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Hypolipidemic and cardioprotective benifits of *Garden cress* seeds in rats compared to atorvastatin

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Abstract
The prevalence of elevated cholesterol and fasting-triglyceride concentrations is 36.7% and 10.2% in Egypt, and an estimated 46% of total fatalities are due to cardiovascular disease. Synthetic drugs with hypolipidemic efficacy are widely used; yet limitations for their use are prices and side effects. Safe and cheap alternatives are therefore natural products. Garden cress possesses several pharmacological properties and has a capability for functional food production. Aim was to explore the impacts of Garden cress seed powder (GSP) on lipid profile and C-reactive protein as a proxy marker of cardiovascular risk in rats fed a high fat diet (HFD) in comparison to atorvastatin. Application as a functional ingredient for incorporation in a real food model as biscuit, pizza and cupcakes was tested. Thirty five Wistar rats were distributed among five groups. Group I (control), group II (on HFD), group III (on HFD and GSP 10%), group IV (on HFD and GSP 15%rats), and group V (on HFD and Lipitor 15mg/kg/day). HFD resulted in significant increase in CRP levels compared to control group. Lipid profile was significantly higher than control rats for total cholesterol (TCh), triglycerides (TG), LDL, while HDL was significantly lower. Both doses of GSP caused significant decrease in CRP. GSP has a hypolipidemic activity and in a dose of 15% it is comparable to atorvastatin 15 mg/kg. Cardioprotective benifits of GSP by significantly reducing CRP levels is the novel finding in the present study.

Keywords; *Lepidium sativum,* medicinal plants, dyslipidemia, surrogates of cardiovascular risk

Introduction
Recently, attempts were made to develop innovative dietary regimens with nutraceutical ingredients to benefit from various health effects and to prevent the chronic disease epidemic. Lepidium sativum or Garden cress (GC), commonly known as Hab-alrashad is related to watercress. GC is found in Egypt and South-West Asia. Since ancient times, GC seeds were used in traditional medicine. GC had not received the attention it deserves despite its large medicinal value and remained a crop that is underutilized [1].

Garden cress seed (GCS) has a range of anti-anemic, antioxidant, and galactogogue pharmacological characteristics and has an enormous ability to create functional foods via supplementation [2, 3].

The safety of ethanolic extract of GCS acute and chronic use in mice has been researched for anti-inflammatory, antipyretic and analgesic properties [4].

Garden cress seed oil (GCSO) is categorized under nuts and oil seeds and contains a good ratio of polyunsaturated fatty acids (PUFA) (± 47%) and monounsaturated fatty acids (MUFA) (± 38%). It includes healthy antioxidants such as vitamin A, E and eugenol, which prevent free radical harm, as well as loaded in health-benefitting omega-3 fatty acids. GCS has been revealed to possess about 23% protein, 28% fat, 30% fiber and 1200 mg/100 g potassium [5, 6].

The existence of substantially high quantities of protein (± 24%), lipids (± 23%), carbohydrates (± 31%), fiber (± 12%), ash (± 7%), and humidity (± 3%) was stated by Zia-Ul-Haq. More ash content suggests that GCS is a useful mineral source. A stable, performance and longer shelf life for seeds are the low moisture content. Higher levels of protein and lipid show that GCS are energy-efficient, [7, 8] and it can be used to manufacture many potential food products with that nutrient core.

Cardiovascular disease (CVD) is the first killer disease in the world. It was recorded that in 2016 there were 41 million fatalities from non-communicable diseases (NCDs), comprising 71% of the general death rate of 57 million. Most of the fatalities have been attributed to four primary NCDs: cardiovascular disease (± 44% of all fatalities from NCDs), cancer (± 22%);

