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Lipids Profile Reduction in Rats Fed (Matthiola Incana) Seed Rich in Omega-3 Fatty Acids

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Abstract

The present study was designed to study the effect of Stock seeds, virgin coconut oil and sunflower oil on hyperlipidemic rats. Forty adult male albino rats were used in this study, weighting (150±10g) were divided into eight groups, five rats each. One of them was kept as a control –ve group, while the other seven groups were fed on the diet plus 1.5% cholesterol for 21 days Stock seeds powder added at percent 2.5% and 5%, virgin coconut and sunflower oil were added at percent 1% and 2% from the main diet. Body weight gain, feed intake, feed efficiency ratio, serum lipid profiles (TG, TC, LDL-c, VLDL-c, HDL-c and AI), serum glucose, serum liver enzymes (ALT, AST and ALP), kidney functions (creatinine, uric acid and urea levels) and complete blood count (RBCs, WBCs, hemoglobin and platelet). From the obtained results it could be concluded that feeding on of Stock seeds powder, virgin coconut oil and sunflower oil caused significant ($P \leq 0.05$) increase in HDL-c, RBCs and hemoglobin, but with significant ($P \leq 0.05$) decreases weight gain and in the rest of the analyses as compared with control (+ve) group, and enhanced the kidney and liver functions with the decrease of ALT, AST, ALP, creatinine, uric acid, urea and serum glucose which reflects the powerful nutraceutical therapeutic effect for feeding on of Stock seeds, virgin coconut oil and sunflower oil for treatment hyperlipidemia in rats.

Keywords*: high lipids, Stock seeds, Plants oils, Rats, Biochemical analysis.*

Introduction

Hyperlipidemia is considered one of the major risk factors causing cardiovascular diseases (CVDs). CVDs accounts for one third of total deaths around the world (Jørgensen et al., 2013).

Hyperlipidemia is an increase in one or more of the plasma lipids, including triglycerides, cholesterol, cholesterol esters and phospholipids and or plasma lipoproteins including very low-density lipoprotein and low-density lipoprotein, and reduced high-density lipoprotein levels Hyperlipidemia relates to increased oxidative stress causing significant production of oxygen free radicals, which may lead to oxidative modifications in low-density lipoproteins, which present a significant function in the initiation and progression of atherosclerosis and associated cardiovascular diseases (Mishra et al., 2011).

It is not a disease but a metabolic disorder that can be secondary to many diseases and can contribute to many forms of disease, most notably cardiovascular disease. Longstanding elevated hypercholesterolemia leads to accelerated atherosclerosis; this can express itself in a number of cardiovascular diseases: coronary artery disease (angina pectoris, heart attacks), stroke and short stroke-like episodes and peripheral vascular disease (Durrington, 1995).

Hypercholesterolemia and hypertriglyceridemia are the main cause of atherosclerosis which is strongly related to is chemic heart disease (IHD) (Brouwers et al., 2012). There is a strong relation between IHD and the high mortality rate. Furthermore, elevated plasma cholesterol levels cause more than four million deaths in a year. Atherosclerosis is a process of arteries hardening due to deposition of cholesterol in the arterial wall which causes narrowing of the arteries. Atherosclerosis and atherosclerosis-associated disorders like coronary, cerebrovascular and peripheral vascular diseases are accelerated by the presence of hyperlipidemia (Wells et al., 2007).

Hyperlipidemia is typically caused by obesity, dietary intake, and other environmental and genetic factors or a combination of both (Bhatnagar et al., 2008). Type 2 diabetes mellitus, alcohol, dialysis, monoclonal gammopathy, hypothyroidism, anorexia nervosa, nephrotic (Schwingshackl et al., 2017).

Virgin coconut oil (VCO) is produced from fresh coconut, which is produced without high heating, so that the important content in coconut oil can be maintained (Aladin et al., 2016). VCO has a rich content of medium chain fatty acids (MCFAs), predominantly lauric acid; others include caproic acid, caprylic acid and capric acid (Mansor, et al., 2012). VCO is known for its medicinal properties as anti-inflammatory, analgesic and hypothermic properties, antimicrobial and VCO showed significant anticoagulant effect (Dumancas et al., 2016).

In addition to many medicinal benefits of VCO which have been mentioned, it has beneficial effects on lipid profile Arunima and Rajamohan (2012), who found that treating rats with VCO compared with copra oil (CO), olive oil (OO), and sunflower oil (SFO) as they were fed with different oils 8% for 45 days the Results showed that VCO feeding was significantly reduced ($P<0.05$) levels of total cholesterol, LDL and VLDL so VCO had the ability to reduce hyperlipidemia.

