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Some of Biological and HistopathologicalEffects of Cooked Red and White Beans (*Phaseolus Vulgaris* L.)Consumption on Obese Rats

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Abstract :

The present study was conducted to investigate some biological effects of cooked red and white beans (*Phaseolus Vulgaris* L.) on obese rats.The obtained results showed that obese-hyperlipidemic rats have significant ($p<0.05$) increase in body weight, % change of body weight, serum levels of total lipids, triglycerides, total cholesterol, LDL-c, blood glucose, leptin hormone (LH), malondialdehyde (MDA)andactivities ofaspartate aminotransferase (AST), alanine aminotransferase (ALT) andalkaline phosphatase (ALP) enzymesas well asvalue of atherogenic index,and have significant ($p<0.05$) decrease inserum HDL-c, insulin, reduced glutathione levels and serum activities of glutathione peroxidase (GPx), superoxide dismutase (SOD) and catalase (CAT) enzymes, compared to those of the normal rats. However, administration of both cooked red and white beans to obese-hyperlipidemic rats significantly ameliorated all of the above parameters, compared to that of untreated obese-hyperlipidemic rats. In addition, the obtained result of histopathological examination showed that obese-hyperlipidemic rats have dilated blood vessel with thick muscle wall and intramuscular hemorrhages in heart. While, vasculitis with thick inflamed wall and perivasculitis with dilated and congested blood vessel in were observed in aorta. However, administration of red or white beans caused partially

improvement in hear and aorta sections, which more detectable in red beans, compared to those in untreated-obese rats.This study concluded that the white or red beans have antiobesity, antihyperlipidemic and antihyperglycemic potential and ameliorate the antioxidant-defense system in rats fed on high- fat diet.

Keywords: Beans (*Phaseolusvulgarisseeds* L) - High-fat diet - Antioxidant Enzymes - Lipid Profile -Lipten Hormone - Histopathological Study

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1. Introduction :

Obesity is one of the most frequently accomplished medical problems **(Kramer and Luke, 2007)**. It is a pathological condition in which excess body fat accumulates to the extent that it may have a reverse effect on health, leading to reduced life expectancy or increased health problems (**Haslam and James, 2005**). The prevalence of obesity has been attributed to the changes in the life style of western societies; especially important among them the consumption of high-fat diets **(Klaus, 2005)**.Obesity is considered malnutrition that may encourage many diseases such as hypertension, cardiovascular diseases, kidney diseases, liver failure and even some cancer types **(Watanabe *et al.*, 2007)**. Imbalanced meal composed of a diet rich in fats causes the development of hepatomegaly **(Oldenburg and Pijl, 2001)**, fatty liver disease **(Altunkaynak, 2005),** dyslipidemia, abdominal obesity **(Innis, 2007)** and splenomegaly **(Altunkaynak*et al.*, 2007)**. Fat is the dietary nutrient with the greatest energy density since it provides 9kcal/g, while carbohydrate and protein provide 4kcal/g. Consequently, increased fat intake promotes high energy consumption (**Schrauwen and Westerterp, 2000**) and considered to be the most important factor that contributes to the current epidemic of obesity **(Bray *et al.*, 2004)**. The continuous consumption of high amounts of fat is directly related to hyperlipidemia in humans. Hyperlipidemia is a major cause for

atherosclerosis, coronary artery, ischemic cerebrovascular and peripheral vascular diseases **(Badimon*et al.*, 2010)**.

Beans (*Phaseolus vulgaris* L.) have a notable place in the folklore throughout the world and in the traditions of many cultures such as its antidiabetic activity **(Carai*et al.*, 2009)**. The species *Phaseolus vulgaris* includes all types of legume seeds normally known as common beans **(Geil** and **Anderson, 1994)**. There is increased gaining attention as a functional or food containing health-giving additives and having medicinal benefit, due to its rich variety of phytochemicals which have health benefits **(N'guessan, 2008)**. The active principle compounds included alkaloids, cyanogenic glycosides, flavonoids, saponins, tannins, terpenes and steroids **(Luka *et al.*, 2013)**. Several researchers reported that common beans enhance anticarcinogenic effects**(Hangen and Bennink, 2002)**, bifidogenic**(Queiroz-Monici*et al.*, 2005)**, antioxidant effects **(Heimler*et al.*, 2005)** and reduce glycemia and glucose absorption in laboratory animals **(Tormo*et al.*, 2004)**.

Nowadays, nutrition as a science needs to extend its basic functions, such the prevention of dietary deficiency, the establishment of nutrition standards and dietary guidelines into the new notion focused on minimizing the risk of diet-related diseases associated with either excess or deficiency of some nutrients. Growing expectations of consumers interested in benefits resulting from nutrition, expected to support the disease control and prevention.

The present study was conducted to investigate some biologicaland histopathologicaleffects of cooked red and white beans consumptionon obese rats.To achieve the aim of this study, body weight and atherogenic index, and serum lipid profile, blood glucose, insulin and leptin hormone, AST and ALT, ALP, malondialdehyde (MDA) and reduced glutathione(GSH) levels and serum activities of glutathione peroxidase GPx, superoxide dismutase (SOD) and catalase (CAT)enzymes were investigated as well as histological structure of heart and aorta were studied.

2. Materials and methods :

Materials:

White and red beansseeds:

Dry white and red beans seeds(*Phaseolus Vulgaris* L.)were purchased from the Agricultural Seeds, Herbs and Medicinal Plants Company, Cairo,Egypt.

Rats and diet :

Forty two male adult albino rats of Sprague-Dawley strain weighing 150 ± 5 g were purchased from the Laboratory Animal Colony, Helwan, Egypt. Basal diet constituents were obtained from El-GomhoryaCompany for Trading Drugs, Chemicals and Medical Instrument, Cairo, Egypt.

Kits:

Kits for biochemical assay of serumtotal lipid (TL), total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-c) and high density lipoprotein cholesterol (HDL-c), blood glucose (BG), insulin, leptin hormone (LH), aspartate aminotransferase (AST) and alanine aminotransferase (ALT), alkaline phosphatase (ALP),malondialdehyde (MDA) and reduced glutathione (GSH) levels, serum activities of glutathione peroxidase (GPx), superoxide dismutase (SOD) and catalase (CAT) enzymes were obtainedfrom the Gamma Trade Company for Pharmaceutical and Chemicals, Dokki, Giza, Egypt.

Methods:

Preparation of beans :

Dry white and red beans were cleaned from foreign materials and washed with tap water to remove possible potential dust. Then, both of them were soaked separately in water for 12 hr. with change soaking water every 2 hr. After 12 hr., soaked beans were cooked in boiling

water (1:5 w/v). The cooked beans were drained to remove the cooked water and dried at 50°C in an oven vacuum for 5 hr. Then, dried cooked beans were ground using grinder mill to obtain beans powder and stored in an air tight container at-10°C until used as feed for the rats.

