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Study the impact of sweet basil extracts (Ocimum basilicum) to reduce blood cholesterol

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Abstract

The study aimed to test the effect of sweet basil extract on hypercholesterolemia. Sweet basil leaves were extracted with distilled water, ethanol and a mixture of distilled water: ethanol (1:3, v/v). Phenolic and flavonoids concentrates were fractionated and identified with High Performance Liquid Chromatograph (HPLC). Two levels of each concentrated extract were used to find its effect on the hypercholesterolemic rats. Six groups of hypercholesterolemic rats received 0.35 or 0.70 g/Kg bw from aqueous, ethanolic and aqueous + ethanolic extracts of sweet basil leaves and were compared with normal control group and hyperlipidemic control group for 30 days. Growth of rats, biochemical biomarks and histopathological examination were evaluated. Results showed that the main phenolic compound was *p*-Hydroxybenzoic acid. Meanwhile, the main flavonoids compound was rutin. The biological experiment showed that second concentration of sweet basil leaves extracts (ASBL2, ESBL2 and AESBL2) had a positive effect to lower the weight of hypercholesterolemic rats. No significant difference was found in relative organs weight among all tested rat groups. Sweet basil leave extracts had a positive effect to decrease serum triglycerides, total cholesterol, LDL-cholesterol and VLDL-cholesterol of HC rats. The Atherogenic index (AI) was decreased due to sweet basil leaves extracts treated rat groups compared to the hyperlipidemic rats group. Sweet basil leaves extracts may play a role in improving the activity of antioxidant enzymes and decrease the lipid peroxidation (malondialdehyde, MDA) and level of H_2O_2 in serum. The serum ALT, AST, ALP and γ-GT activities of the second concentration (0.70 g/Kg bw) of sweet basil leaves extracts had no significant difference compared to NC rats group. There was no significant difference in serum uric acid, urea and creatinine among all tested rat groups received sweet basil leaves extracts and NC rats group except for HC rats group. No histopathological changes were observed in liver of rat groups received second concentration (0.70 g/Kg bw) of sweet basil leaves extracts. In conclusion, the obtained results suggest that sweet basil leaves extracts had an important role in lowering the hyperlipidemia in experimental animals especially the second concentration of sweet basil leaves extracts.

Introduction

Hypercholesterolemia plays an important role in atherosclerosis and related cardiovascular diseases (CVD). Increased plasma levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL) and very low-density lipoprotein cholesterol (VLDL) as well as lowered levels of high-density lipoprotein cholesterol (HDL) have been identified as major risk factors in the development of hypercholesterolemia. A high-cholesterol dietary habit is known to play an important role in the induction of chronic diseases such as hypercholesterolemia and hepatic abnormalities ***(Hsu et al., 2010)***.

A logical strategy, to prevent or to treat atherosclerosis and reduce the incidence of cardiovascular disease events, is to target hypercholesterolemia by drugs and/or dietary intervention ***(Koshy et al; 2001).***With this aim, efforts to develop effective and better hypolipidaemic drugs have led to discovery of natural products and have stimulated the

search for new lipid-lowering agents. Sweet basil (*Ocimum basilicum*), a member of Labiatae (Ocimoideae) is widely used in cooking for its culinary qualities. The chemical constituents of sweet basil are flavonoids, alkaloids, ascorbic acid, terpenoids, tannins, saponin glycosides, and phenolic compounds. Leaves samples showed the presence of two major flavone aglycones, which were identified as salvigenin and nevadensin ***(Grayer et al., 1996).*** This plant is a versatile herb that may be used in an abundant variety of foods. It is excellent in tomato-based dishes, spinach, and large number of squash. It is also widely used in soup and in cream cheese for sandwiches, dips and pasta dishes ***(Miele et al; 2001).*** Sweet basil grows in several regions around the world. Among more than 150 species of the genus *Ocimum*, basil is the major essential oil crop which is cultivated commercially in many countries ***(Sajjadi, 2006).***

Traditionally, basil has been extensively utilized in food as a flavoring agent, and in perfumery and medical industries ***(Telci et al., 2006).*** The leaves and flowering tops of the plant are perceived as carminative, galactogogue, stomachic and antispasmodic in folk medicine ***(Sajjadi, 2006).*** There are many varieties of *Ocimum* sp. and numerous laboratory studies have shown various protective effects including chemopreventive activity ***(Prakash and Gupta, 2000),*** anti-inflammatory activity ***(Klem et al., 2000)***, bactericidal activity ***(Koga et al.,1999),*** a nervous system stimulant effect ***(Maity et al., 2000),*** modulator effect on glutathione and antioxidant enzymes ***(Dasgupta et al; 2004)*** antidiarrheal effects ***(Offiah and Chikwendu, 1999),*** deduced risks of cancer and age-related eye diseases ***(Kopsell et al., 2005)*** and blood-sugar lowering effect ***(Worthley et al.,2007).***

The aim of the current study was to investigate the effect of sweet basil leaves extracts on rats with induced hypercholesterolemia and cardiovascular diseases and determine the suitable concentration to avoid hypercholesterolemia. We compared the liver histology and biochemical markers in hypercholesterolemic induced rats that received sweet basil leaves extracts with the control group.

Materials and Methods

Materials:

Sweet basil (*Ocimum basilicum* L.) leaves, (family: *Lamiaceae* (*Labiatae*), variety: *Mediterranean*). was obtained from the Horticulture Research Institute (HRI), Agriculture Research Center (ARC), Giza, Egypt.

Methods:

Preparation of sweet basil leaves extracts:

The aqueous extract was prepared as follows: The dried leaves (200 g.) was milled and infused 30 min in 1 liter distilled water (100 °C), filtered and the solution obtained was dried in oven under vacuum at 65 °C.

The ethanolic extract was prepared as follows: The dried leaves (200 g.) was milled and extracted by stirring with 1 liter ethanol at 75 °C and 300 rpm for 30 min.

