Anti-Atherosclerotic Activity of Inulin Extracted from *Cichorium Intybus* Roots on Hypercholesterolemic Rats

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Abstract:

Atherosclerosis-associated cardiovascular diseases are main causes leading to high mortality worldwide. Hypercholesterolemic is the primary risk factor of cardiovascular disease (CVD). Inulin is considered as functional food ingredients since they affect physiological and biochemical processes in rats and human beings, resulting in better health and reduction in the risk of many diseases. The main sources of inulin that are used in the food industry are chicory. Rats were divided into two main group, the first group negative control group (5 rats) fed on basal diet for all experimental period as control negative group. Hypercholesterolemic rats were divided into 4 subgroups (5 per each): subgroup 1 served as positive control group, subgroup 2, 3 and 4 were fed basal diet and given orally one receives a daily gavage 0.25, 0.5 and 1g of inulin respectively for 30 days. After the end of experiment period, blood samples were collected for used to the biochemical analysis lipids profile and antioxidant status. The results indicated that treatments with 1g of inulin significantly (p≤0.05) improved lipid profile and compared to positive control group and other treated groups. In Conclusion :The treatment with inulin had ability to decreasing of serum and liver lipid profiles(TC, TG and TL) , LDL, VLDL- Cholesterol, fecal lipid profile and bile acid, and increase level of HDL of serum of hypercholesterolemic rats.

Key words: Chicory root, inulin extract, hypercholesterolemia

Introduction

Atherosclerosis is a chronic inflammatory disease of the arteries, which may ultimately prevent adequate blood flow to target tissues leading to cardiovascular complications including heart attack and stroke. A well-known risk factor in humans is

hypercholesterolemia, i.e., elevated total cholesterol (TC) and low-density lipoprotein cholesterol (LDL c)(WHO, 2017), and other important contributors to this disease includes inflammation, oxidative stress and insulin resistance(Hansson and Libby, 2006), (Gibson and Roberfroid, 1995 ;Hansson, 2005). Foods rich in saturated fat and cholesterol have been linked to elevations in circulating cholesterol level(Orford et al., 2000). Lipid-enriched diets are often used to induce or accelerate the rate of atherosclerotic lesion in murine models of atherosclerosis(Getz and Reardon, 2006).Hypercholesterolemia is one of the main underlying risk factors of atherosclerosis development and is routinely treated by prescription of statins. However, statin treatment lowers total plasma cholesterol levels by approximately 30%(Chan et al., 2014)and only prevents 25–45% of all cardiovascular events (Jukema et al., 2012), indicating the demand for additional therapies. Therefore, early dietary intervention in healthy subjects, using prebiotics, may be a reliable preventive approach to delay the onset and related complications of atherosclerosis(Lara et al., 2007).

A type of dietary fibers that received great attention in the last decade is inulin-type fructans. Inulin is a dietary fiber that meets the three classification criteria for being considered as a prebiotic (Gibson and Roberfroid, 1995), i.e., it is resistant to hydrolysis by human enzymes and therefore minimally absorbed in the gastrointestinal tract, it is fermented by colonic microbiota, and it selective stimulates the growth and/or activity of beneficial colonic bacteria (Videla et al., 2001). Inulin is a soluble non-digestible carbohydrate studied for its health benefits such as improved bowel movements, lowering blood glucose levels and potential lipid modulating effects (Firmansyah et al, 2016). Inulin is naturally available in many types of plants. As ingredient for the food industry native inulin is mainly extracted from chicory roots (Loo, 2012).Evidence is increasingly indicating that inulin exerts favorable effects on a variety of immune-related diseases, such as inflammatory bowel disease (Videla et al., 2001), rheumatoid arthritis (Abhari et al, 2016), and on low-grade chronic inflammation that is associated with cardiovascular disease in humans (Vogt et al, 2015). Furthermore, there are indications that inulin has beneficial effects on hyperlipidemia a in rodents (Rault-Nania et al, 2006). The previous studies in rodents showing beneficial effects of inulin on hyperlipidemia and atherosclerosis were performed in LDL-receptor knockout mice, which are both models characterized by severely hampered lipoprotein remnant metabolism. Also the consumption of inulin has been associated with improved lipid profile and consequently, reduction of cardiovascular risk which inulin can act directly on lipid meta hypothesized that the consumption of inulin-type fructans reduces the de novo synthesis of fatty acids in the liver (Dos Reis et al., 2015).Therefore the present study investigated the influence of inulin extracted from chicory roots to improve lipid profile and atherogenic indices in hypercholesterolemic rats.

