Effect of coconut fruits on some biological changes in obese rats induced

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Abstract

The aim targets of this works is study the effect of coconut kernel powder with different ratio 5,10 and 15% on chemical, biological changes of rats induced obesity. Chemical composition, phenols, flavonoids, DPPH radical assay and fatty acids of coconut powder were studied. In addition, biological parameters were studied. Results showed that it could be concluded that coconut kernel powder is considered as a good source of oil, crude fiber, ash content, protein carbohydrates, total calories and has an antioxidant properties. Also it is a rich source of medium-chain triglyceride, mainly lauric acid, with approximately have the amount of fatty acids. Total saturated fatty acids represent 94.59%. Biological evaluation revealed that obesity rats fed on diet supplemented by coconut powder showed significant decrease ($P \leq 0.05$) of body weight, total lipids, cholesterol, triglyceride, LDLC and VLDLC. In addition, diet containing coconut powder significant decreased ($P \leq 0.05$) in liver enzymes (AST, ALT and LAP) and kidney functions (urea, uric acid and creatinine) of obesity rats. Moreover, increased both serum protein and albumin and lowering blood glucose. Were observed.

INTRODUCTION

Obesity is considered to be a major risk factor for chronic diseases such as cardiovascular, hypertension, type 2-diabetes and some type of cancer. Its prevalence is increasing, with 400 million obese and 1.6 billion overweight adults around the world **(WHO; 2005)**. Although, genetics plays a role in the regulation of body weight, body size and body consumption and the metabolic response to feeding in humans **(Lchihara and Yamada, 2008)** and in animals **(Reuter, 2007 and Speakman et al., 2007)**. The increase in worldwide obesity in a short period of time cannot be explained by genetics; there are individual differences in genetic susceptibility to invironmental factors such as diet **(James 2008; Rosengren and Lissner, 2008)**.

Obesity is a chronic condition characterized by excessive accumulation of body fat, resulting from a positive energy balance which compromises

health **(WHO, 2003)**. There are many contributing factors to obesity, including genetics, lifestyle, environmental factors and microbiota, but diet is still one of the most relevant, both in terms of the number of calories that are consumed as well as the source of those calories.

Coconut is, however, widely available, inexpensive, non-toxic and highly palatable, and consuming a regular intake of good quality coconut oil or another coconut product may become a simple yet important dietary change that may be shown in the future to reduce the risk of Alzheimer's disease **(Fernando et al., 2015)**.

The main targets of this work is study the influence of coconut powder with different ratio on biochemical , biological and histopathological changes of obesity rats. Also, study the efficiency of coconut fruit for increasing weight loss and improving lipid profile in obese rats. In addition, liver, kidney functions and blood glucose of rats were studied.

Approximately 30 - 40 % of coconut meal is formed during the virgin coconut oil production process and may be considered for production of food grade fibre. The effects of fibre on lipid profile depends on source of fibre, levels of incorporation, time duration, physical properties and method of feeding. Coconut virgin oil residue (VOR) and coconut milk residue (MR) incorporated feeds are favourable for lowering increase of serum total cholesterols TC and lowering serum triglycerides TAG concentrations and to increase serum HDL-C at a higher level of incorporation. Therefore both MR and VOR show good potential for use as a source of dietary fibre in functional food preparation **(Yalegama et al., 2013).**

Coconut oil is consist of mostly of mediumchain fatty acids, it composed of about 92% saturated fatty acids with 62-70% long MCFA. The lipid content of coconut, being mostly MCFA, offers an energy source that by passes the usual glucose pathway, in the form of ketone bodies, and without the associated fat deposition often caused by LCFA **(Fernando et al., 2015)**.

Debmandal and Mandal, (2011) noticed that medium chain fatty acids (MSFAs) of coconut oil are easily oxidized to lipids are not stored in the adipose tissue, unlike long-chain fatty acids (LCFAs). Considering this and the fat that coconut oil is rich in (MCFAs) and poor in (LCFAs), the use of extra virgin coconut oil could be effective in the treatment of obesity.

Naghii et al., (2012) assessed coconut oil as a supplement in rat diets, showing that, despite the high content in saturated fat, it seems to be beneficial for cardiovascular health, when consumed in moderation. However, further studies are required to effectively indicate the use of coconut oil as a supplement. **Liau et al., (2011)**. Showed that the use of coconut oil is related to decrease in serum triglyceride, since it contains medium chain fatty acids, which are absorbed and transported through the portal vein to the liver where they are rapidly oxidized, generating energy and also do not take part in the cholesterol cycle and are not accumulated in fat deposits.

