The Potential Effects of Egg White, Soybean Milk and Crestor (*Rosuvastatin calcium*) Medication on Nonalcoholic Steatohepatitis in Rats

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Abstract

Fatty liver disease is a condition in which fat builds up in the liver. There are two foremost types of it: non-alcoholic fatty liver disease (NAFLD) and alcoholic fatty liver disease. NAFLD also known as nonalcoholic steatohepatitis (NASH). It is a metabolic disease-related fatty liver disease with insulin resistance. This study was carried out for finding the effects of Crestor (*Rosuvastatin calcium*) as a medical therapy, and egg white and soybean milk as a dietary treatment of NASH rats. Nonalcoholic steatohepatitis (NASH) was induced by feeding rats for 6 weeks on a high-fat diet. After that, NASH-rats were fed HFD with daily given orally crestor medication (10 mg/kg), egg white, soy milk and the mixture of them (2 ml/100g). The results discovered that the oral administration of soy milk, egg white and the mixture of the three treatments significantly reduces body weight, visceral fat weight, adiposity index of fat, compared to that of the treated NASH rats with crestor and positive group. On the other hand, the improvement in the serum levels of TL, TC, TG, LDL-c, and VLDL-c was more evident in NASH rats treated by the administration of Crestor or the mixture of the three treatments, compared to that treated with soy milk and egg white. While serum levels of HDL-c, TP, Alb, TBL, DBL, IDBL and MDA, and the activities of liver enzymes and antioxidant enzymes in NASH rats treated with soy milk and egg white were more improved, compared to that treated with the Crestor or the mixture of the three treatments. The results of the above biochemical parameters were confirmed by the results of the histopathological examination of the liver. In conclusion, egg white and soy milk induced reduction in body weight and visceral fat, as well as hepatoprotective and antioxidant activities in NASH rats fed on the HFD. However, the study needs more investigation to detect the action mechanism of them.

Keywords: High Fat Diet; Crestor Medication; Fatty Liver; Egg Whit; Soy milk

INTRODUCTION

Liver is a vital organ and gland in the human body, that is accountable for a collection of role that supports digestion, metabolism, detoxification, vitamin storage, immunity, predominant the levels of glucose, iron and cholesterol, blood clotting etc., It is the accountable organ on double blood supply from the portal vein (approximately 75%) and the hepatic artery (approximately 25%) (**Kalra *et al*., 2024**).

The liver always extensively susceptible to multiple external matters such as environmental poison, chemicals, pharmaceutical, and alcohol, whichever all can lead to diverse liver disturbance or damage, and in the latest, may be led to hepatic failure and/or death (**Cooke *et al*., 2010**). In addition, inherits, virus infection and various diseases (as obesity and diabetes), an attack from one's own immune system are the other factors that can permanently damage the liver and the bile duct or manger's life (**Sivakrishnan and Pharm, 2019**).

Fatty liver disease (FLD) is a status in which fat builds up in the liver. There are two foremost types of FLD: non-alcoholic fatty liver disease (NAFLD) and alcoholic liver disease (ALD) (**Tanimine *et al*., 2016**). Non-alcoholic fatty liver disease (NAFLD) is a description by excessive liver fat accumulation, combined with insulin resistance (**Chalasani *et al*., 2018**). NAFLD is also now known as metabolic dysfunction-associated fatty liver disease (MAFLD) (**Eren *et al*., 2022**). Non-alcoholic fatty liver (NAFL) also known as non-alcoholic steatohepatitis (NASH), and NASH-related cirrhosis. NASH is the inflammatory subtype of NAFLD, and it is classified by steatosis, evidence of hepatocyte injury (ballooning), and inflammation with or without fibrosis. NASH-cirrhosis is the presence of cirrhosis with current or previous histological evidence of stenosis or steatohepatitis (**Kang *et al*., 2021**). The second type of FLD is the alcoholic liver disease (AFLD) is a term used to describe fatty liver disease caused by excessive alcohol use. Steatosis starts with fat accumulation in the liver and can progress to acute alcoholic steatohepatitis (**Hamada *et al*., 2022**).

Crestor (*rosuvastatin*) is an HMG Co-A reductase inhibitor, like other statins, is commonly used to lower cholesterol levels. Furthermore, when prescribed in higher doses, it may be used as moderate- or high-intensity statin therapy for patients with atherosclerotic cardiovascular disease to reduce the risk of coronary events and stroke (**Davidson *et al*., 2004**).

Egg white or albumen, constitutes about 60% of the total egg weight, in which water and protein are the main components, followed by carbohydrates, ash and trace amounts of lipids. The ovalbumin, ovotransferrin, ovomucoid, lysozyme and ovomucin are the main egg white proteins and closely related to albumen viscosity. While, the minor egg white proteins are ovoglycoprotein,

ovoinhibitor, cystatin, avidin, ovoflavoprotein, and ovomacroglobulin (ovostatin) (**Abeyrathne *et al*., 2013)**. Egg white is a rich source of high quality proteins with various bioactive peptide fractions (**Nimalaratne *et al*., 2015**). In addition, egg white provides many essential nutrients supporting the development of new life as well as several bioactivities components for the protection of the fertilized egg against microorganisms **(Alabdeh *et al*., 2011).** Recently, Studies have indicated that egg white as a part of common diet, it diminishes liver triglyceride content in rats. It, also, reduces the absorption of dietary triglycerides and cholesterol and promotes the β-oxidation of fatty acids in the liver, bringing about a reduction the triglyceride accumulation in the liver (**Jiang *et al*., 2021**).

Soy milk is an aqueous extract of whole soya beans (Glycine max) and is used as a plant substitute for dairy milk due to its multiple health benefits and nutraceutical potentials has become a popular health beverage. Soy milk has a high content of mono-and polyunsaturated fatty acids, high-quality protein, phosphateidylcholine, B vitamins, calcium, amino acids and the natural antioxidants like isoflavones or phytoestrogens etc. (**Mazumder and Begum, 2016**). In addition, soy milk is non-caloric, cholesterol free and is preferred consumed by the consumers with lactose intolerance (**Chen *et al*., 2012 and Sethi *et al*., 2016**). The current research was performed to find out the effects of crestor (*Rosuvastatin calcium*) as a medical therapy and egg white and soybean milk as a dietary treatment of rats with steatohepatitis-induced.

2. MATERIALS AND METHODS

Materials:

Rats: Forty-five adult male rats (Sprague Dawley Strain), weighing about 200±10g, was obtained from the Laboratory Animal Colony, Helwan, Egypt.

Eggs and Soybean Milk: Eggs and soybean milk were purchased from Agricultural Research Center products outlets, Giza, Egypt.

Crestor (*Rosuvastatin calcium*) Medication: Crestor (*Rosuvastatin calcium*) medication (10 mg) of AstraZeneca Pharmaceuticals LP, Wilmington, DE, IPR Pharmaceuticals, Inc. Canovanas PR., product of Belgium was purchased from the local pharmacy, Cairo, Egypt**.**

Basal Diet Constituents: Basal diet constituents according to AIN 93-M formula were purchased from the El-Gomhorya Company for Pharmaceutical and Chemical, Cairo, Egypt. Corn starch and Dextrin were obtained from Egyptian Starch and Glucose Manufacturing Co., Mostorod, Cairo, Egypt. Soybean oil and sucrose were Agricultural Research Center products outlets, Giza, Egypt.