Preparation of basal :

Basal diet (AIN-93M) was prepared as reported by **Reeves *et al.*,(1993)**. It consisted of casein 20%, soybean oil 5%, choline chloride 0.20%, vitamin mixture 1.0%, mineral mixture 4%, fibers 5%, L-Cystine 0.18%, sucrose 10% and the reminder was corn starch.

Experimental Design:

All rats were housed at a room temperature of 25 ± 2 °C, relative humidity of 50–55% and 12 hr. light/12 hr. dark cycles in animal house of the Faculty of Home Economics, Helwan University, Cairo, Egypt for one week for acclimatization. After acclimatization period (one week), forty two adult male rats were randomized into six equal groups, each of seven rats. Group 1 was fed on basal diet and kept as negative control group. The other five groups were fed on high- fat diet (supplies 59% calories from fat; 21% calories from carbohydrate and 20% calories from protein) for four weeks to induction of obesity and hyperlipidemia according to **Bhatt *et al.*, (2006)**. Thereafter, rats were grouped as following:

Group 2: Rats were fed on HFD and kept as obese-hyperlipidemic rats (positive control group).

Group 3: Rats were fed on HFD enriched with 10% of cooked red beans (CRB).

Group 4: Rats were fed on HFD enriched with 20% of CRB.

Group 5: Rats were fed on HFD enriched with 10% of cooked white beans (CWB).

Group 6: Rats were fed on HFD enriched with 20% of CWB.

At the end of experiment period (8 weeks), animals were fasted for 12-hr., except of water and then rats were sacrificed. Blood samples were collected from the posterior vena cava into dry clean centrifuge tubes and left at room temperature to clot and then centrifuged for 15 minutes at 4000 rpm for serum separation. Serum samples were

carefully aspired using a needle and transferred into dry clean test tubes and frozen at -20°C for biochemical analysis. Heart and aorta were removed,washed with saline solution, dried and immersed in neutral buffered formalin 10% for histopathology examination.

Estimation of feed intake, body weight gain and percent change of body weight :

Feed intake (FI) was determined every day. The changes in body weight were determined by weighing the animals on a balance scalederuring the experiment.Initial body weight (IBW), weight after the first four weeks and final body weight (FBW) at the end of experiment. The biological value of diets was assessed by the determination of its effect on body weight gain (BWG) and percent change of body weight gain were calculated using the following formula:

BWG = Final Body Weight - Initial Body Weight

% change of body weight gain = BWG/IBW X 100

Estimation of lipid profile :

Serum levels of total lipids (TL)and triglyceride (TG) wereestimatedas described by(**Siedel, 1993**), total cholesterol (TC),low-density lipoprotein cholesterol HDL-c and high-density lipoprotein (HDL-c)were determined using method described by (**Young, 1995**).

Estimation of atherogenicindex :

Atherogenic index was calculated according to the formula adopted by **Hostmark *et al.*,(1991)** as follows:

Atherogenic index = (TC-HDL-c) / HDL-c

Estimation of blood glucoselevel :

Fresh serum was used to determine glucose concentration based on colorimetric enzymatic methods using Spectrophotometer DU7400 (Japan) adjusted at 500 nmaccording to**Trinder,** (**1969).**

Estimation of serum insulinlevel :

Serum insulin level was estimated by enzyme amplified sensitivity immunoassay according to**Yallow and Bauman (1983)**.

Estimation of serum leptinhormones level :

Serum leptin was measured by chemiluminescence-based according to the method of **Matsuda*et al.*, (1997)**.

Estimation of serum AST, ALT and ALP :

Serum AST and ALT were carried out by the methods described by **Bergmeyer*et al.*, (1978**) and ALP by **Roy, (1970)**.

Estimation of oxidative stress markers:

Serum MDA was assayed quantitatively in serum using the MDA assay kit by a spectrophotometric method (ABCAM, UK) as described by **Draper and Hadley(1990)**. Serum GSH level was determined according to the method described by **Beutler *et al.*,(1973)** and expressed as nmol/ml. Serum GPx, SOD and CAT activitieswere determined using the pyrogallol autoxidation method described by **Rotruck *et al.*, (1973)**, **Marklund and Marklund(1974)** and **Sinha (1972)**, respectively.

Histopathological Study:

Heart and aorta of all rats was immersed in neutral buffered formalin (10%) for 24 hr. The fixed tissues were processed routinely, embedded in paraffin, sectioned, deparaffinized and rehydrated using the standard techniques according to the method of **Bancroft and Gamble(2002)**. The extent of high-fat diet induced obesity and hyperlipidemia was evaluated by assessing the morphological changes in the heart and aorta sections stained with hematoxylin and eosin (H and E).

Statistical Analysis:

The obtained results were expressed as means ± SD. Data were evaluated statistically with computerized SPSS package program (SPSS 20.00 software for Windows). Significant difference among means was estimated at $p<0.05$ **(Snedecor and Cochran, 1981**).

3. Results :

The present results in Table (1) shows means ± SDof FI, IBW, FBW, BWG and % change of BW values of normal rats, obese-hyperlipidemic rats and treated obese-hypolipidemic rats with CRB and CWB.It revealed that there are no significant differencesof FI and IBW between obese-hyperlipidemic rats and normal rats. After the first four weeks of the experiment, obese-hyperlipidemic rats (positive rats) had significant ($p<0.05$) increase in BW compared to those of the normal rats and no significant changes as compared to those of the other groups. At the end of experiment period, there are significant ($p<0.05$) increasesof FBW, BWG and % change of BW in obese-hyperlipidemic rats, compared to that of the normal rats. Fed rats on high-fat diet enriched with the two different levels of both CRB and CWB have significant reduction in FI, FBW, BWG, % change of BW, compared to those of fed rats on high-fat diet alone (positive rats). These decreases are more pronounced with increasing levels of beans.

Table (2) represents the effect of CRB and CWB on serum TL, TG and TC levels in normal obese-hyperlipidemic rats. Results showed that fed rats on high-fat diethad significant($p<0.05$) increase of serum TL, TG and TClevels, compared to that of normal rats. The two levels of both CRB and CWB caused ($p<0.05$) significant amendments of serum TL, TG and TC levels, compared with that of untreated obese-hyperlipidemic rats. There are no significant differences in serum TL and TC levels among treated rats with CRB and CWB. However, treated obese-hyperlipidemic rats with 20% CRB had significant ($p<0.05$) decrease in serum TG level as compared to that of treated obese-hyperlipidemic rats with CWB.