Coconut flour substituted foods are able to produce high amount of short chain fatty acids (butyric acid), show low glycemic index and maintain lower weight increase **(Trinidad et al., 2006).** Neutral detergent fibre obtained from coconut kernel has the ability to reduce serum total cholesterol, LDL cholesterol and triglycerides concentrations in rats **(Sindurani and Rajamohan, 1998)**. Diabetes mellitus (DM) is a complex metabolic decease associated with impaired insulin secretion, developing insulin resistance as well as B-cell. Dysfunction that leads to abnormal glucose, protein and lipid metabolism **(Farzaei et al., 2015)**.

MATERIALS AND METHODS

Materials:

Coconut fruits were obtained from Agricultural Research Center, Food Technology Research Institute, Giza, Egypt.

Preparation of coconut powder:

Fresh coconut fruits cut into small parts, then dried in an electrical oven at 50^{o}C till dried and crushed into fine powder.

Animals:

Thirty Sprague-Dawely Strain male albino rats were obtained from Ophthalmology Medical Analysis Department, Giza, Egypt.

Methods:

Chemical analysis of moisture, protein, oil, ash, fibers and carbohydrate were determined as described by **AOAC, (2000)**. Results expressed as g/100g fresh weight. Total calories was calculated by **FAO/WHO, (1985)**, results expressed as K.cal/100g. Also, total phenols of aqueous extract were

determined using the method of **Batista et al., (2011)**. the results were expressed as galic acid/g. Meanwhile, total flavonoids were determined according **Batista et al., (2011)**, the results expressed as mg catechin equivalents (CE)/g of equeous extract. DPPH scavenging radical activity (%) of extracts was determined as described by **Hanato et al., (1988)**. Fatty acids were separated and identified by Gas Liquid chromatography (GLC) as described by **Stahle, (1967)**.

Experimental animals:

Diets

Basal diet was prepared according to **AIN, (1993)**. It consisted of 12.5% protein (casine), corn oil 10%, vitamin mixture 1.0%, salt mixture 4.0%, cellulose 5.0%, choline chloride 0.2%, methionine 0.3% and corn starch 67.5%. Meanwhile, diet induced obesity (DIO) was contained 10% cheep tail fat instead by corn oil and supplemented by 5,10 and 15% coconut powder. The composition of vitamin mixture was prepared according **Hegested, (1941)**.

Experimental design:

Thirty albino rats, Sprauge Dawley Strain, weighting 150± 5g. All rats were fed on basal diet for one week to adapted. Rats were divided into five groups (6 rats each). The first group was fed on basal diet as a negative control, the second group was fed on DIO as positive control, the other three groups were fed on DIO supplemented by 5, 10 and 15% coconut powder. At the end of experimental (4 weeks), blood samples of rats were collected according to **Drury and Wallington, (1980)**.

Biological determinations:

Body weight gain (g) and feed efficiency ratio of hypercholesterolemic rats were estimated according **Chapman et al (1959)**. Enzymatic calorimetric determination of triglycerides (mg/dl) was carried out to **Fassati and prencipe, (1982),** total cholesterol (mg/dl) was determined as the method of **Allian, (1974)**, and total lipids (mg/dl) was determined according to **Blank et al., (1967)**. HDL cholesterol (mg/dL) was determined according to the method described by **Lopez – Virella et al., (1977)**. VLDL and LDL (mg/dL) were carried out as **Lee and Nieman, (1996)**.

Also, serum aspartate amino transferase (AST) (μ/l) activity was calorimetrically measured according to the method described by **Yound, (1975)**. Alanine aminotransferase (ALT) (μ/l) activity was determined according to **Reitman and Frankel, (1957)**.

Alkaline phosphatase (ALP) (μ/l) in serum rats was determined as the method of **Moss, (1982)**. In addition, serum uric acid (mg/dl) was determined according to **Barham and Trinder, (1972)**; Serum creatinine (mg/dl) was determined as the method of **Bartles et al., (1972)**, urea (mg/dl) was determined as carried out using chemical kits according to **Trinder, (1969)**. Serum total protein (mg/dl) was determined using the method of **Doumas et al., (1981)**, while serum albumin was carried out to **Doumas et al., (1971)**.

Statistical analysis calculated by **SPSS 1998** program (Version, 19). The data expressed as the means ± standard deviation (SD).

RESULTS AND DISCUSSION

1- Chemical composition of coconut

Table (1) illustrates the chemical composition of fresh coconut fruits (g/100 g on fresh weight basis). Results showed that crude protein was 7.99%, crude fibers 6.2%, moisture content 3.87%, moisture content 1.87%, oil extracted by hexane 60.99%, total carbohydrates 25.23% and total soluble carbohydrates 19.03%. meanwhile, total calories was 681.79 k.cal/100 g fresh weight. These results are in agreement with those obtained by **Arivalagan et al., (2016).** Dry mature coconut kernel contained about 70% lipids **(Konan et al., 2008)**, however, copra dried kernel contained from 65% to 75% oil as reported by **NMCE, (2007).** These lipid are referred to as coconut oil, according to the type of extraction. In addition, the tender coconut water (TCW) contains sugars and sugar alcohols **(Young et al., 2009)**. The coconut residue is made into coconut flour is considered as highly dietary fibers **(Roberfroid, 1997)**. Also, the coconut meal is a rich source of fibers **(Trindad et al., 2006 and Yalegama et al., 2013)**. Besides, it is a good source of minerals **(Effiong et al., 2010)**.