Materials and Methods:

Materials:

Chicory roots (Cichorium intybs) , inulin was isolated from the chicory roots., Pure cholesterol was obtained from Winlab (UK), cholic acid was obtained from Biomark (India) and methyl thiouracil was purchased from Sigma– Aldrich (USA). Any other chemical used was of the highest analytical grade. Kits of total cholesterol (T.C), triglyceride (T.G), were purchase from SPINREACT, S.A.U. Ctra. Santa Coloma, Spain and obtained from El-Gomhoreya company, Cairo, Egypt.

Animals:

Fifty-five adult male Sprague–Dawley rats weighing (120+5 g), at the beginning of the experiment, were obtained from Medical Insects Research Institute, Doki, Cairo, Egypt. Rats were housed individually in wire cages under the normal laboratory conditions. Rats were fed on standard diet for one week as an adaptation period. Diet was introduced to rats in special food cups to avoid scattering of food. Also, water was provided to rats by glass tubes projecting through the wire cages from an inverted bottle supported to one side of the cage. Food and fresh water were provided checked daily. Standard diet was prepared from fine ingredients according to AIN-93 guidelines (Reeves et al., 1993).Body weight were checked every week and fecal were collected every day. All animals were received care in compliance with the Egyptian rules for animal protection.

Methods:

2.1. Extraction of inulin from chicory:

Viable processes for inulin extraction have been reported by Hébette et al., (1998) and Franck, (2002).After cutting the root (207 g) into small pieces, distilled water (150 ml, 70 °C) was added and the root homogenized by blending for 1 minute. An additional 900 ml distilled water(70 °C) was added and heated (with continuous stirring) for 1 hour at 70 °C (the reaction vessel covered with parafilm to prevent excessive evaporation). The solution was filtered through cheesecloth and 0.1 M Ca(OH)2 (70 ml) added to raise the pH to 8, and subsequently lowered to pH 7 using 0.8 M HCl. The pulp was re-suspended in 500 ml distilled water and the process repeated. This cycle required only 3.8 ml 0.1 M Ca(OH)2to raise the pH to 8 and returned to 7 utilizing 0.8 M HCl. Precipitates formed were filtered off and the solutions stored at -10 °C for 7 hours. Upon thawing two distinct layers could be observed, a pasty off white suspension and a dark brown top layer. These were separately concentrated using a rotavapor (40 °C), under reduced pressure, and collected as a dry powder after freeze-drying. A total amount of 1050 ml distilled water was used for the extraction process (Mavumengwana, 2004).

Induction of hypercholesterolemia :

Hypercholesterolemia was induced in obese rat by feeding high cholesterol diet [4% cholesterol (w/w) and 1% cholic acid (w/w)] for 8-weeks (Kameshand Sumathi, 2012). The hypercholesterolemia diet introduced in normal routine rat feed.

Experimental design

Rats were randomly divided into two main group, the first group negative control group (5 rats) fed on basal diet for all experimental period as control negative group. And second group (hypercholesterolemic rats, which the period of 8-weeks was considered as lead-in period to introduce hyperlipidemia in rats). were divided into 4 subgroups (5 per each): subgroup 1 served as positive control group, subgroup 2, 3 and 4 were fed basal diet and given orally one receives a daily gavage 0.25, 0.5 and 1g of inulin respectively for 30 days. After the end of experiment period. The organ (liver) was removed and washed in saline solution, weighted and stored in (10%) formalin solution according to methods described by(Drury and Wallington, 1980).