chronic respiratory illness (± 9%), and diabetes (± 4%) [9].
The prevalence of elevated cholesterol and fasting-triglyceride concentrations is 36.7% and 10.2% for those aged 15 to 65 years in Egypt, and an estimated 46% of total fatalities are due to CVD. This is the result of the nutritional transition and the enhanced incidence of lifestyle risk factors such as lack of fitness, smoking and obesity [10, 11].
The underlying primary cause of CVD is believed to be atherosclerosis, a progressive multifactorial disease of the arterial wall. Central to the pathogenesis of atherosclerosis is the vascular lining deposition of cholesterol. Hyperlipidemia, a significant cardiovascular risk hazard, is one of the major oxidative stresses that generate excess of highly reactive free radicals. (12) Serum cholesterol reduction was demonstrated in successful trials to reduce mortality caused by chronic heart disease. Synthetic drugs with hypolipidemic efficacy are widely used; yet the main reasons for limiting their use are prices and side effects. The safe and cheap alternatives are therefore natural products [13].
One of the most prescribed drug categories worldwide since its incorporation into the market has been hydroxymethyl glutaryl coenzyme A reductase (HMG-CoA) inhibitors (frequently known as statins). A rate limiting step in cholesterol production in the body; HMG-CoA is inhibited by statins. Statin treatment was efficient in reducing LDL concentrations by 20-5%, reducing triglyceride concentrations by 10-20%, and triggering a potential increase in HDL level up to 5-10% [14, 16].
Safety of GCS was assessed in mice to evaluate acute and chronic toxicity; in doses of 0.5–5.0 g/kg body weight and 1.0–10.0-% respectively. GSP had no toxic effect in both instances [17, 18] Higher dose of Lepidium sativum (5-10% of meal) is safe to hepatic and renal functions in mice and acts as an effective cholesterol lowering mediator. Al Hamedan et al, who reported that aquatic extract of GC (20 mg/kg body weight) and GSP added to hypercholesterolemic diet increase weight gain and decrease total cholesterol, LDL cholesterol and triglycerides in rats. Preventing absorption and stimulating excretion of lipids could be caused by GC's hypolipidemic impact. Inhibition of cholesterol formation by GC may be due to the inhibition of HMG-CoA reductase as the rates-limiting enzyme [19-22].
The cardioprotective potential of GSP in albino rats against cardiotoxicity and oxidative stress induced by 5-fluorouracil (5-FU) has been evaluated recently. GSP pre and post treatment significantly enhances all 5-FU modified parameters. The results show that GSP significantly affects the protection of the heart by preserving antioxidant and anti-inflammatory activities against 5-FU-induced cardiotoxicity [23].
The aim of this work was to investigate the effect of GSP on lipid profile and C-reactive protein - surrogate measures of cardiovascular risk - in albino rats fed a high fat diet in comparison to atorvastatin. GSP potential application as a natural additives and functional ingredient for incorporation in a real food model as biscuit, pizza and cupcakes, was also tested. Chemical, physical and organoleptic properties were determined in these different baking blends.

Material & Methods

Collection of Material & Preparation of GSP Sample

Whole garden cress seeds were obtained commercially from Azmour Siwa Company and were cleaned to remove impurities and then were grounded in mixer to fine powder and stored in an airtight container and analyzed in central laboratory in faculty of agriculture, Alexandria University.

Animals and treatment

A total of 35 albino rats (140-180 g) were obtained from the animal house of the faculty of pharmacy and drug manufacturing, Pharos University, Alexandria, Egypt. The rats were acclimated for 3 days before starting the experiment. All animals were housed in standard cages (7 rats/cage), in an air-conditioned rooms at 21-23 °C and 60-65% of relative humidity, and kept on a 12 h light/12 h dark cycle. The animals received humane care in accordance with the Guide for the Care and Use of Laboratory Animals, published by ethics of scientific research committee of Pharos University and fed with standard laboratory diet and tap water ad libitum.

Induction of dyslipidemia: High fat diet (HFD) (46% of calories from fat); 1 kg (standard laboratory diet), 1/2 kg plant margarine rich in saturated fatty acids (SFA) from palm oil, and 1/4 kg lard [24].

Experimental groups and protocol

Rats were divided randomly into 5 groups comprising 7 rats in each group.

Group I: Rats fed on standard laboratory diet without any additives for 8 weeks and served as a negative control group.

Group II: Rats fed on HFD for 8 weeks; i.e. positive control.

Group III: Rats fed on HFD for 4 weeks, and then GSP was added to the HFD by a concentration 10% for 4 weeks.

Group IV: Rats fed on HFD for 4 weeks, and then GSP was added to the HFD by a concentration of 15% for 4 weeks.

Group V: Rats fed on HFD for 4 weeks, and then they received Lipitor 15mg/kg/day by oral gavage in addition to HFD for 4 weeks.

Ingredients of diet

Standard laboratory diet consists of: 60% CHO, 19% protein, 6% fats, 2.3% fibers and more than 5%vitamins and minerals. Energy: 3200Kcal/kg.

Measurements of body weight

Body weights of all groups under investigation were measured twice weekly.

Samples collection: At the end of the experiment, the animals (the control and experimental animals) were sacrificed under pentobarbital anesthesia. Blood samples were collected from aorta after 12 hours fast for biochemical analysis.

Laboratory tests

Samples were tested for CRP, LDL, HDL, triglycerides (TG) and total cholesterol (TCh).

Baking products: 10, 20 and 30% GSP were incorporated in pizza, biscuits and cupcakes formulation on 100g flour basis. Chemical and physical properties were analyzed in central laboratory in faculty of agriculture, Alexandria University. Organoleptic properties were determined in these different baking blends by a panel of 30 students in the Food Processing Course on a 9 point hedonic scale.

Histopathology

The livers were retrieved from all groups; the samples were kept in formalin solution for preparing paraffin sections. Sections were stained with Hematoxylin and eosin (H&E) to study the histological structure of the liver.

Statistical Analysis

Mean and standard deviation were calculated for each studied variable. The interpretation of data obtained was done by analysis of variance (ANOVA) test. Level of significance was accepted at $p<0.05$.