It could be concluded that coconut kernel powder is the rich source of oil, crude fibers, ash content and total carbohydrates, especially soluble carbohydrates.

Table (1): Chemical composition of fresh coconut

Constituents	Percentage (%)
Crude protein	7.99
Ash content	1.92
Crude fibers	6.2
Oil content	60.99
Moisture content	3.86
Total carbohydrates	25.23
Total soluble carbohydrates	19.03
Total calories (k.cal/100g)	681.79

Results expressed as g/100 g on fresh eight basis

2- Antioxidant content of coconut

Table (2) shows total phenols, flavonoids and DPPH radical scavenging activity % of coconut. Results indicated that, total phenols was 0.86 mg as gallic acid equivalent /kg, total flavonoids 0.61 mg as quercetin equivalent /g and DPPH % 86.05%. These results are confirmed by **Marina et al., (2009)** who reported that coconut oil has a rich source of phytochemicals or polyphenols and phenolic acids which are recognized for their antioxidant properties, p.coumaric acid, ferulic, caffeic and catchin acid are the major phenolic acids. **Arlee et al., (2013)** reported that antioxidant levels of the coconut oil were found to be as high or up to twice as high as the control a-tecopherol, it contained the highest total phenolic contents and has an antioxidant properties.

Table (2): Total phenols, flavonoids and DPPH% of fresh coconut fruits powder

Antioxidant	*Total phenols	**Total flavonoids	DPPH (%)
Fresh coconut	0.86	0.61	86.05

* Results expressed as mg/g gallic acid **Results expressed as mg/g quercitin

Data in table (3) shows fatty acid composition of the total extracted oil from coconut kernels. Results showed that there are 12 fatty acids could be identified by GLC analysis, eight saturated and four unsaturated fatty acids.

3- Fatty acids of coconut

It is obvious from the results that only four fatty acids are predominant and constituents 83.89%, while other fatty acids are minor quantities 5.05%. The four major fatty acids are lauric acids $C_{12:0}$ (48.86%), followed by Meristic $C_{14:0}$ (18.23%), cabrylic $C_{8:0}$ (8.59%) and palmatic acid $C_{16:0}$ (8.21%), respectively. The major unsaturated fatty acids was oleic acid

$C_{18:1}$ (4.58%). Meanwhile, Lauric acids $C_{12:0}$ was the predominant of saturated fatty acids. These results are in agreement with those obtained by **Young et al., (2009)**.

Coconut oil is consisted of about 92% saturated fatty acids, with 62-70% medium chain fatty acids and 8.0% unsaturated fatty acids **(Gopala et al., 2010 and Chandrashekar et al., 2010)**. Saturated fatty acids were caproic, caprilic, capric, lauric, myristic, palmatic, stearic and arachidonic acid. Meanwhile, unsaturated fatty acids were palmetoleic, oleic, Linoleic and Gadoleic acid.

It could be concluded that coconut oil is considered as the main source of medium-chain triglyceride (MCTs) mainly lauric saturated fatty acids with approximately half the amount of fatty acids. Total saturated fatty acids represent 94.59%.

4- Biological effects

Body weight gain

The influence of coconut powder on body weight (BW), body weight gain (BWG), food intake (FI) and feed efficiency ratio of obese rats was illustrated in table (7) and fig. (3). The obtained data showed that rats fed on diet induced obesity (DIO) leads to increase body weight and body weight gain as compared to negative control group. At the end of experimental period (8 weeks), BWG of negative control group of rats was recorded 33.1±1.87%, while positive control was 88.3±1.93. Replacement of coconut powder by 5, 10 and 15% induced significant decrease ($P \leq 0.05$) in body weight gain of the obese rats to 75.9±1.67, 60.9±1.85 and 51.2±1.79%, respectively. There were significant decrease ($P \leq 0.05$) in BWG of rats which fed on coconut powder as compared to positive control group, the higher levels of coconut powder, the lower BWG of rats was noticed. The effects of different coconut powder in the control of obesity rats is the main subjects of many studies **(Chan and Elevitch, 2006 and Mohamed et al., 2009)**.