There have also been many scientific reports on the health benefits and nutritional potential of the bioactive compounds of sunflower oil .It is the non-volatile oil extracted from sunflower (Helianthus annus) seeds of the Asteraceae family. The sunflower oil is interesting by its content in linoleic acid. It is a mixture of monounsaturated and polyunsaturated fats with low levels of saturated fats (Madhavi et al., 2015).

Sunflower seed oil is characterized by a high concentration of linolenic acid, moderate level of oleic acid, very low level of linoleic acid, less than 15% of the saturated fatty acids, palmitic and stearic acids and usually less than 1% of acids with fewer than 16 or more than 18 carbon atoms. While lauric, arachidic, behenic, lihnoceric and eicosenoic acids may be present, these acids are of little practical importance. Traces of oxygenated fatty acids also have been found in some sunflower seeds stored for prolonged period (Mikolajczak et al., 1968).

The seed oil and herb tincture are employing for anti-inflammatory, antioxidant, antitumor, anti-asthmatic, antigen, antipyretic, astringent, anti-hypoglycemic effect, cathartic, diuretic, stimulant, vermifuge and antimicrobial activities (Bashir et al., 2015).

Sunflower oil has beneficial effects on lipid profile Basak et al. (2017), who found that the group treated with sunflower oil differed significantly ($P \leq 0.05$)from the control group, and the total triglycerides decreased significantly ($P \leq 0.05$)HDL was significantly increased ($P \leq 0.05$)in the treated group compared with the control group.

Stock seeds is an oilseed of great importance. The stock flower (Matthiola incana, L.) is a species from the Brassicaceae family. It is an ornamental plant (Sanchez et al., 2005). Seeds of the stock flower contain oil rich in linolenic acid (55-65%) that is of medicinal importance (Yaniv et al., 1999).

Chopra et al., (1986), reported that the seeds of Stock acted as a diuretic, expectorant, tonic, stomach tonic and also worked as a cancer treatment.

In addition to it has beneficial effects on lipid profile Yaniv et al., (1999), reported that treating hypercholesterolemia male rats with Stock oil for 6 weeks compared with rats fed a diet containing coconut oil or sunflower oil it significantly reduced TC, TG and LDL-c as Stock seeds contained oil rich (55--65%) in omega-3 (n - 3) linolenic acid and elicited a beneficial effect when fed to animals by reducing cholesterol levels.

The main objective of this work is to study the effect of stock seeds, virgin coconut oil and sunflower oil on the biological, biochemical changes of male white rats infected with high blood lipids and the possibility of improving this condition.

Material And Methods

Materials

1- Source of Stock seeds, virgin coconut oil and sunflower oil: virgin coconut oil and sunflower oil were obtained from the Agriculture Research Center in Giza, Egypt.

Stock ***Seeds was obtained from the Herbal Store in Cairo , Egypt.***
2. Cholesterol powder: Cholesterol powder obtained from Al-Gomhoria Company for Trading Drugs, Chemical and Medical Instruments, Cairo, Egypt.
3. Experimental animals: A total of 40 adult normal male albino rats Sprague Dawley strain weighing 150±10g were obtained from Vaccine and Immunity Organization, Ministry of Health, Helwan Farm, Cairo, Egypt.
Chemical kits: Chemical kits used in this study (TC, TG, HDL-c, LDL-c, VLDL-c, ALT, AST, albumin, globulin, total protein, glucose, creatinine, uric acid, urea) El-Gomhoria company, Cairo, Egypt.

Biological experiments:

Basal diet composition:

The basal diet in the experiment was prepared according to Reeves et al., (1993). It was consisted of 20% protein (casein), 10% sucrose, 4.7% corn oil, 0.20% choline chloride, 1% vitamin mixture, 3.5% salt mixture and 5% fiber (cellulose).

Experimental design:

The experimental was done in the Faculty of Home Economics, Menoufia University, Shebin El-Kom. forty adult male white albino, 10weeks age, weighting (150±10 g) was used in this experiment. All rats were fed on standard diet according to American Institute of Nutrition (AIN) (1993) for 7 days for adaptation and divided into two main groups.
The first main group fed on basal diet as a control negative group (5 rats).
The second main group (hyperlipidemic rats) (35 rats) hyperlipidemia was induced in normal healthy male albino rats by addition of 1.5% cholesterol powder and 10% animal fat for 21 days.
And were divided into 7 sub-groups (5 rats for each group) as the following:
Sub-group 1: Hyperlipidemic rats fed on basal diet as a control positive group.
Sub-group 2: Hyperlipidemic rats fed on basal diet with 2.5% of Stock powder.
Sub-group 3: Hyperlipidemic rats fed on basal diet with 5% of Stock powder.
Sub-group 4: Hyperlipidemic rats fed on basal diet with 1% of virgin coconut oil.
Sub-group 5: Hyperlipidemic rats fed on basal diet with 2% of virgin coconut oil.
Sub-group 6: Hyperlipidemic rats fed on basal diet with 1% of sunflower oil.
Sub-group 7: Hyperlipidemic rats fed on basal diet with 2% of sunflower oil.