Chemicals and Kits: Diethyl ether, formalin and other chemicals used in this study were acquired from the El-Gomhoriya Company for Trading Drugs and Chemicals, Cairo, Egypt. Kits for biochemical assay were purchased from the Gamma Trade Co., for Pharmaceutical and Chemical, Dokki, Egypt.

Methods:

Preparation of Basal Diet: The basal diet (AIN-93M) composed of casein (>85% protein) (14%), dextrinized cornstarch (90–94% tetrasaccharides) (15.5%), soybean oil (4%), mineral mixture (3.5%), vitamin mixture (1%), fiber (5%), sucrose (10%), choline bitartrate (0.25%), L-Cystine (0.18), Tert-Butylhydroquinone (0.80%) and the remainder was corn starch up to 100%. These components were carefully mixed and formulated according to **Reeves *et al.*, (1993).**

Induction of Nonalcoholic Steatohepatitis in Rats: Nonalcoholic steatohepatitis (NASH) was caused by feeding rats for 6 weeks on a high-fat diet including 25% fats, 1% cholesterol, and 0.25% bile salts and 0.5% cholic acid as mentioned by **Zulet *et al.*, (1999)**. After 6 weeks, histopathological investigations for liver of 3 rats were accomplished to assure the incidences of NASH.

Experimental Design and Grouping of Rats: All rats were housed in the animal house at the Faculty of Home Economics, Helwan University in wire cages under adjustment environmental conditions of the temperature (22±4°C), relative humidity (45% to 50%) and light/dark cycle (12/12 hr). The food and water supplies were uninterrupted during the experimental period. Prior to the trial study, rats were kept for a week to acclimatize. Subsequently, rats were randomized into six groups, each with seven rats as follows:

Group 1: Rats were kept as a negative control group (-ve) and fed on the basal diet during the experimental period.

Group 2: Rats with untreated NASH were kept as a positive control group (+ve) and fed on the high fat-cholesterol diet (HFCD) during the experimental period.

Group 3: Rats with NASH were fed on HFCD with daily given crestor medication orally at a dose of 10 mg/kg.

Group 4: Rats with NASH were fed on HFCD and daily given egg white orally at a dose of 2ml/100g of body weight.

Group 5: Rats with NASH were fed on HFCD and daily given soybean milk orally at a dose of 2ml/100g of body weight.

Group 6: Rats with NASH were fed on HFCD and daily given the mixture of Crestor, egg white and soy milk (1:1:1) at a dose of 2ml/100g of body weight.

Determination of FI, BWG and Percent Change in BWG: Food intake (FI) was calculated every day throughout the experimental period (12 weeks). The alteration in body weight was measured by balancing the animals on a balance scale earlier the experiment (initial body weight) and at the end of the experimental period (final body weight). The biological value of the diet was evaluated by the decision of its impact on body weight gain (BWG), while the percent change in body weight gain (BWG %) was calculated using the following formula as referred by **Kratochvílova *et al*., (2002).**

BWG = Final Body Weight (FBW) - Initial Body Weight **(IBW)**

BWG %= BWG/IBW X 100

At the end of the experimental period (12 weeks), rats in all groups were fasted (except from water) for 12 hours, anaesthetized with diethyl ether. Portal vein blood samples were assembly in clean, dry centrifuge tubes and left to coagulate at room temperature. The coagulated blood was centrifuged at 3000 rpm for 15 minutes to get the serum. Then, clear serum samples were taken into the Eppendorf's tubes (1.5 mL) and preserved at –20ºC until they were utilized for biochemical assays. The liver of all rats was carefully removed from each rat following dissection, washed with normal saline for blood removal, weighing and immersed in neutral formaldehyde (10%) for histopathological examination.

Determination of Visceral Fat Weight and Adiposity index: Visceral fat weight (g) and adiposity index were determined as described by **Taylor and Phillips, (1996).**

Biochemical Assessments

Evaluation of Lipid Profile: Serum levels of total lipid (TL), total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-c), and low density lipoprotein cholesterol (LDL-c) were determined using trading reagent kits (Biomed diagnosis, Egypt) as referred by **Zollner and kirsch (1962), Vassault *et al*., (1986), Hostmark *et al*., (1991), Friadwald *et al*., (1972) and Young, (2001),** respectively. While, very low-density lipoprotein cholesterol (VLDL-C) was measured using the following formula: **VLDL-c (mg/ dL) = TG/5**

Evaluation of Liver Functions: The serum activity of Alanine transaminase (ALT), Aspartate aminotransferase (AST), Alkaline phosphatase (ALP) enzymes and gammaglutamyl (GGT) was colorimeters quantified utilizing kits (Diamond Co, Hanover, Germany) in line with the instructions of **Young (1997)** for AST and ALT assay, **Sherwin (1984)** for ALP assay and **Dufour *et al*., (2000)** for GGT assay. The biometrics were quantified using a

spectrophotometer (Hum star 200, automatic biochemistry analyzer, Germany) adjusted at 505 for ALT, AST and ALP, and 510 nm for GGT.

Serum levels of total protein (TP), albumin (Alb), total bilirubin (TBL) and direct (DBL) were quantified colorimetrically using a spectrophotometer (Hum star 200, automatic biochemistry analyzer, Germany) as mentioned by **Tietz (1994)**, **Young (2000)**, **Henry (1991)** and **Burtis and Ashwood (1999)**, respectively.

Evaluation of Malondialdehyde and Activities of Antioxidant Enzymes: The principal method for the determination of oxidative stress was depending on colorimetric by quantifying thiobarbituric acid (TBA) reactivity as malondialdehyde (MDA) in a spectrophotometer adjusted at 534 nm according to the described method by **Ohkawa *et al*., (1979)**.

The activities of antioxidant enzymes (superoxide dismutase (SOD), glutathione (GSH) and glutathione peroxides (GPx)) were determined as referred by the commercial testing kits (Cayman Practice ELISA Kits). The standard procedure to assay the activity of SOD was that the kits utilize an enzyme linked immunosorbent assay duplicate antibody principle. The color modification was calculated spectrophotometrically at 560 nm as referred by **Nishikimi *et al*., (1972)**. The serum activity of GSH and GPx was checked according to the kit's instruction manual as mentioned by **Beutler *et al*., (1963)** and **Paglia and Valentine., (1967)** using spectrophotometrically at 405 nm and 340 nm.

Histopathological Examination: Then, the submerged samples in the formaldehyde were removed, cleaned, washed, and dehydrated in ascending-grade alcohols. Afterward, specimens were cleared in Xylol, fixed and deeply in paraffin mass, sectioned to 4-6 microns in thickness, and stained with the Hematoxylins and Eosin stain for examination as described by **Carleton, (1979).** The histopathological investigation was performed at the Faculty of Veterinary Medicine, Cairo University.

Statistical Analysis: Data were assessed statistically according to the computerized SPSS package program (SPSS 22.00 software for Windows) by one-way analysis of variance (ANOVA). The gained data was stated as Mean ± SD, and the significant difference between means was estimated at $p<0.05$**.**

3. RESULTS

1. Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on FI, FBW, BWG and BWG (%) in rats with NASH: The current results in Table 1 revealed that untreated NASH rats (positive) have a significant ($P<0.05$) decrease in FI compared to that of healthy rats (negative rats). In contrast, NASH rats fed on HFCD with the administration of crestor medication

(10 mg/1000 kg b. wt.) and soy milk, egg white (2 ml/100g b. wt.) and the mixture of the three treatments have no significant changes in FI compared to that of the positive rats and have a significant decrease, compared to the negative group.