Results

Proximate composition of GSP shown in table 1; indicates the existence of significant protein quantities (23.12), a good amount of lipids (30.29), carbohydrates (27.16), fiber (11.99), and moisture (7.44), Table 1.

Table 2 indicates that HFD induced significant elevations ($P < 0.05$) in total cholesterol (153.2±25.2 mg/dL) in comparison to Group I of negative controls (96.5±16.8). While, adding GSP to HFD (15% diet) lessened serum total cholesterol (133.4±26.7), as compared to HFD group, a value that is not significantly different from the effect of atorvastatin (119.8 ± 13.4) *P10 = 0.252*.

GSP in a dose of 10% significantly lowered TG as compared to HFD *P5=0.02*, while GSP in a dose of 15% has almost the same results *P8=0.404*, but Statins were slightly more effective.

Both concentration of garden cress had nearly the same HDL raising effect, while atorvastatin had a more profound effect.

Both supplementary doses of GSP caused significant decrease in CRP *P5=0.001, P6 <0.005* and atorvastatin was slightly more effective.

Table 2: %Weight change, Serum lipid profile and CRP of –ve control, rats fed on HFD, HFD with GSP 10&15%, and HFD with Lipitor® 15mg/kg

The chemical analysis and evaluation of garden cress seed baking products as a functional food was shown in table 3.

Table 4 explains the items of sensory analysis and organoleptic properties of garden cress seed products.

Table 5 compares the cost of garden cress products per100gm.

Figures 1 exhibits baking products: 10, 20and 30% GSP incorporated in pizza, biscuits and cupcakes. Figures 2 and 3 depict the changes elicited by HFD and the effect of treatment groups on the histological structure of the liver.

Discussion

The current research aim was to explore the impacts of Garden cress seed powder on lipid profile and CRP as a proxy marker of cardiovascular risk in rats fed a high fat diet in comparison to atorvastatin. Commonly, the addition of a specific fat in animal diets for studying obesity in rats and mice is used to provide high fat diets ranging from 30–75% of total energy consumption.

Humans have shown that SFAs are more obesogenic than PUFAs and animal studies have endorsed this concept. The obesogenic impact of SFA may be attributed to that they are poorly used for energy, and therefore acylated into TG and stored in fat cells, unlike PUFA and MUFA which are easy to use for energy and thus less stored[24,28].

Our findings indicated that administration of HFD to rats caused an increase in their body weight after 8 weeks by 13.8% compared to –ve control group, while G III (HFD+GSP 10%) showed a drastic increment of 31.2% weight gain in comparison to negative controls. The significant increment in body weight of rats received GSP 10% with HFD, in comparison to their corresponding control groups, may be due to its palatable and spicy taste and this might have led to increase appetite in these rats. (21) G IV (HFD+GSP 15%) showed decrement in body weight by 17.6% in comparison to the HFD treated animals, this was comparable to G V (HFD+ Atorvastatin 15mg/kg) which showed decrement in body weight by 18.1%.

The higher dose of GSP with its peppery and bitter taste might have an influence on the appetite causing the decrement in body weight. These results are almost in accordance with Shukla et al, investigating GCS Sapogenin and Flavonoid extract (LSTS and LSTF) versus Triton x-100 and high cholesterol diet (HCD) provoked hyperlipidemia in mice. (29)

Mainly; a metabolic disorder responsible for atherosclerosis and other related cardiovascular diseases is hyperlipidemia. LDL lowering, plus control of blood cholesterol and triglyceride will eventually diminish progression of CVD. Reduction of serum cholesterol by 1% was hypothesized to re duce the risk of CVD by 2% [30]. Recently several researches focused on the discovery of herbal plants with hypolipidemic activity that could suppress high blood cholesterol [31] without the severe side effects of statins.

Our HFD (46% calories from fat) induced significant elevations in triglycerides, total serum cholesterol and LDL levels as compared to negative controls. Percent change was 153%, 58.7% and 117.6%, for TGs, TCh and LDL-cholesterol levels, respectively. On the other hand HDL decreased by 29% compared to the control group. However, inclusion of GSP in HFD (15% diet) reduced TCh (133.4±26.7), in comparison to positive control group, a value that is not significantly different from the effect of atorvastatin (119.8 ± 13.4) (*P10 = 0.252*). LDL was reduced by this dose comparable to the lowering effect of atorvastatin 15 mg/kg (*P10 = 0.110*).