Table (3): Fatty acid composition of coconut powder

Saturated fatty acids (RAP%)			Un saturated fatty acids (PAR%)		
Caproic	$C_{6:0}$	0.75	palmetoleic	$C_{16:1}$	0.01
Caprjlic	$C_{8:0}$	8.59	Oleic	$C_{18:1}$	4.28
Capric	$C_{10:0}$	6.55	Linoleic	$C_{18:2}$	0.73
Lauric	$C_{12:0}$	48.86	Gadoleic	$C_{20:1}$	0.03
Myristic	$C_{14:0}$	18.23	___		___
Palmatic acid	$C_{16:0}$	8.21	___		___
Stearic acid	$C_{18:0}$	3.32	___		___
Arachedonic	$C_{20:0}$	0.08	___		___
Total saturated fatty acids		94.58	Total unsaturated fatty acids		5.05

(RAP%) Relative area percent

The decrease of either body weight or body weight gain could be attributed that coconut is a rich source of fiber **(Yalegama et al., 2013).** Also, coconut flour contained dietary fiber, cellulose, hemicelluose, lignin, neutral and acid detergent fiber **(Rodrigues and Pinto, 2007 and Ng et al., 2010).**

Moreover, dietary fiber plays an important role in the human health due to its potential to control weight increase **(Ramos et al., 2008).** In addition, coconut kernel contained water soluble galctomannans which are digested in the small intestine and produce short chain fatty acids **(Jensen et al., 1993)**. Whereas, virgin coconut oil has an antioxidant and lipid-lowering **(Arunaksharan et al., 2018)**. Medium chain triglycerides (MCT) present in coconut oil which considered as agents that aid the prevention of obesity or stimulating weight loss **(St-Ong et al., 2003)**. Coconut oil could be effective in the treatment of obesity **(Debmandal and Mandal, 2011)**.

The above-mentioned results revealed that daily food intake of positive control group was recorded 23.6±2.04 g, while it was decreased to 20.1±1.74, 18.4 ±1.53 and 18.1±1.77 g of rats which fed on 5, 10 and 15% coconut powder, respectively. Also, feed efficiency ratio (FER) showed the same trend of results in food intake.

Table (4): Effect of coconut powder on growth performance of obesity rats.

Groups \ Parameters	Initial body weight (g) (IBW)	Final body weight (g) (BW)	Food intake (g) (FI)	Body weight gain (g) (BWG)	Feed Efficiency ratio(FER)
control (–Ve)	$150.0^{a}\pm3.8$	$182.9^{e}\pm3.0$	$13.9^{c}\pm1.4$	$33.0^{b}\pm1.77$	$2.4^{a}\pm0.15$
control (+Ve)	$152.3^{a}\pm3.26$	$240.6^{a}\pm5.83$	$23.6^{a}\pm2.04$	$88.3^{a}\pm1.93$	$3.74^{a}\pm0.11$
obesity rats with 5% coconut powder	$151.2^{a}\pm3.19$	$226.1^{b}\pm5.35$	$20.1^{b}\pm1.74$	$75.9^{b}\pm1.67$	$3.78^{a}\pm0.16$
obesity rats with 10% coconut powder	$152.8^{a}\pm3.52$	$213.7^{c}\pm5.66$	$18.4^{b}\pm1.53$	$60.9^{c}\pm1.85$	$3.31^{b}\pm0.21$
obesity rats with 15% coconut powder	$151.4^{a}\pm3.38$	$202.6^{d}\pm5.14$	$18.1^{b}\pm1.77$	$51.2^{b}\pm1.79$	$2.83^{a}\pm0.17$
L.SD ($p\leq 0.05$)	3.49	5.87	1.89	1.75	0.18

Each value in a column followed by the same letter are not significantly different at ($P\leq0.05$).

5- Lipid profile

Table (5) illustrates the effect of coconut powder on serum total lipids (TL), total cholesterol (TC) and total triglyceride (TG) of obesity rats. The obtained results showed that diet induced obesity (DIO) of rats were significantly increase ($P\leq0.05$) in serum total lipids, cholesterol and triglyceride as compared to negative control group of rats.

Total lipids of positive control group of rats was 423.44±8.13 mg/dL, then decreased to 345.14±6.77; 280.56±5.94 and 239.85±6.07 mg/dL in the groups of rats which fed on coconut powder at 5, 10 and 15%, respectively. This decrease of serum total lipids could be due to the decrease of body weight of rats which fed on coconut powder as shown in the previously results in table (4). In addition, coconut is able to reduce hyperlipidemia **(Salil and Rajamohan, 2001)**.

On the other hand, total serum cholesterol of positive control group of rats was recorded 201.87±5.41 mg/dL, then decreased for rats fed on coconut at ratio 5, 10 and 15% till reached to 166.66±4.69, 140.83±5.16 and 127.26±4.22 mg/dL), respectively. Meanwhile, serum total triglyceride was 195.65±6.23 (mg/dL) in positive control, then decreased to 147.24±5.86, 108.27±5.44 and 89.74±5.39 (mg/dL) of rats fed on 5, 10 and 15% coconut, respectively.