Regarding body weight, the tabulated results showed that NASH rats fed on HFCD have a significant (P<0.05) increase in FBW, BWG and BWG%, compared to that of the normal rats fed on the basal diet. Incorporated, the HFCD with the administration of crestor, soy milk, egg white or their mixture caused significantly reduced (P<0.05) in FBW, BWG and BWG%, compared to the positive control rats. The reduction in FBW, BWG and BWG% was significantly ameliorated by the administration of egg white and the mixture of the three treatments, compared to that of the treated NASH rats with crestor or soy milk.

Table (1): Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on FI, FBW, BWG and BWG (%) in rats with NASH

Parameters / Groups		Parameter as Mean ± SD				
		FI (g)	IBW (after induction of NASH) (g)	FBW (g)	BWG (g)	Chang in BWG (%)
Negative group		12.60±0.89^{a}	253.00±1.22^{b}	301.40±1.34^{f}	48.40±2.19^{d}	19.13±0.94^{c}
Positive group		10.80±0.84^{bc}	332.60±1.82^{a}	422.80±2.77^{a}	90.20±1.10^{a}	27.12±0.35^{a}
Treated NASH rats with:	**Crestor (10mg/kg)**	10.80±0.84^{bc}	333.20±1.30^{a}	403.00±2.12^{b}	69.80±2.11^{b}	20.95±1.00^{b}
	Soymilk (2ml/100)	11.60±0.55^{b}	333.60±0.89^{a}	387.40±1.82^{c}	53.80±1.92^{c}	16.13±0.60^{d}
	EggWhite (2ml/100g)	10.80±0.84^{bc}	333.20±1.30^{a}	379.60±1.14^{d}	46.40±1.14^{d}	13.93±.0.38^{e}
	Mixture(2 ml/100g)	10.40±0.55^{c}	332.40±0.55^{a}	373.20±1.30^{d}	40.80±1.30^{e}	12.27±0.39^{f}

Means with different letters in each row are significantly differs at p< 0.05; **NASH**: Nonalcoholic Steatohepatitis; **FI:** Food Intake; **IBW:** Initial body weight**; FBW:** Final body weight**; BWG:** Body weight gain**; BWG%:** Change in body weight gain%.

2. Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on LW, VFW and AI (%) in Rats with NASH: Table 2 represent the effect of crestor medication, soy milk, egg white and their mixture on liver weight (LW), visceral fat weight (VFW) and adiposity index (%) (AI) in rats with NASH. It was showed that NASH rats fed on HFCD only (positive rats) had a significant (P<0.05) increase in LV, VFW g and AI, compared to that of the rats fed on the normal basal diet (normal rats). While, feeding NASH rats on HFCD with the administration of crestor, soy milk and egg white or their mixture has a significant (P<0.05) decrease in LW (g) and VFW (g), compared to NASH rats

feeding on HFCD only. In addition, the administration with soy milk, egg white, or the mixture the three treatment caused significant reduction in AI, while the he administration of crestor caused no significant change, compared to the positive group.

Table (2): Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on LW, VFW and AI (%) in Rats with NASH

Groups / Parameters		Parameter as Mean ± SD		
		LW (g)	**VFW (g)**	**AI %**
Negative group		9.78±0.13^{d}	8.12±0.18^{f}	2.70±0.05^{c}
Positive group		11.32±0.19^{a}	14.30±0.21^{a}	3.38±0.04^{a}
Treated groups with	**Crestor (10 mg/kg)**	10.58±0.11^{b}	12.58±0.11^{b}	3.33±0.33^{a}
	Soymilk (2 ml/100g)	10.22±0.19^{c}	11.40±0.12^{c}	2.94±0.03^{b}
	Egg White (2 ml/100g)	10.56±0.09^{b}	10.38±0.13^{d}	2.72±0.05^{c}
	Mixture (2 ml/100g)	10.08±0.09^{c}	9.74±0.25^{e}	2.74±0.19^{bc}

Means with different letters in each row are significantly differs at p< 0.05; **NASH**: Nonalcoholic Steatohepatitis; **LW:** Liver Weight; **VFW:** Visceral Fat Weight**; AI%:** Adiposity Index %

3. Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on TL, TC, TG, LDL-c, HDL-c and VLDL-c in Rats with NASH: In the case of the serum lipid profile, the parameters of serum TL, TC, TG, LDL-c, HDL-c, and VLDL-c levels were used to check the effect of crestor medication, soy milk, egg white or their mixture on NASH rats. The results in Table 3 revealed that NASH rats fed on HFCD have a significant (P<0.05) increase in the serum concentrations of TL, TC, TG, LDL-c, HDL-c, and VLDL-c, and a decrease in HDL-c levels, compared to that of the normal rats fed on a normal basal diet. In contrast, the administration of crestor, soy milk and egg white or their mixture caused a significant amendment in the serum levels of the above parameters, as compared to that of NASH rats fed on the HFCD alone (positive rats).

The rate of improvement in the serum levels of TL, TC, TG, LDL-c, and VLDL-c was more evident with the administration of crestor or the mixture of the three treatments (soy milk and egg white), while serum HDL-c, levels were more improved treated NASH rats with soy milk and egg white.

Table (3): Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on TL, TC, TG, LDL-c, HDL-c and VLDL-c in Rats with NASH

Parameters / Groups		TL (mg/dl)	TC (mg/dl)	TG (mg/dl)	LDL-C (mg/dl)	HDL-C (mg/dl)	VLDL-C (mg/dl)
Negative group		388.29±1.49^{e}	104.75±1.75^{f}	94.07±0.73^{e}	200.86±1.57^{f}	38.53±0.93^{a}	18.81±0.15^{e}
Positive group		644.28±1.38^{a}	187.28±1.79^{a}	163.79±1.82^{a}	456.57±2.29^{a}	43.27±0.57^{d}	32.76±0.36^{a}
Treated groups with	**Crestor (10 mg/kg)**	414.29±1.98^{d}	119.04±1.76^{e}	95.5±1.36^{d}	283.00±1.83^{e}	36.40±0.79^{c}	19.10±0.27^{d}
	Soymilk (2 ml/100g)	416.86±1.36^{c}	146.11±1.53^{c}	121.82±1.35^{c}	300.14±1.68^{d}	38.33±0.12^{a}	24.36±0.27^{c}
	Egg White (2 ml/100g)	520.43±1.18^{b}	184.39±1.22^{b}	141.29±0.82^{b}	318.71±3.19^{b}	37.67±2.08^{a}	28.26±0.15^{b}
	Mixture (2 ml/100g)	416.82±1.68^{c}	125.93±1.37^{d}	96.28±1.22^{d}	306.43±3.26^{c}	36.6±1.97^{b}	19.26±0.24^{d}

Means with different letters in each row are significantly differs at p< 0.05; **NASH:** Nonalcoholic Steatohepatitis; **TL**: Total Lipid; **TG:** Triglycerides; **TC:** Total Cholesterol; **LDL**: Low Density Lipoprotein; **HDL**: High Density Lipoprotein; **VLDL-C:** Very Low-Density Lipoprotein Cholesterol**.**

4. Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on AST, ALT, ALP and GGT in Rats with NASH: The tabulated results in Table (4) outlined that untreated rats with NASH have a significant increase in the serum activity of AST, ALT, ALP and GGT enzymes, compared to the normal rats. Whilst the treated NASH rats by the oral administration of crestor medication, soy milk, egg white and their mixture caused significant (P<0.05) reductions in the serum activity of AST, ALT, ALP and GGT enzymes, compared to untreated NASH rats. The significant improvement in the activities of liver enzymes (AST, ALT, ALP and GGT) were reported in NASH rats treated with soy milk and egg white, compared to that treated with the crestor or the mixture of the three treatments.