The aqueous + ethanolic extract was prepared as follows: dried leaves (200 g.) was milled and extracted by stirring with 1 liter of a mixture of distilled water and ethanol (1:3, v/v) at 75 °C and 300 rpm for 30 min.

The extracts (ethanolic or aqueous + ethanolic extracts) were then filtrated through filter paper. The filtrates were collected and dried using a rotary evaporator at 40 °C.

Dried powder of each of the obtained extraction was transferred into plastic dark bottles and stored at -20 °C. Dilutions of 0.35 and 0.70 % (w/w) were prepared by dissolving the dried extracts in distilled water on the day of experiment.

Determination and identification of phenolic and flavonoids fraction of sweet basil leaves extracts using HPLC:

Phenolic and flavonoids fraction of sweet basil leaves extracts were identified determined by the method described by ***(Schieber et al., 2001).*** A high performance liquid chromatography system equipped with a variable

wave length detector (Agilant ,Germany) 1100, autosampler, quaternary pump degasser and column compartment. Analyses were performed with a C_{18} reverse phase packed stainless-steel column (Zorbax ODS 5 μm 4.6 × 250 mm). HPLC method started with linear gradient at a flow rate of 1.0 ml / min with mobile phase of water / acetic acid (98: 2 v/v, solvent A) and methanol / acetonitril (50: 50, v/v, solvent B), starting with 5 % B and increasing B to levels of 30% at 25 min, 40% at 35 min, 52% at 40 min, 70% at 50 min, 100% at 55 min. The initial conditions were re-established by 5 min wash in both solvents. All chromatograms were plotted at 2^· nm to estimated phenolic acids and at 330 nm for flavonoids. All components were identified and quantified by comparison of peak areas with external standards.

Animals and treatments:

48 adult male *albino* rats weighing 200 g ± 5g were obtained and housed in the Ophthalmology Research Institute, Giza, Egypt. The rats were kept under normal health laboratory conditions and fed basal diet for one week. Water and basal diet were provided *ad libitum*. After one week, the animals were divided into eight groups (each six rats) as follows:

Group 1: Normal control (NC), received basal diet.
Group 2: Hyperlipidemic control (HC), received hypercholesterolemic diet (HCD).
Group 3: Aqueous extract of Sweet Basil Leaves (0.35 g/Kg bw) and received as HCD, (ASBL1).
Group 4: Aqueous extract of Sweet Basil Leaves (0.70 g/Kg bw) and received as HCD, (ASBL2).
Group 5: Ethanolic extract of Sweet Basil Leaves (0.35 g/Kg bw) and received as HCD, (ESBL1).
Group 6: Ethanolic extract of Sweet Basil Leaves (0.70 g/Kg bw) and received as HCD, (ESBL2).
Group 7: Aqueous + Ethanolic extract of Sweet Basil Leaves (0.35g/Kg bw) and received as HCD, (AESBL1).
Group 8: Aqueous + Ethanolic extract of Sweet Basil Leaves (0.70g/Kg bw) and received as HCD, (AESBL2).

The sweet basil leaves extracts were orally given to rats daily by using stomach tube. Rats were given free access to food and water for 30 days.

Preparation of hypercholesterolemic diet (HCD):

HCD consists of basal diet 81.8%, cholesterol 2%, lard 16% and cholic acid 0.2% ***(Harnafi et al., 2009).***

Growth of rats:

The rats were weighed at zero time and twice a week, total feed intake of each rat was weighed and feed efficiency ratio was calculated as gain of rat weight / total feed intake, g. At the end of the experimental period, rats were weighed and sacrifice by diethyl ether. The carcasses were dissected. Liver was cut off, washed in saline solution (NaCl 0.09 %) and weighed. The liver weight percentage was calculated as follows (Weight of liver/ total body weight x 100).

Biochemical parameters assays:

At the end of the experimental period, blood samples were collected from the animals' eye plexuses. Each sample was divided into two patches the first one was collected into heparinized tubes to obtain the plasma and the second into a free coagulation dry clean centrifuge glass tube to prepare the serum. Blood samples were left for 15 min. at room temperature, and then the tubes were centrifugated for 15 min. at 3000 rpm and the clean supernatant serum samples were frozen at -20 °C until analysis.

Serum triglycerides, total cholesterol, HDL-cholesterol and LDL-cholesterol were determined by using the methods described by ***Fossati and Prencipe (1982), Wastson (1960), Assmann (1979)*** and ***Wieland and Seidel (1983)***, respectively. Serum VLDL-cholesterol was calculated according to ***Wallach (1992)*** using the following equation: VLDL-cholesterol = (Serum triglycerides/5). Moreover, Athergenic index (AI) was calculated as follows: AI = (VLDL-cholesterol + LDL- cholesterol)/HDL- cholesterol ***(Harnafi et al., 2009).***

The activity of superoxide dismutase (SOD) and catalase (CAT) of rats' blood plasma were estimated according to ***Marklund and Marklund (1974)*** and ***Luck (1971)***. Erythrocytes glutathione peroxides (GPx) and glutathione-S-transferases (GST) activities were estimated according to **Rotruck *et al.*, (1973)** and **Habig *et al.*, (1974)**. Moreover, lipid peroxidation level (malondialdehyde, MDA) and hydrogen peroxide (H_2O_2) were determined according to the method showed by ***Meltzer et al., 1997*** and ***Lacy et al., 1998***.

Alanine aminotransferase (ALT; EC 2.6.1.2) and aspartate aminotransferase (AST; EC 2.6.1.1) activities were assayed by the method of ***Bergmeyer and Harder (1986).*** Alkaline phosphatase (ALP; EC 3.1.3.1) activity was measured at 405 nm by the formation of paranitrophenol from para-nitrophenylphosphate as a substrate using the method of **Varley *et al.*, (1980)**. Gamma -GT was measured according to the method described by ***Szasz (1969)***. Creatinine, urea and uric acid were determined by using the methods described by ***Larsen (1972), Patton and Crouch (1977) and Caraway (1955)***.