The same trend of results are in agreement with those obtained by **Salil and Rajamohan, (2001)** who found that coconut had significantly lower levels of serum cholesterol, triglyceride and phospholipids. The

hypocholesterolimic Also, Epidemiological studies suggested that high intakes of dietary fiber reduced the risk of coronary heart disease **(Despress et al., 1996)**.

It could be concluded that total lipids, cholesterol and triglyceride of serum lipids were significantly decrease ($p \leq 0.05$) in obesity rats which fed on coconut powder, the lower values of total lipids, triglycerides and total cholesterol of serum lipids of rats as compared to positive control.

Table (5): Effect of coconut powder on TC, TG and TL of obesity rats.

Lipid profile / Groups	Total cholesterol (TC) (mg/dL)	Total Lipids (TL) (mg/dL)	Total triglyceride (TG) (mg/dL)
control (–Ve)	$94.1^{e} \pm 2.60$	$195.1^{e} \pm 3.2$	$86.1^{d} \pm 2.55$
control (+Ve)	$201.87^{a} \pm 5.41$	$423.44^{a} \pm 8.13$	$195.65^{a} \pm 6.23$
obesity rats with 5% coconut powder	$166.66^{b} \pm 4.69$	$345.14^{b} \pm 6.77$	$147.24^{b} \pm 5.86$
obesity rats with 10% coconut powder	$140.83^{c} \pm 5.16$	$280.56^{c} \pm 5.94$	$108.27^{c} \pm 5.44$
obesity rats with 15% coconut powder	$127.26^{d} \pm 4.22$	$239.85^{d} \pm 6.07$	$89.74^{d} \pm 5.39$
L.SD ($p \leq 0.05$)	**5.76**	**7.83**	**6.37**

Each value in a column followed by the same letter are not significantly different at ($P \leq 0.05$).

Table (6) illustrates the influence of coconut fruit powder on serum of high density lipoprotein (HDL) , low density lipoprotein (LDL) and very low density lipoprotein (VLDL) of the obesity rats. The results indicated that serum of HDL, LDL and VLDL values were significantly increase ($P \leq 0.05$) in positive than negative control group, while serum of HDL of positive control was recorded 43.92±2.34 mg/dL and increased to 45.25±2.48, 46.36±1.99 and 48±2.35mg/dL for rats fed on diet containing 5, 10 and 15% of coconut powder , respectively.

In contrast, serum LDL value of positive control group was recorded 126.34±5.61 mg/dL and decreased to 92.75±3.78, 70.35±5.61 and 58.64±3.18 mg/dL for rats fed on 5, 10 and 15% coconut, respectively. Also, serum VLDL showed the same trend of results of LDL.

The obtained results showed that serum VLDL of positive control group was 31.61±1.83 mg/dL and decreased to 28.66±1.76, 24.12±1.55 and 20.38±1.73 mg/dL for rats fed on 5, 10 and 15% coconut powder,

respectively. These results are in agreement with those found by **Rajamohan and Kurup, (1996); Salil and Rajamohan, (2001) and Trindad et al., (2006)**.

It is obvious from the abovementioned results , that diet of obesity rats supplemented by coconut powder showed significantly decrease ($P \leq 0.05$) in both serum LDL and VLDL values as compared with positive control group of rats, while HDL was significantly increase.

Such results may be due to that, coconut has an antioxidant contents such as total phenolics and flavonoids as illustrated in the previously results in table (1). In addition, natural antioxidants could prevent oxidative damage in various health disorders with oxidative stress **(Khaki et al., 2013)**. Phenolic compounds are protective agents, reducing the oxidative damage in the human body and retarding chronic disease, as well as LDL play an important role of atherosclerosis **(Shen et al., 2010)**. Also, flavonoids were free radical scavengers and can prevent, LDL cholesterol oxidation in vitro **(Folsom et al., 1999)**.

Moreover, coconut oil has very low cholesterol than animal protein and medium chain triglycerides are easily digested, absorption is faster and contributes to formation of LDL which transport cholesterol **(Dayrite, 1995)**.

It could be concluded that diet supplemented by coconut powder caused a significant decrease ($P \leq 0.05$) in both LDLC and VLDC as compared with positive control and increased HDLC, i.e improved lipid profile of obesity rats.

Table (6): Effect of coconut powder on HDL, LDL and VLDL of obesity rats.