Biological Evaluation:

Body weight gain (BWG), feed intake (FI), and feed efficiency ratio (FER):
During the experimental period (28 days) the net feed intake was daily recorded, while body weight was weekly recorded. The net feed intake and gained body weight were used

for the calculation of feed efficiency ratios (FER) according to Chapman et al., (1959) as follow:

$$FER\% = \frac{Body\ weight\ gain\ (g)}{Food\ intake\ (g)} \times 100$$

Blood sampling:

After fasting for 12 hours, blood samples in initial times were obtained from retro orbital vein, while it obtained from hepatic portal vein at the end of each experiment. Blood samples were collected into a dry clean centrifuge glass tubes and left to clot in water bath (37°C) for 30 minutes, then centrifuged for 10 minutes at 4000 rpm to separate the serum, which were carefully aspirated and transferred into clean cuvette tube and stored frozen in deep freezer till analysis according to method described by Schermer (1967).

Biochemical analysis:

Determination of blood glucose

Serum blood glucose was measured using the modified kinetic method according to Kaplan, (1984) by using kit supplied by spin react. Spain.

Liver functions

Determination of alanine amino transferase (ALT) (GPT)

ALT activities were measured in serum using the modified kinetic method of Tiez, (1976) by using kit supplied by Human, Germany.

Determination of aspartate amino transferase (AST) (GOT)

AST activities were measured in serum using the modified kinetic method of Henry, (1974) by using kit supplied by human, Germany.

Kidney functions

Determination of urea nitrogen

Urea was determination in serum using the modified kinetic method or liquicolor of Patton and crouch, (1977) by using kit supplied by Human, Germany.

Determination of creatinine

Serum creatinine was measured using the modified kinetic method according to Henry, (1974) by using kit supplied by Human, Germany.

Determination of uric acid:

Serum uric acid was measured using the modified kinetic method according to Schultz, (1984)by using kit supplied by Human, German.

Lipid's profile

Determination of total cholesterol (T.C)

Serum cholesterol was measured using the modified kinetic according to Richmond, (1973) by using kit supplied by Hu Germany.

Determination of triglycerides (T.G)

Serum triglycerides (T.G) were measured using the modified kinetic method according to the method described by Fossati and Prencipe, (1982) by using kit supplied by Spinreact, Spain.

Determination of high-density lipoprotein cholesterol (HDL-c)

Serum high density lipoprotein cholesterol (HDL-c) was measured using the modified kinetic method according to Allain, (1974) by using kit supplied by Human, Germany.

Determination of very low-density lipoprotein cholesterol (VLDL-c)

Serum very low-density lipoprotein cholesterol (VLDL-c) was calculated as mg/dl according to Lee and Nieman, (1996) equation:

VLDL-c Concentration mg/dl = $\frac{T.G}{5}$

Determination of low-density lipoprotein cholesterol (LDL-c)

Serum low density lipoprotein cholesterol (LDI-c) was calculated as mg/dl according to Castelli et al., (1977) equation:

LDL Concentration mg/dl = Total Cholesterol – HDL-c – VLDL-c

Calculation of atherogenic index:

The VLDL + LDL / HDL ratio: this index was calculated according to the formula of Kikuchi et al., (1998)

Statistical analysis

The data were analyzed using a completely randomized factorial design (SAS, 2002) when a significant main effect was detected; the means were separated with the student-Newman-Keuls Test. Differences between treatments of ($P \leq 0.05$) were considered significant using Costat Program. Biological results were analyzed by One Way ANOVA.

Results and Discussion

Data presented in Table (1) and show the effect of Stock powder, virgin coconut oil and sunflower oil on serum triglycerides (T.G) and serum total cholesterol (T.C) of hyperlipidemic rats. The obtained results indicated that the higher serum triglycerides levels recorded for positive control group, while negative control group recorded the lower value with significant differences. The mean values were 206.80a±0.20 and 79.35h±0.84 mg/dl, respectively.

On the other hand, the highest serum triglycerides levels of treated groups recorded for 2.5% Stock powder, while the lowest value recorded for 2% virgin coconut oil with significant differences. The mean values were 163.20b±0.88 mg/ dl and 105.30g±0.07 mg/ dl, respectively. These results are in agree with Adeyemi et al., (2020), who found

that treating rats with VCO has a beneficial effect in lowering lipid profile. It reduced TG and TC. so VCO had the ability to reduce hyperlipidemia. Also, Go et al., (2014), reported that treating hypercholesterolemic rats with sunflower oil did significantly reduce serum TG. Nevin and Rajamohan (2004), reported that treating hypercholeterolemic male rats with VCO it significantly reduced TG.