Biochemical assays:

The serum levels triglyceride (TG), total cholesterol (TC). High-density lipoprotein cholesterol(HDL-c) were measured with an automatic analyzer using a diagnostic kit for each according to Fossati and Prencipe, (1982); Allain et al., (1974); and Burstein et al., (1980) respectively. Low-density lipoprotein cholesterol (LDL-c) was calculated from Friedewald formula LDL = TC - HDL - TG/5.0 (mg/dL) (Friedewald et al., 1972). The Atherogenic ratios were calculated as follows: Atherogenic Index of Plasma (AIP) = log TG/HDLc, Cardiac risk ratio (CRR), = TC/HDLc Castelli's Risk Index (CRI) = LDLc/HDLc Atherogenic Coefficient (AC) = (TC– HDLc)/HDLc according to(Bhardwaj et al., 2013) and Atherogenic fraction (AF) was calculated as the difference between TC and HDL-C according to(Aguilar et al., 2011).

Analyses of hepatic and fecal lipid profile:

The content of hepatic total lipids, triglyceride and total cholesterol were analyzed as described previously (Bligh and Dyer, 1959; Shahdat et al., 2004 and Hossain et al., 2011). Hepatic total lipids, triglyceride and total cholesterol content were expressed as mg/dl of protein. Bile acid of feces were extracted by the method of Tokunaga et al ., (1986), and the extracted solutions were used to determine bile acid concentration enzymatically by the method of(Mashige et al., 1981).

Statistical analysis:

Results were expressed as the mean ± SD. Data for multiple variable comparisons were analyzed by one-way analysis of variance (ANOVA) using SPSS for Windows, version 19.0 (SPSS Inc., Chicago, IL, USA). For the comparison of significance between groups, Duncan's test was used as a post hoc test according to the statistical package program (Artimage and Berry, 1987).

Result and discussion:

Data presented in Table (1) illustrated the effect of inulin on serum lipid profile of normal and hypercholesteremic rats. The results showed that there was high significantly increase(P≤0.05) in the levels of total lipid(T.L), triglyceride (T.G), Total cholesterol(T.C), low density lipoprotein (LDL) and very low-density lipoprotein (VLDL)for hypercholesterolemic rats which received basal diet alone (positive control) during duration of the experimental compared to those of the normal control rats (negative control group) and other treated hypercholesterolemic groups while HDL had opposite trend. Similar trend was observed for (Makni et al., 2008) who reported that cholesterol- enriched diet resulted in a significant increase in total cholesterol, total lipids, phospholipids and triacylglycerol in plasma and liver and LDL-C levels, with decreased circulating HDL-C, thus providing a model for dietary hyperlipidemia .Any changes in the levels of lipids make the individuals more inclined to develop atherosclerotic cardiovascular diseases as well as endothelial dysfunction (Parinita, 2012). The treatment of 1g of inulin was more effective in reducing the levels of TL , TG and TC compared to other concentrations of inulin. Serum TG concentrations were even light significant difference reduced in rats fed on inulin .5 g and inulin .25 g which were (85.2 , 114.6 mg respectively. Similar changes in the TC which were 144.54 and 146.18 mg respectively .These findings are supported by Guo et al., (2012) who showed that the intake of inulin resulted in a significant decrease in total cholesterol, LDL.C and triglyceride concentrations in the serum of hyperlipidemic patients treated with inulin compared with controls. Inulin is a soluble and viscous compound that can increase the thickness of the unstirred layer of the small intestine and thus inhibit the absorption of cholesterol (Dikeman et al ., 2006).The proposed hypocholesterolemia mediation effect by inulin is via two mechanisms: decreasing the cholesterol level absorbed by intestinal epithelial cells and improving the production of short-chain fatty acids fermented by intestinal bacterial microflora (Ooi and Liong, 2011) . No significant (P≥ 0.05) differences were observed in LDL and VLDL between hypercholesteremic rats treated with .25g and 1g of inulin and then HDL and LDL between hypercholesteremic rats treated with .5g of inulin . Also, the treatment of 1g was more effective (P≤0.05) in the raising the levels of HDL and reducing the levels of LDL and VLDL compared to other concentrations. The result of this study came in accordance with that reported by(Kim