As shown in Table (3), marked significant ($p<0.05$) increase of serum LDL-c level and AI and decrease in serum HDL-c level in obese-hyperlipidemic rats, compared with those of normal rats. High-fat diet enriched with CRB or CWB significantly ($p<0.05$) decreased serum LDL-c level and AI, and increased serum HDL-c level compared to those fed on HFD alone.

Table 1: FI, IBW, BW and FBW (mean ± SD) of normal and obese-hyperlipidemic rats

Groups	FI (g/d)	IBW (g)	BW After First 4 weeks (g)	FBW (g)	BWG (g)	% change of BW
G 1: Normal control rats	21.43 ±1.99[a]	153.43 ±1.27[a]	194.70 ±0.76[b]	234.57 ±0.98[e]	81.14 ±2.04[d]	52.90 ±1.74[d]
G2: Obese-hyperlipidemic rats	19.71 ±2.63[a]	153.14 ±1.07[a]	214.57 ±0.79[a]	265.14 ±0.90[a]	112.00 ±0.82[a]	73.07 ±0.97[a]
G 3:HFD+10% CRB	17.71 ±1.38[b]	153.00 ±1.29[a]	214.57 ±0.79[a]	254.57 ±1.13[b]	101.57 ±1.51[b]	66.40 ±1.43[b]
G 4:HFD+20% CRB	16.43 ±1.51[b]	153.00 ±1.29[a]	214.71 ±0.49[a]	240.57 ±0.79[d]	87.57 ±1.99[c]	57.27 ±1.78[c]
G 5:HFD+10% CWB	17.00 ±1.29[b]	153.14 ±1.07[a]	214.43 ±0.79[a]	255.14 ±1.07[b]	102.00 ±1.41[b]	66.61 ±1.28[b]
G6:HFD+20% CWB	15.86 ±1.07[b]	153.00 ±1.29[a]	215.00 ±0.57[a]	241.86 ±1.21[c]	88.86 ±1.95[c]	58.14 ±1.26[c]

Means with different letters in same row are significantly different at $p< 0.05$.

Table 2: Serum TL, TG and TC levels (mean ± SD) of normal and obese-hyperlipidemic rats

Groups	TL (mg/dl)	TG (mg/dl)	TC (mg/dl)
G 1: Normal control rats	208.23±1.38[c]	84.77±2.92[e]	78.03±0.80[c]
G2: Obese-hyperlipidemic rats	474.26±0.77[a]	259.93±1.9[a]	102.53±1.11[a]
G 3: HFD +10% CRB	307.43±2.05[b]	124.97±1.30[b]	89.47±1.7[b]
G 4: HFD + 20% CRB	208.56±1.54[c]	89.80±0.85[d]	78.04±0.73[c]
G 5:HFD + 10% CWB	308.59±0.85[b]	124.77±1.73[b]	90.04±0.90[b]
G6:HFD + 20% CWB	208.44±1.11[c]	94.36±0.72[c]	78.87±0.92[c]

Means with different letters in the same row are significantly different at $p< 0.05$.

Table 3: Serum LDL-c, HDL-c levelsand AI (mean ± SD) of normal and obese-hyperlipidemic rats

Groups	LDL-c(mg/dl)	HDL-c(mg/dl)	AI
G 1: Normal control rats	12.04±0.59^{d}	57.30±1.48^{a}	0.37±0.02^{d}
G2: Obese-hyperlipidemic rats	66.84±1.08^{a}	34.53±0.18^{c}	1.97±0.04^{a}
G 3:HFD +10% CRB	30.56±0.65^{b}	45.21±0.63^{b}	0.98±0.04^{c}
G 4:HFD +20% CRB	13.23±0.77^{c}	57.10±0.59^{a}	0.37±0.02^{d}
G 5:HFD+10% CWB	31.13±1.00^{b}	44.64±0.84^{b}	1.02±0.05^{b}
G6:HFD+20% CWB	13.39±0.67^{c}	57.44±0.74^{a}	0.37±0.02^{d}

Means with different letters in same row are significantly different at p< 0.05.

Results in Table (4) showed significant (p<0.05) increase of blood glucose (BG) and serum leptin hormones (LH) levelsand decrease in serum insulin level in obese-hyperlipidemic rats, compared with those of the normal rats. Treated obese-hyperlipidemic rats with the two different levels of both CRB and CWB induced significant (p<0.05) amelioration in blood glucose, insulin and leptin hormone levels, compared with those of the untreated obese-hyperlipidemic rats (positive control group). There are no significant changes among treated groups of both levels of CRB and CWB.

Table 4: Blood glucose, insulin and leptin hormones levels (mean ± SD) of normal and obese-hyperlipidemicrats

Groups	BG (mg/dl)	Insulin (ng/ml)	Leptin (ng/ml)
G 1: Normal control rats	209.14±0.9^{c}	2.97±0.0^{a}	2.89±0.12^{c}
G2: Obese-hyperlipidemic rats	287.14±1.01^{a}	0.96±0.04^{c}	6.47±0.11^{a}
G 3:HFD +10% CRB	235.00±0.82^{b}	2.43±0.01^{b}	4.40±0.10^{b}
G 4:HFD +20% CRB	208.29±1.25^{c}	2.97±0.01^{a}	2.44±0.11^{d}
G 5:HFD+10% CWB	235.43±0.79^{b}	2.42±0.01^{b}	4.47±0.08^{b}
G6:HFD+20% CWB	208.71±1.11^{c}	2.97±0.01^{a}	2.49±0.07^{d}

Means with different letters in the same row are significantly different at p< 0.05.

Results in Table (5) revealed significant ($p<0.05$) increase of serum AST, ALT and ALP levels in obese-hyperlipidemic rats (positive control rats) compared to those of the normal control rats. Administration of different levels of CRB and CWB resulted in a significant ($p<0.05$)reduction in the serum activity of AST, ALT and ALP enzymes compared to those of the positive control group.

Table 5: Serum of activities AST, ALT and ALP enzymes(mean ± SD) of normal and obese-hyperlipidemicrats

Groups	AST (U/L)	ALT (U/L)	ALP (U/L)
G 1: Normal control rats	16.86±2.12^{c}	12.29±1.25^{d}	33.43±2.44^{d}
G2: Obese-hyperlipidemic rats	27.71±2.14^{a}	20.57±2.30^{a}	50.57±2.94^{a}
G 3:HFD +10% CRB	21.86±2.04^{b}	17.00±1.41^{bc}	44.71±2.14^{b}
G 4:HFD +20% CRB	16.71±2.06^{c}	15.57±1.27^{c}	34.86±2.12^{cd}
G 5:HFD+10% CWB	20.00±1.15^{b}	17.86±1.07^{b}	45.43±1.62^{b}
G6:HFD+20% CWB	17.29±1.89^{c}	16.43±1.27^{bc}	36.57±2.07^{c}

Means with different letters in the same row are significantly different at $p< 0.05$.