These results were in line with Al Hamedan who recorded the GCS extract and powder protection impact on hypercholesterolemic rats evidenced by a significant lower value of serum cholesterol, triglycerides, LDL level, cholesterol/ HDL, LDL / HDL. Furthermore; Amawi et al reported that GCS extract administered orally for four weeks to hypercholesterolemic and diabetic rats resulted in better lipid profile and reduction in blood glucose level. (20, 22) Similar observations were made by a number of workers, demonstrating a hypolipidemic effect of GSP in experimental animals. The modified profile of lipid in cisplatin injection rats was revealed by Halaby et al, after supplemented with 5% and 10% GSP. (32) Of notice in the current study is that inclusion of GSP in HFD (10% diet) resulted in TCh and LDL-cholesterol levels even higher than the HFD treated animals; this might be explained by the fact that they showed a drastic increment of 17.4% in body weight higher than the HFD treated group and an increased consumption of HFD. Yet GSP in a dose of 10% significantly lowered TG as compared to HFD *P5=0.02*, and the higher dose had almost the same results *P8=0.404*.

In the current research, negative control group livers showed healthy hepatic tissue. HFD group livers displayed areas of degenerated hepatocytes. There were large hepatic vacuoles with an appearance of signet rings, Figures 2. Liver of G III/GSP 10% diet treated rats showed normal histological pattern in spite of their higher body weight and high TCh. Normal liver histology was detected in G IV/ GSP 15% diet and G IV/ Lipitor, Figures 3.

In the JUPITER research, a high CRP was shown to identify a greater risk group using statins to avoid incidents of vascular disease and enhance survival [33]. High sensitivity CRP was lowered and cardiac incidents were effectively reduced by 62% after statins treatment. Baseline CRP concentrations are correlated with a higher risk of stroke in patients with acute coronary syndromes [34]. Generally, the classification strata of elevated risk were > 3 mg / L and low risk < 1 mg / L whereas intermediate risk was regarded in rates of 1 to 3 mg / L. [35] Obesity, overweight and high concentrations of CRP are considered variables that pose a significant health threat particularly for cardiovascular diseases. Increased risk of myocardial infarction, ischemic stroke and peripheral arterial disease was linked to concentrations of CRP above 10 mg / l. These levels are also expected to predict the incidence of cardiovascular disease in patients [36].

In comparison to a healthier cautious diet Fung et al discovered a positive correlation between CRP and the Westernized diet. Although CRP was not related to individual foods or nutrients, it is usually suggested that elevated fat consumption, in particular saturated and trans fatty acids, and inflammation are positively associated [37].

Study finding indicated that HFD resulted in a significantly tremendous elevation in CRP levels (30.3 ±9.9 mg/L) compared to control group (1.2 ± 0.61 mg/L) P1<0.005. Both supplementary doses of GSP caused significant decrease in CRP and atorvastatin was slightly more effective; 60, 70, 80% decrease respectively for GSP 10%, GSP 15% and atorvastatin.

Liver toxicity is evidenced to be a probable side effect of a great number of drugs [38]. In addition to the adverse impacts on liver and muscle, kidney inadequacy, and hyperthyroidism; synthetic hypolipidemics are costly. Moreover, serum transaminases are elevated, which can be a significant side effect in connection with atorvastatin treatment [39].

Clinical proof obviously supports the use of statins for the prevention of cardiovascular disease. In January 2014, however, the United States FDA issued guidance on statin hazards documenting the following: “statin benefit is indisputable, but they need to be taken with care and knowledge of their side effects”. Of statin hazards; myopathy is a quite prevalent experience for patients, it ranges from clinically quiescent myalgia to life-threatening, rare rhabdomyolysis [40].

In light of their powerful protective impacts against CVD, this presents a significant challenge for clinicians. Thus, before initiating treatment with statins, a reasonable screening of patients at risk or those with adverse impacts should be conducted by a clinician and alternative methods should be considered when such adverse effects occur. GSP could ameliorate the dysmetabolic disturbances induced by HFD. GSP in the studied dose of 10% increase body weight significantly in rats. However, it decreased CRP and triglycerides close to other treatment groups. Its use in humans should be accompanied by a calorie controlled healthy diet with modifications in the type of fat; like the therapeutic lifestyle change diet (TLC diet).

The sensory characteristics of the products were analyzed on a 9 point hedonic scale. The color, taste and texture of garden cress seed products were desirable. The result of sensory evaluation showed that the incorporation of garden cress seed flour up to 10% was found to be most acceptable to obtain cupcake, while for biscuit and pizza, both 10% and 20% were acceptable.

During the present investigation it was observed that moisture; protein and ash content of different treatments with GCSP (10, 20, and 30%) were increased compared with control while the content of fat decreased (table 3). The moisture content of garden cress seed product samples was higher than those of control because garden cress seed powder is hygroscopic in nature. Higher dose of supplementation could improve nutritional quality.

Many supplementation studies have been conducted on garden cress seeds and its products producing good results; especially the modification and enrichment of snacks. The seeds are not common among the population, despite being rich in nutrients. Bitterness of the seeds is one of the factors which significantly decrease its acceptability [41-42].

The cost of food products was calculated in Egyptian pounds at the time of preparation of the experiment. Total cost of control group for cupcake, biscuits and pizza were 5.40, 3 and 9.8 LE/100 gm respectively. Fortification with 10% of GSP cost was comparable to control.

