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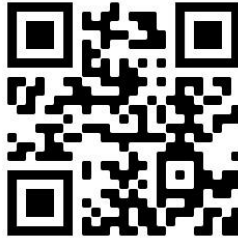
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# Effect of Soybean Flour, Coconut Oil and Magnesium on Rats Suffering from Non-Alcoholic Fatty Liver

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**Abstract:** The current research was conducted to study the effect of low-fat soybean flour, virgin coconut oil, magnesium, and all of them on rats suffering from non-alcoholic fatty liver disease (NAFLD). Sixty adult male albino rats Sprague Dawley Strain were included in this study. The rats were categorized into two main groups. The first main group (12 rats) was divided into two subgroups: "subgroup one" (6 rats) which were on basal diet BD, while " subgroup two" (6 rats) fed on a basal diet containing low-fat soybean flour which provided the diet with 14% protein, these groups used as a control negative groups (-ve). The second main group (48 rats) was on a high-fat diet (HFD) for 8 weeks to induce non-alcoholic fatty liver disease. Non-alcoholic fatty liver disease rats were randomly assigned to eight equal subgroups. The best results were recorded for the NAFLD groups which were treated with HFD containing low-fat soybean flour as the source of protein with replacing 20% coconut oil instead of 20% sheep tallow and supplemented with a 1000 mg magnesium/ kg diet. In conclusion, based on the findings of the current study, low-fat soybean flour, virgin coconut oil and magnesium are beneficial in fatty liver treatment.

**Keywords :** liver enzymes - lipid profile - kidney function - non-alcoholic fatty liver - superoxide dismutase.

## Introduction

A fatty liver disease which is a metabolic disorder, is commonly correlated with severe obesity as well as elevated levels of lipid in the blood (**Day, 2011 and Ganz et al., 2014**). Non-alcoholic fatty liver disease (NAFLD) is a prevalent type of chronic liver disease in the world associated with obesity, insulin resistance, and metabolic syndrome (**Lazo and Clark, 2008**). Some studies have revealed that fatty liver disease is induced by a high-fat diet (**Ganz et al., 2014 and Jacobs et al., 2002**). In this respect, **Fabbrini et al., (2010)** demonstrated that, extreme accumulation of triglycerides in the liver > 5% of the liver size or weight is the major distinguishing feature of NAFLD.

An investigation of **Jenkis and Kendall, (2003)** reported that one of the health benefits of soybean is a decrease in renal filtration, proteinuria, and renal acid load, and thus a reduced risk of kidney disease in type 2 diabetes when animal protein is replaced with soybean. On the other hand, (**McGraw et al., 2016**) also illustrated that Soy protein provides defense against low-density lipoprotein cholesterol, free radical damage, endothelial damage.

Coconut oil is an edible oil that has been used in many countries for thousands of years. Moreover, coconut oil has a long shelf life and 76° F melting point, in addition to being utilized in baking industries. Some researchers reported that there are no adverse impacts on people's health consuming diets that are high in coconut oil (**Thampan, 1998**). Virgin Coconut Oil (VCO) is considered a functional food usually used as a kind of traditional medicine and dietary supplement in several tropical regions. VCO includes medium-chain fatty acids as well as polyphenol antioxidants. In addition, VCO biological impact is traced back to its elevated antioxidant content like caffeic acid, ferulic acid, syringic acid, catechin, and epigallocatechin (**Illam et al., 2017 and Marina et al., 2009**). On the other hand, **Arunima and Rajamohan, (2012)** indicated that VCO enhances hepatic lipid metabolism in rats and increases fatty acid catabolism rates. While, (**Lekshmi Sheela et al., 2016**) reported that, 52% of VCO are medium-chain fatty acid "Lauric acid", this acid increases the

oxidative metabolism and contributes significantly to reducing lipid accumulation.

Magnesium can be a factor in both alcoholic and nonalcoholic liver diseases. **Rivlin, (1994) and Young *et al.*, (2003)** reported that individuals who drink large proportions of alcohol are exposed to elevated risks of magnesium deficiency and a significant reduction in magnesium homeostasis in the liver. Some studies have reported that, magnesium intake improves insulin resistance, decreases metabolic syndrome risk and decreases mortality risk resulting from liver disease also those with hepatic steatosis (**Song *et al.*, 2006; Champagne, 2008 and Wu *et al.*, 2017**). Therefore, the present study attempted to examine the impact of a soybean, coconut oil, and magnesium diet on non-alcoholic fatty liver rats.

## Materials and Methods

### Materials

- Vitamins, casein, minerals, cellulose, magnesium, and choline chloride were obtained from Al-Gomhoria Company for Trading Drugs, Chemicals, and Medical Instruments, Cairo, Egypt.
- Soybean and virgin coconut oil (*Cocos nucifera* L) were obtained from Agricultural Research Center, Giza, Egypt.
- Kits for biochemical analysis were obtained from Alkan for pharmaceutical and chemical Dokki, Egypt.
- Sixty adult male albino rats Sprague-Dawley Strain weighing ( $180 \pm 10$ g) were obtained from Helwan farm of experimental animals, Ministry of Health and Population, Helwan, Cairo, Egypt.

## Methods

### Soybean chemical analysis

Moisture, total protein, fat, ash, and fiber in low-fat soybean flour were identified according to (A.O.A.C. 1990), whereas total carbohydrates were estimated by deference.

### Fatty acid composition of coconut oil

Fatty acids compositions of virgin coconut oil were identified according to Gunstone *et al.*, 1994 and Yeshajahu, (1994).

### Experimental Design

Sixty adult male albino rats Sprague Dawley Strain weighing  $180 \pm 10$ g was used in this study. The rats were divided into two main groups: The first main group (12 rats) was divided into two subgroups "subgroup one" fed on basal diet BD containing 14% casein as a source of protein (Reeves *et al.*, 1993), while the "subgroup two" fed on a basal diet containing soybean powdered which provided the diet with 14% protein, these groups used as a control negative groups (-ve). The second main group (48) rats were on a high-fat diet (HFD) containing (carbohydrate 55.6% "starch 21.6%, sucrose 30% and fructose 4%"), (Fat 27.5% "soybean oil 2.5%, hydrogenated oil 5% and sheep tallow 20%"), protein of casein 8.2%, cellulose 4.2%, salt mixture 3.5% and vitamin mixture 1% for 8 weeks to induce non-alcoholic fatty liver disease (NAFLD) according to (Zarghani *et al.*, 2016). Rats that suffer from NAFLD were divided into (8 subgroups). The first subgroup was on HFD and used as a positive control group (+ve)<sup>1</sup>, whereas the second subgroup was on HFD containing the amount of protein from low-fat soybean flour and used as a positive control group (+ve)<sup>2</sup>, the third subgroup was fed on HFD containing all amount of protein from low-fat soybean with replacing 10% coconut oil instead of 10% sheep tallow, the fourth subgroup was fed on HFD containing all amount of protein from low-fat soybean with replacing 20% coconut oil instead of 20% sheep tallow, the fifth subgroup fed on HFD containing all amount