In case of serum total cholesterol levels, it could be concluded that the higher serum cholesterol levels recorded for positive control group, while negative control group recorded the lowest value with significant differences. The mean values were 198.60a±0.80 and 94.09h±0.36 mg/dl, respectively. For treated groups, the highest serum total cholesterol levels recorded for 2.5% Stock powder, while the lowest value recorded for 2% virgin coconut oil with significant differences. The mean values were 172.17b±0.50 and 112.10g±0.60 mg/dl, respectively. These results are in agree with Narayanankutty et al., (2018), reported that treating hepatosteatosis male rats with VCO has significantly reduced levels of total cholesterol and triacylglycerols. . Also, Arunima and Rajamohan (2012), who found that treating rats with VCO compared with copra oil (CO), olive oil (OO), and sunflower oil (SFO) as they were fed with different oils 8% for 45 days the Results showed that VCO feeding was significantly reduced ($P \leq 0.05$) levels of total cholesterol, LDL and VLDL so VCO had the ability to reduce hyperlipidemia. Basak et al., (2017), who found that the sunflower oil treated group significantly ($P \leq 0.05$) differ from that of control group. The total triglyceride was decreased significantly ($P \leq 0.05$) in the treated group compared to control.

Table (1): Effect of matthiola incana powder, virgin coconut oil and sunflower oil on serum triglycerides (TG) and serum total cholesterol (TC) of hyperlipidemic rats:

Groups	Triglycerides (TG) mg/dl	Total cholesterol (TC) mg/dl
G_1 Control negative	79.35^{h}±0.84	94.09^{h}±0.36
G_2 Control positive	206.80^{a}±0.20	198.60^{a}±0.80
G_3 (2.5% stock powder)	163.20^{b}±0.88	172.17^{b}±0.50
G_4 (5% stock powder)	147.80^{c}±0.32	158.60^{c}±0.21
G_5 (1% virgin coconut oil)	131.40^{d}±0.19	130.05^{e}±0.40
G_6 (2% virgin coconut oil)	105.30^{g}±0.07	112.10^{g}±0.60
G_7 (1% sunflower oil)	128.60^{e}±0.54	149.20^{d}±0.71
G_8 (2% sunflower oil)	115.05^{f}±0.05	126.40^{f}±0.35
LSD	0.86	0.91

Each value represents mean±standard deviation (n = 3). Mean under the same column bearing different superscript letters are different significantly (p ≤ 0.05).

Data presented in Table (2) show the effect of stock powder, virgin coconut oil and sunflower oil on high density lipoprotein cholesterol (HDL-c), low density lipoprotein

cholesterol (LDL–c) and very low-density lipoprotein cholesterol (VLDL–c) levels of hyperlipidemic rats. The highest mean value of HDL-c levels recorded for negative control group, while positive control group recorded the lowest value with significant differences. The mean values were 56.30a±0.80 and 32.26f±0.72 mg/dl, respectively.

On the other hand, the higher mean value for (HDL-c) levels of treated groups recorded for 2% virgin coconut oil, while the lower value recorded for 2.5% stock powder with significant differences. The mean values for another treated group were 53.64b±0.96 and 45.70e±0.31 mg/dl, respectively. These results agreed with Narayanankutty et al., (2017), reported that treating hepatosteatosis male rats with VCO increased improving HDL-c level (53.5%) and reducing hepatic and serum triglycerides. Also, Arunima and Rajamohan, (2012), reported that Animals fed VCO showed increase in HDL compared to other groups that fed copra oil (CO), olive oil (OO), and sunflower oil (SFO). Also, Basak et al., (2017), who reported that the sunflower oil treated group showed HDL was increased significantly ($P \leq 0.05$) in the treated group compared to control.

Data also indicated that the higher mean value of LDL-c levels recorded for positive control group, while negative control group recorded the lower value with significant differences. The mean values were 124.98a±0.04 and 21.92h±0.61 mg/dl, respectively.

On the other hand, the highest mean value of levels of LDL-c for treated groups recorded for 2.5% stock powder, while the lowest value recorded for 2% virgin coconut oil with significant differences. The mean values were 93.83b±0.01 and 37.40g±0.37 mg/dl, respectively. These results agreed with Adeyemi et al., (2020), who reported that treating hypercholeterolemic male rats with VCO for four weeks by Using different concentrations of it (VCO 200 +VCO 400 and +VCO 600) it significantly reduced TC, TG and LDL-c.