Tables (6) represent the results of serum MDA and GSH levels in normal rats, obese-hyperlipidemic rats and treated obese-hyperlipidemic rats with CRB and CWB. In comparison to the normal control rats, obese-hyperlipidemic rats had significant ($p<0.05$) increase of serum MDA and decrease of serum GSH levels. Administration of different levels of both CRB and CWB to obese-hyperlipidemic rats caused significant ($p<0.05$)ameliorate of serum MDA and GSH levels. There are no significant changes in serum MDA and GSH between treated groups with CRB and CWB.

As shown in Table (7), clears marked significant ($p<0.05$) decrease of serum glutathione peroxidase (GPx), superoxide dismutase (SOD) and catalase (CAT) activities in obese-hyperlipidemic rats, compared with those of the normal rats. The administrations of different

levels of CRB or CWB induced significant ($p<0.05$) increase of serum GPx, SOD and CAT activities, compared to that of untreated obese-hyperlipidemic rats.There is no significant changes of serum GPx, SOD and CAT activities between treated rats with CRB and CWB.

Table 6: Serum MDA and GSH levels (mean ± SD) of normal and obese-hyperlipidemic rats

Groups	MDA(nmol/l)	GSH(nmol/l)
G 1: Normal control rats	31.10±0.72^{c}	175.62±0.48^{a}
G2: Obese-hyperlipidemic rats	82.06±0.64 ^{a}	49.97±0.44^{c}
G 3:HFD +10% CRB	51.21±0.74^{b}	107.27±0.29^{b}
G 4:HFD +20% CRB	30.69±0.30^{c}	175.21±0.56^{a}
G 5:HFD+10% CWB	51.00±0.56^{b}	107.01±0.34^{b}
G6:HFD+20% CWB	30.61±0.19^{c}	175.50±0.55^{a}

Means with different letters in the same row are significantly different at $p< 0.05$.

Table 7: Serum activities of GPx and SOD and CAT enzymes (mean ± SD)of normal and obese-hyperlipidemic rats

Groups	GPx (mmol/dl)	SOD (U/ml)	CAT(mmol/dl)
G 1: Normal control rats	25.63±0.43^{a}	6.97±0.41^{a}	69.94±0.54^{a}
G2: Obese-hyperlipidemic rats	10.59±0.45^{c}	2.89±0.04^{c}	42.94±0.57^{c}
G 3:HFD +10% CRB	16.73±0.33^{b}	3.60±0.23^{b}	58.49±0.59^{b}
G 4:HFD +20% CRB	25.64±0.29^{a}	6.96±0.50^{a}	69.79±0.25^{a}
G 5:HFD+10% CWB	16.51±0.35^{b}	3.73±0.15^{b}	58.13±0.67^{b}
G6:HFD+20% CWB	25.44±0.19^{a}	6.67±0.17^{a}	69.41±0.50^{a}

Means with different letters in the same row are significantly different at $p< 0.05$.

Photomicrograph of heart sections of normal rats (negative control group) showed normal heart muscle and blood vessel (Fig. 1).

Heart sections of obese-hyperlipidemic rats (positive control group) revealed dilated blood vessel with thick muscle wall (Fig. 2) and intramuscular hemorrhages (Fig. 3). On the other hand, photomicrograph of heart sections of treated obese-hyperlipidemic rats with 10 %CRB showing edema with atrophied myocardial muscles (Fig. 4). Heart sections from treated obese-hyperlipidemic rats with 20% CRBrevealed few leucocytic cells infiltrationasas shown in Fig. (5). Heart sections from treated obese-hyperlipidemic rats with 10 and 20% of CWBnoticeddilated and congested blood vessel (Fig. 6) and congested blood vessel (Fig. 7), respectively.

Photomicrograph of aorta sections from rats of the normal control rats showed no histological changes (Fig. 8). Aorta sections of obese-hyperlipidemic rats (positive control group) noticed vasculitis with thick inflamed wall and perivasculitis with dilated and congested blood vessel (Fig. 9). Photomicrograph of aorta sections from treated obese-hyperlipidemic rats with 10 % CRBrevealed peri vascular edema as shown in Fig. (10). No histological changes were observed in aorta sections from treated obese-hyperlipidemic rats with 20 % CRB. On the other hand thick walled dilated vessel was observed in treated obese-hyperlipidemic rats with 10 % CWB (Fig. 11) and slight thickening in the wall in treated obese-hyperlipidemic rats with 20 % CWB (Fig 12).

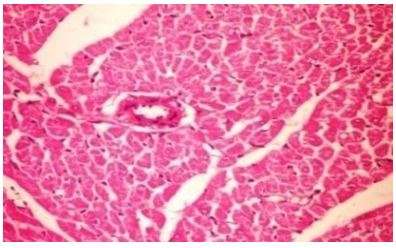

Fig. 1: Photomicrograph of heart sections from the normal control group showing apparently normal heart muscle and blood vessel (H & E x 400).

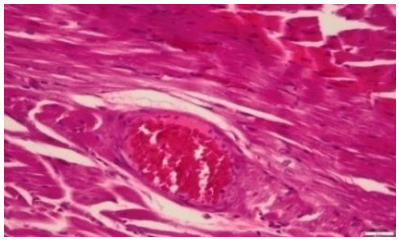

Fig. 2: Photomicrograph of heart sections from obese-hyperlipidemic rats (positive control group) showing dilated blood vessel with thick muscle wall (H & E x 400).

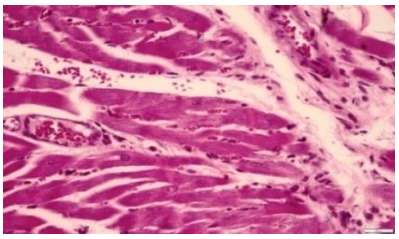

Fig. 3: Photomicrograph of heart sections from obese-hyperlipidemic rats (positive control group) showing intramuscular hemorrhages (H & E x 400).

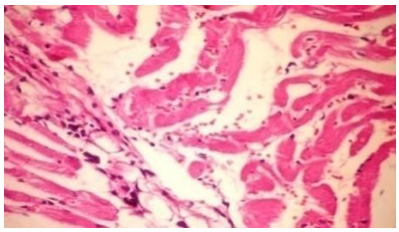

Fig. 4: Photomicrograph of heart sections from treated obese-hyperlipidemic rats with 10% CRB showing edema with atrophied myocardial muscles (H & E x 400).