Products supplemented by GSP are more economical compared to the prices of specialized food products for diabetics and obese in the form of snacks and cookies. Instead of buying high- calorie, ready- made foods that are popular among them healthier version of these snacks can be tried at home. Similar findings were documented by others.

Table 1: Chemical analysis of GSP:

Sample	Moisture**%**	**Protein%**	**CHO%**	**Fats%**	**Fiber%**	**Dry matter%**
GSP	7.44	23.12	27.16	30.29	11.99	92.56

Table 2: %Weight change, Serum lipid profile and CRP of –ve control, rats fed on HFD, HFD with GSP 10&15%, and HFD with Lipitor® 15mg/kg:

Test	**G I/-ve control**	**G II/+ ve control**	**G III/GSP 10%**	**G IV/ GSP 15%**	**G V/ Atorvastatin**
%Weight change after 8 weeks	**50.8%**	**64.6%**	**82%**	**47%**	**46.5%**
TCh mg/dL Mean ± SD	96.5 ±16.8	153.2 ±25.2 P1<0.005*	180.0 ±23.6 P2<0.005* P5=0.064	133.4 ±26.7 P3=0.009* P6=0.178 P8=0.005*	119.8 ±13.4 P4=0.015* P7=0.009* P9<0.005* P10=0.252
TG mg/dL Mean ± SD	55.0 ±14.6	139.0 ±32.7 P1=<0.005*	102.1 ±16.1 P2<0.005* P5=0.02*	108.2 ±22.1 P3<0.005* P6=0.062 P8=0.565	75.2 ±11.6 P4=0.014* P7<0.005* P9=0.004* P10=0.004*
HDL mg/dL Mean ± SD	41.2 ±4.6	29.2 ±5.1 P1=0.001*	36.0 ±5.8 P2=0.088	33.2 ±5.8 P3=0.015*	46.8 ±7.1 P4=0.107

			P5=0.043*	P6=0.201 P8=0.404	P7<0.005* P9=0.009* P10=0.002*
LDL mg/dL Mean ± SD	44.2 ±16.7	96.2 ±24.3 P1=0.001*	123.5 ±23.0 P2<0.005* P5=0.051	78.4 ±25.1 P3=0.011* P6=0.205 P8=0.004*	57.9 ±18.9 P4=0.178 P7=0.007* P9<0.005* P10=0.110
CRP mg/L Mean ± SD	1.2 ± 0.61	30.3 ±9.9 P1<0.005*	11.1 ±4.6 P2<0.005* P5=0.001*	9.3 ±3.2 P3<0.005* P6<0.005* P8=0.398	5.1 ±1.3 P4=<0.005* P7<0.005* P9=0.006* P10=0.009*
P1, p2, p3, p4: P value for comparing between G l and other groups for ANOVA P5, p6, p7: P value for comparing between GII and other groups for ANOVA P8, p9: P value for comparing between GIII and other groups for ANOVA P10: P value for comparing between GIV and GV for ANOVA *: statistically significant at $p \leq 0.05$ for ANOVA					

Table 3: Chemical analysis of garden cress products:

Product	Moisture%	Protein%	Ash%	Fat%	Total energy/100gm
Cupcakes					
Control	14.23	11.11	0.65	7.66	378 Kcal
10%	28.24	8.61	1	7.67	321 kcal
20%	30.71	10.40	1.71	9.81	318 kcal
30%	31.27	10.44	2.1	9.86	316 kcal
Biscuits					
Control	1.56	5.8	1.75	18.8	480 kcal
10%	2.31	5.98	2.61	18.21	470 kcal
20%	2.45	6.61	2.89	18.05	468 kcal
30%	3.34	6.91	3	17.91	463 kcal
Pizza					
Control	12.34	9.95	1.93	19.50	440 kcal
10%	12.88	10.56	2.48	18.22	429 kcal
20%	13.50	10.67	2.55	17.28	374 kcal
30%	13.88	10.88	2.78	17.21	370 kcal

Table 4: Sensory analysis of garden cress seed products:

(Products)	Color	Odor	Texture	Taste	Over all acceptability	P (AVOA)
Pizza						
Control	8.4 ± 0.62	8.3 ± 0.97	8.5 ± 0.57	8.6 ± 0.62	8.5 ± 0.57	0.0001
10%	7.9 ± 1.15	7.5 ± 1.05	8.1 ±0.75	7.8 ± 0.60	8.1 ± 0.80	
20%	7.8 ± 0.77	7.3 ± 1.17	7.5 ±1.18	7.4 ± 1.12	7.5 ± 1.15	
30%	7.0 ± 0.80	6.1 ± 1.28	6.9 ± 0.95	6.4 ± 1.32	5.9 ± 1.11	
Biscuits						
Control	7.5 ± 1.59	8.0 ±0.94	8.2 ±0.72	7.3 ± 1.71	7.8 ± 1.08	0.0001
10%	6.6 ± 1.72	6.2 ± 2.08	6.8 ±1.95	5.8±2.56	6.3 ± 1.93	
20%	6.1 ± 2.04	6.4 ± 2.57	6.7 ±2.54	5.4±2.56	5.8± 2.44	
30%	5.4± 1.37	5.0 ± 2.40	5.8 ±1.78	4.1±1.54	4.8± 1.46	
Cupcakes						
Control	8.2 ± 1.04	7.6 ± 1.43	7.6 ± 1.10	7.6±1.60	8.2 ±1.06	0.0001
10%	6.4 ± 2.12	5.7 ± 2.54	6.1 ± 2.18	5.52 ± 1.78	7.4 ± 1.60	
20%	5.5 ± 1.80	4.0± 2.13	5.1±1.83	4.8 ± 2.43	6.3 ± 1.49	
30%	4.1 ± 2.20	3.1 ± 1.56	3.9 ± 2.23	3.1 ± 2.14	3.3± 2.34	