of protein from low-fat soybean flour, this diet supplemented with 500 mg magnesium/ kg diet, the sixth subgroup fed on HFD containing all amount of protein from low-fat soybean, this diet supplemented with 1000 mg magnesium/ kg diet, the seventh subgroup fed on HFD containing all amount of protein from low-fat soybean with replacing 10% coconut oil instead of 10% sheep tallow and supplemented with 500 mg magnesium/ kg diet. The eighth subgroup was fed on HFD containing all amount of protein from low-fat soybean with replacing 20% coconut oil instead of 20% sheep tallow and supplemented with 1000 mg magnesium/kg diet. By the end of the experimental duration (6 weeks), the animals were fasted overnight, and then sacrificed under very light ether anesthesia. Serum was withdrawn from the hepatic portal vein of each rat. Serum was carefully separated by centrifugation of the blood sample. Then, they were maintained at - 20°C until analysis. Livers were carefully enucleated from all mice via necropsy, rinsed with saline (0.9%), dried with a filter paper and weighed independently.

### Biochemical analysis

Determination of serum glucose was done according to (Trinder, 1969). Leptin hormone was determined by Leptin ELISA Kit based on the method proposed by Guillaume and Bjorntorp (1996). Total cholesterol was according to Allain *et al.*, (1974). Triglycerides were according to Fossati and prencipe (1982), whereas High density lipoprotein cholesterol (HDL-C) was according to (Burstein, 1970). Low and very low density lipoprotein-cholesterol (LDL-c and VLDL-c) were determined according to Friedwald *et al.*, (1972). Aspartate Aminotransferase (AST) and Alanin Aminotransferase (ALT) were according to Henry (1974). Alkaline phosphates (ALP) were based on the method of Belfield and Goldberg (1971). Liver [superoxide dismutase (SOD), catalase (CAT), as well as decreased glutathione (GSH) activities] were determined based on the methods demonstrated by (Aebi, 1984; Beauchamp and Fridovich 1971 and [Paglia & Valentine 1967](#)), respectively.

## Statistical Analysis

Results of biological evaluation of each group were statistically analyzed (mean  $\pm$  standard deviation and one-way ANOVA test) using SAS package and compared with each other using the suitable test (least significant differences at  $P < 0.05$  (SAS, 1996).

## Results and Discussion

### Chemical analysis of low fat soybean flour

The findings displayed in Table (1) demonstrate the chemical analysis of low-fat soybean flour. The moisture, protein, fat, ash, fiber, and carbohydrates were 4.00, 46.30, 6.62, 4.83, 10.106 and 28.114 g/100g, respectively.

**Table (1) Chemical analysis of low fat soybean flour (g / 100g dry weight)**

Nutrients	(g/100g DW)
Moisture	4.00
Protein	46.30
Fat	6.62
Ash	4.83
Dietary Fiber	10.106
Carbohydrate by deference's	28.144

### Fatty acid composition of coconut oil

The fatty acid composition of coconut oil is presented in table (2). Saturated fatty acid (SFA) were 92.25%, lauric acid (C12:0) was the major SFA presented in coconut oil (49.77%), followed by myristic C14:0 (13.21%), caproic C10:0 (9.95%),



caprylic C8:0 (9.61%), palmitic C16:0 (8.23%), stearic C18:0 (1.08%) and arachidic C20:0 (0.4%), respectively. The oleic acid C18:1 was 5.92% and linoleic acid C18:2 was 1.83% in the coconut oil.

**Table (2) Fatty acid composition of coconut oil (g/100g)**

Fatty Acids		Value(%)
Caprylic	C8:0	9.61
Caproic	C10:0	9.95
Lauric	C12:0	49.77
Myristic	C14:0	13.21
Palmitic	C16:0	8.23
Stearic	C18:0	1.08
Oleic	C18:1	5.92
Linoleic	C18:2	1.83
Arachidic	C20:0	0.4
<b>Saturated Fatty Acids [SFA]</b>		92.25
<b>Mono-unsaturated Fatty Acids [MUFA]</b>		5.92
<b>Poly-unsaturated Fatty Acids [PUFA]</b>		1.83

In similar study, **Shahidi, (2005)** stated that coconut oil contains 65% medium-chain fatty acids. **Nik et al., (2009)**, demonstrated that coconut oil is a natural source of medium-chain triglycerides (MCTs) with nearly 60% of the total oil content being MCTs. The term MCT mentions triglyceride which is constituted of glycerol and three saturated fatty acids with a chain length of 6-12 carbons. In contrast, the authors mentioned that MCTs have a beneficial effect on human health. **Bhatnagar et al., (2009)** found that, coconut oil has elevated concentrations of saturated fatty acids (SFA) ( $\approx 93\%$ ). Nonetheless, coconut oil also

has medium chain fatty acids (MCFA) (C6:0, C8:0, C10:0, C12:0) ( $\approx 60\%$ ), especially C12:0 ( $\approx 50\%$ ).

### **Effect of soybean diet, coconut oil and magnesium on feed intake, body weight gain % and liver weight/body weight% of rats Suffering from non-alcoholic fatty liver disease**

The effect of soybean diet, coconut oil, and magnesium on feed intake, body weight gain %, and liver weight/body weight% of rats suffering from non-alcoholic fatty liver disease is presented in Table (3). Non-significant differences in the mean value of feed intake was observed between the negative control group fed on the basal diet (control - ve)<sup>1</sup> and the negative control group fed on the soybean diet (control -ve)<sup>2</sup>.

The mean values of feed intake in the positive control groups fed on a high fat diet containing casein or soybean flour as sources of protein (control +ve)<sup>1&2</sup> decreased significantly ( $P \leq 0.05$ ), as compared to the negative control groups which were fed on normal diets that contain casein or soybean flour as sources of protein (control -ve)<sup>1&2</sup>. On the other hand, non-significant changes in the mean values of feed intake in all treated groups, as compared to the positive control groups (control +ve)<sup>1&2</sup>.

