSO5%) at p <0.05. Injection of triton induced hyperlipidemia characterized by drastic increase in hepatic levels of TG,TC,vLDL-c,LDL-c, and a marked significant decrease in hepatic level of HDL-c as compared to control group at p<0.05. Treatment of hyperlipidemic rats with SSO5% and SSO10% showed hypolipidemic activity as they decreased hepatic level of TG, TC, vLDL-c, and LDL- c significantly, while HDL-c was increased significantly as compared to hyperlipidemic rats at p<0.05. The decreased levels in liver lipid profile by treatment of sesame oil that due to dietary sesame lignans greatly increased the hepatic activity of fatty acid oxidation enzymes, including carnitine palmitoyltransferase, acyl-CoA dehydrogenase, acyl-CoA oxidase, 3-hydroxyacyl-CoA dehydrogenase, enoyl-CoA hydratase, and 3-ketoacyl-CoA thiolase. Dietary sesamin also increased the activity of 2, 4-dienoyl-CoA reductase and delta3, delta2-enoyl-CoA isomerase, enzymes involved in the auxiliary pathway for beta-oxidation of unsaturated fatty acids dose-dependently (Sirato et al, 2001). Examination of hepatic mRNA levels using specific cDNA probes showed a sesamin-induced increase in the gene expression of mitochondrial and peroxisomal fatty acid oxidation enzymes. Among these various enzymes, peroxisomal acyl-CoA oxidase and bi functional enzyme gene expression were affected most by dietary sesamin (15- and 50-fold increase by the 0.5% dietary level). Sesamin-induced alterations in the activity and gene expression of carnitine palmitoyltransferase I and acyl-CoA oxidase were in parallel with changes in the mitochondrial and peroxisomal palmitoyl-CoA oxidation rate,

respectively (Akimoto et al, 1993). In contrast, dietary sesamin decreased the hepatic activity and mRNA abundance of fatty acid synthase and pyruvate kinase, the lipogenic enzymes. However, this lignan increased the activity and gene expression of malic enzyme, another lipogenic enzyme. Also this was associated with the down-regulation of sterol regulatory element-binding protein-1, a transcriptional factor that regulates the lipogenic enzyme gene expression. These changes may reduce the hepatic triacylglycerol synthesis and consequently decrease the assembly and secretion of triacylglycerol-rich lipoproteins by the liver. An alteration in hepatic fatty acid metabolism may therefore account for the serum and liver lipid-lowering effect of SSO in the rat (Ashakumary et al, 1999).

This result was agreed with that obtained by Bhaskaran et al, (2006) who noticed significant reduction in hepatic level of TC, LDL and TG levels in mice when the atherogenic diet was reformulated with the same concentration of sesame oil.

### **3.3 Effect of sesame oil on liver weight, liver lipid percent in induced hyperlipidemic rats.**

The data concerned liver weight and liver lipid percent (table 3) showed that, liver weight was significantly increased in hyperlipidemic rats as compared to control group. This could be a consequence of higher fat content which come in harmony with data obtained by Tirlangi Teja et al,(2013) and Taha et al , (2012) who founded that liver weight and liver lipid % after injection of triton were significantly increased. The obtained result illustrated that SSO before induction of hyperlipidemia did not alert liver lipid % which indicated safety benefits of SSO. However, when used for treatment of hyperlipidemic rats caused a significant decrease of liver lipid % and that may be due to hypolipidemic effect as demonstrated in this study. This data also showed that liver weight increased significantly in group III. The same pattern were established by Sugano et al,( 1990) that was a similar but lesser degree of liver enlargement has been observed in rats fed with 10% sesame oil. Since there was no accumulation of lipids, except for a slight increase in phospholipid within the normal range, the overall functions of the liver are expected to have been normal. These results may be attributed to the presence of omega -6-polyunsaturated fatty acid which are inflammatory when used in large amount that may cause NAFAD (nonalcoholic fatty acid disease) which may lead to increasing in liver weight in the feed supplemented

with sesame oil (Jacob et al. 1996 ; Gaíva et al. 2003) .In contrast Satchithanandam et. Al (1996) observed that liver lipid level was significantly higher in the 24% sesame oil-fed group, compared with levels in the group fed the control diet.