Table (4): Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on AST, ALT, ALP and GGT in Rats with NASH.

Parameters / Groups		ALT (u/L)	AST (u/L)	ALP (u/L)	GGT (u/L)
Negative group		17.00±1.67^{e}	20.00±2.38^{e}	298.32±1.40^{f}	36.50±2.02^{c}
Positive group		88.32±2.44^{a}	116.07±0.84^{a}	615.61±2.29^{a}	45.25±2.98^{a}
Treated groups with	**Crestor(10mg/kg)**	51.67±1.26^{b}	63.56±0.97^{b}	525.04±2.33^{b}	41.75±2.67^{b}
	Soymilk(2ml/100g)	28.50±1.32^{d}	39.50±1.32^{d}	402.68±1.97^{e}	36.25±1.01^{c}
	EggWhite(2ml/10g)	28.25±2.32^{d}	39.50±2.02^{d}	409.70±1.60^{d}	34.75±2.38^{c}
	Mixture(2 ml/100g)	46.50±2.67^{c}	51.75±2.39^{c}	484.86±2.34^{c}	40.00±1.32^{b}

Means with different letters in each row are significantly differs at p< 0.05; **NASH:** Nonalcoholic Steatohepatitis; **AST:** Aspartate Aminotransferase; **ALT**: Alanine Aminotransferase; **ALP**: Alkaline Phosphatase; **GGT**: γ-glutamyl trans peptidase.

5. Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on TP, Alb, TBL, DBL and IDBL in Rats with NASH: As recorded in Table (5), the results indicated that the serum concentration of TP, Alb was reduced significantly (P<0.05), while serum levels of TBL, DBL and IDBL increased significantly in NASH rats fed on the HFCD, compared to the normal rats. In addition, NASH rats fed on the HFCD with incorporated of oral administration of Crestor medication or the mixture of crestor, soy milk, egg white have no significant change in serum level of TP, while the oral administration of soy milk, egg white caused a significant increase (P<0.05), compared to the positive rats. On the other hand, oral administration of crestor medication, soy milk, egg white and their mixture significantly (P<0.05) ameliorates serum levels of Alb, TBL, DBL and IDBL (except crestor with DBL), compared with the untreated NASH rats fed on the HFCD only (positive group). The superior result in the serum concentration of TP, Alb, TBL, DBL and IDBL was shown in the treated group by soy milk or egg white, compared to that treated with crestor medication or the mixture of the three treatments.

Table (5): Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on TP, Alb, TBL, DBL and IDBL in Rats with NASH.

Groups	Parameters	TP (gm/dl)	Alb (gm/dl)	TBL (gm/dl)	DBL (gm/dl)	IDBL (gm/dl)
Negative group		7.12±0.10^{a}	4.22±0.21^{a}	0.69±0.01^{e}	0.18±0.01^{d}	0.51±0.01^{d}
Positive group		6.59±0.17^{b}	3.80±0.13^{c}	1.36±0.04^{a}	0.36±0.01^{a}	1.00±0.05^{a}
Treated groups with	**Crestor (10 mg/kg)**	6.75±0.08^{b}	3.99±0.10^{b}	1.14±0.05^{b}	0.36±0.01^{a}	0.78±0.06^{b}
	Soymilk (2ml/100g)	7.27±0.25^{a}	4.13±0.14^{ab}	0.78±0.01^{d}	0.31±0.01^{c}	0.48±0.02^{d}
	Egg Whit (2ml/100g)	7.12±0.17^{a}	4.23±0.04^{a}	0.80±0.01^{d}	0.30±0.01^{c}	0.50±0.02^{d}
	Mixture (2ml/100g)	6.60±0.13^{b}	4.00±0.07^{b}	0.94±0.05^{c}	0.33±0.01^{b}	0.61±0.05^{c}

Means with different letters in each row are significantly differs at p< 0.05; **NASH:** Nonalcoholic Steatohepatitis**; TP:** Total Proteins; **ALb**: Albumen; **TBL**: Total Bilirubin; **DBL:** Direct Bilirubin; **IDBL:** Indirect Bilirubin**.**

6. Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on MDA, SOD, GSH and GPx in Rats with NASH: Table 6 represents the results of lipid peroxidation as indicated by serum MDA level and activity of SOD, GSH and GPx in normal rats, untreated NASH-rats and treated NASH-rats with the oral administration of crestor medication, soy milk, egg white and their mixture. In comparison to normal rats, untreated NASH rats (positive rats) have a significant increase (P<0.05) in serum levels of MDA and decrease in serum activities of SOD, GSH and GPx. However, oral administration of Crestor medication, soymilk, egg white and their mixture encourages a significant (P<0.05) decrease in serum MDA level and increase in the activity of SOD, GSH and GPx enzymes compared to the positive control group. The good result in serum concentration of MDA and activity of antioxidant enzymes

was shown in the NASH group that treated by soy milk and egg white as compared to the other treated group with crestor or the mixture of the three treatments.

Table 6: Effect of Crestor Medication, Soy Milk, Egg White and Their Mixture on MDA, SOD, GSH and GPx in Rats with NASH.

Groups \ Parameters		MDA (u/ml)	SOD (u/ml)	GSH (u/ml)	GPx (u/ml)
Negative group		65.32±1.25^{f}	990.11±1.57^{a}	2.90±1.70^{b}	59.50±2.98^{a}
Positive group		165.46±1.52^{a}	514.78±1.15^{f}	1.85±1.27^{d}	28.00±1.66^{f}
Treated groups with	**Crestor (10 mg/kg)**	115.78±1.12^{b}	684.89±1.67^{e}	2.10±1.11^{c}	30.75±1.75^{e}
	Soymilk (2 ml/100g)	99.00±1.27^{d}	945.57±1.13^{b}	3.08±2.13^{a}	48.00±2.39^{b}
	Egg White(2 ml/100g)	87.50±1.19^{e}	906.00±1.54^{c}	2.82±1.03^{b}	43.50±2.75^{c}
	Mixture (2 ml/100g)	113.75±1.46^{c}	735.00±1.15^{d}	2.21±1.14^{c}	35.75±1.98^{d}

Means with different letters in each row are significantly differs at $p< 0.05$; **NASH:** Nonalcoholic Steatohepatitis**; MDA:** Malondialdehyde; **SOD**: Superoxide Dismutase; **GSH**: Reduced Glutathione; **GPX**: Glutathione Peroxidase.