Histopathological study: Tissue specimens from rats' liver were fixed in 10% neutral buffered formalin solution. The fixed specimens were trimmed, washed and dehydrated in ascending grades of alcohol, cleaned in xylene, embedded in paraffin then sectioned (4-6 micron) and stained with hematoxyline and eosin according to ***Bancroft et al., (1996)***. The degree of hepatic injury was estimated using an ordinal scale modified from **Palaa and Charbonneau (1994)**.

Statistical analysis: Each parameter was analyzed separately by using one-way analysis of variance (ANOVA). For determining differences between groups, Duncan test was used. All p values ≤ 0.05 were considered significant. ***(Bouveresse et al., 2011).***

Results

Results of fractionation and determination of phenolic compounds by high performance liquid chromatography (HPLC) are presented in Table (1). The data illustrated the phenolic compounds of sweet basil leaves extracts contains pyrogallo, protocatchic acid, caffeic acid, rosmarinic acid, catechein, catechol, vanillic acid, ferulic acid, *p*-Hydroxybenzoic acid and caffeine acid. It could be noticed that there was a remarkable variation in the amount of phenolic compounds of different extracts due to the extraction method. *p*-Hydroxybenzoic acid showed the highest amount of phenolic compounds. Meanwhile, caffeine resulted in the lowest amount of phenolic compounds.

Data in Table (2) shows the flavonoids compounds of sweet basil leaves extracts. Rutin is considered the main compound of flavones. Quercitrin, kampferol, apigenin, naringenin and quercetin were found in the different extracts with a fair deference in amounts of these compounds. The data show a slight difference among different extracts of sweet basil leaves. Rutin flavonoids compound was found to be the highest value while quercetin was in traces.

Data presented in Table (3) show the initial weight, final weight, weight and relative weight of liver, heart and kidney among groups. There was a significant difference in final weight of rats among the tested groups. It could be noticed that the lowest weight in rat groups received ASBL2, ESBL2 and AESBL2. It means that concentration of sweet basil leaves extracts at 0.70 g/Kg body weight reduced the weight of hypercholesterolemic rats. Low concentration of sweet basil extracts decreased the weight of hypercholesterolemic rats by 10 %. There was no significant difference in relative weight of liver, heart, and kidney among rat groups received all extracts of sweet basil compared to the NC. In contrast, the HC rats group showed highest final weight, relative weight of liver, heart, and kidney.

Data in Table (4) shows the lipid profile of different groups' rats that received sweet basil leaves extracts. There was significant increase in all parameters rats fed hypercholesterolemic diet. Sweet basil extracts significantly decreased the measured parameters. The ASBL1, ESBL1 and AESBL1 rat groups had about 40 % less serum triglycerides HC rat groups. Meanwhile, the second concentration (0.70 g Kg/ bw) of sweet basil leaves extracts decreased serum

triglycerides to 50 % and there was no significant deference among these extracts and NC. The same trend is noticed in serum total cholesterol, HDL-cholesterol, LDL-cholesterol, and VLDL-cholesterol. The AI was significantly different in rat groups that received ASBL2, ESBL2, AESBL2 compared to NC rats group. However, the AI in ASBL2 was considered the nearest rats group in the AI the NC. AI value decreased approximately 62.5 % in rat groups that received the first concentration of sweet basil leaves extracts (0.35 g/Kg bw). The HC rats group had the highest AI value.

Data in Table (5) illustrated serum SOD, catalase, GST, GP_x, MDA and H_2O_2 of rats in various sweet basil leaves extracts treatment groups. The data clear that the hypercholesterolemic diet had a negative effect on the activity of antioxidant enzymes. There was a significant decreased in serum SOD, catalase and GP_x activities of rats fed on HC diet. In contrast, the level of MDA, hydrogen peroxide (H_2O_2) and the activity of GST had significantly increased in rats fed on HC diet. Sweet basil leaves extracts improved the negative effect of HC diet. Concerning to the ASBL1, ESBL1 and AESBL1 rat groups, there were significant increase in serum SOD, catalase and GP_x activities (72, 78 and 71% respectively) compared to HC rats group. In the ASBL2, ESBL2 and AESBL2 rat groups a positive increase in serum SOD, catalase and GP_x activities was observed. There was no significant difference in the level of MDA, hydrogen peroxide (H_2O_2) and the activity of GST among rat groups received ASBL2, ESBL2 and AESBL2 and NC.

The liver function tests of the tested rat groups are shown in Table (6). There was significant increase in liver function tests in HC rats group compared to all rat groups and NC rats group. The serum ALT, AST, ALP and γ-GT activities of the second concentration (0.70 g/Kg bw) of sweet basil leaves extracts had no significant difference compared to NC rats group. Moreover, the first concentration (0.35 g/Kg bw) of sweet basil leaves extracts had a significant decrease of serum ALT, AST, ALP and γ-GT activities compared to HC rats group.

Data of the kidney function tests (Table 7) revealed clearly that hypercholesterolemic diet had a negative effect on serum uric acid, urea and creatinine of tested rats. There was no significant difference among all tested rat groups received sweet basil leaves extracts and NC rats group.

Histopathological examination of liver (Fig. 1) showed no apparent histopathological changes in liver of NC rats group no (Slide1). Meanwhile, liver of HC rats group revealed cytoplasmic vacuolization of hepatocytes (Slide 2). The liver of rats received ASBL1 illustrated cytoplasmic vacuolations of hepatocytes (Slide 3). However, the liver of rats received ESBL1 showed congestion of central veins and kupffer cells activation (Slide 5). There was kupffer cells activation in liver of rats which received AESBL1 (Slide 7). In contrast, the liver of rats that received ASBL2, ESBL2 and AESBL2 (0.7 g/kg b.w.) showed no histopathological changes (Slide 4, 6 and 8).