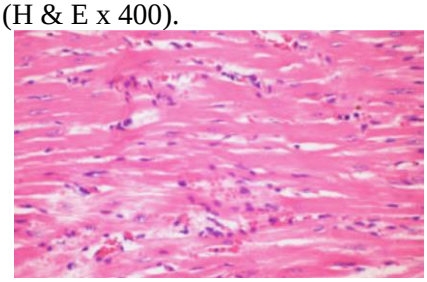

Fig. 5: Photomicrograph of heart sections from treated obese-hyperlipidemic rats with 20% CRB showing few leucocytic cells infiltration (H & E x 400).

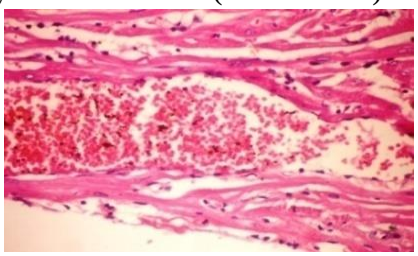

Fig. 6: Photomicrograph of heart sections from treated obese-hyperlipidemic rats with 10% CWB showing severely dilated and congested in blood vessel (H&E x 400).

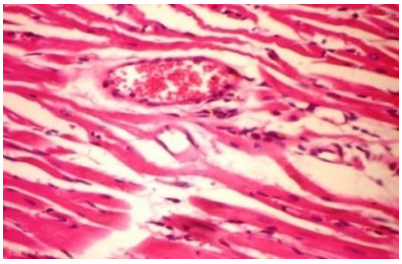

Fig. 7: Photomicrograph of heart sections from treated obese-hyperlipidemic rats with 20% CWB showing congested blood vessel (H&E x 400).

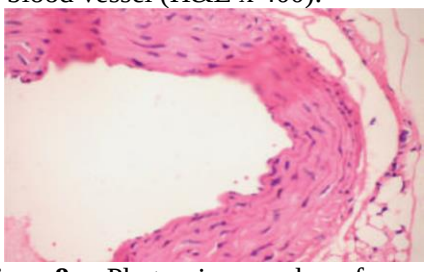

Fig. 8: Photomicrograph of aorta sections from the normal control group showing no histopathological changes (H & E x 400).

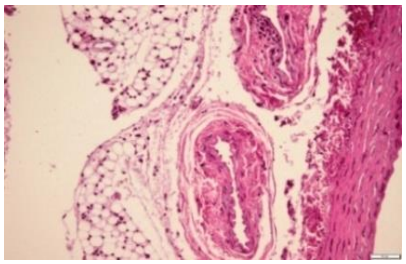

Fig. 9: Photomicrograph of aorta sections from obese-hyperlipidemic rats (positive control group) showing vasculitis with thick inflamed wall and perivasculitis with dilated and congested blood vessel (H&E x 200).

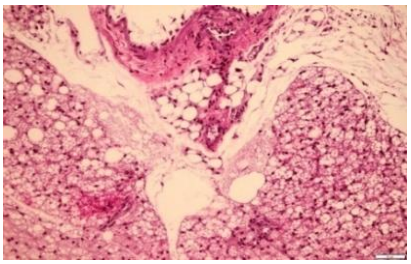

Fig. 10: Photomicrograph of aorta sections from treated obese-hyperlipidemic rats with 10% CRB showing peri vascular edema (H&E x 200).

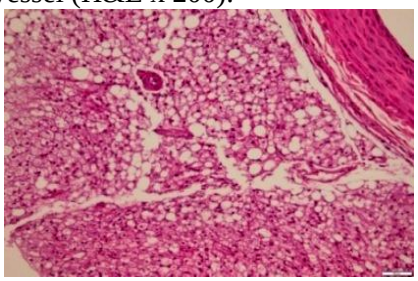

Fig. 11: Photomicrograph of aorta sections from treated obese-hyperlipidemic rats with 10% CWB showing thick walled dilated vessel (H&E x 200).

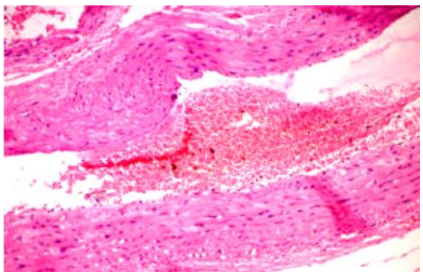

Fig. 12: Photomicrograph of aorta sections from treated obese-hyperlipidemic rats with 20 % CWB showing slight thickening in the wall (H & E x 200)**.**

4. Discussion :

The present study was conducted to investigate some biological and histopathological effects of cooked red and white beans on obese rats.

The present result has revealed that rats exposed to high-fat diet for 4 weeks have a significant increase in body weight, and therefore

verifying the obese status.At the end of experimental period, although there was a significant difference in the body weights between the high-fat and normal diet groups, there is no significant change in the daily food intake of animals.This observation provides the fact that an increase in body weight is independent of the amount of food consumed by the animals, but the content of the diet. The present result is in accordance with **Ramulu *et al.*,(2011)** who showed no significant differences in the amount of food intake between control and experimental groups fed high-fat diet. According to **Kusnoki *et al.*,(2000)**, high fat diet is considered to be an important factor in the development of obesity, leading to accumulation of body fat even in the absence of an increase in caloric intake in fed rats with high fat diet. Recently, several researchers indicated that high-fat diet being high in energy is harmful and therefore leading to overweight than rats fed normal diet **(Macfarlane and Macfarlane, 2012** and**Haghshenas*et al.*, 2014).**High-fat diet enriched with both CRB and CWB at 100 g/kg diet (10%) and 200 g/kg diet (20%) conversely caused a remarkable reduction in food intake,body weight gain and % change of body weight when compared to the high-fat diet alone.The reduction in food consumed and body weight was more pronounced with increasing levels of beans. The present result suggested that CRB and/or CWB supplementation at the two different levels are capable of minimizing body weight gain, concomitantly helping in maintaining the original body weight. This result is in accordance with the results reported fromprevious studies where administration of *Phaseolusvulgaris* dry extract resulted in dose-dependent decreases infood intake and body weight **(Carai*et al.*, 2011)**.Additionally, **Tormo*et al.*,(2006)** and **Pusztai *et al.*,(2008)**reported that extracts of beans as well as some of their isolated ingredients reduce food intake and body weight in obese rats. **Nilsson *et al.*, (2013)** reported that a regular diet including beans correlates with a lower risk of overweight and obese in men and women.In addition, there is evidence that a bean extract used by humans lowers body weight, percentage of fat and waist and hip circumference **(Preuss, 2009)**. The effectiveness of white beans in reducing food intake