Table 5: The cost of garden cress products per100gm:

Products	Control	10%	20%	30%
Cupcakes	5.40 LE	6.78 LE	8.20 LE	9.60 LE
Biscuits	3 LE	3.45 LE	3,95 LE	4.25 LE
Pizza	9.87 LE	10.32 LE	11.72 LE	13.125 LE

Fig 1: 10, 20 and 30% GSP incorporated in pizza, biscuits and cupcakes.

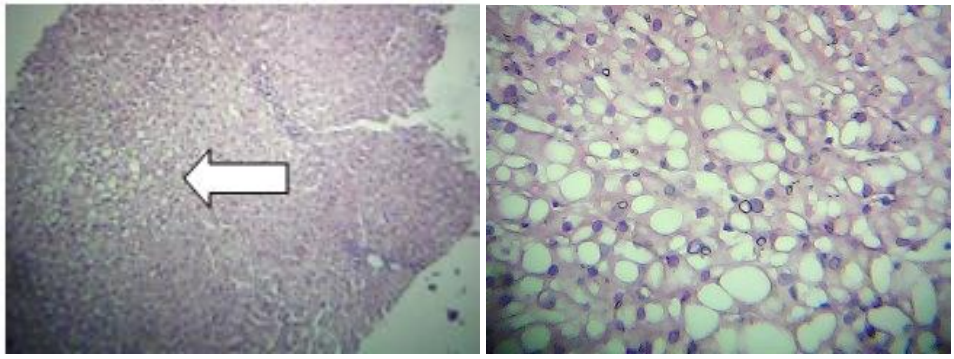

Fig 2: Changes elicited by HFD in the liver (positive control) Left: The low-power view shows moderate steatosis. Right: The high-power view shows swollen hepatocytes with macrovesicular steatosis and signet ring profile.

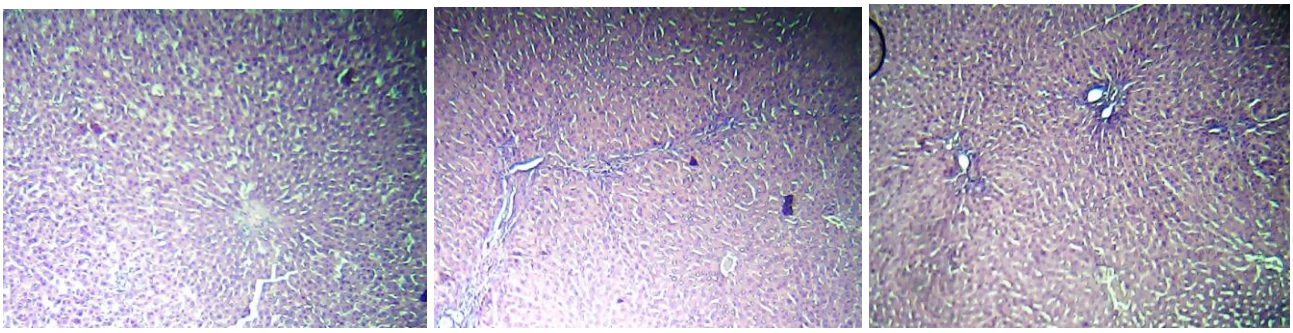

Fig 3: The effect of treatment groups on the histological structure of the liverLeft: Liver of G III/GSP 10% diet treated rats showing normal hepatocytes. Middle: Normal liver histology of G IV/ GSP 15% diet. Right: Normal liver histology of G IV/ Lipito.

Conclusions

Our HFD 46% confirmed the induction of dyslipidemia, which was indicated by an altered lipid profile in positive controls. GSP has an antihyperlipidemic activity. It significantly reduces lipid parameters and enhances the good cholesterol. Moreover, in a dose of 15% it has an effect that is comparable to atorvastatin in a dose of 15 mg/kg. Novell in the present research; is the potential cardioprotective effect of GSP that could be in part due to its antioxidant and anti-inflammatory activities through significant lowering of CRP levels.