Histopathological Examination of Liver: Microscopic examination of liver sections from the normal group revealed normal structure of hepatic parenchyma as shown in Photo 1. In contrast, as shown in Photo 2, the liver sections of NASH rats from group 2 (positive rats) have marked steatosis in several examined sections, portal inflammatory cell infiltration with variable inflammatory cell infiltration in the hepatic sinusoids, as well as marked degeneration of the hepatic parenchyma (Photo 3). Liver sections from NASH rats from group 3 treated with crestor showed vacuolation and steatosis in several examined sections (Photo 4). In addition, few sections showed multifocal inflammatory foci. (Photo 5). As shown in Photo 6, the highest protective effect was noticed in the liver sections of NASH rats (group 4) which treated with soy milk. Liver sections of NASH rats from group 5 treated with egg white revealed mild improvement with mild vacuolation of hepatic parenchyma (Photo 7). Meanwhile, the liver sections of NASH rats from group 6 treated with the mixture of the three treatments exhibited mild vacuolation of the hepatic tissue (Photo 8). Periportal hepatic steatosis was also observed and characterized by the presence of empty fat globules pushing the nucleus to the periphery with the characteristic signet ring appearance (Photo 9).

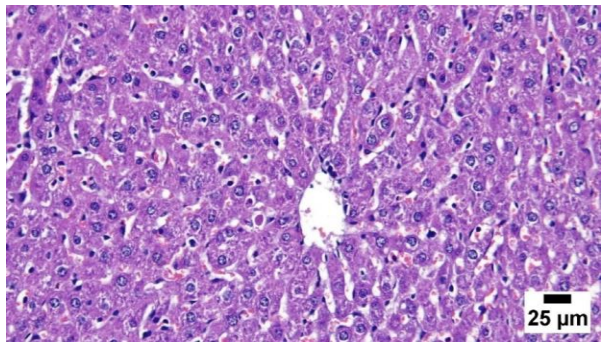

Photo 1: Photomicrograph of liver sections of negative rats (group 1) showing the normal histological architecture of hepatic lobule (H & E).

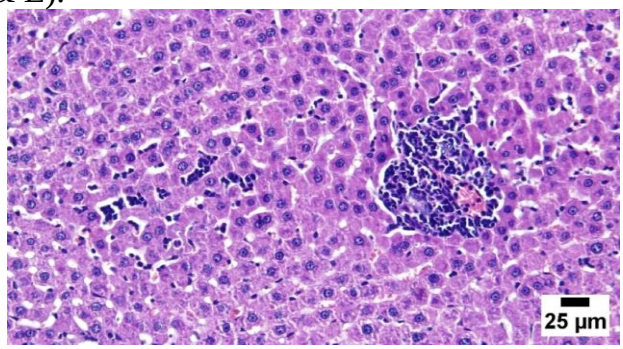

Photo 2: Photomicrograph of liver sections of positive rats (group 2) showing steatosis in several examined sections portal inflammatory cells infiltration with variable inflammatory cells infiltration in the hepatic sinusoids (H&E).

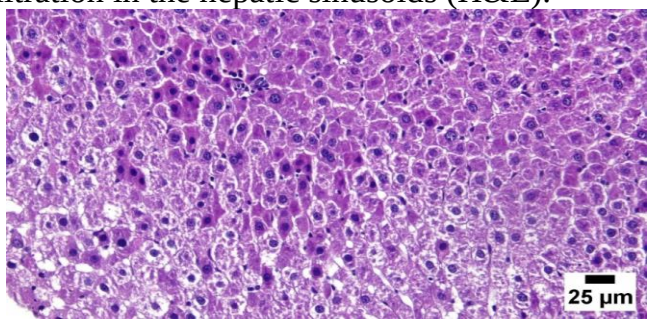

Photo 3: Photomicrograph of liver sections of positive rats (group 2) showing degeneration of the hepatic parenchyma (H&E).

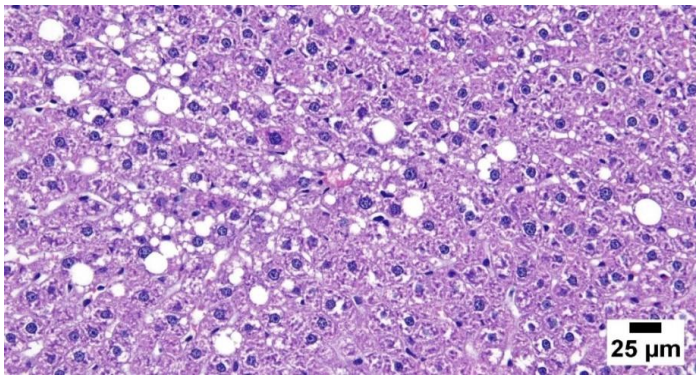

Photo 4: Photomicrograph of liver sections of NASH rats (group 3) treated with crestor showing vacuolation and steatosis of the hepatic tissue (H&E).

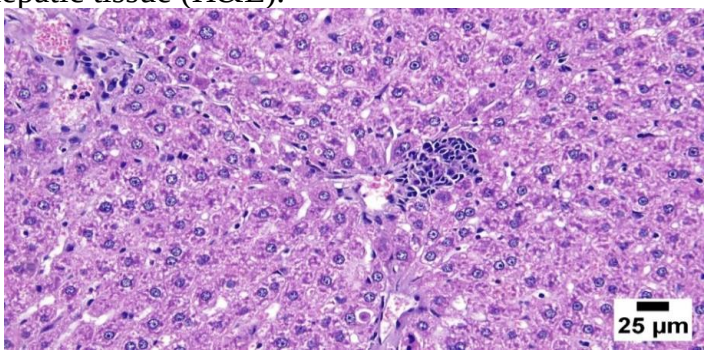

Photo 5: Photomicrograph of liver sections of NASH rats (group 3) treated with crestor showing focal inflammatory cells aggregation (H&E).

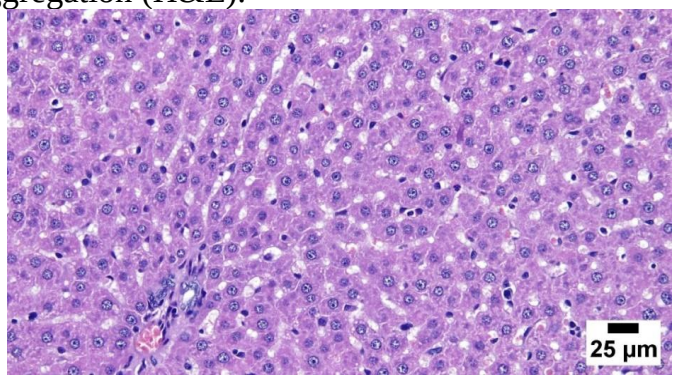

Photo 6: Photomicrograph of liver sections of NASH rats (group 4) treated with soy milk showing apparently normal hepatic parenchyma (H&E).

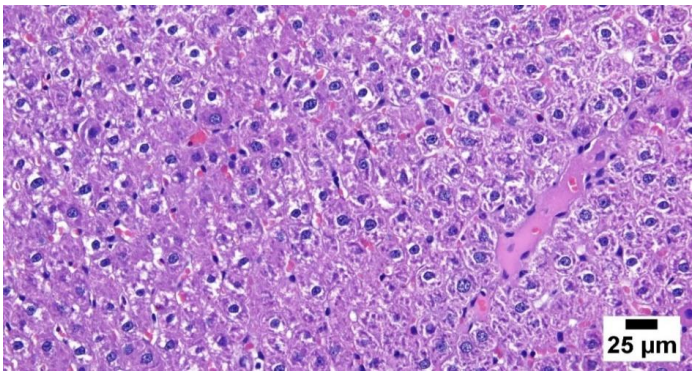

Photo 7: Photomicrograph of liver sections of NASH rats (group 5) treated with egg white showing mild vacuolation of hepatic parenchyma (H&E).