**Table (3) Effect of soybean diet, coconut oil and magnesium on feed intake, body weight gain % and liver weight/body weight% of rats suffering from non-alcoholic fatty liver disease**

Parameters		Feed intake (g/day/rat)	BWG%	Liver weigh / body weight%
Groups				
Control (-ve) <sup>1</sup> fed on basal diet		21.292 <sup>a</sup> ± 1.226	19.712 <sup>g</sup> ± 1.154	2.758 <sup>f</sup> ± 0.082
Control (-ve) <sup>2</sup> fed on soybean diet		20.884 <sup>a</sup> ± 1.223	18.634 <sup>g</sup> ± 1.212	2.678 <sup>f</sup> ± 0.072
Control (+ve) <sup>1</sup> fed on basal diet		17.598 <sup>b</sup> ± 0.839	31.735 <sup>a</sup> ± 1.214	3.895 <sup>a</sup> ± 0.047
Control (+ve) <sup>2</sup> fed on soybean diet		17.528 <sup>b</sup> ± 0.756	29.268 <sup>b</sup> ± 0.925	3.482 <sup>b</sup> ± 0.047
NAFLD group fed on HFD containing soybean flour as a source of protein,	with 10% CO instead of 10% ST	17.684 <sup>b</sup> ± 0.884	26.036 <sup>c d</sup> ± 1.473	3.289 <sup>c</sup> ± 0.052
	with 20% CO instead of 20% ST	17.616 <sup>b</sup> ± 0.633	23.650 <sup>e</sup> ± 1.786	3.071 <sup>d</sup> ± 0.055
	and supplemented with 500 mg Mg /kg diet	17.718 <sup>b</sup> ± 0.246	27.100 <sup>c</sup> ± 1.477	3.359 <sup>c</sup> ± 0.063
	and supplemented with 1000 mg Mg/kg diet	17.812 <sup>b</sup> ± 1.132	25.228 <sup>d</sup> ± 0.849	3.186 <sup>d</sup> ± 0.067
	with 10% CO instead of 10% ST and supplemented with 500 mg Mg/kg diet	17.638 <sup>b</sup> ± 0.954	23.255 <sup>e</sup> ± 0.621	3.063 <sup>d</sup> ± 0.082
	with 20% CO instead of 20% ST and supplemented with 1000 mg Mg/kg diet	17.984 <sup>b</sup> ± 0.456	21.445 <sup>f</sup> ± 0.453	2.905 <sup>e</sup> ± 0.045

CO: Coconut Oil

ST: Sheep Tallow

Mg: Magnesium

Means with different letters in each column are significantly different at  $P < 0.05$ .

The data in the previous table indicate non-significant changes in the mean value of body weight gain% (BWG%) that were recorded between the negative control groups (control -ve)<sup>1</sup> vs. (control -ve)<sup>2</sup>. In contrast, the BWG% of the positive control group (fatty liver disease group) fed on high-fat diet containing soybean flour as a source of protein decreased significantly  $p \leq 0.05$ , as compared to the positive control group (fatty liver

disease group) fed on high-fat diet containing casein as a source of protein.

Non- alcoholic fatty liver disease NAFLD groups treated with high-fat diet HFD containing soybean flour as a source of protein (with replacing 10% and 20% coconut oil instead 10% and 20% sheep tallow), or HFD containing soybean flour supplemented with (500 & 1000 mg Mg/kg diet) or (HFD containing soybean flour with replacing 10% coconut oil instead of 10% sheep tallow and supplemented with 500 mg Mg/kg diet) and / or (HFD containing soybean flour with replacing 20% instead 20% sheep tallow and supplemented with 1000 mg Mg/kg diet) showed a significant decrease ( $P \leq 0.05$ ) in BWG%, as compared to the positive control groups (control +ve)<sup>1&2</sup>.

The NAFL disease group demonstrated a marked decline in BWG% recorded for, as they were fed on HFD containing soybean flour as a source of protein (with replacing 20% coconut oil instead of 20% sheep tallow and supplemented with 1000 mg Mg/kg diet), followed by the group which fed on HFD that contains soybean flour (with replacing 10% coconut oil instead of 10% sheep tallow and supplemented with 500 mg Mg/kg diet) and the group fed on HFD containing soybean flour (with replacing 20% coconut oil instead of 20% sheep tallow), respectively.

Liver weight / body weight% of positive control groups (control +ve)<sup>1&2</sup> showed a significant increase  $P \leq 0.05$ , as compared to the negative control groups (control -ve)<sup>1&2</sup>. All treated groups with the two levels of (coconut oil, magnesium and their combinations) recorded significant decrease  $P \leq 0.05$ , as compared to the positive control groups (control +ve)<sup>1&2</sup>. The highest decrease in liver weight/body weight% was noticed for the NAFLD group which was fed on HFD containing soybean flour (with replacing 20% coconut oil instead of 20% sheep tallow and supplemented with 1000 mg Mg/kg diet).

On the other hand, **Bhandari et al., (2010)** found that feeding albino rats on high-fat diet increased the mean values of

body weight gain, visceral fat pad weight and heart weight, as compared to the rats which fed on a normal diet. The obtained results revealed that, soybean flour, coconut oil and magnesium improved weight gain and the weight of liver in NAFLD rats by decreasing them compared to the positive control groups. In this respect, **Caldwell *et al.*, (2005)** reported that, feeding obese Wistar fatty rats on soybean protein diet for 3 weeks decreased the mean value of body weight and triacylglycerols in the plasma and liver, as compared to Wistar fatty rats fed on casein diet. Moreover the current study was in general agreement with **Shahidi, (2005)** stated that coconut oil is composed of 65% medium chain fatty acids that are rapidly metabolized in the liver for energy production and are not engaged in the biosynthesis as well as cholesterol transportation, elevating HDL-c and decreasing the ratio between LDL to HDL. In addition, this oil does not result in obesity as it does not precipitate in fatty tissues. Therefore, the researcher was reported that medium-chain fatty differed in their metabolism from all the long-chain fatty acids, whether unsaturated or saturated. Mg supplement does not impact the enzymes of liver; nonetheless, weight loss may contribute to enhancing fatty liver disease (**Karandish *et al.*, 2013**).

### **Effect of soybean diet, coconut oil and magnesium on serum glucose and leptin hormone of rats suffering from non-alcoholic fatty liver**

The effect of soybean diet, coconut oil, and magnesium on serum glucose and leptin hormone of rats suffering from non-alcoholic fatty liver disease presented in Table (4). The values displayed in this table demonstrate that the mean values of serum glucose and leptin hormone in the negative control group<sup>1</sup> which was fed on the basal diet did not change significantly, as compared to the negative control group<sup>2</sup> which fed on a soybean diet. While these parameters decreased significantly  $P \leq 0.05$  in NAFLD group (control +ve group)<sup>2</sup> which fed on HFD containing soybean diet as a source of protein, as compared to NAFLD group (control +ve group)<sup>1</sup> which fed on the HFD containing casein as the source of protein.