The Result of sensory evaluation showed that the incorporation of garden cress seed flour up to 10% for cupcakes, and 20% to obtain biscuit, and pizza, were found to be most acceptable with improved nutritional quality and good sensorial attributes. The developed supplemented products contained significantly higher moisture, protein, fiber content and were lower in fat content. GSP is a promising multipurpose medicinal source whereas further clinical trial is required to prove its efficacy.

References

1. Hernandez BJE, Leon J. (eds.). Neglected crops: 1492 from a different perspective. FAO plant production and protection series. Food and Agriculture Organization of United Nations, Rome. 1994; 26:307-10. http://www.fao.org/docrep/018/t0646e/t0646e.pdf.
2. Gokavi SS, Malleshi NG, Guo M. Chemical composition of garden cress (*Lepidium sativum*) seeds and its fractions and use of bran as a functional ingredient. Plant Foods for Human Nutrition. 2004; 59(3):105-111.
3. Mali RG, Mahajan SG, Mehta AA. Lepidium sativum (garden cress) a review of contemporary literature and medicinal properties. Oriental Pharmacy and Experimental Medicine. 2007; 7(4):331-335
4. Al-yahya MA, Mossa JS, Ageel M, Rafatullah S. Pharmacological and safety evaluation studies on *Lepidium sativum* L., Seeds. Phytomedicine. 1994; 1:155-159.
5. Raghavendra RH, Akhilender NK. Eugenol and n-3 rich garden cress seed oil as modulators of platelet aggregation and eicosanoids in Wistar albino rats. The Open Nutraceuticals Journal. 2011; 4:144-150.