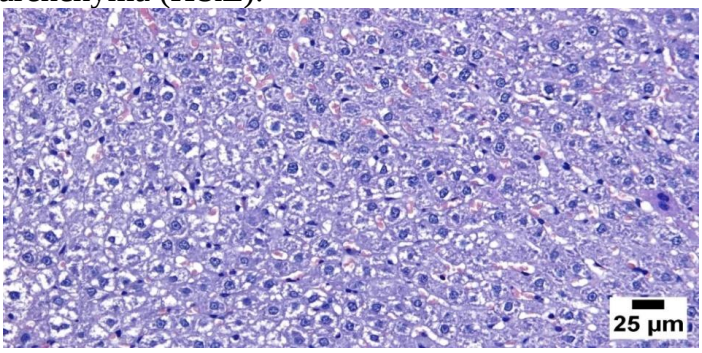

Photo 8: Photomicrograph of liver sections of NASH rats (group 6) treated with the mixture of the three treatments showing mild vacuolation of the hepatic parenchyma (H&E).

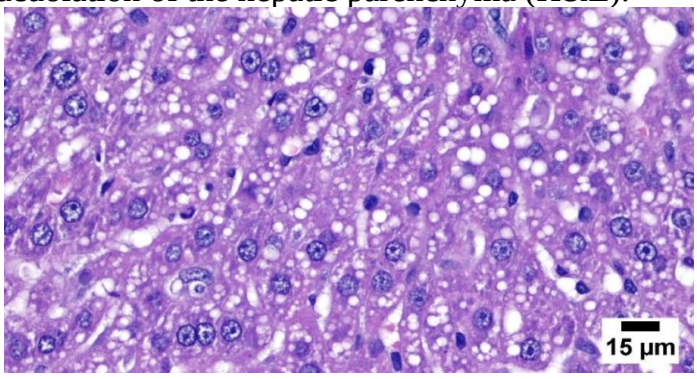

Photo 9: Photomicrograph of liver sections of NASH rats (group 9) treated with the mixture of the three treatments showing empty fat globules pushing the nucleus to the periphery with the characteristic signet ring appearance (H&E).

4. DISCUSSION

Non-alcoholic fatty liver disease (NAFLD) known as metabolic disorder is a condition in which fat builds up in the liver combined with insulin resistance (**Eren *et al*., 2022**). The multiplicity of the disease variety from simple liver fat accumulation which known as hepatic steatosisto and more severe sequence as nonalcoholic steatohepatitis, cirrhosis and hepatocellular carcinoma (**Younossi *et al*., 2016**). The existence of the disease is joined with obesity and is present in up to 91% of acutely obese patients (**Machado *et al*., 2006**). High-fat diet intake give authorization to animals to develop obesity, hyperglycemia, hyperinsulinemia, hypertension and liver injury which is similar to the phenotype observed in humans with NAFLD (**White *et al*., 2013**). Therefore, the intake of a high-energy density, high-fat diet (HFD) is considered to be one of the main factors for the initiation of NAFLD in rats. In the existing study, the impact of crestor (*Rosuvastatin calcium*) as a medicinal, and egg white and soybean milk as a dietary treatment contrary to high fat cholesterol diet (HFCD) -induced nonalcoholic steatohepatitis in rats was examined. That effect has been audited by exploring their effect on some biological and biochemical parameters such as FI, changes in body weight, visceral fat weight (VFW) and adiposity index (AI), lipid profiles, liver functions, lipid peroxidation levels and the activities of antioxidant enzymes as well as histopathological examination of liver tissues.

The obtained data revealed that untreated steatohepatitis-rats fed on HFCD alone had significant increases ($P<0.05$) in FBW, BW, liver weight (LW), VFW and AI, and decrease in FI compared to that of the normal rats fed on a basal diet alone. As shown from the obtained results shows that the increases in BW are independent of the amount of food consumed by the rats. These outcomes were in arrangement with **Rezq, (2017)** and **Ogungbemi *et al*., (2017)** who found a significant increment in BW and no significant change in FI of positive rats fed on HFCD, comparable to negative rats fed on the basal diet. Further, **Abdulrahman *et al*., (2020)** informed that

feeding rats on HFD caused a significant increase in BW, compared to that of feeding rats on the basal diet. As well **Deng *et al*., (2019)** showed a significant increase in the liver weight of untreated steatohepatitis-rats fed on HFCD had, compared to normal rats.

As suggested by the obtained results of increasing in VFW and AI, it confirms that obesity is characterized by augmented adipose tissue weight that consequence from both increasing fat cell number and fat cell size (**Lafontan and Langin, 2009**). It known that abiogenesis is a component of the adipocyte differentiation action from pre-adipocyte precursors into mature fat cell with the development and increase of subcellular lipid bead (**Ali *et al*., 2013**). This process is associated with the development of obesity. As well, excess energy uptake and lower energy expenditure derive in abnormal disproportionate growth of white adipose tissue (WAT), which can lead to the progress of obesity in rats (**Jo *et al*., 2009**). These results agreed with the previous results of **Ibrahim *et al*., (2022)** who reported that feeding rats on HFC caused a significant increase in VFW and AI, compared to normal rats.

As well the current results showed significant increase in serum TL, TC, TG, LDL-c, VLDL-c, TBL, DBL, IDBL and MDA levels, and activities of AST, ALT, ALP and GGT enzymes, and decrease in serum TP, Alb and HDL-c levels, and antioxidant enzymes (SOD, GSH and GPx) in untreated NASH-rats, compared to that of the normal rats fed on a basal diet alone. Furthermore, histopathological investigation of the liver sections of NASH-rats (positive rats) possess a steatosis in multiple examined sections, and pronounced portal inflammatory cell infiltration with variable inflammatory cell infiltration in the hepatic sinusoids, as well as deterioration of the hepatic parenchyma. This outcome were in alignment with **Kusunoki *et al*., (2000)** who proved the incidence of dyslipidemia in rodents fed on a high-fat diet. These consequence confirmed by the results of **Sumiyoshi *et al*., (2006)** who reported that HFD admission bring to the induction of hyperlipidemia, hypertension, glucose intolerance and atherosclerosis. Furthermore, **Rezq and El-Khamisy, (2011)** exhibit that nourishing rats on the HFD results in

dyslipidemia characterized by the increasing in serum TL, TG, TC, VLDL, and LDL-c and decreasing HDL-c levels. As well, **Rezq *et al*., (2017)** and **Ibrahim *et al*., 2022)** exhibit that feeding rats on the HFD results in significant increase in serum concentrations of TG, TL, TC and LDL-c and decrease HDL-c levels.