and body weight may be based on the presence of two lectins: phytohaemoagglutinin and α–amylase inhibitors **(Ishimoto *et al.*, 1995)**. Lectins bind to the intestinal brush border, stimulating the release of colecystokinin and glucagon-like peptides that modulate food intake **(Baintner*et al.*, 2003)**.*Phaseolus vulgaris* has been considered the best source of α amylase inhibitor **(Fantini*et al.*, 2009)**. Specifically, inhibition of the pancreatic enzyme α- amylase delays gastric emptying, producing satiety and in turn decreasing food intake **(Jain *et al.*, 1991)**. Additionally,daily feeding of raw white bean extract markedly reduce the food intake in rats with access to a starch-enriched diet, an effect that is associated with a reduction in body weight gain **(Tormo*et al.*, 2006)**. On the other hand, beans are slowly digested and have a high proportion of non-digestible carbohydrates, which can be fermented in the large intestine. Non-digested carbohydrates that reach the colon include resistant starch, soluble and insoluble dietetic fibre and non-digestible oligosaccharides **(Reynoso-Camacho *et al.*, 2006)**. In addition to, common bean has high fibre content **(Cruz-Bravo *et al.*, 2011)** which is strongly prevents obesity and is inversely associated with body fat and body mass index at all levels of fat intake **(Slavin, 2004)**. High-fiber foods have much less energy density compared with high-fat diet and can displace energy. Eating equal amount of high-fiber food increases satiety. The bulking and viscosity properties of dietary fiber are mainly responsible for the influencing satiety **(Burton-Freeman, 2000)**.

Dyslipidemia is another important lineament in the manner of development of obesity which is characterized by hyperlipidemia, hypertriglyceridemia with increased level of LDL-c and VLDL-c **(Klop*et al.*, 2013)**. Chronic dyslipidemia has been characterized as a major risk factor for cardiovascular disease and atherosclerosis **(Mbikay, 2012)**. In the present study high-fat diet exposure resulted in significant increase of serum TL, TG, TC, LDL-c levels and atherogenic index with decrease serum HDL-c level. In addition, histopathological results showed that consumption of high-fat diet may play a crucial role in the pathogenesis ofdilated blood vessel with thick muscle wall and intramuscularhemorrhages in heart and vasculitis with thick inflamed

wall and perivasculitis with dilated and congested blood vessel in aorta. The present results are in accordance with **Park *et al.*(2004)** and **Rezq and El-Khamisy(2011)** who showed that high-fat diet supplementation results in dyslipidemia changes by increased serum TG, VLDL, TC and LDL-c and decreased serum HDL-c levels. Additionally, **Puskas*et al.*, (2004)** demonstrated that excess cholesterol in the bloodstream can form plaque in artery walls. The cholesterol or plaque build-up causes arteries to become thicker, harder and less flexible, slowing down and sometimes blocking blood flow to the heart and results in a heart attack.When there is too much LDL-c in the blood, it is deposited inside the blood vessels, where it can build up to hard deposits and cause atherosclerosis **(Ma, 2004)**. Further, atherogenic index is regarded as a marker for various cardiovascular disorders; the higher the value, the higher the risk of developing cardiovascular disease and vice versa **(Takasaki, 2005)**. The dyslipidemia in rats fed high-fat diet may be attributed to the activity of lipoprotein lipase which was augmented in hypercholesterolemic animals. Lipase transforms VLDL-c to LDL-c lead to increase serum concentration of LDL-c **(Tebib*et al.*, 1994)**. An uptake of LDL-c is depended on receptors in plasmatic membrane and these are reduced in number when the cell has enough cholesterol. Further, the alteration of lipid profile induced by high-fat diet might be the activation of gastric lipases, intestinal fat absorption and the lipolysis **(Saravanan and Ponmurugan, 2012)**. In contrast, high-fat diet supplemented with the two different levels of both CRB and CWB induced significant attenuation in serum TL, TG, TC, and LDL-c levels, and atherogenic index and increased serum HDL-c level in obese-hyperlipidemicrats. These improvements were found to be in a dose dependent manner.Thus, it can be concluded that CRB and CWBpossess antihyperlipidemic and cardioprotective potential.**Marzolo*et al.*,(1993)** showed that long-term feeding with beans decreases serum levels of cholesterol and LDL-c in humans, so it protects against cardiovascular diseases. A number of intervention studies have found that the consumption of cooked or canned dry beans reduce serum TC and LDL-c levels and therefore the risk of developing cardiovascular diseases in

men**(Winham*et al.*, 2007 and Zhang *et al.*, 2010)**.Also, **Kabagambe*et al.*, (2005)** reported that individuals who consume approximately 79–92 g of cooked dry beans/d are associated with lower risk of myocardial infarction compared with non-consume individuals.**Zhu *et al.*, (2012)** reported that dietary bean reduced circulating levels of TC and LDL-c in rats.Thecholesterol lowering effect of beans has been showed by **Lujan *et al.*,(2008)** and suggested that the mechanism of action may be due to the possible effects of soluble fiber, saponins, tannins andproteins.In addition to, the common bean had high fibre content **(Cruz-Bravo *et al.*, 2011)**especially, soluble dietary fiber **(Anderson *et al.*, 1994)** which had been shown to reduce blood cholesterol in epidemiologic **(Brown *et al.*, 1999)**, clinical **(Anderson *et al.*, 1990)**, and animals **(Rosa *et al.*, 1998)** studies.Fiber content of beans prolongs the postprandial presence of intestinally derived lipoproteins and increases the cholecystokinin response to the meal leading to hypocholesterolemic effect **(Bourdon *et al.*, 2001)**.Also, beans are good source of viscous polysaccharides which lower plasma cholesterol in humans and contribute to the reduction in risk of cardiovascular disease associated with diets high in fiber-rich foods **(Brown *et al.*, 1999)**.**Olivia *et al.*, (2013)** revealed that the extract of white beans had significant lowering effect on serum TG, TC, LDL-c, and increase HDL-c concentrations. Additionally,the high amount of phytochemicals in white beans probably accounts for their cholesterol lowering effect and may contribute to the vast pharmacological properties **(Obohe*t al.*, 2010)**. Also, **Carew *et al.*, (2003)** observed a reduction in serum TC and TG levels after treatment with beans and attributed the effect to the availability of saponins. Saponins have been shown to reduce cholesterol by forming insoluble complexes with cholesterol and bile, making them unavailable for absorption.