and Shin, 1998) reported that rats fed chicory extract and inulin diets had significantly higher serum high density lipoprotein (HDL) cholesterol and generally lower low density lipoprotein (LDL) cholesterol concentrations, thus significantly greater ratios of HDL/LDL cholesterol compared with the controls. Many human and animal experiments have demonstrated that inulin appears to be more effective in decreasing the level of triglyceridemia than cholesterolemia in hypertriglyceridemic conditions. With regard to the mechanism of action, it is suggested that oral inulin can modify the insulin levels and the absorption of macronutrients, as well as increase the production of fermentation end products and change the gut's peptide production (Roberfroid, 2007).

Table (1):Effect of inulin on serum lipid profile of normal and hypercholesterlemic rats

| | Negative control group | Hypercholesterolemic groups | | |
| | | Inulin(g) | | |
		Positive control group	0.25	0.5	1.0
TL (mg/dl)	$349.1^d \pm 13.9$	$491.4^a \pm 6.2$	$466.8^b \pm 8$	$451.0^b \pm 22.5$	$405.1^c \pm 12.5$
TG (mg/dl)	$75.0^d \pm 3.5$	$135.2^a \pm 3.7$	$114.6^b \pm 9.98$	$85.2^c \pm 0.8$	$84.2^c \pm 3.3$
TC (mg/dl)	$120.0^d \pm 4.1$	$156.0^a \pm 1.67$	$146.7^b \pm 4.19$	$144.5^b \pm 2.3$	$135.5^c \pm 2.3$
HDL (mg/dl)	$39.2^a \pm 1.51$	$25.8^d \pm 1.31$	$33.5^c \pm 1.84$	$36.2^b \pm 1.5$	$38.8^a \pm 1.7$
LDL (mg/dl)	$65.6^d \pm 4.2$	$103.2^a \pm 2.99$	$90.2^b \pm 6.17$	$91.34^b \pm 2.7$	$79.8^c \pm 2.0$
VLDL(mg/dl)	$15.0^d \pm 0.71$	$27.0^a \pm 0.74$	$22.9^b \pm 1.99$	$17.0^c \pm 0.15$	$16.8^c \pm 0.7$

Data are expressed as mean ± SD. Values within a row having different superscripts are significantly different (p ≤ 0.05); where the small letters indicate significant among dietary treatment groups as indicated by one-way ANOVA followed by Duncan's multiple range test (a > b > c > d > e).

The results of table (2) showed the effect of Inulin on atherogenic indices of control and hypercholesterlemic rats in normal and hypercholesterlemic rats. It is known that high levels of cholesterol and LDL-cholesterol (LDLc) have an important role in the pathogenesis of atherosclerosis. A significant (P≤0.05) increase in an atherogenic indices and LDL-c/HDL.-c Ratio (HTR) in normal and hypercholesterolemic rats. A significant increase (P≤0.05) in an atherogenic Index (AI), cardiac risk ratio (CRR), Castelli's Risk Index (CRI), Atherogenic fraction (AF) and Atherogenic coefficient (AC) were observed in hypercholesterolemic rats compared to normal rats. However, HTR of hypercholesterolemic rats were lower (P≤0.05) than normal rats. These results could be attributed high serum levels of TC, TG, LDL-c and VLDL.-c as well as lower levels of HDL which was observed in hypercholesterolemic rats. The present results were in