**Table (4) Effect of soybean diet, coconut oil and magnesium on serum glucose and leptin hormone of rats suffering from non-alcoholic fatty liver**

Parameters		Glucose mg/dl	Leptin mg/dl
Groups			
Control (-) <sup>1</sup> fed on basal diet		75.826 <sup>g</sup> ± 3.859	3.360 <sup>h</sup> ± 0.142
Control (-) <sup>2</sup> fed on soybean diet		71.484 <sup>g</sup> ± 3.110	3.058 <sup>h</sup> ± 0.140
Control (+) <sup>1</sup> fed on basal diet		150.579 <sup>a</sup> ± 3.423	13.125 <sup>a</sup> ± 0.496
Control (+) <sup>2</sup> fed on soybean diet		140.410 <sup>b</sup> ± 2.250	10.656 <sup>b</sup> ± 0.443
NAFLD group fed on HFD containing soybean flour as a source of protein,	with 10% CO instead of 10% ST	125.936 <sup>d</sup> ± 4.437	8.933 <sup>d</sup> ± 0.458
	with 20% CO instead of 20% ST	112.886 <sup>e</sup> ± 2.800	7.030 <sup>f</sup> ± 0.156
	and supplemented with 500 mg Mg/kg diet	133.634 <sup>c</sup> ± 2.543	9.505 <sup>c</sup> ± 0.335
	and supplemented with 1000 mg Mg/kg diet	125.744 <sup>d</sup> ± 3.675	8.035 <sup>e</sup> ± 0.246
	with 10% CO instead of 10% ST and supplemented with 500 mg Mg/kg diet	109.937 <sup>e</sup> ± 4.475	7.221 <sup>f</sup> ± 0.294
	with 20% CO instead of 20% ST and supplemented with 1000 mg Mg/kg diet	97.840 <sup>f</sup> ± 5.187	5.159 <sup>g</sup> ± 0.244

CO: Coconut Oil

ST: Sheep Tallow

Mg: Magnesium

Means with different letters in each column are significantly different at  $P < 0.05$ .

All NAFLD treated groups with two levels of (coconut oil, magnesium, and their combination) showed a significant decrease  $P \leq 0.05$  in serum glucose and leptin hormone, as compared to the positive control groups (control +ve)<sup>1&2</sup>. The highest improvement in serum glucose and leptin hormone was recorded for the NAFLD group treated with (HFD containing soybean flour as a source of protein with replacing 20% CO instead of 20% sheep tallow and supplemented with 1000 mg Mg/kg diet), followed by the groups fed on HFD containing soybean flour as a

source of protein with 10% CO instead of 10% sheep tallow and supplemented with 500 mg Mg/kg diet, and the group which was treated with HFD containing soybean flour as a source of protein with 20% CO instead of 20% sheep tallow, respectively.

From these results we can observe the following: Feeding NAFLD rats on high-fat diet that contains casein or soybean flour as sources of protein increased serum glucose and leptin hormone significantly, as compared to the negative control groups which were fed on normal diets containing casein or soybean flour, in this respect (**Bhandari et al., 2010**) reported that albino rats treated with high-fat diet caused marked elevation in leptin, serum glucose as well as insulin in contrast to normal mice that were on normal diet. In contrast, the obtained results showed that soybean flour, coconut oil and magnesium diminished leptin hormone and serum glucose median values in NAFLD rats. In this respect, **Jenkins et al., (1981)** demonstrated that, soybean flour contains important nutrients including, complex carbohydrates, protein, dietary fiber, oligosaccharides, phytosterol, saponin, lecithin, isoflavone, phytic acid, trypsin inhibitor, and minerals. Complex carbohydrates and dietary fiber contents contribute to lower glycemic indexes, which benefit diabetic individuals and reduce the risk of developing diabetes. These results in line with **Thampan, (1994)** whom stated that, coconut oil contributes to the energy supply of cells as coconut oil can be absorbed easily without the need for additional (insulin or enzymes). Therefore, coconut oil improves insulin excretion and the utilization of glucose in the blood. **Kochikuzhyil et al., (2010)** reported that in a study of streptozocin-induced diabetic mice that were fed different fat types, rats fed with coconut oil had best results for their glucose level compared to palm oil and groundnut oil. The researcher recommended that insulin sensitivity improved as a result of improved triglycerides in the same rats. **Iranloye et al., (2013)** illustrated that virgin coconut oil has a hypoglycemic action and increases insulin excretion. In addition, it improves oxidative stress-induced in type I diabetes in male rats.

Magnesium is an important cofactor of glucose movement in the cell and carbohydrate metabolism, which participates in insulin cellular activity. The authors referred to that, it is considered magnesium low intake is a risk factor for diabetes (Lopez-Ridaura *et al.*, 2004). On the other hand, (Liu, *et al.*, 2019)<sup>a</sup> reported that, dietary magnesium administration (50 mg/mL in drinking water) for six weeks led to decrease blood glucose, improved mitochondrial function and decreased oxidative stress in rats with diabetes. Furthermore, Liu, *et al.*, (2020) showed that magnesium supplementation positively enhanced the activity of insulin receptor activity and insulin sensitivity in type 2 diabetes.

### **Effect of soybean diet, coconut oil and high level of magnesium on serum cholesterol and triglycerides of rats suffering from non-alcoholic fatty liver**

The effect of soybean diet, coconut oil, and magnesium on lipid profile including serum (cholesterol, triglycerides, high-density lipoprotein-cholesterol HDL-c, low-density lipoprotein-cholesterol LDL-c, and very low-density lipoprotein-cholesterol VLDL-c) of rats suffering from non-alcoholic fatty liver disease presented in Table (5 and 6). Total cholesterol and triglycerides of normal rats on the normal diet containing soybean flour as a source of protein decreased significantly  $P \leq 0.05$ , as compared to normal rats fed on a basal diet containing casein as a source of protein, the same trend was observed when comparing the NAFLD group fed on high-fat diet which contains soybean (control +ve)<sup>2</sup>, as compared to NAFLD group fed on high-fat diet which contains casein (control +ve)<sup>1</sup>. On the other hand, the mean values of total cholesterol and triglycerides in the positive control groups (control +ve)<sup>1&2</sup> increased significantly  $P \leq 0.05$ , as compared to these parameters in the negative control groups (control -ve)<sup>1&2</sup>.

All treated groups with soybean diet containing coconut oil, magnesium and their combination improved the mean values of



serum cholesterol and triglycerides, as compared to the positive control groups. The highest improvement of these parameters recorded for NAFLD groups which were fed on HFD containing soybean flour as a source of protein, with replacing (20% coconut oil CO instead of 20% sheep tallow ST and supplemented with 1000 mg magnesium /kg diet) and the group fed on the same diet with replacing (10% CO instead of 10% ST and supplemented with 500 mg Mg/kg diet), respectively.