Non-alcoholic fatty liver disease is a major generated of hepatic dysfunction. It is combined with metabolic conditions and complications, such as obesity, diabetes, cardiovascular disease and other diseases. (**Shetty and Syn 2019**). As well, NAFLD are generated from free fatty acids of the blood, de novo lipogenesis, and dietary fat intake. The metabolic processes resulting from HFD can cause oxidative stress in mitochondria and the endoplasmic reticulum, as well as induce de novo lipogenesis and inflammation This action expedite the progress of NAFLD (**Yang *et al*., 2019**). Also, **Deng *et al*., (2019)** demonstrated that histopathological results from liver sections of rats fed an HFD for eight weeks developed NAFLD, described as hepatic steatosis. The lipotoxicity in liver and undue fat accumulation caused malfunctions in several metabolic pathways, proven insulin resistance related to raised circulating levels of lipids and the metabolic change in fatty acid employment and intracellular cue. The protein kinase C and the JNK-1 pathways were maybe implicated as mechanisms for lipotoxicity-induced insulin resistance in nonadipose tissue organs, such as liver and muscle (**Yazıcı and Sezer, 2017**).

Considering that the high effectiveness of liver enzymes in the blood is the optimal measure of liver dysfunction, their elevated levels in the blood can be utilized to predict inflammatory alteration in the liver (**Singh and Sharma, 2011**). Generally liver function examination such as ALT, AST, ALP, GGT, TP, Alb, TBL, DBL, IDBL and other markers can help find out the hepatic injury, and the elevation pattern can help classify injury detection (**Ribeiro *et al*., 2019**). As mentioned by **LaBrecque et *al*., (2014)** AST, ALT, GGT and other markers of liver injury are usefully surrogate quantify of NAFLD. The metabolic operation consequent from HFD can generate oxidative stress in mitochondria and the endoplasmic

reticulum, as well as encourage de novo lipogenesis and inflammation in liver cells (**Yang *et al*., 2019**). The obtained results concurred with **Cho et al., (2014)** who comparison of biochemical markers in fatty liver patients revealed that glucose, TP, AST, ALT and TG were higher. Also, **Sanyal et al., (2015)** indicated that NAFLD was significantly associated with higher ALT and GGT levels in defective glucose tolerance and T2DM patients. As well, **Al Shammari, (2020)** founded that HFD caused significant increase in serum ALT, AST and ALP enzymes as compared to negative control group. Recently, **(Huang *et al*., (2022)** reported the high fat diet significantly elevated the levels of TG, TC, LDL-c, AST, and ALT and lowered HDL-c in male mice (**Huang *et al*., 2022**).

Obesity in mice or rats of feeding on HFD has been shown to be one of the disorders that decline antioxidant capacity (**Asayama *et al*., 2001**) and antioxidant defense by lowering the activities of antioxidant enzymes (CAT, GPx and GSH) (**Carmiel-Haggai *et al*., 2005**). The current research gives an optimum relation between serum lipid peroxidation products as pointed out by MDA and the activity of antioxidant enzymes, which perform a vital role in the antioxidant system in NASH-rats. It exhibits that NASH-rats fed on HFCD possess a significant increment of serum MDA level, and lower serum activities of SOD, GSH and GPx enzymes, compared to normal rats fed on the basal diet. A possible mechanism for the formation of free radicals may be the initiation of b-adrenergic receptors informed for obesity-prone rats. This might raise lipolysis to produce free fatty acids which are capable to decouple the mitochondrial phosphorylation and more create free radicals (**Turrens, 1997**). The decline in serum antioxidant enzymes, as found in untreated NASH-rats, can lead to the excessive presence of superoxide and peroxyl radicals, which in turn create hydroxyl radicals, consequent in the initiation and reproduction of further lipid peroxidation products. As well HFD consumption releases free fatty acids by the action of lipoprotein lipase to increase serum TG and causes lipotoxicity as lipids and their metabolites develop oxidative stress (**Zhang *et al*., 2007**).

The existing results agreed with **Amirkhizi *et al*., (2007)** who showed increases in the production of reactive oxygen species with reduced antioxidant defense mechanisms in humans and animal obesity. Dyslipidemia in obese rats fed HFD participates in the modification of oxidant-antioxidant equilibrium, implying increment the bioavailability of free fatty acids and lipid peroxidation (**Amirkhizi *et al.*, 2007**). Therfore, dyslipidemia has been regarded as served factors to induce oxidative stress in obesity (**Leopold and Loscalzo, 2008**). **Denisenko and Novgorodtseva (2013)** demonstrated that fed animals on HFD inhibits the activity of blood antioxidant enzymes and elevate MDA. Further, **Jiang *et al*., (2016)** revealed that after eight weeks of HFD feeding, ALT, AST, TG, TC, LDL-C, FFA, MDA in liver tissue were evaluated liver tissues of NAFD-rats, while SOD and GSH were lower compared to normal rats. Likewise, **Rezq, (2017)** and **Ibrahim *et al.*, (2022)** informed that fed rats for four weeks on HFD markedly decreased activities of GSH, GPx, SOD and CAT enzymes, and increased serum MDA level, compared to normal rats fed on the basal diet. **Deng *et al.*, (2019)** revealed that untreated steatohepatitis-rats fed on HFCD had a significant decrease in the levels of SOD, GSH and increase in MDA in the liver.

With regard to the fulfillment of crestor (*Rosuvastatin calcium*) as a medical therapy as well as soybean milk and egg white as a dietary treatment on rats with steatohepatitis-induced. The attained results demonstrated that the incorporated of HFCD with the oral administration of crestor, soy milk, egg white and their combination resulting in significantly ameliorates ($P<0.05$) in BWG, LW (g) and VFW (g), AI, and serum levels of TL, TC, TG, LDL-c and VLDL-c, HDL-c, Alb, TBL, DBL, IDBL (except crestor with DBL), MDA and activities of liver and antioxidant enzymes. The most ameliorates in the BW, LW, VF, AI, liver functions and antioxidant enzymes were exhibited in the treatment steatohepatitis-rats with soy milk and egg white. While, the rate enhancement in the serum levels of TL, TC, TG, LDL-c, and VLDL-c was more evident with the administration of crestor or the mixture of the three treatments

(crestor, soy milk and egg white). The results of the biological and biochemical examination agreed-upon with the result of the histological investigation of liver sections.

Rosuvastatin is a lipid-lowering agent that by competitive examination inhibits 3-hydroxy-3-methylglutaryl coenzyme A reductase. It exhibits the highest effectiveness in the lowering of TG, TC, and LDL, compared with other agents at the similar doses (**Vijan and Hayward, 2004**). It has been informed that rosuvastatin ameliorates hepatic insulin resistance in rodents and humans (**Fraulob *et al*., 2012**). Additionally, Rosuvastatin diminishing the risk of cardiovascular disease, vascular reactive oxygen species (ROS) generation independently of cholesterol reduction (**Puccetti *et al*., 2011**) and employ several pleiotropic effects and accomplish significant enhancement in endothelial role consequence (**Parson *et al*., 2010**). The attained results arranged with the obtained results by **Seif el-Din *et al*., (2015**) who mentioned that the treatment of NAFLD-HFD by rosuvastatin caused a significant improvement in body weight normal but not significant effect with NAFLD-HFD control group. As well, administration of rosuvastatin nearly significantly improved serum levels of ALT, AST, ALP, GGT and lipid profile, compared with the positive group. In the interim, hepatic MDA level, and activity of SOD and GSH were enhanced. Furthermore, histologically, hepatic steatosis was diminished, and inflammation was markedly ameliorated with lowering of TNF-α and TGF-βWhile, **Valero-Muñoz *et al*., (2014)** stated that rosuvastatin did not modify body weight or the weight of the adipose packages in HFD rat.