Discussion

Sweet basil is widely used in medicine in many countries because it has antioxidant compounds. Phenolic and flavonoid compounds help to protect and treat certain diseases. In the present study, data of the phenolic compounds in sweet basil leaves extracts are in agreement with those observed by ***Sgherri et al. (2010)***. They cited that the aqueous extract of sweet basil leaves contains many phenolic acids such as: Protocateuic acid, *p*-Hydroxybenzoic acid, vanillic acid, caffeic acid, *p*-coumaric acid and ferulic acid. Moreover, ***Lee and Scagel (2009)*** illustrated that rosmarinic acid was identified in fresh basil leaves. Rosmarinic acid was the main compound found in both leaves and stems. On the other hand, ***Kwee and Niemeyer (2011)*** found that the main compound of methanolic sweet basil leaves extract (15 cultivars) was caffeic acid.

From the flavonoid compounds, rutin showed the highest amount of tested sweet basil leaves extracts. ***Sofic et al. (2010)*** showed that the rutin was found in sweet basil leaves extract. Rutin was extracted with the hot water and the

rutin amount was 6.4 (mg/g) in supernatant. However, ***Yang et al. (2008)*** cited that the kampferol and apigenin were not detected in sweet basil hydrolysis sample.

In the present study, the HC rat group had the highest final weight and relative weight of liver, heart, and kidney. ***Nakamura and Tonogai (2002) and Tong et al., (2012)*** illustrated that the hypercholesterolemic diet significantly increased the body weight and weight liver ratio. On the other hand, ***Harnafi et al. (2009)*** found that the hypercholesterolemic rats had significant decrease in body weight, liver, kidney and heart weights of rats when rats received aqueous sweet basil extract (ABE). Ethanolic extract of sweet basil extract had a positive effect on weight loss in hypercholesterolemic rats ***(Juntachote et al., 2007).*** These results are in agreement with the obtained results.

It is long known that there is a relationship between the high prevalence of cardiovascular diseases (atherosclerosis, diabetes, hypertension.) and abnormalities in lipid metabolism. In addition, a positive correlation between dietary fat, hyperlipemia and incidence of coronary diseases has been established and documented. Moreover, it is well established that the traditional Mediterranean diets were associated with low incidence and prevalence of cardiovascular diseases. Modification of diet composition can prevent cardiovascular events and improve metabolic disorders relative to western diet ***(Watts et al., 1996).*** In view of previous evidence that many foods, plant food and spices such as sunflower, virgin-olive and fish oils ***(Aguilera et al., 2002)*** celery (*A. graveolens*) and garlic (*Allium sativum*) successfully prevented hyperlipidemia and atherosclerosis ***(Heidarian et al., 2011),*** we assessed the effect of sweet basil (*O. basilicum*) one of the plant foods for its beneficial effect on serum and liver lipid profiles. The results show that the sweet basil leaves extracts decrease AI. ***Harnafi et al., (2009)*** cited that *O. basilicum* may contain polar products able to lower plasma lipid concentrations and might be beneficial in treatment of hyperlipidemia and atherosclerosis. Administration of sweet basil leaves extracts provides a beneficial action in rat lipid metabolism regarding to the reduction of atherogenic index (AI). In fact, the AI was decreased in sweet basil leaves extracts treated rat groups comparatively to the hyperlipidemic one. Similar results were reported by others when studying the hypolipidemic effect of natural products ***(Cherng and Shih, 2005).*** This ameliorative action could be due to the serum lipid-lowering activity of extract. It is also desirable to have higher serum HDL and lower LDL-cholesterol to prevent atherogenesis, since there is a positive correlation between an increased LDL-C/HDL-C ratio and the development of atherosclerosis and related cardiovascular events.

The current study findings include increased activity of antioxidant enzymes in rat groups treated with sweet basil leaves extracts. ***Kusamran et al. (1998)*** found that the rats fed on dietary sweet basil leaves showed a significant decrease in the plasma level of Glutathione S-Transferase activity (GST). Moreover, ***Thiruchenduran et al. (2011)*** cited that increased oxidant stress, resulting from both increased oxygen free radical production and decreased nitric oxide generation, appears to play an important role in the chronic inflammatory responses to hypercholesterolemia and atherosclerosis. ***Dasgupta et al.,(2004)*** reported an improvement of antioxidant enzymes and significant reduction of lipid peroxidation and lactate dehydrogenase formation of rats that received 200 and 400 mg/kg body weight of hydroalcoholic extract (80% ethanol, 20% water) of the fresh leaves of *Ocimum basilicum.*

Moreover, Basil leaf extract was highly effective in inhibiting carcinogen-induced tumor. ***Lee et al., (2005)*** showed that sweet basil had antioxidant activities because it had aroma constituents [3,7- dimethyl-1,6-octadien-3-ol (linalool; 3.94 mg/g), 1-methoxy-4-(2-propenyl) benzene (estragole; 2.03 mg/g), methyl cinnamate (1.28 mg/g), 4-allyl-2-methoxyphenol (eugenol; 0.896 mg/g), and 1,8-cineole (0.288 mg/g). In the same trend, ***Juntachote et al. (2007)*** found that the supplementation of cooked ground pork with dried. Basil powder and its ethanolic extract significantly inhibited the formation of thiobarbituric acid-reactive substances (TBARS) and peroxide value.

Our results show marked improvement in liver function of rats that received sweet basil leaves extracts compared to HC rats group. On the other hand, ***(Otunola et al., 2010)*** found that significant increase in the level of AST,

ALT, ALP and GGT activities in rats fed with the hypercholesterolemic (HPC) diet for eight weeks. Moreover, Meera *et al.,* (2009) found that the ethanolic extract of sweet basil (100 mg/Kg b.w.) returned the serum AST, ALT, ALP and GGT enzyme activities to the normal range for the goat treated with H_2O_2 or CCl_4. Moreover, ***Sakr et al. (2011)*** showed that treating rats with CCl_4 and aqueous leaves extract of sweet basil led to an improvement, in both histopathological and biochemical alterations induced by CCl_4.

