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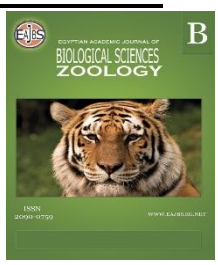

Influence of Honeydew Melon, (*Cucumis melo* L.) Fruit Fractions on The Diabetic Hypercholesterolemic Rats

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ABSTRACT

This study aimed to investigate the effect of replacing corn starch in the standard diet of diabetic hypercholesterolemic rats with 3, 6 and 9% levels of honeydew melon fruit powder, peeled honeydew melon fruit powder and honeydew melon peels powder (melon fractions) on the body weight gain, insulin, organs weight, blood glucose, total cholesterol, LDL, triglyceride, kidney functions, liver functions, catalase enzyme, glutathione S-transferase enzyme and malondialdehyde. Data showed diabetes and hypercholesterolemia caused a significant increase in body weight gain, organs weight, blood glucose, total cholesterol, LDL, triglyceride, kidney functions, liver functions and malondialdehyde as well as a significant decrease in insulin, catalase enzyme and glutathione S-transferase enzyme of rats. The feeding with any level of melon fractions restored the normal liver weight and AST level in diabetic hypercholesterolemic rats to the normal values in negative control rats. Also, feeding with 9% melon fractions restored the normal body weight gain, insulin, kidney weight, heart weight, urea, creatinine, ALT, catalase enzyme, glutathione S-transferase enzyme and malondialdehyde in diabetic hypercholesterolemic rats to the normal values in negative control rats. Although glucose, total cholesterol, LDL, triglyceride, and ALP in diabetic hypercholesterolemic rats were decreased by increasing the melon fraction levels but did not return to normal values in negative control rats.

INTRODUCTION

Hypercholesterolemia is a genetic disease characterized by high low-density lipoprotein cholesterol concentration that increases cardiovascular risk and causes premature death (Nordestgaard et al., 2013). Diabetes mellitus is a metabolic disease characterized by hyperglycemia and hypoinsulinemia or insulin resistance leading to a lot of complications (Deepthi *et al.*, 2017; Lotfy *et al.*, 2017). Oxidative stress plays an essential role in the development of diabetic complications (Asmata *et al.*, 2016). The antioxidants played a protective role against the risk of oxidative stress (Cemek et al., 2008; Forouzani *et al.*, 2013). In general, the prevalence of diabetes mellitus is lower in people with hypercholesterolemia than in the general population (Besselian *et al.*, 2015), suggesting a relationship between glucose and lipid metabolism. Research on natural

products and their anti-hypercholesterolemia and anti-diabetes mellitus has become a major topic of interest in recent years.

Honeydew melon, (*Cucumis melo* L.) belongs to the family Cucurbitaceae and is a creamy yellowish oval shape fruit. Honeydew melon is rich in vitamin C, riboflavin, thiamine, pro-vitamin A, and folic acid (Eitenmiller *et al.,* 1985; Laur and Tian, 2011). The soluble solids, sucrose, total sugars, β-carotene, and 5-methyltetrahydrofolic acid varied in different parts of the fruit (Lester, 2008). In recent years, many studies were done on different plants of this family due to their useful medicinal properties such as analgesic, anti-inflammatory, antioxidant, antiulcer, anticancer, antimicrobial, diuretic, and antidiabetic properties (Ismail *et al.,* 2010; Milind and Kulwant, 2011; Ibrahim and Abd El-Maksoud, 2018; El-Sayed *et al.,* 2021; White1 *et al.,* 2021). To the best of our knowledge, there are no reports on the use of honeydew melon fruit fractions for feeding diabetic hypercholesterolemic rats. Therefore, this study aimed to investigate the effects of replacing corn starch in the standard diet of diabetic hypercholesterolemic rats with 3, 6 and 9% levels of honeydew melon fruit powder, peeled honeydew melon fruit powder and honeydew melon peels powder (melon fractions) on the body weight gain, insulin, organs weight, blood glucose, total cholesterol, LDL, triglyceride, kidney functions, liver functions, catalase enzyme, glutathione S-transferase enzyme and malondialdehyde.

MATERIALS AND METHODS

Fresh honeydew melon fruits (*Cucumis melo* L.) were obtained from a local market in Shibin El-Kom, Egypt. Honeydew melon fruits were cleaned under running tap water. Honeydew melon fruits were cut into halves, the seeds were removed by hand and the pulp was sliced. The peels were taken after scrubbing off the pulpy portion from the peels. Honeydew melon fruit, peeled honeydew melon fruit and honeydew melon peels (melon fractions) were cut into small pieces followed by soaking in 0.1% sodium metabisulphite solution for 30 min. The pieces of honeydew melon fractions were dried at 45°C overnight in an electric draught oven. The dried melon fractions were ground to pass through a 60-mesh sieve using a grinder then put in an airtight container and kept at 5°C until use.