### **3.4. Effect of sesame oil on liver functions tests in induced hyperlipidemic rats.**

Results regarding with liver enzymes as shown in table (4) revealed that hyperlipidemic rats had elevated liver assessment of liver function can be made by estimating the activities of serum AST, ALT, ALP and GGT as compared to control, these enzymes originally present in higher concentration in cytoplasm, when there is hepatopathy, these enzymes leak into blood stream in conformity with the extent of liver damage (Venukumar and Latha 2004). The results of sesame oil treatment indicated that sesame oil was more effective for the treatment of high-fat diet toxicity through its ability to decrease the elevated activity of ALT and AST at the cellular level, as SSO contain some powerful antioxidants (IP-6, phytate, lignin, pinoresionoly, vitamin E, Lecithin, myristic acid and linolate) which may prevent free radical formation and scavenge free radicals that already formed. The present finding sesame treated group is well agreed with Morris (2002). While Horis et al (1991) activity of serum GOT and GPT remained unchanged.

### **3.5. Effect of sesame oil on adiponictin, thyroid hormones and body weight in induced hyperlipidemic rats**

Results in table (7) showed that hyperlipidemia (group IV) causing a significant reduction in serum levels of T3, T4 and not significant reduction in adiponectin hormones as compared to control group, that agreed with Dhingra et al (2003) who found that T3 and T4 concentration decreased significantly on HFD feeding in respective to control group. However Lotfi et al, (2012) reported that was no any changes in T3 or T4 in cholesterol feeding animals. The majority of the thyroid hormone circulates bound to Thyroxine Binding Globulin (TBG), with the remainder bound to trans thyretin and serum albumin. TBG has the highest affinity for thyroid hormone, and is induced byestrogen (Glinoyer, 1997). So, the elevation in serum T3 and T4 may due to estrogenic effect of SSO. Also SSO is a source of tyrosine, an amino acid which uses to manufacture thyroid hormone. The key enzymes involved in the activation and inactivation of thyroid hormones (iodothyronine deiodinases) are also selenoproteins. Selenium is an integral part of selenoproteins and critical enzymes

in thyroid hormone synthesis making this an essential micronutrient. As SSO consider selenium rich oil, so it may lead to improve circulating thyroid hormones. Also it contains healthy fatty acids that protect thyroid gland and keep it functioning normally (Sharma et al, 2014). While alterations between group II, III in T3 and T4 as a significant increase in T4 and a significant decrease in T3 in group III and not significant decrease in T3 in group II may be due to perturbations in thyroid hormone levels secondary to alterations in peripheral metabolism have received far less clinical attention. Several syndromes, such as "euthyroid sick syndrome" (ESS) and "low T3 syndrome," (Euthyroid sick syndrome (ESS), sick euthyroid syndrome (SES), non-thyroidal illness syndrome (NTIS) or low T3 high T4 syndrome is a state of adaptation or dysregulation of thyrotropic feedback control (Dietrich et al, 2012) where the levels of T3 and/or T4 are at unusual levels, but the thyroid gland does not appear to be dysfunctional. This condition is often seen in starvation, critical illness or patients in intensive care unit. The most common hormone pattern in sick euthyroid syndrome is a low level of circulating T3, with normal/ slightly elevated T4, and either normal or slightly suppressed TSH levels. This pattern of altered thyroid hormones is now generally agreed to be a result of impairment in extra-thyroidal peripheral metabolism. Causes of euthyroid sick syndrome include a number of acute and chronic conditions, including pneumonia, fasting, starvation, anorexia nervosa, sepsis, trauma, cardiopulmonary bypass, malignancy, stress, heart failure, hypothermia, myocardial infarction, chronic renal failure, cirrhosis, and diabetic ketoacidosis (Kohrle et al. 1988 ; Aatif et al. 2008). In our study that may be due to hypothermia and fasting as the experiment done in winter season.

Available experimental data suggest that adiponectin and thyroid hormones have biological interaction in vivo. Adiponectin receptors gene expression levels in the adipose tissue of treated animals have positive correlations with thyroid hormones concentrations. Their results suggest that AdipoR1 and AdipoR2 gene expression is regulated by thyroid hormones in hypo- and hyperthyroidism (Saifi et al, 2013) that was improved by our study as table (8) illustrated. As the result number above the median line the same under it.

#### 4- CONCLUSION:

From the results of this study, it is illustrated that SSO reserved the hyperlipidemia induced by triton in rats. SSO 5% has better effect than SSO 10% to improve hyperlipidemia and as hepatoprotective, and we needed more researches to discover more about that queen oil.

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