accordance with those of (Dehghanet al., 2013) observed inulin consumption caused a significant decrease in TC(12.90%), TG (23.60 %), LDL-c (35.30 %), LDL-c/HDL-c ratio (16.25%) and TC/HDL-c ratio (25.20%) in intervention compared to control group. The inulin supplementation significantly increased HDL-c (19.90%) compared to the control group after adjusting for dietary intakes and baselines values .On the other hand, Atherogenic index (AI) and (AIP) were significantly reduced (P≤0.05) in hypercholesterolemic rats treated with 0.25 g , 0.5 g and 1g compared with hypercholesterolemia rats which untreated (positive control group (0g inulin) while HTR had an opposite trend. However, treatments with 0.25g of inulin had not significantly decreased (P≤0.05) in CRR, CRI, AF and AC in hypercholesterolemic rats in comparison to hypercholesterolemic rats untreated (positive control group). Treatment with 0.5g and 1g of inulin had not significantly decreased (P≥0.05) in between CRR and AC and CRI and AF in hypercholesterolemic rats in comparison to hypercholesterolemic rats untreated (positive control group).Moreover, the highest reduction (P≤0.05) in AI, CRR , CRI, AF , AIP and AC and the highest elevation (P≤0.05) in HTR were observed in hypercholesterolemic rats treated with 1g of inulin compared to other concentrations.

Table (2) :Effect of Inulin on atherogenic indices of control and hypercholesterlemic rats .

	Negative control group	Hypercholesterlemic groups Inulin(g)		
		Positive control group	0.25	0.5	1.0
AI	2.06 [e] ± .2	5.04 [a] ±0.36	3.4 [b] ±.26	3.02 [c] ± .19	2.5 [d] ± .16
CRR (mg/dl)	3.06 [e] ±.2	6.04[a] ±0.36	4.4 [b] ± .27	4.02 [c] ±.19	3.49 [d] ±.16
CRI (mg/dl)	1.68 [e] ±.13	4 [a] ± 0.32	2.7[b] ±.3	2.5[b] ±.19	2.08 [c] ±.13
AF	80.76 [d] ± 4.8	130.2[a]±2.7	113.2[b]±4.9	108.4[b]± 2.77	96.66 [c] ± 2.34
HTR%	.33 [a] ± .02	.16 [e]± .01	0.23 [d] ± .1	.25 [c]± .02	.29 [b] ± .01
AIP	.28 [e] ± .03	.7 [a] ± .03	0.34 [d] ± .02	.37 [c] ± .02	.53 [b] ± .04
AC (mg/dl)	2.06 [e] ±.21	5.04[a] ± .36	3.38 [b] ± .26	3.02 [c] ±.19	2.5 [d] ±.16

Data are expressed as mean ± SD. Values within a row having different superscripts are significantly different (p ≤ 0.05); where the small letters indicate significant among dietary treatment groups as indicated by one-way ANOVA followed by Duncan's multiple range test (a > b > c > d > e). Atherogenic Index (AI) = log TG/HDLc, Cardiac risk ratio (CRR) = TC/HDLc, Castelli's Risk Index (CRI) = LDLc/HDLc, Atherogenic fraction (AF) was calculated as the difference between TC and HDL-C, HTR = HDL/TC Ratio and Atherogenic Coefficient (AC) = (TC– HDLc)/HDLc .