Biological Evaluation:

Experimental Animals:

One Hundred and ten male albino rats (150g ±5) were obtained from the Research Institute of Ophthalmology, Animal House Department, Giza, Egypt. Rats were housed in wire cages under normal laboratory conditions and fed a standard diet for a week as an adaptation period. Diets and water were provided ad-labium and checked daily.

Experimental Groups:

Rats were randomly divided into two main groups, the first, the negative control group (n=10), fed a standard diet according to Reeves *et al.,* (1993) and the second group (n=100) induced for diabetic hypercholesterolemic rats. Hypercholesterolemia was induced in rats by the addition of cholesterol powder, bile salt, and tallow to the standard diet in percentages of 1.5%, 0.25%, and 10%, respectively for about 21 days according to Wu *et al.* (2015). Total cholesterol was determined in 15% of all animals to ensure the induction of hypercholesterolemia. Hypercholesterolemic rats were given a single dose of intraperitoneal injection of 125 mg/kg BW of alloxan dissolved in distilled water after 8-12h fasting (Triastuti *et al.,* 2009). After 72h of alloxan injection, fasting blood glucose level was measured to confirm hyperglycemia. Experimental animals with fasting blood glucose levels over 130 mg/dl were considered diabetic (Sedigheh *et al.,* 2011).The second group (n=100) was divided into 10 subgroups, with 10 rats per subgroup. The first

subgroup is positive control fed a standard diet. Second, third and fourth subgroups fed the standard diet replaced with 3, 6, and 9% of honeydew melon fruit powder. The fifth, sixth and seventh subgroups fed the standard diet replaced with 3, 6, and 9% of peeled honeydew melon fruit powder. The eighth, ninth, and tenth subgroups fed the standard diet replaced with 3, 6, and 9% of honeydew melon peels powder. Melon fraction levels were replaced with corn starch in the standard diet. The experiment period was 30 days.

Blood Sampling:

Blood samples were collected from arteries of the eye and were analyzed for blood glucose at 0, 15, and 30 days and at 0 and 30 days for other analysis. The blood samples were placed in dry clean centrifuge tubes and allowed to clot for 1-2h at room temperature. The serum was then removed by centrifuging at 1500g for 10 min. The clear supernatant serum was kept at - 20°C until analysis (Schermer, 1967).

Biochemical Analysis:

Plasma insulin level was assayed by Enzymatic Linked Immune Sorbent Assay Kit as described by Nakagawa *et al.* (1973). Blood glucose was estimated according to Rojas *et al.,* (1999). Total cholesterol (TC) was determined according to Allain *et al.,* (1974). Low-density lipoprotein (LDL) was calculated according to the methods of Lee and Nieman (1996). Triglycerides (TG) were determined according to Fossati and Prencipe (1982). Urea and creatinine levels were determined according to the method described by Gutmann and Bergmeyer (1974) and Houot (1985), respectively. Alkaline phosphatase (ALP), alanine aminotransferase (ALT), and aspartate aminotransferase (AST) enzymes were measured according to the methods described by Varley *et al.,* (1980), Bergmeyer and Harder (1986), and Kachmar and Moss (1987), respectively. Catalase, glutathione S-transferase and malonaldehyde were determined according to the methods described by Aebi (1974), Mannervik and Gutenberg (1981) and Jentzsch *et al.,* (1996), respectively.

Statistical Analysis:

Data are presented as mean ± SD. Initial weight, final weight, body weight gain and organs weight of normal and diabetic hypercholesterolemic rats were analyzed by one-way analysis of variance. Two-way completely randomized designs were used for other data. An analysis of variance was conducted using Costat version 6.311 (Copyright 1998-2005, CoHort software). When a significant main effect was detected, the means were separated with the Student Newman Keuls test. The predetermined acceptable level of probability was 5% *(*$p \leq 0.05$) for all comparisons.

RESULTS AND DISCUSSION

The initial weight, final weight, and body weight gain of positive control rats were higher ($P \leq 0.05$) than negative control rats. However, the opposite trend was observed for plasma insulin content (Table 1). Alloxan causes diabetes by a partial deterioration of the β cells in the pancreas followed by a reduction in the amount of insulin produced (Ighodaro *et al.,* 2017). Similar body weight gain was reported by El-Sayed et al., (2021) for obese rats. Hosny (2017) reported that rats fed a high-fat diet showed a significant increase in body weight gain. Ibrahim and Abd El-Maksoud (2018) reported that diabetes caused a significant decrease in plasma insulin levels in rats.