Lipid profile / Groups	High density lipoproteins (HDL) (mg/dL)	Low Density lipoproteins (LDL) (mg/dL)	Very Low Density lipoproteins (VLDL) (mg/dL)
control (–Ve)	$48.0^{a} \pm 2.25$	$28.65^{e} \pm 2.11$	$17.0^{e} \pm 1.5$
control (+Ve)	$43.92^{c} \pm 2.34$	$126.34^{a} \pm 5.61$	$31.61^{a} \pm 1.83$
obesity rats with 5% coconut powder	$45.25^{bc} \pm 2.48$	$92.75^{b} \pm 3.78$	$28.66^{b} \pm 1.76$
obesity rats with 10% coconut powder	$46.36^{b} \pm 1.99$	$70.35^{c} \pm 3.81$	$24.12^{c} \pm 1.55$
obesity rats with 15% coconut powder	$48.24^{a} \pm 2.35$	$58.64^{d} \pm 3.18$	$20.38^{d} \pm 1.73$
L.SD ($p \leq 0.05$)	**2.15**	**4.52**	**1.58**

Each value in a column followed by the same letter are not significantly different at ($P \leq 0.05$).

6- Liver functions

Table (7) shows the effect of coconut powder on enzyme activity of liver, serum asparatate aminotransferase (AST), serum alanine aminotransferase (ALT) and serum alkaline phosphatase (ALP). Liver function tests that evaluate the presence of liver disease or damage **(Henery, 2001).** AST enzyme is found in high concentrations of liver cells (16-40µL) and it is important for metabolism of alanine, amino acids. An increase in serum AST levels may indicate liver disease **(Nanji et al., 2003)**. The level of serum ALT in the blood are low (8-54 µL). Higher than of ALP may indicate liver disease or damage **(Henery, 2001)**.

The obtained results indicated that serum AST, ALT and ALP levels of positive control group of rats was significantly increase ($P \leq 0.05$) than negative control group. On the other hand, serum AST of positive control group was recorded 122.76±5.73 µ/L then decreased to 92.54±4.64, 71.62±3.98 and 65.9±4.59 (µ/L) in serum rats which fed on diet supplemented by 5, 10 and 15%, respectively. While, serum ALT was 65.17±3.15, (µ/L) in positive control then decreased to 49.4 ± 2.69, 31.16±2.74 and 23.49±2.55 (µ/L) in rats fed on coconut powder with the percentage 5, 10 and 15% , respectively. Results showed that serum ALP of positive control was 97.26 ±4.96 ((µ/L) then decreased to 80.78±3.99, 75.67 ±3.85 and 71.42 ± 4.14 (µ/L), for rats fed on coconut powder with 5, 10 and 15% , respectively.

It is evident from the results that, there were significantly decrease ($P \leq 0.05$) of serum AST, ALT and ALP levels as compared with positive control group of rats due to the addition of coconut powder to the diet, the higher percentage of coconut, the lower levels of these enzymes was observed. i.e. improved these liver functions. This decrease of liver enzymes may be due to that coconut is a good source of antioxidant contents as illustrated in the previously results in table (5). Also, these results were in agreement with those noticed by **Trindad et al., (2003).** Dietary fibers has the ability to bind with bile acids and prevents its reabsorption in the liver, thus inhibit cholesterol synthesis.

Diet supplemented by coconut powder was improved liver functions and significantly reduced ($p \leq 0.05$) the activity of AST, ALT and ALP enzymes of obesity rats.

Table (7): Effect of coconut fruits powder on AST, ALT and ALP of obesity rats.

Liver functions / Groups	Aspartate aminotransferase (AST) (IU / L)	Alanine Aminotransferase (ALT) (IU/L)	Alkaline Phosphatase (ALP) (IU/L)
control (–Ve)	$62.9^{d} \pm 1.88$	$23.8^{d} \pm 1.91$	$69.9^{c} \pm 4.18$
control (+Ve)	$122.76^{a} \pm 5.73$	$65.17^{a} \pm 3.15$	$97.26^{a} \pm 4.96$
obesity rats with 5% coconut powder	$92.54^{b} \pm 4.64$	$49.40^{b} \pm 2.69$	$80.78^{b} \pm 3.99$
obesity rats with 10% coconut powder	$71.62^{c} \pm 3.98$	$31.16^{c} \pm 2.74$	$75.67^{bc} \pm 3.85$
obesity rats with 15% coconut powder	$65.91^{d} \pm 4.59$	$23.49^{d} \pm 2.55$	$71.42^{c} \pm 4.14$
L.SD ($p \leq 0.05$)	**3.81**	**2.37**	**4.23**

Each value in a column followed by the same letter are not significantly different at ($P \leq 0.05$).

7- Kidney functions

Table (8) illustrates the influence of coconut powder on kidney functions (Serum urea, uric acid and creatinine) of rats. The results revealed that serum urea, uric acid and creatinine of positive control group of rats were significantly increase ($p \leq 0.05$) than negative control group. Serum urea of positive control was recorded 30.86 ± 2.90 mg/dL. Diets supplemented by coconut powder at 5, 10 and 15% caused a decrease to 24.27±2.45, 20.15±2.85 and 19.39±2.10 (mg/dL), respectively. On the other hand, serum, uric acid of positive control was 49.94±2.63 mg/dL then decreased to 35.58±3.24, 33.37±2.5 and 30.91±2.68 mg/dL, by the addition of coconut powder at 5, 10 and 15%, respectively. Meanwhile, serum creatinine of positive control was 1.59±0.35 (mg/dL), then decreased to 0.98±0.24, 0.74±0.21 and 0.68±0.26 (mg/dL) for rats fed on diet supplemented by coconut powder at 5,10 and 15%, respectively.