The effect of Inulin on liver lipid profile of control and hypercholesterlemic rats showed in Table (3) .The results showed that there was high significantly increase(P≤0.05) in the levels of hepatic total lipid(T.L), hepatic triglyceride (T.G) and hepatic total cholesterol(T.C) for hypercholesterolemic rats which received basal diet alone (positive control) compared to those of the normal control rats (negative control group) and other treated hypercholesterolemic groups . The treatment of 1g of inulin were more effective (p≤0.05) in reducing the levels of hepatic T.L compared to other treatments, followed by .5g inulin, followed by .25g of inulin. Also, the treatment of 1g and .5g of inulin were more effective to improve the level of hepatic T.G and T.C compared to other treatments which were similar, followed by .25g inulin treatment .The obtained results agree with the previous findings by(Dos Reis et al ., 2015)who observed that animals and humans fed diets containing inulin-type fructans had lower serum levels and/or hepatic triglycerides (TG) .Similar trend was observed for(Sugatani et al., 2012)who reported that dietary inulin alone was effective to prevent the development of hepatic steatosis.

Table (3) :Effect of Inulin on liver lipid profile of control and hypercholesterlemic rats .

	Negative control group	Hypercholesterlemic groups Inulin(g)			
		Positive control group	0.25	0.5	1.0
hepatic TL(mg/dl)	220[e]±3.8	446.6[a]±4.3	406[b] ± 11.9	342 [c] ±1.6	290 [d] ± 15.8
hepatic TG (mg/dl)	75[d]±4.1	125.6[a]±12.1	105 [b] ± 4.1	90.8 [c] ± 5	83.8 [c] ± 3.2
hepatic TC (mg/dl)	7.12[c]±0.4	17.8 [a]±3.7	13.7[b] ± 1.9	10 [c] ± 2.5	8 [c] ± 2.3

Data are expressed as mean ± SD. Values within a row having different superscripts are significantly different (p ≤ 0.05); where the small letters indicate significant among dietary treatment groups as indicated by one-way ANOVA followed by Duncan's multiple range test (a > b > c > d > e).

Data in table (4) recorded the effect of Inulin on fecal lipid profile excretion and fecal bile acid of normal and hypercholesteremic rats. Cholesterol can be eliminated from the body as fecal neutral sterols and as fecal bile acids. Bile acids are produced as a result of metabolic conversion of cholesterol in the liver and are secreted into the intestine via the biliary pathway(Mistry et al., 2018).Bile acids are a group of gut microbiota-derived metabolites involved in various metabolic diseases, which are stored in the gallbladder and released into the intestine to facilitate the absorption of dietary lipids and fat-soluble vitamins (Parseus et al., 2017).

The results indicated that there were a significant decrease (P ≤0.05) in fecal TC, TG , VLDL , LDL and fecal bile acid in hypercholesterolemic group which received basal

diet alone (positive control group) as compared to negative control and another treated group. The levels of TC, TG, LDL-c and VLDL-c in fecal are significantly (P≤0.05) increased in hypercholesterolemic rats treated with different concentrations of inulin compared with untreated hypercholesterolemic rats (positive control group). Also, the treatment of 1 gm inulin was more effective (p≤0.05) in increasing the levels of fecal TC, TG, LDL-c , VLDL.-c and fecal bile acid compared to other concentrations, this increase in fecal cholesterol excretion in rats fed inulin might be caused by a reduction in cholesterol absorption might result in higher cholesterol catabolism in the liver, which causes lower plasma cholesterol concentration. This results was agreed with(Nakamura et al., 2017)who observed that fecal excretion of lipids markedly potentiated by Fructooligosaccharides (FOS) consumption thus indicated the suppression effect of dietary FOS to the high-fat diet-induced body fat accumulation, and intestinal absorption of dietary fat. Similar trend was observed forDos Reiset al. (2015) who observed that rats fed extract and inulin had significantly greater fecal lipid, cholesterol and bile acid excretions than those fed fiber-free diets, Also who reported that inulin-type fructans do not seem to be able to bind the bile acids present in the intestinal lumen , however the fermentation of inulin-type fructans in the intestinal mucosa leads to the production of organic acids, reducing the pH in the intestinal lumen. Thus, the bile acids become less soluble and may be eliminated with the feces, which reduces their intestinal absorption. These findings are supported by Catryet al. (2018), who reported that prebiotics can impact the composition of the intestinal microbiota potentially resulting in altered bile acid profiles.