**Table (5) Effect of soybean diet, coconut oil and high level of magnesium on serum cholesterol and triglycerides of rats suffering from non-alcoholic fatty liver**

Parameters		Cholesterol	Triglycerides
		mg/dl	
Groups			
Control (-) <sup>1</sup> fed on basal diet		86.051 <sup>h</sup> ± 3.647	50.586 <sup>f</sup> ± 3.390
Control (-) <sup>2</sup> fed on soybean diet		79.919 <sup>i</sup> ± 3.401	45.724 <sup>g</sup> ± 2.611
Control (+) <sup>1</sup> fed on basal diet		173.719 <sup>a</sup> ± 4.617	100.301 <sup>a</sup> ± 6.933
Control (+) <sup>2</sup> fed on soybean diet		163.088 <sup>b</sup> ± 4.847	93.245 <sup>b</sup> ± 5.024
NAFLD group fed on HFD containing soybean flour as a source of protein.	with 10% CO instead of 10% ST	149.156 <sup>c</sup> ± 2.132	85.116 <sup>c</sup> ± 3.518
	with 20% CO instead of 20% ST	131.722 <sup>e</sup> ± 2.926	72.412 <sup>d</sup> ± 3.367
	and supplemented with 500 mg Mg /kg diet	144.180 <sup>d</sup> ± 2.803	82.220 <sup>c</sup> ± 1.892
	and supplemented with 1000 mg Mg/kg diet	135.358 <sup>e</sup> ± 3.527	68.650 <sup>d</sup> ± 3.471
	with 10% CO instead of 10% ST and supplemented with 500 mg Mg/kg diet	126.919 <sup>f</sup> ± 3.104	70.321 <sup>d</sup> ± 1.467
	with replacing 20% CO instead of 20% ST and supplemented with 1000 mg Mg/kg diet	109.280 <sup>g</sup> ± 3.279	57.701 <sup>e</sup> ± 1.851

CO: Coconut Oil

ST: Sheep Tallow

Mg: Magnesium

Means with different letters in each column are significantly different at P < 0.05.

## Effect of soybean diet, coconut oil and high level of magnesium on serum lipoprotein-cholesterol of rats suffering from non-alcoholic fatty liver

High density, low density and very low-density lipoprotein – cholesterol (HDL-c, LDL-c and VLDL-c) of NAFLD groups which were treated with soybean diets containing CO, magnesium and their combination presented in Table (6). Non-significant change in the mean value of serum HDL-c was observed among the healthy groups which were fed on normal diets containing casein and soybean flour as sources of protein, while the healthy group which was on diet containing soybean flour (control –ve)<sup>2</sup> induced significant decrease  $P \leq 0.05$  in the mean values of serum LDL-c and VLDL-c, as compared to the healthy group fed on a basal diet (casein diet) (control –ve)<sup>1</sup>. The same trend was observed between the positive control groups (control +ve)<sup>1&2</sup>. NAFLD groups on HFDs containing soybean flour as a source of protein, (with 10% CO instead of 10% ST), or (with 20% CO instead of 20% ST), or (supplemented with 500 mg Mg /kg diet), or (supplemented with 1000 mg Mg /kg diet), or (with 10% CO instead of 10% ST and supplemented with 500 mg Mg/kg diet) and/or (with 20% CO instead of 20% ST and supplemented with 1000 mg Mg/kg diet) led to a significant increase in the mean value of serum HDL-c and decrease the mean values of serum (LDL-c & VLDL-c), as compared to the positive control groups (control +ve)<sup>1&2</sup>.

**Table (6) Effect of soybean diet, coconut oil and high level of magnesium on serum lipoprotein-cholesterol of rats suffering from non-alcoholic fatty liver**

Parameters		HDL-c	LDL-c	VLDL-c
		mg/dl		
Control (-) <sup>1</sup> fed on basal diet		53.798 <sup>a</sup> ± 1.757	22.152 <sup>g</sup> ± 1.372	10.117 <sup>f</sup> ± 0.678
Control (-) <sup>2</sup> fed on soybean diet		55.533 <sup>a</sup> ± 2.495	15.241 <sup>h</sup> ± 1.512	9.145 <sup>g</sup> ± 0.522
Control (+) <sup>1</sup> fed on basal diet		22.508 <sup>f</sup> ± 2.999	131.150 <sup>a</sup> ± 1.828	20.060 <sup>a</sup> ± 1.386
Control (+) <sup>2</sup> fed on soybean diet		24.530 <sup>f</sup> ± 2.873	119.908 <sup>b</sup> ± 2.760	18.649 <sup>b</sup> ± 1.004
NAFLD group fed on HFD containing soybean flour as source of protein,	with 10% CO instead of 10% ST	30.469 <sup>e</sup> ± 2.571	101.664 <sup>c</sup> ± 79.435	17.023 <sup>c</sup> ± 0.703
	with 20% CO instead of 20% ST	37.804 <sup>c</sup> ± 1.541	79.435 <sup>e</sup> ± 2.090	14.482 <sup>d</sup> ± 0.673
	and supplemented with 500 mg Mg /kg diet	32.170 <sup>d,e</sup> ± 2.553	95.567 <sup>d</sup> ± 4.148	16.443 <sup>c</sup> ± 0.378
	and supplemented with 1000 mg Mg/kg diet	39.373 <sup>b,c</sup> ± 1.356	82.254 <sup>e</sup> ± 2.652	13.729 <sup>d</sup> ± 0.694
	with 10% CO instead of 10% ST and supplemented with 500 mg Mg/kg diet	34.174 <sup>d</sup> ± 2.550	78.681 <sup>e</sup> ± 4.382	14.064 <sup>d</sup> ± 0.293
	with 20% CO instead of 20% ST and supplemented with 1000 mg Mg/kg diet	42.382 <sup>b</sup> ± 2.513	55.358 <sup>f</sup> ± 4.551	11.539 <sup>e</sup> ± 0.370

CO: Coconut Oil

ST: Sheep Tallow

Mg: Magnesium

Means with different letters in each column are significantly different at P &lt; 0.05.

The highest improvement in serum lipoprotein recorded for NAFLD group fed on HFDs containing soybean flour as a source of protein with replacing 20% CO instead of 20% ST and supplemented with 1000 mg Mg/kg diet. This treatment increased the mean value of serum HDL-c by about 72.776% and decreased the mean values of serum LDL-c and VLDL-c by about 53.83%

and 38.125%, respectively than that of the positive control group fed on HFD containing soybean flour as a source of protein.

From the results in table (5 and 6) it can be concluded that, feeding NAFLD rats on high-fat diet containing casein or soybean flour as sources of protein increased serum cholesterol, triglycerides, LDL-c and VLDL-c, as compared to the negative control groups which were fed on normal diets containing casein or soybean flour, while HDL-c decreased. These results are in agreement with (**Zhang *et al.*, 2019**) Who found that, after feeding rats on high-fat diet in order to trigger non-alcoholic fatty liver disease, total cholesterol, triglycerides and low density lipoprotein serum levels were markedly increased, while the high density lipoprotein – cholesterol levels were markedly decreased in contrast to the normal control group ( $P<0.01$ ).