6. Moser BR, Shah SN, Winkler-Moser JK, Vaughn SF, Evangelista RL. Composition and physical properties of cress (*Lepidium sativum* L.) and field pennycress (*Thlaspi arvense* L.) oils. Industrial Crops and Products. 2009; 30:199-205.
7. Zia-Ul-Haq M, Ahmad S, Calani L, Mazzeo T, Rio DD, Pellegrini N, Feo VD. Compositional study and antioxidant potential of Ipomoea Hederacea Jacq and *Lepidium sativum* L. seeds. Molecule. 2012; 17: 10306-21.
8. Shail, Manjari D, Neeraj K, Gupta LN, Nutritional importance of *Lepidium sativum* L. (Garden cress/Chandrashoor):A Review. Pharmacy and Analytical Research. 2016; 5(1):152-160.
9. World Health Statistics, 2018. https://apps.who.int/iris/bitstream/handle/10665/272596/9789241565585-eng.pdf
10. Attia et al. Country report Egypt – October 2014. Available from: https://www.escardio.org/static_file/Escardio/Subspecialty/EACPR/egypt-country-report.pdf
11. WHO and ARE-Ministry of Health & Population: Egypt National STEPwise Survey of Non Communicable Diseases Risk Factors 2011-2012, 2.
12. Nelson RH. Hyperlipidemia as a Risk Factor for Cardiovascular Disease. Prim Care. 2013; 40(1): 195-211.
13. Elmahdi B, El-Bahr SM. Influence of Dietary Supplementation of Fenugreek (*Trigonella foenum-graecum* L.) on Serum Biochemical Parameters of Rats Fed High Cholesterol Diet. International Journal of Biological Chemistry. 2015; 9:1-10.
14. Taylor F, Huffman MD, Macedo AF *et al*. Statins for the primary prevention of cardiovascular disease. Cochrane Database Syst Rev, 2013, 1(1).
15. Huang WC, Lin TW, Chiou KR *et al*. The effect of intensified low density lipoprotein cholesterol reduction on recurrent myocardial infarction and cardiovascular mortality. Acta Cardiol Sin. 2013; 29(5):404-12.
16. Odden MC, Pletcher MJ, Coxson PG *et al*. Cost-effectiveness and population impact of statins for primary prevention in adults aged 75 years or older in the United States; statins for primary prevention in U.S. adults aged 75 years or older. Ann Intern Med. 2015; 162(8):533-41.
17. Bafeel SO, Ali SS. The potential liver toxicity of Lepidium sativum seeds in albino rats. Res Biol Sci. 2009; 4:1250-1258
18. Datta PK, Diwaker BT, Viswanatha S, Murthy KN, Naidu KA. Safety evaluation studies on Garden cress (*Lepidium sativum* L.) seeds in Wistar rats. Int J Appl Res Nat Prod. 2011; 4:37-43
19. Althnaian T. Influence of dietary supplementation of Garden cress (*Lepidium sativum* L.) on liver histopathology and serum biochemistry in rats fed high cholesterol diet. javar. 2014; 1(4):216-223.
20. Al-Hamedan WA. Protective Effect of *Lepidium sativum* L. Seeds Powder and Extract on Hypercholesterolemic Rats. Journal of American Science. 2010; 6(11): 873-879.
21. Chauhan K, Sharma S, Agarwal N, Chauhan S, Chauhan B. A study on potential hypoglycemic and hypolipidemic effects of *Lepidium sativum* (Garden Cress) in Alloxan induced diabetic rats. American Journal of Pharm Tech Research. 2012; 2:522-535.
22. Amawi K, Aljamal A. Effect of Lepidium Sativum on Lipid Profiles and Blood Glucose in Rats. Journal of Physiology and Pharmacology Advances, 2012; 2(8): 277-281.
23. Mohamed ET, Safwat GM. Evaluation of cardioprotective activity of *Lepidium sativum* seed powder in albino rats treated with 5-fluorouracil. Beni-Suef university journal of basic and applied sciences. 2016; 5:208-215.
24. Buettner R, Scholmerich J, Bollheimer LC. High fat diets: modeling the metabolic disorders of human obesity in rodents. Obesity. 2007; 15:798-808.
25. Moussavi N, Gavino V, Receveur O. Could the quality of dietary fat, and not just its quantity, be related to risk of obesity? Obesity. 2008; 16:7-15.
26. Ailhaud G, Massiera F, Weill P, *et al*. Temporal changes in dietary fats: role of n-6 polyunsaturated fatty acids in excessive adipose tissue development and relationship to obesity. Prog Lipid Res. 2006; 45:203-6.
27. Piers LS, Walker KZ, Stoney RM, *et al*. Substitution of saturated with monounsaturated fat in a 4-week diet affects body weight and composition of overweight and obese men. Br J Nutr. 2003; 90:717-727.
28. Silva APS, Guimaraes DED, Mizurini DM. *et al*. Dietary fatty acids early in life affect lipid metabolism and adiposity in young rats. Lipids. 2006; 41:535-541.
29. Shukla A.K, Bigoniya P, Soni P. Hypolipidemic Activity of *Lepidium sativum* Linn. Seed in Rats. IOSR Journal of Pharmacy and Biological Sciences. 2015; 10:13-22.
30. Jain KS, Kathiravan MK, Somani RS, Shishoo CJ. The biology and chemistry of hyperlipidemia. Bioorg Med Chem. 2007; 15(14):4674-99.
31. Konda Vg, Madhavi E, Ruckmani A, Venkataramana Y. A Review on Medicinal Plants with Potential Hypolipidemic Activity. International Journal of Pharma and Bio Sciences. 2013; 4(4):729-740.
32. Halaby MS, Farag MH, Mahmoud SAA. Protective and curative effect of garden cress seeds on acute renal failure in male albino rats. Middle East J Appl Sci 2015; 5(2):573-86.
33. Ridker PM, Danielson E, Fonseca FAH *et al*. for the JUPITER Study Group. Rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein. N Engl J Med. 2008; 359:2195-207.
34. Hermusa L, Lefrandtb JD, Tioc RA, Breek JC, Zeebregts CJ. Carotid plaque formation and serum biomarkers. Atherosclerosis. 2010; 213:21-9.
35. Montgomery JA, Brown JR. Metabolic biomarkers for predicting cardiovascular disease. Vascular Health and Risk Management. 2013; 9:37-45
36. Ramdas J, Jella V. Elevated C reactive protein levels in obese individuals with metabolic syndromes. Int J Adv Med. 2016; 3(2):162-165.
37. Fung TT, Rimm EB, Spiegelman D, Rifai N. Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk. Am J Clin Nutr. 2001; 73:61-67.
38. Magni P, Macchi C, Morlotti B, Sirtori CR, Ruscica M. Risk identification and possible countermeasures for muscle adverse effects during statin therapy. European Journal of Internal Medicine. 2015; 26: 82-88.
39. Macedo AF, Taylor FC, Cases JP. Unintended effects of statins from observational studies in the general population: systemic review and meta-analysis. BMC Med. 2014; 12:51.
40. Farag MM, Mohamed MB, Youssef EA. Assessment of hepatic function, oxidant/antioxidant status, and

histopathological changes in rats treated with atorvastatin-Effect of dose and acute intoxication with acetaminophen. Hum Exp Toxicol. 2015; 34(8):828-37.
41. Deshmukh YR, Thorat SS, Mahaska SR. Influence of Garden Cress Seed (*Lepidium sativum* L.) Bran on Quality Characteristics of Cookies. Int. J Curr. Microbiol. App. Sci. 2017; 6 (9):586-593.
42. Jain T, Grover K, Gill NK. Impact of garden cress supplemented biscuits on nutritional profile of malnourished and anemic school children (seven–nine years). Nutrition & Food Science. 2017; 47:553-66.
43. Jain T, Grover K, Grewal IS. Development and Sensory Evaluation of Ready to Eat Supplementary Food Using Garden Cress (*Lepidium sativum*) Seeds. Journal of Applied and Natural Science. 2016; 8:1501-1506.
44. Elizabeth GK, Poojara RH. Organoleptic Attributes of Garden Cress Seed Incorporated Snacks Suitable for Adolescents. IJFANS. 2014; 3:126-129.