Feeding different levels of melon fractions reduced ($P \leq 0.05$) the final weight and body weight gain and increased plasma insulin content of diabetic hypercholesterolemic rats as compared to positive control rats. This effect may be related to the role of melon fractions in reducing body weight gain. A similar decrease in body weight gain was reported by El-Sayed *et al.,* (2021) for obese rats fed citron melon pulp juice, seed powder, and peel powder.

Body weight gain was decreased (P≤0.05) by increasing the level of melon fruit fractions in the diet of diabetic hypercholesterolemic rats. On contrary, the plasma insulin level was increased (P≤0.05) by increasing the level of melon fruit fractions in the diet of diabetic hypercholesterolemic rats. No difference (P>0.05) was observed in plasma insulin content among the same level of melon fractions.

The diabetic hypercholesterolemic rats fed on melon fruit, peeled melon fruit and melon peels at 6% and 9% replacement levels for 30 days led to restoring the normal body weight gain value of negative control rats. However, the diabetic hypercholesterolemic rats fed on melon fruit, peeled melon fruit and melon peels at 9% replacement levels for 30 days led to restoring the normal plasma insulin value of negative control rats.

Table 1: Effect of melon fractions at different levels on body weight gain of normal and diabetic hypercholesterolemic rats.

Groups	Initial weight	Final weight	Body weight gain	Insulin
	(g)		(%)	(ng/ml)
Negative control rats	155.75±8^{e}	183.04±8^{e}	17.52±1de	20.25±1.2^{a}
Positive control rats	237.42±6^{a}	307.17±6^{a}	29.38±2^{a}	4.98±0.6^{f}
Melon fruit				
3%	195.35±3^{d}	238.62±9^{c}	22.15±4^{b}	12.14±1.1^{e}
6%	201.19±8^{cd}	242.33±8^{c}	20.45±3^{c}	17.65±0.8^{cd}
9%	209.82±7^{bc}	243.16±6^{c}	15.89±2^{ef}	20.19±1.8^{a}
Peeled melon fruit				
3%	211.56±6^{bc}	257.24±7^{b}	21.59±2^{b}	11.45±0.7^{e}
6%	217.42±9^{b}	258.75±6^{b}	19.01±4^{d}	16.76±1.5^{cd}
9%	220.91±7^{b}	255.25±8^{b}	15.54±2^{f}	19.35±1.2^{ab}
Melon peels				
3%	193.54±7^{d}	230.85±4^{d}	19.28±1^{c}	10.49±0.9^{e}
6%	196.88±5^{d}	231.47±8^{d}	17.57±3^{d}	15.92±1.2^{d}
9%	202.12±4^{cd}	232.66±3^{d}	15.11±2^{f}	18.26±0.8^{abc}

Means in the same column with different letters are significantly different (p≤ 0.05)

Heart, liver, and kidney weights of positive control rats were higher (P≤0.05) than negative control rats (Table, 2). Kumar *et al.,* (2012) reported that feeding a high-fat diet for 28 days resulted in a significant increase in organ weight. Lecumberri *et al.,* (2007) reported that rats fed high cholesterol diet showed a significant increase in organ weight.

Feeding different levels of melon fractions reduced (P≤0.05) the liver, kidney, and heart weights of diabetic hypercholesterolemic rats as compared to positive control rats. White *et al.,* (2021) reported that liver and kidney weights of diabetic or high-fat mice treated with bitter melon powder were reduced as compared with control mice. Yoon *et al.,* (2017) observed a significant reduction in liver weight for high-fat mice treated with the bitter melon extract.

The diabetic hypercholesterolemic rats fed on melon fractions at any replacement level for 30 days led to restoring the normal liver weight of negative control rats. The diabetic hypercholesterolemic rats fed on melon fractions at a 9% replacement level for 30 days led to restoring the normal heart, and kidney weights of negative control rats.

Table 2: Effect of melon fractions at different levels on organs weight of normal diabetic hypercholesterolemic rats.