Creatinine is the major waste product of creatine metabolism. Free creatinine appears in blood serum. Urea and uric acid are the principal waste products of protein catabolism. Data in Table (7) illustrate that different concentrate levels of sweet basil leaves extracts improved the kidney function of hyperlipidemic rats. ***Muntner et al. (2000), Baylis (2005) and Balarini1 et al. (2011)*** illustrated that both hypercholesterolemia and aging contribute to the loss of renal function. They found when the mice and old rats fed hypercholesterolemic diet (HCD), serum urea and creatinine were significantly increased and the percentage of increase was higher in old rat than mice. In the contract, ***Rasek et al. (2012)*** found that *Ocimum Basilicum* hydroalcoholic extract decreased the serum uric acid, urea and creatinine in wistar rats.

In conclusion, our results suggest that sweet basil leaves extracts had an important role in lowering the hyperlipidemia in blood especially the second concentration of sweet basil leaves extracts. In fact, flavonoids and tannins a heterogeneous group of ubiquitous plant polyphenols, have exhibited a variety of pharmacological activities, including the hypolipidemic effect. The possible mechanism underlying hypolipidemia of these phytochemicals results generally in three points: firstly these phenolic compounds increase hepatic LDL receptor activity ***(Dasgupta et al., 2004).*** Secondly, tannins and flavonoids are known to reduce cholesterol synthesis via the suppression of hydroxymethyl glutaryl-CoA (HMG-CoA) reductase ***(Jung et al., 2006).*** Finally, these molecules inhibit the major enzyme involved in cholesterol metabolism, acyl CoA cholesterol acyl transferase (ACAT), ***(Lee et al., 2006).*** *O. basilicum* may contain polar products able to lower plasma lipid concentrations and might be beneficial in preventing hyperlipidemia and related cardiovascular diseases **(Harnafi *et al.*, 2009)**. Moreover, sweet basil had high levels of nutritionally important carotenoids, ***(Kopsell et al., 2005).***

Table (1)
Phenolic fractions of sweet basil leave extracts using HPLC (mg/g. dry weight)

	ASBL	ESBL	AESBL
Pyrogallol	0.79	0.62	0.69
Protocatchuic	0.84	0.77	0.78
Caffeic	0.30	0.29	0.32
Rosmarinic acid	0.27	0.26	0.33
Catechein	0.79	0.75	0.81
Catechol	0.28	0.32	0.26
Vanillic acid	0.34	0.31	0.29
Ferulic acid	0.34	0.36	0.37
p-Hydroxybenzoic acid	1.27	1.21	1.18
Caffeine	0.10	0.11	0.10

ASBL= Aqueous extract of Sweet Basil ESBL= Ethanolic extract of Sweet
AESBL= Aqueous + Ethanolic extract of

Table (2)
Flavionods fractions of sweet basil leave extracts using HPLC (mg/g. dry weight)

	ASBL	ESBL	AESBL
Rutin	1.45	1.38	1.97
Quercitrin	0.44	0.36	0.46
Kampferol	0.09	0.14	0.12
Apigenin	0.14	0.21	0.18
Naringenin	0.13	0.07	0.09
Quercetin	--	0.01	0.02

ASBL= Aqueous extract of Sweet Basil ESBL= Ethanolic extract of Sweet
AESBL= Aqueous + Ethanolic extract of

Table (3)
Growth and relative organ weights of hypercholesterolemic rats received two concentrates of sweet basil leaves extracts

Rat groups	Initial weight (g)	Final weight (g)	Liver weight (g)	Relative liver weight	Heart weight (g)	Relative heart weight	Kidney weight (g)	Relative kidney weight
NC	165.3 ± 5.15^{a}	208.07 ± 2.11^{cd}	6.65 ± 0.70^{b}	3.20 ± 0.31^{b}	1.13 ± 0.11^{b}	0.54 ± 0.06^{b}	1.54 ± 0.06^{b}	0.74 ± 0.03^{b}
HC	164.9 ± 5.63^{a}	236.88 ± 4.08^{a}	8.40 ± 0.37^{a}	3.12 ± 0.10^{a}	1.94 ± 0.04^{a}	0.82 ± 0.01^{a}	2.34 ± 0.12^{a}	0.99 ± 0.02^{a}
ASBL1	165.1 ± 3.83^{a}	217.83 ± 4.02^{b}	6.94 ± 0.44^{b}	3.19 ± 0.14^{b}	1.20 ± 0.07^{b}	0.55 ± 0.04^{b}	1.63 ± 0.05^{b}	0.75 ± 0.02^{b}
ASBL2	165.6 ± 4.87^{a}	208.10 ± 4.34^{cd}	6.68 ± 0.43^{b}	3.21 ± 0.22^{b}	1.14 ± 0.06^{b}	0.55 ± 0.02^{b}	1.54 ± 0.12^{b}	0.74 ± 0.04^{b}
ESBL1	165.5 ± 6.19^{a}	216.15 ± 5.79^{bc}	6.97 ± 0.53^{b}	3.22 ± 0.16^{b}	1.20 ± 0.08^{b}	0.56 ± 0.02^{b}	1.66 ± 0.17^{b}	0.77 ± 0.06^{b}
ESBL2	165.6 ± 1.22^{a}	208.78 ± 5.00^{cd}	6.57 ± 0.46^{b}	3.17 ± 0.18^{b}	1.14 ± 0.11^{b}	0.54 ± 0.04^{b}	1.53 ± 0.02^{b}	0.73 ± 0.02^{b}
AESBL1	165.1 ± 3.43^{a}	213.38 ± 6.0^{bcd}	6.90 ± 0.32^{b}	3.23 ± 0.20^{b}	1.15 ± 0.14^{b}	0.54 ± 0.05^{b}	1.59 ± 0.15^{b}	0.74 ± 0.06^{b}
AESBL2	165.0 ± 3.86^{a}	207.14 ± 3.57^{d}	6.35 ± 0.14^{b}	3.06 ± 0.11^{b}	1.13 ± 0.05^{b}	0.54 ± 0.03^{b}	1.56 ± 0.10^{b}	0.75 ± 0.06^{b}
LSD ($P \leq 0.05$)	7.81	7.82	0.78	0.32	0.15	0.06	0.19	0.08

NC =Normol HC = Hyperlipidemic control ASBL1= Aqueous extract of Sweet Basil Leaves (0.35 g/Kg
ASBL2= Aqueous extract of Sweet Basil Leaves ESBL1= Ethanolic extract of Sweet Basil Leaves (0.35 g/Kg
ESBL2= Ethanolic extract of Sweet Basil Leaves AESBL1= Aqueous + Ethanolic extract of Sweet Basil Leaves
AESBL2= Aqueous + Ethanolic extract of Sweet

Each value represents the mean ± SE. The mean values with different superscript alphabets indicate significant differences ($P \leq 0.05$) using LSD test.