It is evident from the results that, there were significant decreased ($P \leq 0.05$) in serum urea, uric acid and creatinine as compared with positive control group due to the addition of coconut powder, the higher percentage of coconut powder, the lower levels of serum urea, uric acid and creatinine was observed. The improving of kidney functions may be due to that coconut is considered as a good source of antioxidant contents such as total phenols and flavonoids as shown in the previously, results in table (2).

Generally, it could be concluded that diet supplemented by coconut powder caused lowering of serum urea, uric acid and creatinine and also improved kidney functions of obesity rats.

Table (8): Effect of coconut powder on urea, uric and creatinine of obesity rats.

Kidney functions / Groups	Uric acid [mg / dl]	Urea [mg / dl]	Creatinine [mg / dl]
control (–Ve)	$32.1^{c} \pm 2.07$	$17.9^{d} \pm 2.46$	$0.67^{c} \pm 0.08$
control (+Ve)	$49.93^{a} \pm 2.63$	$30.86^{a} \pm 2.90$	$1.59^{a} \pm 0.35$
obesity rats with 5% coconut powder	$35.58^{b} \pm 3.24$	$24.27^{b} \pm 2.45$	$0.98^{b} \pm 0.24$
obesity rats with 10% coconut powder	$33.37^{b} \pm 2.50$	$20.15^{c} \pm 2.85$	$0.74^{b} \pm 0.21$
obesity rats with 15% coconut powder	$30.91^{c} \pm 2.68$	$19.39^{cd} \pm 2.10$	$0.68^{cd} \pm 0.26$
L.SD ($p \leq 0.05$)	**3.46**	**2.64**	**0.21**

Each value in a column followed by the same letter are not significantly different at ($P \leq 0.05$).

8- Serum proteins

Table (9) shows the effect of coconut fruit powder on serum total protein (TP), albumin (AL) and bilirubin (BL) of obesity rats. The obtained results revealed that both serum total protein and albumin of rats which fed on diet induced obesity were recorded significantly decrease ($P \leq 0.05$) than negative control which fed on basal diet, while serum bilirubin was significant increase.

It is obvious from the results that total protein of positive control group of rats was 6.84±0.85 (mg/dL) and increased to 8.62±0.80, 10.01 ± 0.94 and 10.63 ± 0.96 (mg/dL) for rats fed on diet supplemented by coconut powder with 5, 10 and 15%, respectively. Also, serum total albumin showed the same trend of results.

On the other hand, serum total albumin of positive control was recorded 3.82±0.38 (mg/dL) and increased to 4.74 ± 0.49. 5.36±0.41 and 5.90±0.40 (mg/dL) for rats fed on 5, 10 and 15% coconut powder, respectively.

It is worthy to mention that, the higher percentage of coconut powder, the higher levels of both total serum protein and albumin was noticed. i.e. coconut powder improved both serum protein and albumin of obesity rats.

In contrast, serum bilirubin of positive control group of rats was 3.25 ± 0.42 mg/dL , then decreased to 2.52 ± 0.42, 1.37 ±0.33 and 1.18 ± 0.28

mg/dL, for rats fed on diet contained 5,10, and 15%, respectively. It is evident from the results that there were significantly decrease ($p \leq 0.05$) as the addition of coconut powder increased. Results also showed that there were. Significantly increase in either serum protein or albumin of rats due to the addition of coconut powder. This increase may be due to phenolics and flavonoids as shown in the previously results in table (5). Moreover, coconut has highly nutritious or functional food due to its rich source of vitamins, minerals and dietary fibers **(Fernando et al., 2015)**.

9- Serum glucose

Table (10) shows the effect of coconut powder on serum glucose (mg/dL) of obesity rats during the experimental period. The obtained results indicated that serum glucose of positive control group of rats was significant increased ($P \leq 0.05$) than negative control group. At the end of experimental period, serum glucose of positive control was recorded 156.25±2.38 mg/dL, then decreased to 104.53±1.93, 97.28±2.06 and 88.36±2.14 mg/dL for rats fed on diet supplemented by coconut powder at 5, 10 and 15%, respectively. The higher percentage of coconut powder, the lower level of serum glucose was observed. It is worthy to mention that, serum glucose an improved as the time of experimental proceeded. The same trend of results are confirmed by **Trindad et al., (2003)**.