Groups	Organ's weight (g)		
	Heart	Liver	Kidney
Negative control rats	0.29±0.0^{g}	3.01±0.3^{b}	0.59±0.1^{ef}
Positive control rats	0.55±0.0^{a}	3.72±0.2^{a}	1.11±0.1^{a}
Melon fruit			
3%	0.42±0.1^{bc}	3.12±0.2^{b}	0.86±0.1^{b}
6%	0.35±0.0^{def}	3.09±0.1^{b}	0.70±0.1^{cd}
9%	0.31±0.0^{fg}	3.00±0.1^{b}	0.58±0.1^{ef}
Peeled melon fruit			
3%	0.46±0.0^{b}	3.18±0.2^{b}	0.92±0.1^{b}
6%	0.38±0.0^{cd}	3.12±0.1^{b}	0.85±0.1^{b}
9%	0.32±0.0^{efg}	3.00±0.1^{b}	0.65±0.1^{de}
Melon peels			
3%	0.36±0.1^{de}	3.07±0.6^{b}	0.73±0.1^{c}
6%	0.34±0.0^{def}	3.05±0.4^{b}	0.70±0.1^{cd}
9%	0.29±0.0^{g}	2.98±0.1^{b}	0.52±0.1^{f}

Means in the same column with different letters are significantly different (p≤ 0.05)

The blood glucose content of positive control rats was higher (P≤0.05) than negative control rats during the experimental period (Table, 3). Similar results were reported by El-Sayed *et al.,* (2021) for obese rats and White *et al.,* (2021) for insulin resistance and type 2 diabetic rats.

Table 3: Effect of melon fractions at different levels on blood glucose of normal diabetic hypercholesterolemic rats

Groups	Experiment period (day)			Means[1]
	0	15	30	
Negative control rats	80.61	81.15	85.13	82.30±1.7^{h}
Positive control rats	298.61	300.34	310.93	303.29±7.5^{a}
Melon fruit				
3%	296.89	205.58	154.71	219.06±5.9^{c}
6%	298.39	178.33	115.82	197.51±5.7^{e}
9%	299.72	151.67	105.33	185.57±3.8^{f}
Peeled melon fruit				
3%	318.33	219.42	175.26	237.67±5.1^{b}
6%	315.00	195.12	152.94	221.02±4.2^{c}
9%	314.67	167.53	126.74	202.98±6.2^{d}
Melon peels				
3%	299.14	170.27	135.82	201.74±6.1^{d}
6%	300.32	152.31	101.56	184.73±5.3^{f}
9%	305.71	137.29	94.12	179.04±7.4^{g}
Means[2]	284.31±5.6^{a}	178.09±4.5^{b}	141.67±4.1^{c}	

[1]Means in the same column with different letters are significantly different (p≤ 0.05)
[2]Means in the same row with different letters are significantly different (p≤ 0.05)

Blood glucose of diabetic hypercholesterolemic rats was decreased (P≤0.05) by increasing melon fraction levels and the experiment periods. The highest reduction in blood glucose content was observed in the rats fed melon peels diets. However, the lowest reduction in blood glucose content was observed in the rats fed peeled melon fruit. El-Sayed *et al.,* (2021) reported that citron melon peels powder at 5% recorded as the best treatment for reducing glucose levels as compared with other groups. White *et al.,* (2021) reported that diabetic rats treated with bitter melon on a 50 g/kg diet have a lower glucose level than other groups.

Although melon fractions at different levels and the experiment periods reduced blood glucose content in the diabetic hypercholesterolemic rats, their values were still higher than in the negative control rats. Treating diabetic hypercholesterolemic rats with melon fraction diets for longer than 30 days may be led to glucose content returning to negative control rat value.

Total cholesterol, LDL, and triglyceride contents of positive control rats were higher (P≤0.05) than negative control rats (Table, 4). Similar results were reported by El-Sayed *et al.,* (2021) for obese rats. Ibrahim and Abd El-Maksoud (2018) reported that diabetes caused a significant increase in the total cholesterol, LDL, and triglyceride levels in rats. During diabetes, insulin deficiency or insulin resistance increases the level of circulating free fatty acids by enhancing the activity of lipase.

Table 4: Effect of melon fractions at different levels on cholesterol and triglycerides of normal diabetic hypercholesterolemic rats.

Melon fractions	Negative control rats	Diabetic hypercholesterolemic rats				Means[1]
		Positive control rats	3%	6%	9%	
TC						
Melon fruit	103.15	255.22	185.12	150.32	132.17	165.19±8b
Peeled melon fruit	103.15	255.22	181.21	165.89	150.58	171.21±9[a]
Melon peels	103.15	255.22	174.87	142.93	125.01	160.24±9[c]
Means[2]	103.15±3[e]	255.22±1[a]	180.40±4[b]	153.05±3[c]	135.92±2[d]	
LDL						
Melon fruit	34.93	183.39	103.42	85.48	69.28	95.30±6b
Peeled melon fruit	34.93	183.39	115.54	106.37	71.52	102.35±4[a]
Melon peels	34.93	183.39	92.71	71.89	59.16	88.42±7[c]
Means[3]	34.93±3[e]	183.39±3[a]	103.89±9[b]	87.91±7[c]	66.65±8[d]	
Triglycerides						
Melon fruit	61.12	165.32	77.14	70.15	63.87	87.52±4b
Peeled melon fruit	61.12	165.32	84.15	74.25	69.30	90.83 ±5[a]
Melon peels	61.12	165.32	70.84	66.11	59.41	84.56±3[c]
Means[4]	61.12±1[e]	165.32±3[a]	77.38±2[b]	70.17±4[c]	64.19±4[d]	