Table (4)
Lipid profile of hypercholesterolemic rats received two concentrates of sweet basil leaves extracts.

Rat groups	Triglycerides (mg/dl)	T.cholesterol (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL	A I
NC	99.64 ± 6.92^{c}	68.65 ± 4.22^{e}	36.06 ± 2.21^{d}	12.67 ± 0.77^{b}	19.93 ± 1.38^{c}	0.90 ± 0.020^{e}
HC	224.46 ± 10.73^{a}	194.04 ± 11.77^{a}	45.25 ± 3.13^{a}	119.17 ± 27.24^{a}	44.89 ± 2.15^{a}	3.60 ± 0.488^{a}
ASBL1	138.5 ± 5.80^{b}	96.51 ± 4.21^{b}	39.25 ± 2.05^{bcd}	29.59 ± 1.84^{c}	27.70 ± 1.16^{b}	1.46 ± 0.071^{b}
ASBL2	111.33 ± 9.89^{c}	81.73 ± 2.99^{cd}	42.03 ± 1.50^{ab}	17.45 ± 1.08^{b}	22.26 ± 1.98^{c}	0.95 ± 0.055^{de}
ESBL1	137.26 ± 9.66^{b}	93.50 ± 4.71^{b}	41.37 ± 1.27^{abc}	24.68 ± 1.84^{c}	27.45 ± 1.93^{b}	1.26 ± 0.045^{bcd}
ESBL2	115.3 ± 12.70^{c}	73.30 ± 6.45^{de}	36.02 ± 3.17^{d}	13.71 ± 1.65^{b}	23.06 ± 2.54^{c}	1.02 ± 0.006^{cde}
AESBL1	135.83 ± 6.40^{b}	90.41 ± 4.39^{bc}	38.73 ± 1.89^{bcd}	24.44 ± 1.76^{c}	27.17 ± 1.28^{b}	1.33 ± 0.034^{bc}
AESBL2	109.27 ± 5.76^{c}	72.76 ± 3.79^{de}	37.60 ± 0.67^{cd}	13.31 ± 3.90^{b}	21.85 ± 1.15^{c}	0.98 ± 0.033^{de}
LSD ($P \leq 0.05$)	15.281	10.25	3.71	17.0	3.05	0.31

Each value represents the mean ± SE. The mean values with different superscript alphabets indicate significant differences ($P \leq 0.05$) using LSD test

NC =Normol control HC = Hyperlipidemic control
ASBL1= Aqueous extract of Sweet Basil Leaves (0.35
ASBL2= Aqueous extract of Sweet Basil Leaves (0.70 g/Kg
ESBL1= Ethanolic extract of Sweet Basil Leaves (0.35
ESBL2= Ethanolic extract of Sweet Basil Leaves (0.70
AESBL1= Aqueous + Ethanolic extract of Sweet Basil
AESBL2= Aqueous + Ethanolic extract of Sweet Basil

Table (5)
Activity of antioxidant enzymes, malondialdehyde (MDA) and H_2O_2 concentration in serum of hypercholesterolemic rats received two concentrations of sweet basil leaves extracts.

Rat groups	SOD (U/ml)	Catalase (U/ml)	GST (U/g Hb)	GPx (U/g Hb)	Malondialdehyde (mmol/ml)	H_2O_2 (mmol/ml)
NC	271.65 ± 8.12^{a}	182.15 ± 4.56^{a}	4.75 ± 0.25^{d}	155.5 ± 7.75^{a}	3.32 ± 0.65^{c}	0.129 ± 0.0106^{c}
HC	67.33 ± 2.41^{e}	86.99 ± 5.58^{d}	24.1 ± 1.61^{a}	65.17 ± 3.69^{d}	16.9 ± 2.39^{a}	0.204 ± 0.0055^{a}
ASBL1	196.04 ± 10.3^{d}	142.17 ± 7.15^{c}	13.3 ± 1.72^{b}	107.7 ± 7.22^{c}	10.4 ± 0.79^{b}	0.175 ± 0.0092^{b}
ASBL2	248.43 ± 9.80^{b}	157.77 ± 9.42^{b}	5.21 ± 0.35^{d}	135.3 ± 8.55^{b}	4.39 ± 0.32^{c}	0.141 ± 0.0066^{c}
ESBL1	197.97 ± 13.7^{d}	143.65 ± 2.23^{c}	12.5 ± 1.58^{b}	107.3 ± 4.33^{c}	10.3 ± 1.29^{b}	0.171 ± 0.0067^{b}
ESBL2	220.43 ± 12.1^{c}	167.47 ± 13.89^{b}	6.29 ± 0.40^{d}	147.0 ± 7.96^{ab}	4.72 ± 0.43^{c}	0.133 ± 0.0055^{c}
AESBL1	193.7 ± 7.38^{d}	142.42 ± 4.41^{c}	10.2 ± 0.50^{c}	114.7 ± 7.21^{c}	8.42 ± 1.04^{b}	0.164 ± 0.0060^{b}
AESBL2	222.60 ± 11.7^{c}	163.79 ± 6.89^{b}	5.34 ± 0.25^{d}	143.6 ± 7.15^{ab}	4.29 ± 0.31^{c}	0.129 ± 0.0061^{c}
LSD ($P \leq 0.05$)	17.29	13.08	1.80	12.0	1.92	0.013