Data also indicated that the higher mean value of VLDL-c levels recorded for positive control group, while negative control group recorded the lower value with significant differences. The mean values were 41.36a±0.04 and 15.87h±0.17 mg/dl, respectively.

On the other hand, the highest mean value of VLDL-c levels of treated groups recorded for 2.5% stock powder, while the lowest value recorded for 2% virgin coconut oil with significant differences. The mean values were 32.64b±0.18 and 21.06g±0.01 mg/dl, respectively. These results agreed with Nevin and Rajamohan, (2004), reported that treating hypercholeterolemic male rats with VCO it significantly reduced LDL, and VLDL cholesterol levels compared to copra oil. Also, Yıldırım et al., (2014), reported that the serum triglyceride and VLDL cholesterol levels were significantly decreased in only the cocoa butter and sunflower oil ($P \leq 0.05$) as compared to control.

Table (2): Effect of stock powder, virgin coconut oil and sunflower oil on lipid profile of hyperlipidemic rats

Groups	HDL-c (mg/dl)	LDL-c (mg/dl)	VLDL-c(mg/dl)
G_1 Control negative	56.30^{a}±0.80	21.92^{h}±0.61	15.87^{h}±0.17
G_2 Control positive	32.26^{f}±0.72	124.98^{a}±0.04	41.36^{a}±0.04
G_3 (2.5% stock powder)	45.70^{e}±0.31	93.83^{b}±0.01	32.64^{b}±0.18
G_4 (5% stock powder)	47.91^{d}±0.17	81.13^{c}±0.02	29.56^{c}±0.06
G_5 (1% virgin coconut oil)	48.21^{d}±0.60	55.56^{e}±0.24	26.28$^{d±}$0.04
G_6 (2% virgin coconut oil)	53.64^{b}±0.96	37.40^{g}±0.37	21.06^{g}±0.01
G_7 (1% sunflower oil)	46.08^{e}±0.22	77.40$^{d±}$0.38	25.72^{e}±0.11
G_8 (2%sunflower oil)	50.19^{c}±0.12	53.21^{f}±0.22	23.00^{f}±0.01
LSD	**0.99**	**0.54**	**0.17**

Each value is represented as mean±standard deviation (n = 3). Mean under the same column bearing different superscript letters are different significantly (p < 0.05). HDL-c = High density lipoprotein cholesterol. LDL-c = Low density lipoprotein cholesterol. VLDL -c = Very low-density lipoprotein cholesterol.

Data presented in Table (3) show the effect of stock powder, virgin coconut oil and sunflower oil on atherogenic index (AI) and atherogenic fraction (AF) levels of hyperlipidemic rats. It's clear to notice that the higher atherogenic index levels (AI) levels recorded for positive control group, while negative control group recorded the lower value with significant differences. The mean values were 0.81a±0.009 and 0.15h±0.002, respectively. On the other hand, the highest atherogenic index (AI) of treated groups (hyperlipidemic groups) recorded for 2.5% stock powder, while the lowest value recorded for 2% virgin coconut oil with significant differences. The mean values were 0.55b±0.002 and 0.29g±0.008, respectively. These results agreed with Yuiwarti, et al., (2018), who reported that treating these hypercholeterolemic male rats VCO was effective in reducing TG, TC, LDL and VLDL levels compared to virgin olive oil group and control group, so VCO had the ability to reduce hyperlipidemia. Hence, it is obvious that lowering the lipid levels could reduce the risk of cardiac heart disease (CHD) by regression of atherosclerosis.

Data presented in Table (4) show the effect of stock powder, virgin coconut oil and sunflower oil on glucose level of hyperlipidemic rats. It's clear to notice that the highest glucose levels recorded for positive control group, while negative control group recorded the lowest value with significant differences. The mean values were 238.00a±0.38 and 120.90h±0.06 mg/dl, respectively.

On the other hand, the highest glucose levels of treated groups recorded for group 3 (2.5% stock powder), while the lowest value recorded for group 8 (2% sunflower oil) with significant differences. The mean values were 174.00b±0.14 and 134.50g±0.42 mg/dl, respectively. These results are in agree with Adeyemi et al., (2020), reported that treating

hypercholeterolemic male rats with VCO that was used concentrations (200,600 and 400 mg/kg) it was significantly reduced blood glucose. Also, Yıldırım et al., (2014), reported that sunflower oil decreases the serum glucose level compared to control group in rats The mean value in sunflower oil group was 120.33±15.57 mg/dl. On the other hand, the mean value in control groups recorded for serum glucose was 137.08±7.86 mg/dl.