Treating NAFLD rats with a soybean diet containing coconut oil, magnesium and their combination improved lipid profile, as compared to the positive control groups. In this respect (**Razzeto *et al.*, 2015**) suggest that replacement of soybean flour instead of casein in normocaloric and hypercaloric diets decreased triglycerides and improved fatty acids profile in liver of rats. The results coincided with that reported by **Ascencio *et al.* (2004)** whom suggested that hepatic triglycerides were decreased by feeding on soy protein or elevated-fat diets that contain soy protein in contrast to rats fed casein or elevated-fat diets that contain casein. The authors demonstrate that soybean flour reduces the risk of hepatic triglyceride deposition, and this impact is more obvious in hypercaloric groups. **Watzinger *et al.*, (2020)** and **Kouris-Blazos and Belski (2016)** showed that, an reverse correlation between the legumes consumption legumes and liver fat content. The researchers reported that, regular consumption of legumes may have beneficial effects in the prevention of cardiovascular disease and diabetes.

Regarding coconut oil, **Nurul-Iman *et al.*, (2013)** explained that consumption of coconut oil reduces tissue lipid levels, enhances the anti-thrombotic effects, promotes low cholesterol

levels, increases antioxidant activity, and inhibits lipid peroxidation in mice. The present results confirmed the data reported by **Sundram *et al.*, (1994)** whom found that, when consumed the individuals which were suffering from moderate cholesterol levels with a diet containing coconut oil, the levels of total and LDL cholesterol decreased, as compared to those consuming safflower oil and butter. Epidemiological studies could not detect the relationship between consumption coconut oil and the prevalence of cardiovascular disease (**Dayrit, 2003**). While (**Mensink *et al.*, 2003**) reported that, coconut oil appears to enhances the ratios between low-density lipoprotein cholesterol: high-density lipoprotein cholesterol and the ratio between total cholesterol: high density lipoprotein-cholesterol, both important markers of cardiovascular health.

Coconut oil contains ~ 65% medium chain fatty acids that are rapidly metabolized in the liver top resulting in energy production and are not involved in biosynthesis and cholesterol transportation. Consequently, coconut oil enhanced the level of HDL-c and decreased the ratio between LDL-c to HDL-c. Moreover, this oil does not result in overweight as is not accumulated in adipose tissues. Thus, medium chain fatty differ in their metabolism from all long-chain fatty acids, whether they are unsaturated or saturated (**Shahidi, 2005**).

Concerning the relationship between magnesium (Mg) and the lipid profile, **Cambray *et al.*, (2020)** reported that, Mg levels are correlated with heart health. While, **Zheltova *et al.*, 2016 and Scibior *et al.*, (2013)** reported that Mg is a major antioxidant, Mg deficiency has been correlated with elevated oxidative stress biomarkers and lipid peroxidation.

**Corica *et al.*, (2016)** in their clinical studies demonstrated that people with decreased magnesium levels have diminished HDL-cholesterol levels; nonetheless, they have elevated levels of triglycerides and total cholesterol (**Guerrero-Romero and Rodriguez-Moran 2002 and Song *et al.*, 2007**). On the contrary (**Bo and Pisu, 2008**) reported that, Mg decreases triglycerides and

increases high-density lipoprotein (HDL) through raised lipoprotein lipase activity, which catabolizes triglyceride lipoproteins and produces HDL. On the other hand, **(Rosanoff and Seelig, 2004)** reported that, magnesium inhibits (3-hydroxy-3-methyl-glutaryl-coenzyme A reductase "HMGCoA reductase", the rate-limiting enzyme for cholesterol synthesis, such as statin drugs, is fundamental to lecithin cholesterol acyl transferase activity. Therefore, it reduces low-density lipoprotein (LDL) triglycerides and elevates the levels of HDL.

### **Effect of soybean diet, coconut oil and high level of magnesium on liver enzymes of rats suffering from non-alcoholic fatty liver**

The effect of soybean diet, coconut oil and magnesium on liver enzymes including (Aspartate Aminotransferase AST, Alanine Aminotransferase ALT and Alkaline phosphates ALP) of rats suffering from non-alcoholic fatty liver disease is presented in Table (7). Feeding normal rats on a normal diet that contains soybean flour as a source of protein showed non-significant changes in the mean values of serum ALT and ALP, while AST enzyme decreased significantly ( $P \leq 0.05$ ), as compared to normal rats fed on basal diet. On the other hand, the NAFLD group fed on HFD that contains soybean flour as source of protein recorded a significant decrease ( $P \leq 0.05$ ) in liver enzymes (AST, ALT and ALP), as compared to the NAFLD group fed on HFD that contains casein as a source of protein.

**Table (7) Effect of soybean diet, coconut oil and high level of magnesium on liver enzymes of rats suffering from non-alcoholic fatty liver**

Parameters		AST	ALT	ALP
		U/l		
Control (-) <sup>1</sup> fed on basal diet		66.659 <sup>h</sup> ± 2.668	22.942 <sup>g</sup> ± 1.069	106.627 <sup>g</sup> ± 5.106
Control (-) <sup>2</sup> fed on soybean diet		62.808 <sup>i</sup> ± 2.372	19.652 <sup>g</sup> ± 0.999	105.596 <sup>g</sup> ± 5.790
Control (+) <sup>1</sup> fed on basal diet		120.596 <sup>a</sup> ± 1.718	79.290 <sup>a</sup> ± 2.527	210.182 <sup>a</sup> ± 6.719
Control (+) <sup>2</sup> fed on soybean diet		109.711 <sup>b</sup> ± 2.404	72.496 <sup>b</sup> ± 2.198	197.357 <sup>b</sup> ± 4.231
NAFLD group fed on HFD containing soybean flour as source of protein	with 10% CO instead of 10% ST	93.597 <sup>d</sup> ± 2.462	60.427 <sup>c</sup> ± 3.378	182.478 <sup>c</sup> ± 3.621
	with 20% CO instead of 20% ST	79.095 <sup>f</sup> ± 3.687	52.008 <sup>e</sup> ± 2.489	166.136 <sup>e</sup> ± 4.417
	and supplemented with 500 mg Mg/kg diet	97.228 <sup>c</sup> ± 3.599	63.030 <sup>c</sup> ± 3.591	185.312 <sup>c</sup> ± 2.548
	and supplemented with 1000 mg Mg/kg diet	84.701 <sup>e</sup> ± 2.516	56.989 <sup>d</sup> ± 3.312	172.109 <sup>d</sup> ± 5.258
	with 10% CO instead of 10% ST and supplemented with 500 mg Mg/kg diet	85.032 <sup>e</sup> ± 2.787	51.251 <sup>e</sup> ± 3.176	164.712 <sup>e</sup> ± 4.406
	with 20% CO instead of 20% ST and supplemented with 1000 mg Mg/kg diet	72.972 <sup>g</sup> ± 2.224	42.606 <sup>f</sup> ± 3.148	146.753 <sup>f</sup> ± 3.026

CO: Coconut Oil

ST: Sheep Tallow

Mg: Magnesium

Means with different letters in each column are significantly different at  $P < 0.05$ .