Each value represents the mean ± SE. The mean values with different superscript alphabets indicate significant differences ($P \leq 0.05$) using LSD test

NC =Normol control HC = Hyperlipidemic control
ASBL1= Aqueous extract of Sweet Basil Leaves (0.35
ASBL2= Aqueous extract of Sweet Basil Leaves (0.70 g/Kg
ESBL1= Ethanolic extract of Sweet Basil Leaves (0.35
ESBL2= Ethanolic extract of Sweet Basil Leaves (0.70
AESBL1= Aqueous + Ethanolic extract of Sweet Basil
AESBL2= Aqueous + Ethanolic extract of Sweet Basil

Table (6)
Alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP) and gamma glutamyl transferase (γ-GT) of hypercholesterolemic rats received two concentrations of sweet basil leaves extracts

Rat groups	ALT (U/L)	AST (U/L)	ALP (U/L)	γ GT (U/L)
NC	34.57 ± 2.22^{c}	42.36 ± 3.56^{d}	48.68 ± 3.28^{c}	4.48 ± 0.36^{d}
HC	65.91 ± 3.46^{a}	73.34 ± 2.83^{a}	88.11 ± 4.88^{a}	8.79 ± 1.15^{a}
ASBL1	50.04 ± 4.50^{b}	52.87 ± 4.43^{b}	64.73 ± 2.73^{b}	6.55 ± 0.413^{b}
ASBL2	36.29 ± 3.63^{c}	42.61 ± 3.51^{d}	52.22 ± 3.13^{c}	4.67 ± 0.440^{d}
ESBL1	47.84 ± 4.24^{b}	51.14 ± 4.37^{bc}	65.78 ± 5.44^{b}	6.16 ± 0.658^{bc}
ESBL2	35.97 ± 2.37^{c}	44.15 ± 5.21^{cd}	52.30 ± 3.17^{c}	4.84 ± 0.594^{d}
AESBL1	45.58 ± 3.07^{b}	53.19 ± 3.16^{b}	69.02 ± 4.26^{b}	6.28 ± 0.549^{b}
AESBL2	37.59 ± 4.26^{c}	43.27 ± 4.64^{d}	51.27 ± 3.36^{c}	5.05 ± 0.771^{cd}
LSD ($P \leq 0.05$)	6.16	6.99	6.73	1.15

Each value represents the mean ± SE. The mean values with different superscript alphabets indicate significant differences ($P \leq 0.05$) using LSD test

NC =Normol control HC = Hyperlipidemic control
ASBL1= Aqueous extract of Sweet Basil Leaves (0.35
ASBL2= Aqueous extract of Sweet Basil Leaves (0.70 g/Kg
ESBL1= Ethanolic extract of Sweet Basil Leaves (0.35
ESBL2= Ethanolic extract of Sweet Basil Leaves (0.70
AESBL1= Aqueous + Ethanolic extract of Sweet Basil
AESBL2= Aqueous + Ethanolic extract of Sweet Basil

Table (7)
Serum uric acid, urea and creatinine of hypercholesterolemic rats received two concentrations of sweet basil leaves extracts

Rat groups	Uric acid (mg/dl)	Urea (mg/dl)	Creatinine (mg/dl)
NC	5.63 ± 0.466^{b}	21.10 ± 2.67^{b}	0.636 ± 0.055^{b}
HC	9.19 ± 1.367^{a}	26.17 ± 3.29^{a}	0.931 ± 0.055^{a}
ASBL1	6.30 ± 0.954^{b}	22.44 ± 0.91^{ab}	0.643 ± 0.032^{b}
ASBL2	6.30 ± 0.634^{b}	20.96 ± 1.94^{b}	0.603 ± 0.020^{b}
ESBL1	6.69 ± 0.778^{b}	21.97 ± 1.93^{b}	0.546 ± 0.066^{b}
ESBL2	5.91 ± 0.669^{b}	21.22 ± 2.09^{b}	0.573 ± 0.045^{b}
AESBL1	6.27 ± 0.456^{b}	21.54 ± 2.52^{b}	0.613 ± 0.065^{b}
AESBL2	5.65 ± 0.554^{b}	21.52 ± 1.27^{b}	0.580 ± 0.088^{b}
LSD ($P \leq 0.05$)	1.36	3.80	0.098

Each value represents the mean ± SE. The mean values with different superscript alphabets indicate significant differences ($P \leq 0.05$) using LSD test

NC =Normol control HC = Hyperlipidemic control
ASBL1= Aqueous extract of Sweet Basil Leaves (0.35
ASBL2= Aqueous extract of Sweet Basil Leaves (0.70 g/Kg
ESBL1= Ethanolic extract of Sweet Basil Leaves (0.35
ESBL2= Ethanolic extract of Sweet Basil Leaves (0.70
AESBL1= Aqueous + Ethanolic extract of Sweet Basil
AESBL2= Aqueous + Ethanolic extract of Sweet Basil