Table (3): Effect of stock powder, virgin coconut oil and sunflower oil on atherogenic index (AI) and atherogenic fraction (AF) of hyperlipidemic rats

Groups	Atherogenic index (AI) Ratio	Atherogenic fraction (AF) Ratio
G_1 Control negative	0.15 ^{h}±0.002	37.79 ^{h}±0.44
G_2 Control positive	0.81 ^{a}±0.009	166.34 ^{a}±0.08
G_3 (2.5% stock powder)	0.55 ^{b}±0.002	126.47 ^{b}±0.19
G_4 (5% stock powder)	0.498 ^{c}±0.001	110.69 $^{c\pm}$0.04
G_5 (1% virgin coconut oil)	0.435 ^{e}±0.005	81.84 ^{e}±0.2
G_6 (2% virgin coconut oil)	0.29 ^{g}±0.008	58.46 ^{g}±0.36
G_7 (1% sunflower oil)	0.447 ^{d}±0.003	103.12 ^{d}±0.49
G_8 (2%sunflower oil)	0.36 ^{f}±0.001	76.21 ^{f}±0.23
LSD	**0.008**	**0.51**

Each value represents mean±standard deviation. Mean under the same column bearing different superscript letters are different significantly (P≤0.05) AI: Atherogenic Index. AF: Atherogenic Fraction.

Table (4): Effect of stock powder, virgin coconut oil and sunflower oil on Glucose level of hyperlipidemic rats

Groups	Glucose (mg/dl)
G_1 Control negative	120.90^{h}±0.06
G_2 Control positive	238.00^{a}±0.38
G_3 (2.5% stock powder)	174.00$^{b\pm}$0.14
G_4 (5% stock powder)	170.10$^{c\pm}$0.57
G_5 (1% virgin coconut oil)	158.6$^{e\pm}$0.32
G_6 (2% virgin coconut oil)	141.9$^{f\pm}$0.63
G_7 (1% sunflower oil)	168.05$^{d\pm}$0.85
G_8 (2% sunflower oil)	134.50$^{g\pm}$0.42
LSD	**0.84**

Each value is represented as mean±standard deviation (n = 3). Mean under the same column bearing different superscript letters are different significantly (p≤ 0.05).

From table (5) data show the effect of stock powder, virgin coconut oil and sunflower oil on liver functions (ALT, AST and ALP) of hyperlipidemic rats. It's clear to notice that the highest ALT levels recorded for positive control group, while negative control group

recorded the lowest value with significant differences. The mean values were 127.57a±0.56 and 36.94h±0.32 U/L, respectively.

On the other hand, the highest ALT levels of treated groups recorded for 2.5% stock powder, while the lowest value recorded for 2% sunflower oil with significant differences. The mean values were 101.29b±0.25 and 61.33g±0.80 U/L, respectively. These results agreed with Yıldırım et al., (2014), reported that the serum enzymes activity levels (ALP, AST and ALT) were significantly decreased in only the cocoa butter and sunflower oil groups as compared to control the mean values in cocoa butter were 274.83±32.63, 200.00 and 64.50 U/L, respectively. The mean values in sunflower oil were 268.94±39.76, 151.00 and 61.00 U/L, respectively.

On the other hand, the mean values in control groups recorded for ALP 312.58±31.39, value recorded for AST 194.00 and value recorded for ALT 70.00 U/L. Sinaga, et al., (2020), they reported that treating male rat with virgin coconut oil to measure hepatic oxidative stress and antioxidant defenses after maximum physical activity caused AST and ALT levels of liver of the VCO-1, VCO-2, and VCO-4 groups were significantly lower than the Control group ($p \leq 0.05$).The decrease in AST and ALT levels in this study due to antioxidant activity and the content of polyphenol compounds found in VCO. These results agreed with Famurewa et al., (2017), reported that treating male rats with 10% VCO supplemented diet significantly decreased ALT and AST activity Compared to mice fed repeatedly heated palm kernel oil (HPO).

With regard to AST, the higher levels recorded for positive control group, while negative control group recorded the lower value with significant differences. The mean values were 266.66a±0.14 and 116.17g±0.20 U/L, respectively. On the other hand, the highest AST levels of treated groups (hyperlipidemic groups) recorded for 2.5% stock powder, while the lowest value recorded for 2% sunflower oil with significant differences. The mean values were 218.76b±0.31 and 130.79g±0.70 U/L, respectively. These results agreed with Yıldırım et al., (2014), reported that the serum enzymes activity levels (ALP, AST and ALT) were significantly decreased in only the cocoa butter and sunflower oil groups as compared to control the mean values in cocoa butter were 274.83±32.63, 200.00 and 64.50 U/L, respectively. The mean values in sunflower oil were 268.94 ± 39.76, 151.00 and 61.00 U/L, respectively. On the other hand, the mean values in control groups recorded for ALP 312.58±31.39, value recorded for AST 194.00 and value recorded for ALT 70.00 U/L. Famurewa et al., (2017), reported that treating male rats with 10% VCO supplemented diet significantly decreased ALT and AST activity Compared to mice fed repeatedly heated palm kernel oil (HPO).