Feeding the NAFLD groups on high-fat diets HFDs that contain soybean flour as a source of protein, with (10% and 20% coconut oil instead of 10% and 20% sheep tallow) or with supplemented with 500 and 1000 mg Mg /kg diet) or (with 10% coconut oil instead of 10% sheep tallow and supplemented with 500 mg Mg / kg diet), and / or (with 20% coconut oil instead of 20% sheep tallow and supplemented with 1000 mg Mg / kg diet) improved all liver enzyme parameters than that of the positive control groups. The mean values of AST, ALT and ALP enzymes

decreased gradually with increasing coconut oil, magnesium and their combination.

The highest decrease in liver enzymes (AST, ALT and ALP) was observed in the NAFLD group fed on HFD containing soybean flour as a source of protein with (20% coconut oil instead of 20% sheep tallow and supplemented with 1000 mg Mg/kg diet) followed by NAFLD group fed on the same diet with replacing (10% coconut oil instead of 10% sheep tallow and supplemented with 500 mg Mg/kg diet), respectively.

From these results, we can observe the following: Feeding rats on high-fat diet to induce NAFLD caused an increase in AST, ALT and ALP enzyme mean values. Whereas, treating NAFLD rats with soybean flour, coconut oil, magnesium and their mixture improved all liver enzyme parameters (AST, ALT and ALP), as compared to the positive control groups. In this respect, (**Goorani et al., 2019**) demonstrated that a high-fat diet-induced significant increase ( $p \leq 0.05$ ) of the concentrations of ALP, AST, ALT, GGT, total and conjugated bilirubin in Wistar male rats, as compared to the control group. These results in line with **Panchal et al., (2011)** whom evaluated high fat/ high cholesterol diets, and reported increased liver weight, fat deposition, inflammation, and fibrosis with elevated plasma activity of liver enzymes.

Regarding the effect of soy on liver enzymes **Liu et al., (2017)** whom suggested that isoflavones found in soybean flour reduce fat deposits in the liver through decreasing adipogenesis and lipogenesis and activating the expression of PPAR- $\alpha$  to potentiate fatty acid oxidation in the liver. Hence, soy isoflavones can improve the development of NAFLD via reducing ALT enzyme and improving the liver structure.

With regard to coconut oil, **Nandakumarani et al., (2009)** concluded that administration of coconut oil increased antioxidant enzyme activity “superoxide dismutase” SOD, which is known to be protective against reactive oxygen species. In the similar study **Abd El-Fattah and Barakat, (2013)** reported that, the significant



decrease in the levels of ALT, AST, ALP enzymes and bilirubin in coconut oil administered animals might be due to decreased leakage of the enzymes in liver cells. This suggests that coconut oil could repair the hepatic injury and/or restore the cellular permeability, thus reducing the toxic effect of 2, 4-D induced liver toxicity and preventing enzymes leakage into the blood circulation. **Zakaria et al., (2011)** suggested that coconut oil protects the structural integrity of the liver cell membrane, so coconut oil works to protect the liver, which in turn leads to inhibition of increased liver enzymes in the blood. In this respect, **Mohammed et al., (2020)** found that, feeding rats that suffer from hypothyroidism on diets containing (7.5% and 10% coconut oil) decreased the mean values of AST and ALT significantly, as compared to the positive control group.

While it was found that there is a relationship between the consumption of magnesium and liver enzymes. "In this regard, **Liu et al., (2019)** <sup>b</sup> reported that magnesium is a vital action involved in many cellular processes. Magnesium deficiency is commonly associated with liver diseases and may result from low nutrient uptake. Magnesium supplementation can improve liver function in certain liver diseases. Moreover the current study was in general agreement with **Adachi and Brenner (2005)** also reported that, many cirrhosis patients to have a long history of alcohol intake, and magnesium deficiency is universally recognized in chronic alcoholics.

In a rat model of cirrhosis, serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were significantly lower following oral magnesium administration (**Paik et al., 2011**). While **Li et al., (2018)** suggest that high intake of magnesium may be associated with reduced risks of fatty liver disease and prediabetes.

**Effect of soybean diet, coconut oil and high level of magnesium on liver antioxidant enzymes of rats suffering from non-alcoholic fatty liver**

The effect of soybean diet, coconut oil and magnesium on antioxidant enzymes including (reduced glutathione GSH, superoxide dismutase SOD and catalase CAT, activities) of rats suffering from non-alcoholic fatty liver disease is presented in Table (8). Treating normal rats with a normal diet containing soybean flour as a source of protein showed a significant increase  $P \leq 0.05$  in the mean values of liver GSH-Px and CAT, compared to the normal rats fed on basal diet. While SOD showed non-significant changes between them. The same trend was observed when comparing the NAFLD group (Positive control groups). The two HFD groups (positive control groups) showed a significant decrease  $P \leq 0.05$ , as compared to the negative control groups.

**Table (8) Effect of soybean diet, coconut oil and high level of magnesium on liver antioxidant enzymes of rats suffering from non-alcoholic fatty liver**

Parameters		GSH-Px	SOD	CAT
		U/mg protein		
Control (-) <sup>1</sup> fed on basal diet		0.677 <sup>b</sup> ± 0.013	5.770 <sup>a</sup> ± 0.254	26.250 <sup>b</sup> ± 0.240
Control (-) <sup>2</sup> fed on soybean diet		0.747 <sup>a</sup> ± 0.009	5.878 <sup>a</sup> ± 0.269	29.150 <sup>a</sup> ± 0.240
Control (+) <sup>1</sup> fed on basal diet		0.234 <sup>h</sup> ± 0.014	2.896 <sup>g</sup> ± 0.147	10.250 <sup>h</sup> ± 0.140
Control (+) <sup>2</sup> fed on soybean diet		0.313 <sup>g</sup> ± 0.015	2.994 <sup>g</sup> ± 0.068	14.700 <sup>g</sup> ± 0.260
NAFLD group fed on HFD containing soybean flour as a source of protein.	with 10% CO instead of 10% ST	0.404 <sup>e</sup>	3.784 <sup>e</sup>	18.250 <sup>e</sup>
	with 20% CO instead of 20% ST	± 0.007	± 0.070	± 0.260
	and supplemented with 500 mg Mg/kg diet	0.518 <sup>d</sup>	4.588 <sup>c</sup>	21.900 <sup>c</sup>
	and supplemented with 1000 mg Mg/kg diet	± 0.008	± 0.046	± 0.210
	with 10% CO instead of 10% ST	0.353 <sup>f</sup>	3.436 <sup>f</sup>	16.350 <sup>f</sup>
	and supplemented with 500 mg Mg/kg diet	± 0.006	± 0.105	± 0.190
	with 20% CO instead of 10% ST	0.431 <sup>e</sup>	4.132 <sup>d</sup>	20.100 <sup>d</sup>
	and supplemented with 500 mg Mg/kg diet	± 0.009	± 0.103	± 0.140
with 20% CO instead of 20% ST	0.544 <sup>d</sup>	4.466 <sup>c</sup>	21.500 <sup>c</sup>	
and supplemented with 1000 mg Mg/kg diet	± 0.046	± 0.252	± 0.100	
		0.642 <sup>c</sup> ± 0.042	5.054 <sup>b</sup> ± 0.168	25.00 <sup>b</sup> ± 0.90

CO: Coconut Oil

ST: Sheep Tallow

Mg: Magnesium

Means with different letters in each column are significantly different at  $P < 0.05$ .