Soybean-derived products such as soy milk are recognized as the functional foods since they are a rich source of isoflavones including genistein, daidzein, and glycitein as well as bioactive peptides, unsaturated fatty acids, and fiber (**Sato *et al*., 2017**). Administration of genistein decreased lipid accumulation in the livers and ameliorated fatty liver, enhanced insulin sensitivity, lipid profiles, liver injury, histological abnormalities and activated the antioxidant activity, reduced the pre-inflammatory cytokines, IL-6

and TNF-α, and avoided oxidative damage in the high-fructose induced insulin-resistant rats (**Salih *et al*., 2009**). Additionally, **Xiao and Hendry, (2022)** demonstrated that both soy protein and isoflavones was effective in lowering liver and blood lipids, improving glucose tolerance and insulin sensitivity and reducing liver steatosis. Soy genistein and daidzein could inhibit oleic acid-induced intracellular lipid accumulation in human HepG2 liver cell lines (**Huang *et al*., 2016**). The study of **Gudbrandsen *et al*., (2009)** proved that obese rats with nonalcoholic fatty liver and treated with soy proteins showed improved liver inflammation biomarkers such as TNF-alpha, AST, ALT and IL-1. As well incorporation of soy protein or soy isoflavones in the diet enhanced hepatic and blood lipid profiles by reducing TG, TC and LDL levels and raising the ratio of HDL /LDL cholesterol ratio in both human (**Moradi *et al*., 2020**) and animal studies (**Ascencio, *et al*., 2004**). In addition increasing evidence exhibits that soy intake had advantageous effects in patients with NAFLD (**Eslami *et al*., 2019**) and reduced the formation and accumulation of hepatic lipid globule and enhanced liver steatosis in experimental animal of NAFLD (**Hakkak *et al*., 2018**). As mentioned by **Yang et al., (2011**) feeding soy protein-containing diet diminished hepatic lipid depots of TG and TC, reduced plasma MDA and body fat accumulation in rats with high-fat induced NASH. As well, other study commented that dietary soy protein decreased high-fat caused steatosis in the liver of rats (**Badger *et al*., 2008**), and diminished hepatic steatosis and diacylglycerols (**Panasevich *et al*., 2017**). In addition **Li et al., (2023**) reported that liver function indices (AST and ALT), fatty liver indicators (TG and TC), that oxidative stress indices (MDA) and antioxidant enzyme (SOD) were significantly improved in the NAFLD-group treated with the soybean, compared to the untreated NAFLD-group.

Egg white is a part of common diet and essentially composed of high-quality protein contains various essential amino acids (**Sato *et al*., 1992**). In comparative to milk and soybean proteins, egg white protein has higher net protein utilization and significantly

augmented the muscle mass, diminished the total and subcutaneous fat in rats, reducing the overall obesity (**Niibo, 2019**). It was found that egg white protein diminished the absorption of dietary TG and cholesterol in rats (**Xu *et al*., 2013**) and stimulate the β-oxidation of fatty acids in the liver, bringing approximately a lowering in the accumulation of TG in the liver (**Shirouchi *et al*., 2019**). Additionally, **Garcés-Rimón *et al*., (2016)** informed that the consumption of egg white hydrolyzed with pepsin significantly lowered the hepatic steatosis, plasma concentration of free fatty acids and oxidative stress in the obese rats. The physiological effects of proteins are related to their digestion and absorption (**Pan *et al*., 2007**). These results indicate that eating egg white protein hydrolyzed by neutral protease has a better effect on the improvement of fatty liver than eating the untreated protein (**Xu *et al*., 2013**).

5. CONCLUSION

Finally, the current study concluded that egg white and soy milk induced reduction in body weight and visceral fat, as well as hepatoprotective and antioxidant activities in NASH rats fed on the HFD. However, the study needs more investigation to detect the action mechanism of them. As well as, further more study on the action mechanism of soy milk and egg white on NASH-patients.

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تأثير بياض البيض وحليب فول الصويا وأدوية الكريستور (روسوفاستاتين كالسيوم) على تحسين التهاب الكبد الدهني غير الكحولي في الفئران

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الملخص العربي

الكبد الدهني هو حالة مرضية تتميز بتراكم الدهون في خلايا الكبد. كما يوجد نوعان رئيسيان هما مرض الكبد الدهني الغير كحولي ومرض الكبد الدهني الكحولي. ويعرف ايضا مرض الكبد الدهني باسم التهاب الكبد الدهني غير الكحولي. يرتبط مرض الكبد الدهني بالعديد من أمراض التمثيل الغذائي و مقاومة الأنسولين. أجريت هذه الدراسة لمعرفة تأثير دواء كريستور (روسوفاستاتين كالسيوم) كعلاج طبي، وبياض البيض وحليب فول الصويا كعلاج غذائي للفئران المصابة بالتهاب الكبد الدهني غير الكحولي. تم إحداث التهاب الكبد الدهني غير الكحولي بتغذية الفئران لمدة ٦ اسابيع علي نظام غذائي غني بالدهون. بعد ذلك، تم إعطاء الفئران المصابة بالتهاب الكبد الدهني الغير كحولي الكريستور عن طريق الفم يوميا (١٠مجم/ كجم من وزن الفئران) وبياض البيض وحليب الصويا وخليط المعاملات الثلاثة (٢مل / ١٠٠جم من وزن الفئران). اظهرت النتائج ان تناول حليب الصويا وبياض البيض وخليط العلاجات الثلاثة عن طريق الفم يقلل بشكل كبير من وزن الجسم ووزن الدهون ومؤشر السمنه للدهون كمقارنة بالفئران المصابة بالتهاب الكبد الدهني الغير الكحولي المعالجة بالكريستور والمجموعة الايجابية. من ناحية اخري، كان التحسن في مستويات الكوليسترول والدهون الكلية والجلسريدات الثلاثية ومستوي الكوليسترول المنخفض الكثافة والمنخفض الكثافة جدا اكثر وضوحا في الفئران المصابة بالتهاب الكبد الدهني الغير كحولي التي عولجت بالكريستور او خليط العلاجات الثلاثة، مقارنة بتلك المعالجه مع حليب الصويا وبياض البيض. في حين ان تركيزات السيرم من البروتين الكلي والكوليسترول المرتفع الكثافة والبيليروبين الكلي والمباشر والغير مباشر والالبومين والمالونديالدهيد وانشطة انزيمات الكبد والانزيمات المضادة للاكسدة في الفئران المصابة بالتهاب الكبد الدهني الغير كحولي و المعالجة بحليب الصويا وبياض اليض كانت اكثر تحسنا مقارنة بتلك المعالجة بالكريستور او خليط من العلاجات الثلاثة. تم تاكيد نتائج المؤشرات البيوكيميائية المذكورة اعلاه من خلال نتائج الفحص النسيجي المرضي للكبد. في الختام، ادي بياض البيض وحليب الصويا الي انخفاض في وزن الجسم والدهون فضلا عن انشطة حماية الكبد وتحسن انشطة الانزيمات المضادة للاكسدة في الفئران المصابة بالتهاب الكبد الدهني الغير كحولي التي تتغذي علي نظام غذائي عالي الدهون .

الكلمات المفتاحية : نظام غذائي عالي الدهون – ادوية كريستور - الكبد الدهني - بياض البيض – حليب الصويا