In the current study data revealed that significant increased in serum liver AST, ALT and ALP levels in feeding rats with high fat-diet. The increased in serum AST and ALT, ALP levels may be attributed to excessive release from the damaged liver cells as a result of hyperlipidemia into the blood circulation (**Arkkila*et al.*, 2001)**. In addition, **Amin and Nagy, (2009)** indicated that serum AST and ALT

concentrations were significantly higher in the high fat diet rats compared to the normal control rats. Recently**Rezq, (2012)** and **de Castro *et al.*, 2013** reported that high fat-diet significantly increase serum AST, ALT and ALP levels. In contrast, feeding rats with high fat-diet supplemented with the two different levels of CRB and CWB produced significant decrease in serum AST, ALT and ALP levels compared to that of the feeding rats with high fat diet only. This result was agreed with **Luka *et al.*, (2013)** who showed that aqueous extracts of *Phaseolus vulgaris* L. at 400 mg/kg body weight significantly reduce the values of serum ALT, AST and ALP levels whencompared to high values of the enzymes in diabetes rats.This effect may be related to antioxidant properties of bean seeds. The high concentration of antioxidants of *Phaseolus vulgaris*may be protecting the liver from the CCL_4 induced fibrotic effect **(Salinas-Moreno *et al.*, 2005)**. Also, (**Pourmorad*et al.*, (2006**) reported that the hepatoprotective action of *Phaseolus* seeds extracts may be attributed to the presence of the flavonoids which provides maximum conjugation with free radical species generated, thereby, reducing the number of free radicals and extent of cellular damage by decreasing lipid peroxidation, scavenging super oxide radicals and maintaining level of GSH.

Insulin resistance in humans can be linked to lifestyle and can be noticed more as a cause of lipid deposition in a caloric excess **(Unger and Scherer, 2010**). Thus, excessive caloric intake can lead to hyperinsulinemia, which raises sterol regulatory element-binding protein- 1c expression in beta cells, resulting in increaselipogenesis and obesity **(Muhlhausler and Smith, 2009)**.Insulin resistance is associated with a number of metabolicdisorders such as obesity, hyperlipidemia, and hypertension. High-fat diet intakes were shown to contribute to syndromessuch as hyperlipidemia, glucose intolerance, hypertension,and atherosclerosis **(Sumiyoshi *et al.*, 2006)**.Numerous evidences showed in experimental animals, high-fat diets resulted indisturbance in glucose metabolism and impaired glucosetolerance**(Vessby, 2000 and Lichtenstein and Schwab, 2000)**.The present results showed significant increase of serum glucose level and decrease of serum insulin level in

rats fed on high-fat diet, compared to that fed on normal basal diet. This result agreed with **Kusunoki *et al.*,(2000)** who showed hyperglycaemia, dyslipidaemia and hyperinsulinaemia in rodents fed a high-fat diet. **Srinivasan*et al.*,(2004)** revealed that the feeding onhigh-fat diet for a period of 30 days presentshyperglycemia as shown by increased levels of serum glucose, insulin and insulin resistance. **Huang *et al*. (2004)** found that feeding high-fat diet results in decrease of insulin secretion (hypoinsulenemia). Some previous studies revealed that hyperinsulinemia and insulin resistance are common features of obesity in humans **(Kay *et al.*, 2001)** and experimental animals **(Amin and Nagy, 2009)**.Also, **Saravanan*et al.*,(2014)** showed significant increase in bodyweight andserum glucose, lipid profileand decrease of serum insulin levels inhigh-fat diet rats. In contrast, the present results showed recover in blood glucose and insulin levels in treated obese-hyperlipidemic rats with the two different levels of either CRB or CWB.These results are consistent with those of previous studies which revealed that extracts of beans and/or some of their isolated ingredients have been reported to reduce glycemia in lean and obese rats **(Tormo*et al.*, 2004 and Pusztai*et al.*, 2008)**. Repeated daily administration of raw white bean extract markedly reduced the glucose levels **(Fantini*et al.*, 2009).**The in vivo effect of a supplemented diet with black bean flour on rats with streptozotocin-induced diabetes showed significant decrease in glucose level **(Hernández-Saavedra*et al.*, 2013)**.**Feregrino-Pérez *et al.*,(2008)** reported that starch in beans is slowly digested and reduce postprandial response to insulin. Also, **Carai*et al.*,(2009)** observed that *Phaseolus vulgaris* extracts reduced postprandial glycemia in a similar way as metformin drug. There are two mechanisms of action may be proposed for the reducing effect of *Phaseolus vulgaris* on glycemia. Both these mechanisms are based on the presence of lectins**(Ishimoto *et al.*, 1995)**. Lectins together with arcelins possess high degree of amino acid sequence similarity **(Lee *et al.*, 2000)** and specifically, inhibit the pancreatic enzyme α- amylase and suppresses starch metabolism, resulting in a decrease in glycemia**(Santimone*et al.*, 2004)**. The α-amylase inhibitor isoform 1 (α-AI1) has been extracted from *Phaseolus*

*vulgaris*and used in diverse commercial products against obesity and diabetes in humans **(Barrett and Udani, 2011).**

Leptin is a common protein produced by the adipose tissue and highly correlates with body fat, suggesting that obese persons are insensitive to endogenous leptin production. It is a key fat-derived regulator of food intake and energy expenditure and its secretion levels are usually positively correlated with the extent of the triglyceride stores in adipocytes **(Staiger, and Haring, 2005)**. Adipocytes secrete a variety of peptide hormones called adipocytokines such as leptin, adiponectin, visfatin, resistin, tumour necrosis factor-α and interleukin-6, which play a role in energy regulation **(Garg, 2006)**.In the present study, result showed that serum leptin level was increased significantly in the high-fat diet control group compared with that of the normal control group. Substitution of dietary carbohydrate for fat has been shown to increase plasma leptin**(Weigle*et al.*, 2003)**. The present experimental diet consisted of more fat and this might have accounted for the elevated levels of leptin, consistent with literature reports **(Handjieva-Darlenska and Boyadjieva, 2009)**. **Huang *et al*., (2004)** and **Saravanan*et al*., (2014)** showed that rats fed on high fat-diet had high serum leptin hormone level when compared with those fed on normal basal diet.Treated obese-hyperlipidemic rats with CRB and CWB results results in significant decreased of serum leptin level as compared to untreated obese-hyperlipidemic rats. The decrease in plasma leptin concentration has been reported following energy restriction **(Dubuc*et al.*, 1998)**. These observations suggested that the decrease of serum leptin levels after CRD and CWB supplementation may be attributable to their effect on the decrease of food intake and body weight and consequently the decrease of lipid accumulation in the adipocytes.