All NAFLD groups treated with coconut oil, magnesium and their combination showed a significant increase  $P \leq 0.05$  for these antioxidant enzymes, as compared to the positive control groups. The data in this table revealed that, the mean values of the liver GSH-Px, SOD, and CAT increased with increasing the levels of coconut oil, magnesium, and the high levels of coconut oil and magnesium in the combination group. The best results for liver antioxidant enzymes recorded for the NAFLD group fed on HFD contain soybean flour as a source of protein, with 20% coconut oil instead of 20% sheep tallow and supplemented with 1000 mg Mg/kg diet, followed by NAFLD groups fed on the same diet, (with 10% coconut oil instead of 10% sheep tallow and supplemented with 500 mg Mg/kg diet) and the group treated with 20% coconut oil instead of 20% sheep tallow), respectively.

From these results, it can be observed that feeding rats on HFD to induce NAFLD decreased the mean values of GSH-Px, SOD, and CAT in the liver. These results agree with (**Pan *et al.*, 2006**) which reported that feeding rats on a high-fat diet caused a significant increase in the level of malondialdehyde in the liver, while SOD and GSH-Px activities were decreased in the liver, as compared to the control group.

Treating rats with soybean flour as a source of protein increased the mean values of GSH-Px and CAT in the liver, while SOD did not change, as compared to rats treated with casein. In this respect (**Li and Zhang, 2017**) reported that, soy isoflavone SIF is the main active ingredient in soybeans and possesses a high antioxidant activity.

Feeding rats suffering from NAFLD on HFD, with coconut oil instead of sheep tallow increased the mean values of these antioxidants. In this respect, **Arsang *et al.*, (2020)** reported that, virgin coconut oil can be useful in the treatment of fatty liver by reducing lipids and increasing antioxidants. On the other hand, **Nevin and Rajamohan (2006)** showed that a diet containing coconut oil led to significant increases and decreases in

antioxidant enzymes and lipid peroxidation in rats, respectively. **Babu et al., (2014)** showed that virgin coconut oil contains phenolic compounds that increase antioxidant activity and reduce lipid index and blood pressure. Some researchers reported that, phenolic compounds significantly decreased inflammatory cytokines, enhanced antioxidant potency, and reduced interleukin-6 production (**Gauliard et al., 2008 and Haghdoost-Yazdi et al., 2016**). Coconut oil has a substantial effect on the antioxidant profiles, as compared to fish oil (**Attia et al., 2018**). On the other hand, **Bhatnagar et al., (2009)** reported that coconut oil supplementation increases total tocopherols. Tocopherols are essential antioxidants that protect the cell membrane from free radicals (**Attia et al., 2006**).

NAFLD rats that fed on HFD supplemented with magnesium, had increased antioxidants enzymes, compared to the NAFLD rats fed on HFD only. (**Zheltova et al., 2016 and Scibior et al., 2013**) reported that Mg is an important antioxidant, deficiency of Mg has been related to an increase in biomarkers of oxidative stress, and to an elevation in lipid peroxidation. Whereas, **Wenshuai et al., (2018)** suggest that the high intake of magnesium may be associated with lower odds of having fatty liver disease and prediabetes. Also **Tao and Fulda, (2021)** suggested that a high intake of magnesium may reduce the odds of having significant liver fibrosis.

Finally, based on the findings of the current study, low-fat soybean flour, virgin coconut oil and magnesium are beneficial in fatty liver treatment. The best results were recorded for the NAFLD groups which treated with HFD containing low-fat soybean flour as a source of protein with 20% coconut oil instead of 20% sheep tallow and supplemented with 1000 mg magnesium/kg diet.

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## تأثير دقيق فول الصويا، زيت جوز الهند، و الماغنسيوم علي الفئران التي تعاني من مرض الكبد الدهني غير الكحولي

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### الملخص العربي

تم إجراء هذا البحث لدراسة تأثير دقيق فول الصويا قليل الدهون وزيت جوز الهند البكر والماغنيسيوم وخليطهم على الفئران التي تعاني من مرض الكبد الدهني غير الكحولي وذلك من خلال بعض التقديرات الغذائية والكيميائية الحيوية .تم تقسيم الفئران إلى مجموعتين رئيسيتين: المجموعة الأولى (عددها 12 فأراً) قسمت إلى مجموعتين فرعيتين ["المجموعة الفرعية الأولى" تم تغذيتها على غذاء اساسي ، بينما تغذت "المجموعة الفرعية الثانية" على غذاء اساسي يحتوي على دقيق فول الصويا قليل الدهون يمد هذا الغذاء بـ 14% بروتين] ، وتم استخدام هذه المجموعات كمجموعات ضابطة سلبية. المجموعة الرئيسية الثانية (عددها 48 فأراً) تم تغذيتها علي غذاء عالي الدهون لمدة 8 أسابيع لإحداث مرض الكبد الدهني غير الكحولي. تم تقسيم فئران مرض الكبد الدهني غير الكحولي بشكل عشوائي إلى ثماني مجموعات فرعية متساوية. تم تسجيل أفضل النتائج للمجموعة المصابة بالكبد الدهني التي تم معاملتها بغذاء عالي الدهون والمحتوية على دقيق فول الصويا قليل الدهون كمصدر للبروتين مع استبدال 20% زيت جوز الهند بدلاً من 20% دهن غنم والمدعمة بـ 1000 مجم ماغنيسيوم / كجم غذاء. ختاماً و بناءً على نتائج الدراسة الحالية نستنتج أن دقيق فول الصويا قليل الدهون وزيت جوز الهند البكر والماغنيسيوم وخليطهم يحسن المضاعفات أو الأعراض الجانبية التي تنتج عن مرض الكبد الدهني غير الكحولي.

**الكلمات المفتاحية:** إنزيمات الكبد - صورة دهون الدم - وظائف الكلى - تدهن الكبد غير الكحولي - سوبر أكسيد ديسميوتيز .