In case of ALP the higher levels recorded for positive control group, while negative control group recorded the lower value with significant differences. The mean values were 309.68a±0.45 U/L and 137.30h±0.15 U/L, respectively.

On the other hand, the highest ALP levels of treated groups recorded for group 3 (2.5% stock powder), while the lowest value recorded for group 8 (2% sunflower oil) with significant differences. The mean values were 280.60b±0.06 and 187.47g±0.73 U/L.

Table (5): Effect of stock powder, virgin coconut oil and sunflower oil on liver functions of hyperlipidemic rats

Groups	ALT (U/L)	AST (U/L)	ALP (U/L)
G_1 Control negative	36.94^{h}±0.32	116.17^{h}±0.20	137.30^{h}±0.15
G_2 Control positive	127.57^{a}±0.56	266.66^{a}±0.14	309.68^{a}±0.45
G_3 (2.5% stock powder)	101.29^{b}±0.25	218.76^{b}±0.31	273.64^{d}±0.53
G_4 (5% stock powder)	67.8^{f}±0.13	191.30^{c}±0.11	265.01d^{c}±0.16
G_5 (1% virgin coconut oil)	100.27^{c}±0.48	187.28^{d}±0.23	280.60^{b}±0.06
G_6 (2% virgin coconut oil)	77.28^{d}±0.19	154.27^{f}±0.88	203.10^{f}±0.75
G_7 (1% sunflower oil)	73.82^{e}±0.66	162.10^{e}±0.45	242.42^{e}±0.81
G_8 (2% sunflower oil)	61.33^{g}±0.80	130.79^{g}±0.70	187.47^{g}±0.73
LSD	**0.83**	**0.79**	**0.93**

Each value represents mean±standard deviation (n = 3). Mean under the same column bearing different superscript letters are different significantly (p ≤ 0.05).

Data presented in Table (6) show the effect of stock powder, virgin coconut oil and sunflower oil on kidney functions (serum urea, uric acid and creatinine) of hyperlipidemic rats. It's clear to notice that the higher serum urea levels recorded for positive control group, while negative control group recorded the lower value with significant differences. The mean values were 69.00a±0.50 and 20.70h±0.14 mg/dl, respectively.

On the other hand, the highest serum urea levels of treated groups recorded for 2.5% stock powder, while the lowest value recorded for 2% sunflower oil with significant differences. The mean values were 64.30b±0.81 and 36.17g±0.19 mg/dl, respectively. Yıldırım et al., (2014), reported that the effect of the cocoa butter and sunflower oil on kidney functions (serum uric acid and creatinine) all treated groups showed significant improvement in kidney functions level compared to control group sunflower oil recorded in both urea, uric acid and creatinine levels 2.83±0.28 and 0.48ab mg/dl respectively.

Control group recorded in both urea, uric acid and creatinine levels 2.17±0.15 and 0.53a mg/dl respectively. In case of uric acid there were increase in value of sunflower oil group and cocoa butter group compared to control group. Famurewa et al., (2017) reported that treating male rats with 10% VCO supplemented diet significantly prevented the HPO-induced nephrotoxicity evident by prominent decreases ($p \leq 0.05$).in urea, creatinine and uric acid in comparison to the HPO control group.

Data also showed that the highest serum uric acid levels recorded for positive control group, while negative control group recorded the lowest value with significant differences. The mean values were 6.70a±0.21 mg/dl and 3.10d±0.60 mg/dl, respectively. On the other hand, the highest serum uric acid levels of treated groups recorded for 2.5% stock powder, while the lowest value recorded for 2% sunflower oil with significant differences. The mean values were 5.60b±0.08 and 3.82c±0.40 mg/dl, respectively. Sinaga et al., (2019), they reported that treating male rat with virgin coconut oil while they are doing their most physical activity due to the low levels of urea and creatinine.
There were no significant differences between 2.5% and 5% stock powder, 1% virgin coconut oil and 1% sunflower oil group. Also, there were no significant differences between 2% virgin coconut oil and 2% sunflower oil group.
Data also indicated that the higher serum creatinine levels recorded for positive control group, while negative control group recorded the lower value with significant differences. The mean values were 1.45a±0.13 and 0.50c±0.01 mg/dl, respectively.
On the other hand, the highest serum creatinine levels of treated groups recorded for 2.5% stock powder, while the lowest value recorded for 2% sunflower oil with significant differences. The mean values were 1.14b±0.13 and 0.67c±0.18 mg/dl, respectively. Yıldırım et al., (2014), reported that the effect of the cocoa butter and sunflower oil on kidney functions (serum uric acid and serum creatinine) all treated groups showed significant improvement in kidney functions level compared to control group sunflower oil recorded in creatinine level 0.48ab mg/dl. control group recorded creatinine level 0.53a mg/dl. In case of creatinine was decrease in sunflower oil group. This result is agreement with this study. Sinaga et al., (2019), reported that the VCO administration can reduce levels of urea and creatinine when rats perform maximum physical activity.