The present study provided a perfect correlation between serum lipid peroxidation products as indicator by MDA and level of GSH and activity of antioxidant enzymes which play an important role in the antioxidant system.It showed that rats fed on high-fat diet induced significant increase in serum MDA level, and decrease in serum GSH

level and activities of GPx, SOD and CAT enzymes, compared to that fed on normal basal diet. The decrease in serum activity of antioxidant enzymes, as seen in obese-hyperlipidemic rats, can lead to the excessive availability of superoxide and peroxyl radicals, which in turn generate hydroxyl radicals, resulting in the initiation and propagation of more lipid peroxidation products. High-fat diets results in the release of free fatty acids by the action of lipoprotein lipase with increase serum triglycerides and cause lipotoxicity, which results in insulin receptor dysfunction. The release of excessive free fatty acids provokes lipotoxicity, as lipids and their metabolites create oxidative stress **(Zhang, *et al.*, 2007)**.The present result agreed with **Amirkhizi *et al.*, (2007)** who showed increase in the production of reactive oxygen species as well as reduced antioxidant defense mechanisms in both humans and animal models of obesity. Further, lipid alterations have been considered as contributory factors to oxidative stress in obesity. Hypertriglyceridemia results in obese rats participate in the alteration of oxidant-antioxidant balance, suggesting increase the bioavailability of free fatty acids and lipid peroxidation **(Leopold and Loscalzo, 2008)**.Hyperlipidemia induces oxidative stress and increase lipid peroxidation **(Moussa, 2008)**. Recently, **Denisenko and Novgorodtseva (2013)** showed inhibits activity of blood antioxidant enzymes and elevate lipid peroxidation (MDA) in fed animals on high fat diet. In contrast, feeding rats on high-fat diet enriched with the two different levels of CWB and CRB significantly ameliorate antioxidant system in rats as showed by decreased serum MDA level and increased GSH level and elevate serum activity of antioxidant enzymes.This result was in convention with **Venkateswaran and Pari (2002)** who showed that been extract causes significant increase in serum GSH level and activity of SOD, CAT and GPx enzymes in the liver and kidneys tissues of rats. Also, *Phaseolus vulgaris* extract was showed to inhibit free radical production and lipid peroxidation and activates antioxidant enzymes in liver and kidneys of rats with STZ-induced diabetes **(Kyznetsova*et al.*, 2015)**. Thus CRB and CWB have the ability to normalize the elevated lipid peroxidation and improved susceptibility to oxidative stress

associated with depletion of antioxidants in obese-hyperlipidemic rats. The antioxidant properties of RB and WB might be attributed to its content of phenolic and flavonoid compounds.Preliminary phytochemical studies of white and black beans (*Phaseolus vulgaris*) shows the presence of alkaloids, saponins, glycosides, tannins, and flavonoids and phenolic compounds. They do not differ significantly in their contents of glycosides, soluble carbohydrate and tannins but not alkaloids, flavonoids, and saponins**(Olivia *et al.,* 2013)**.The most widespread flavonoid group in beans is proanthocyanidins**(Reynoso-Camacho *et al.,* 2006)**. Phenolic compounds are efficient scavenger of free radicals as well as transition metal ion chelating agents. Flavonoids possess a chemical structure with particular hydroxyl position in the molecule that is considered to be involved in proton donating and radical scavenging mechanism **(Hou*et al.,* 2003)**. Flavonoids, phenolic acids and tannins can terminate oxidative chain reactions by eliminating free radical intermediaries and inhibiting other oxidation reactions **(Jeon*et al.,* 2012)**.

5. Conclusions :

From this study, it may be concluded that the *P. vulgaris* seeds (white or red) have antiobesity, antihyperlipidemic,antihyperglycemic potential,cardioprotective potential and ameliorate the antioxidant-defense system in rats that feed on the high-fat diet. However, regular intake of *Phaseolus Vulgaris*using it in enriching food products may enhances functional foods with the improvement of health status.

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بعض التأثيرات البيولوجية والهستوباثولوجية لاستهلاكالفاصوليا الحمراء والبيضاء المطهية علي الفئران المصابة بالسمنة

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الملخص العربى:
أجريت الدراسة لمعرفة بعض التأثيرات البيولوجية والهستوباثولوجيةلاستهلاك الفاصوليا الحمراء والبيضاء المطهية علي الفئران المصابة بالسمنة. وقد أظهرت النتائج أن الفئران المصابةبالسمنة وارتفاع دهون الدم لديهم زيادة معنوية كبيرة في وزن الجسم والنسبة المئوية للزيادة في الوزن، و كذلك في تركيزات الدهون الكلية، الجلسريدات الثلاثية، الكولستيرول،الليبوبروتيناتمنخفضة الكثافة، سكر الدم، هرمون الليبتين، المالون داي الدهيد ومؤشر تصلب الشرايين ونشاط انزيمات الاسبارتيزامينوترانسفييريز،الالنينامينوترانسفيريز، الالكنين فوسفاتييز. بينما وجد إنخفاض ملحوظ في تركيز،الليبوبروتينات مرتفعة الكثافة، الانسولين، الجلوتاثيون المختزل وأنشطة انزيمات الجلوتاثيونبيروكسيديز، السوبر اوكسيد ديسميوتيزوالكتاليز في سيرم الدم وذلك بالمقارنة بالفئران الطبيعية.في حين ان الفئران المصابة بالسمنة وإرتفاع في دهون الدم وتغذت علي الفاصوليا الحمراء اوالبيضاء المطهية لديها تحسن ملحوظ في المؤشرات البيوكيميائية السابقة كمقارنة بالفئران المصابة بالسمنة بارتفاع دهون الدم وغير معالجة. كما اظهرت نتائج الفحص الهستوباثولوجي ان الفئران المصابة بالسمنة وارتفاع في دهون الدم وجود تمدد بالاوعية الدموية وزيادة السمك مع وجود نزيف في جدار عضلة القلب. كما وجد تمدد والتهاب وزيادة في سمك الشريان الاورطي. في حين ان التغذية علي الفاصوليا الحمراء والبيضاء تؤدي الي تحسن جزئي في القلب والشرين الورطي كما اظهرت نتائج الفحص الهستوباثولوجي. وإستخلصت نتائج هذه الدراسة ان الفاصوليا الحمراء و البيضاء لها تأثير مضاد للسمنة وارتفاع دهون الدم ومخفض لسكر الدم وتحسن فيمستوي مضادات الاكسدة في الفئران التي تغذت علي نظام غذائي عالي في الدهون.

الكلمات المفتاحية: الفاصوليا البيضاء والحمراء – الغذاء العالي في الدهون - الانزيمات المضادة للاكسدة – دهون الدم – هرمون الليبتين– الفحص الهستوباثولوجي.