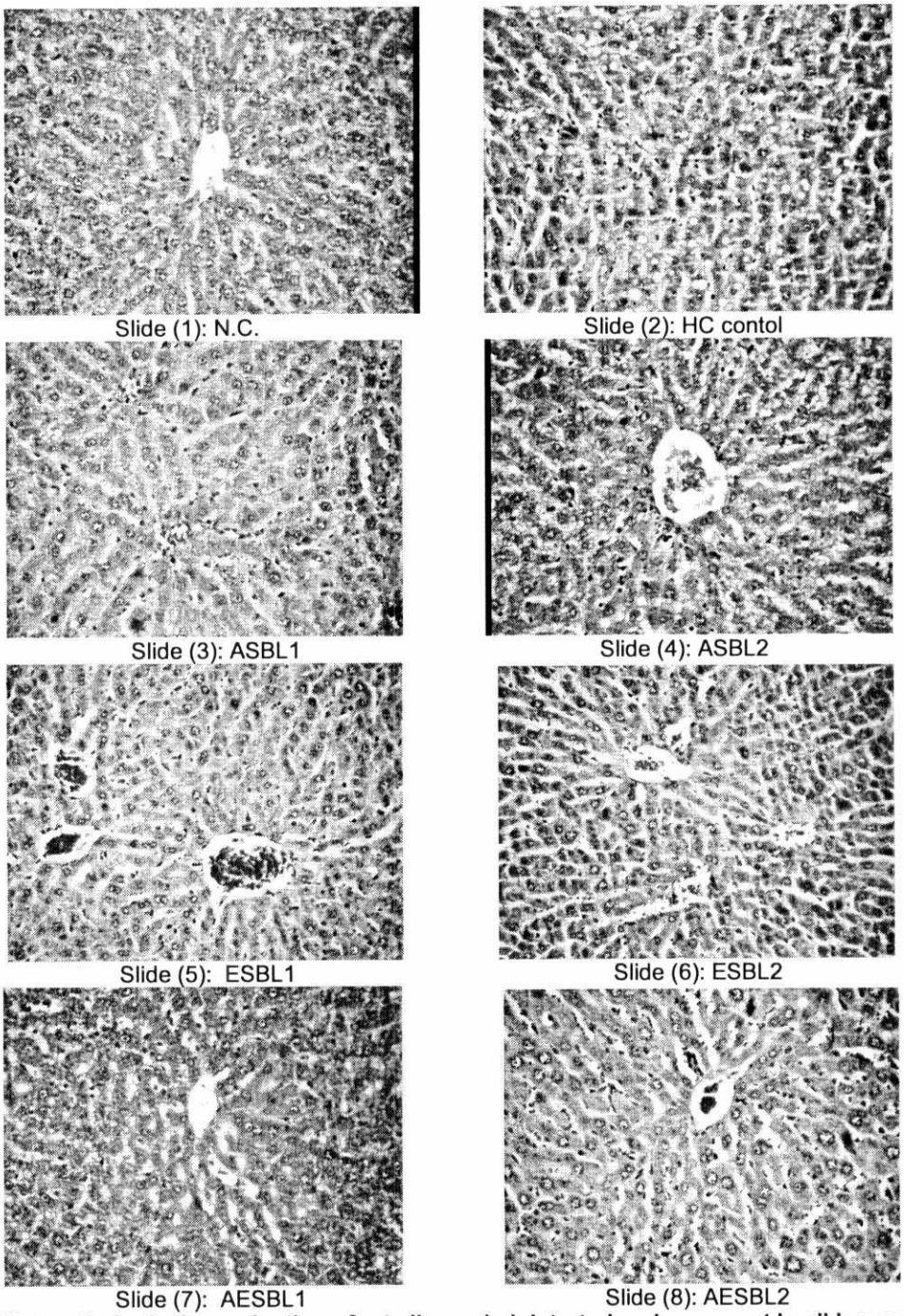

Fig. (1): Histopathological examination of rats liver administrated various sweet basil leaves extracts

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دراسه أثر مستخلصات الريحان على خفض كوليستيرول الدم

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الملخص العربي

هدف الدراسة هو اختبار أثر مستخلصات الريحان على خفض نسبة الكوليسترول فى حيوانات التجارب للحد من ارتفاع الكوليسترول و اثاره السيئه على صحه الأنسان. تم تحضير ثلاثة مستخلصات (مائى- ايثانولى – مائى و ايثانولى بنسبة ٣: ١) من أوراق الريحان. تم التعرف على تركيز الفينولات و الفلافينويد باستخدام كروماتوجرافى السوائل. تم أعداد تركيزان من كل مستخلص بعد تجفيف المستخلصات الأصلية حيث تم اذابتها فى حجم معين من الماء للوقوف على مدى تأثيرها فى خفض كوليسترول الدم. تم اعطاء ستة مجموعات من الفئران المغذاه على وجبه عاليه من الكوليسترول جرعه يوميه من تركيزان (٠,٣٥- ٠,٧٠ جرام/ كجم وزن جسم) لكل من المستخلص المائى – ايثانولى – مائى و ايثانولى من أوراق الريحان و مقارنتها بمجموعه ضابطه تتغذى على وجبه قياسيه خاليه من الكوليسترول و مجموعه تتغذى على الوجبه العاليه الكوليسترول لمده ٣٠ يوم. أظهرت النتائج أن الأعلى تركيز فى المركبات الفينوليه هو التجربه البيولوجيه أثبتت أن التركيز الثانى (٠,٧٠ جرام/ rutin. و أن الأعلى تركيز فى الفلافينويد هو *p*-Hydroxybenzoic acid كجم وزن جسم) من مستخلصات أوراق الريحان له تأثير ايجابى على خفض وزن الجسم. لا يوجد فروق معنويه فى الوزن فى الوزن النسبى للأعضاء ما بين مجاميع الفئران المختبره. triglycerides, total cholesterol, LDL-cholesterol and VLDL-cholesterol المستخلصات لها تأثير ايجابى لخفض ال Athergenic لسيرم الفئران المغذاه على وجبه عاليه من الكوليسترول و كذا خفض index (AI) و مستوى بيروكسيد الهيدروجين فى السيرم. لا يوجد أختلاف معنوى فى نشاط أنزيمات الكبد بين (malondialdehyde, MDA) المجاميع المختبره بأعطاء التركيز الثانى (٠,٧٠ جرام/ كجم وزن جسم) من مستخلصات أوراق الريحان و بين مجموعه ضابطه تتغذى على وجبه قياسيه خاليه من الكوليسترول . لا يوجد أختلاف معنوي فى وظائف الكليه ما بين المجاميع المختبره. أظهرت الدراسه الهستوباثولجيه أن التركيز الثانى (٠,٧٠ جرام/ كجم وزن جسم) من مستخلصات أوراق الريحان له القدره على التخلص من الأثر السلبى للوجبه عاليه الكوليسترول. و يتضح من ذلك أن المستخلصات الثلاث لأوراق الريحان لها القدره على خفض الكوليسترول فى الفئران المغذاه على وجبه عاليه من الكوليسترول و خصوصا التركيز الثانى.