Table (4) :Effect of Inulin on fecal lipid profile excretion and fecal bile acid of normal and hypercholesterlemic rats .

	Negative control group	Hypercholesterlemic groups Inulin(g)		
		Positive control group	0.25	0.5	1
TC (mg/dl)	20[a] ± 1.6	8.5 [d] ± .79	9.48[cd] ± 1.8	11.12 [c] ± 1.7	16.5 [b] ± 1.9
TG (mg/dl)	23.2[a]± 1.6	7.4[e] ± 1.7	10.11 [d] ± 1.7	17.96 [c]± 1.5	20.6 [b] ± 1.98
VLDL(mg/dl)	4.6[a]± .48	1.4[e] ±.32	2 [d] ± .33	3.6 [c] ±.3	4.1 [b] ±.38
LDL (mg/dl)	8[a]±1.4	4.2[b] ± .8	4.44[b]± 1.6	4[b] ± 2	7.0 [a] ± 3
Fecal Bile acid (nmol/mg)	0.38 [a] ± .02	0.15 [e] ± .02	0 .2[d] ±. 03	0.23 [c] ±.01	0. 3[b]± .02

Data are expressed as mean ± SD. Values within a row having different superscripts are significantly different (p ≤ 0.05); where the small letters indicate significant among dietary treatment groups as indicated by one-way ANOVA followed by Duncan's multiple range test (a > b > c > d > e).

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النشاط المضاد لتصلب الشرايين للأنيولين المستخلص من جذور الهندباء على الفئران المصابة بارتفاع كوليسترول الدم

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تعد أمراض القلب والأوعيه الدمويه المرتبطه بتصلب الشرايين من الأسباب الرئيسيه المؤديه الى إرتفاع معدل الوفيات فى جميع أنحاء العالم. وارتفاع كوليستيرول الدم عامل الخطرالرئيسى لأمراض القلب والأوعيه الدمويه. يعتبر الأنيولين من مكونات الأغذيه الوظيفيه والتى يكون لها تأثيرعلى العمليات الفسيولوجيه والبيوكميائية فى الفئران والبشر فى تحسين الصحه والحد من مخاطر العديد من الأمراض. والمصادر الرئيسيه للأنيولين التى تستخدم فى صناعه الأغذيه هى الهندباء .تم تقسيم الفئران إلى مجموعتين رئيسيتين المجموعه الأولى (5 فئران) والتى تغذت على الوجبه الأساسيه طول فتره التجربه كمجموعه ضابطه سالبه .قسمت الفئران المصابه بارتفاع كوليسترول الدم إلى أربع مجموعات فرعيه (5 فئران).المجموعه الفرعيه الأولى مجموعه ضابطه موجبه والمجموعات الفرعيه الثانيه والثالثه والرابعه تم تغذيتهم على الوجبه الأساسيه بالإضافهإلى الأنيولين الذى أعطى عن طريق الفم(25, -5 , -1جم) على التوالى يوميا لمده 30 يوم.وبعد إنتهاء فتره التجربه , تم جمع عينات الدم لإجراء التحاليل البيوكميائيه دهون الدم وحاله مضادات الأكسده .وأشارت النتائج أن المعامله 1جم أنيولين حسنت معنويا دهون الدم عند مقارنتها بالمجموعه السالبه ومجموعات المعاملات الأخرى . الخاتمه : المعامله بالأنيولين أدت إلى خفض الدهون فى السيرم والكبد (TC, TG, TL) وLDL, VLDL كوليستيرول ودهون البراز وأملاح الصفراء وزياده مستوى HDL فى السيرم للفئران المصابه بارتفاع كوليسترول الدم .

الكلمات الافتتاحيه :جذور الهندباء، مستخلص الأنولين، ارتفاع الكوليستيرول