Table (6): Effect of stock powder, virgin coconut oil and sunflower oil on kidney functions of hyperlipidemic rats

	Urea (mg/dl)	Uric acid (mg/dl)	Creatinine (mg/dl)
G_1 Control negative	20.70 ^{h}±0.14	3.10 ^{d}±0.60	0.50 ^{c}±0.01
G_2 Control positive	69.00 ^{a}±0.50	6.70 ^{a}±0.21	1.45 ^{a}±0.13
G_3 (2.5% stock powder)	64.30 ^{b}±0.81	5.60 ^{b}±0.08	1.14 ^{b}±0.05
G_4 (5% stock powder)	51.70 ^{d}±0.45	5.44 ^{b}±0.18	0.69 ^{c}±0.22
G_5 (1% virgin coconut oil)	57.60 ^{c}±0.20	4.90 ^{b}±0.30	0.81 ^{c}±0.13
G_6 (2% virgin coconut oil)	40.60 ^{f}±0.75	4.00 ^{c}±0.02	0.67 ^{c}±0.18
G_7 (1% sunflower oil)	44.50 ^{e}±0.32	5.12 ^{b}±0.46	0.77 ^{c}±0.03
G_8 (2% sunflower oil)	36.17 ^{g}±0.19	3.82 ^{c}±0.40	0.63 ^{c}±0.15
LSD	0.84	0.58	0.23

Each value represents mean±standard deviation (n = 3). Mean under the same column bearing different superscript letters are different significantly (p ≤ 0.05).

There were no significant differences between negative control, 5% stock powder, 1% virgin coconut oil, 2% virgin coconut oil, 1% sunflower oil and 2% sunflower oil group.

Conclusion

In conclusion, our results showed that feeding experimental animals with stock seeds, virgin coconut oil and sunflower oil caused a significant ($P \leq 0.05$) increase in HDL-c, RBCs and hemoglobin, but with a significant decrease ($P \leq 0.05$) in weight gain compared to the group control (+ve), kidney and liver function was enhanced with decreased ALT, AST, ALP, creatinine, uric acid, urea and blood glucose reflecting the strong therapeutic effect of feeding on of stock seed, virgin coconut oil and sunflower oil for the treatment of hyperlipidemia in rats.

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تقليل دهون الدم في الفئران المغذاة على بذور المنثور الغني بالأوميجا 3

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صممت الدراسة الحالية لدراسة تأثير مسحوق بذور المنثور وزيت جوز الهند البكر وزيت عباد الشمس على الفئران المصابة بارتفاع دهون الدم. تم استخدام أربعين فأر من ذكور الفئران البيضاء في هذه الدراسة يتراوح أوزانها (150±10) وتم تقسيمها الى 8 مجموعات 5 فئران في كل مجموعة، تركت إحداها كمجموعة ضابطة سالبة، أما المجموعات السبعة الأخرى تم تغذيتها على الوجبة الاساسية بالإضافة إلى 1.5% من الكوليسترول وذلك لمدة 21 يوم. تم إضافة مسحوق بذور المنثور بنسبة 2.5% , 5% من الوجبة الاساسية و أيضا تم إضافة كل من زيت جوز الهند البكر و زيت عباد الشمس بنسبة 1% , 2% من الوجبة الأساسية. من النتائج التي تم الحصول عليها يمكن استنتاج أن التغذية على مسحوق بذور المنثور وزيت جوز الهند البكر وزيت عباد الشمس أدت عند مستوى معنوية 5% زيادة في البروتين الدهني عالي الكثافة وكرات الدم الحمراء والهيموجلوبين وانخفاض في باقي التحاليل وذلك مقارنة بالمجموعة الضابطة الموجبة وأيضا تعزيز وظائف الكلى والكبد عن طريق خفض إنزيمي ALTو AST وخفض إنزيم ALP واليوريا والكرياتينين وحمض اليوريك وخفض مستوى سكر الدم. هذه النتيجة تعكس التأثير التغذوى العلاجي لمسحوق بذور المنثور وزيت جوز الهند البكر وزيت عباد الشمس على الفئران المصابة بارتفاع دهون الدم.

الكلمات المفتاحية: ارتفاع دهون الدم – بذور النباتات – زيوت النباتات - الفئران - التحاليل الكيميائية والحيوية.