Table (9): Effect of coconut powder on total protein (TP), albumin (AL) and bilirubin (Bil)of obesity rats.

Parameter / Groups	Total protein (TP) [mg /dl]	Albumin (AL) [mg /dl]	Bilirubin (Bil) [mg /dl]
control (–Ve)	10.1^{a} ±1.14	6.01^{a} ± 0.33	1.05^{c} ± 0.26
control (+Ve)	6.84^{d} ±0.85	3.82^{c} ± 0.38	3.25^{a} ± 0.53
obesity rats with 5% coconut powder	8.62^{c} ± 0.80	4.74^{b} ± 0.49	2.52^{b} ± 0.42
obesity rats with 10% coconut powder	10.01^{a} ± 0.94	5.36^{ab} ± 0.41	1.37^{bc} ± 0.33
obesity rats with 15% coconut powder	10.63^{a} ± 0.96	5.90^{a} ± 0.40	1.18^{c} ± 0.28
L.SD ($p \leq 0.05$)	**0.97**	**0.39**	**0.36**

Each value in a column followed by the same letter are not significantly different at ($P \leq 0.05$).

The intake of dietary fiber might increase the hypoglycemic effects, there was a decrease in the blood sugar as well as a drop in the serum insulin levels. This decrease may be due to the biding of glucose by the fiber

which a decrease in insulin production by the pancreas **(Rani and Rajamohan, 2006)**.

Also, high dietary fiber of foods could reduce blood glucose and insulin responses **(Collier et al., 1988)** and patients with diabetes mellitus **(Wolever et al., 1992)**. Glycemic index of coconut decreased with increasing coconut percentage **(Trindad et al., 2003)**.

It could be concluded that diet supplemented by coconut powder caused lowering blood glucose during experimental period. Also, there were significantly decrease ($P \leq 0.05$) in blood glucose of obesity rats.

Table (10): Effect of coconut powder on serum glucose of obesity rats.

Blood glucose / Groups	Serum glucose (Mg /dl)		
	Zero time	**2 weeks**	**4 weeks**
control (–Ve)	$86.01^{aA} \pm 1.14$	$6.32^{eA} \pm 1.69$	$85.8^{dA} \pm 2.0$
control (+Ve)	$132.46^{aC} \pm 2.09$	$141.35^{aB} \pm 2.54$	$156.25^{aA} \pm 2.38$
obesity rats with 5% coconut powder	$130.56^{aA} \pm 2.80$	$113.51^{bB} \pm 2.05$	$104.53^{bC} \pm 1.93$
obesity rats with 10% coconut powder	$132.71^{aA} \pm 2.94$	$106.92^{cB} \pm 2.18$	$97.28^{cC} \pm 2.06$
obesity rats with 15% coconut powder	$130.49^{aA} \pm 2.96$	$96.74^{dB} \pm 1.86$	$88.36^{d} \pm 2.14$
L.SD ($p \leq 0.05$)	**2.83**	**2.17**	**2.06**

Each value in a column followed by the same letter are not significantly different at ($P \leq 0.05$).

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تاثير ثمار جوز الهند على بعض التغيرات البيولوجية في الفئران المصابة بالسمنة

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المستخلص

يهدف البحث إلى دراسة التركيب الكيميائي، مضادات الأكسدة، الأحماض الدهنية في جوز الهند وكذلك دراسة تأثير التغذية بجوز الهند المجفف بنسب 5، 10، 15% على التغيرات البيولوجية في الفئران المصابة بالسمنة وقد دلت النتائج على ما يلي: يعتبر جوز الهند مصدر جيد لكل من الزيت والألياف والمعادن والبروتين والكربوهيدرات والسكريات الذاتية والعنبولات الكلية والفلافوتويدات فضلاً على أنه مصدر غني بالأحماض الدنية الغير مشبعة (94.54%) وخصوصاً حمض اللوريك. أدت التغذية باستخدام جوز الهند إلى حدوث إنخفاض معنوي عند مستوى المعنوية 0.05 في كل من وزن الجسم في الفئران، الليبيدات الكلية، الدهون الثلاثية، الكوليسترول عالي ومنخفض الكثافة في الفئران المصابة بالسمنة بالمقارنة بالمجموعة الضابطة الموجبة. لوحظ إرتفاع معنوي في كل من البروتينات الكلية والألبومين في سبرم دم الفئران. أيضاً حدث إنخفاض معنوي عند مستوى المعنوية 0.05 في كل من نشاط إنزيمات الكبد (ALP, ALT, AST)، مما أدى لتحسين وظائف الكبد، وكذلك إنخفاض اليوريا وحمض اليوريك والكرياتين مما أدى لتحسين وظائف الكلى فضلاً عن إنخفاض مستوى سكر الدم في الفئران المصابة بالسمنة بالمقارنة بالمجموعة الضابطة الموجبة.