[1]Means in the same column with different letters are significantly different (p≤ 0.05)
[2-4]Means in the same row with different letters are significantly different (p≤ 0.05)

The total cholesterol, LDL, and triglyceride levels were significantly (P≤0.05) decreased by increasing melon fraction levels. The highest reduction in total cholesterol, LDL, and triglyceride levels were observed in the rats fed melon peels diets. However, the lowest reduction in total cholesterol, LDL, and triglyceride levels were observed in the rats fed peeled melon fruit. Data showed that melon fractions could improve lipid metabolism and thus lower complications of coronary heart diseases and atherosclerosis. El-Sayed *et al.,* (2021) reported that citron melon peels powder at 5% recorded as the best treatment for reducing total cholesterol, LDL, and triglyceride levels as compared with other groups. Yoon *et al.,* (2017) reported that LDL and total cholesterol levels in high-fat

diet mice treated with bitter melon extract for 12 weeks were reduced as compared with other groups.

Although melon fractions at different levels reduced total cholesterol, LDL, and triglyceride levels in the diabetic hypercholesterolemic rats, their values were still higher than in the negative control rats. Treating diabetic hypercholesterolemic rats with melon fraction diets for longer than 30 days may be led to total cholesterol, LDL, and triglyceride levels returning to negative control rat values.

Table 5: Effect of melon fractions at different levels on kidney functional of normal diabetic hypercholesterolemic rats

Melon fractions	Negative control rats	Diabetic hypercholesterolemic rats				Means[1]
		Positive control rats	3%	6%	9%	
Urea						
Melon fruit	30.37	48.14	40.81	36.14	31.23	37.34 ±2[a]
Peeled melon fruit	30.37	48.14	41.28	36.96	32.16	37.78±3[a]
Melon peels	30.37	48.14	41.76	37.44	33.12	38.17±2[a]
Means[2]	30.37±3[d]	48.14±2[a]	41.28±1[b]	36.85±1[c]	32.17±2[d]	
Creatinine						
Melon fruit	0.56	0.92	0.82	0.71	0.56	0.71±0.1[a]
Peeled melon fruit	0.56	0.92	0.84	0.73	0.57	0.72±0.1[a]
Melon peels	0.56	0.92	0.83	0.74	0.57	0.72±0.1[a]
Means[3]	0.56±0.1[d]	0.92±0.1[a]	0.83±0.1[b]	0.73±0.1[c]	0.57±0.1[d]	

[1]Means in the same column with different letters are significantly different ($p \leq 0.05$)
[2-3]Means in the same row with different letters are significantly different ($p \leq 0.05$)

Urea and creatinine contents of positive control rats were higher ($P \leq 0.05$) than negative control rats (Table 5). Similar results were reported by Aly-Aldin *et al.,* (2015) for hypercholesterolemic rats. Barakat and Mahmoud (2011) reported that feeding rats with a cholesterol-enriched diet caused a significant increase in serum urea.

Urea and creatinine contents were significantly ($P \leq 0.05$) decreased by increasing the melon fraction levels in the diabetic hypercholesterolemic rat diets. Non-significant ($P > 0.05$) differences in urea and creatinine contents were observed among rats fed on melon fractions. Rats fed 9% melon fractions restored the urea and creatinine contents of negative control rats.

The ALP, ALT, and AST enzymes of positive control rats were higher ($P \leq 0.05$) than negative control rats (Table, 6). Hassan et al., (2015) reported that the high levels of AST, ALT, and ALP in diabetic rats indicate a loss of functional safety of the hepatic cell membranes including hepatocellular damage. Ibrahim and Abd El-Maksoud (2018) reported that ALP, ALT, and AST enzymes in rats were increased by diabetes.

The ALP, ALT, and AST enzymes were significantly ($P \leq 0.05$) decreased by increasing melon fraction levels. The highest reduction in ALP enzyme was observed in the rats fed melon peels diets. However, the lowest reduction in ALP enzyme was observed in the rats fed peeled melon fruit. Non-significant ($P > 0.05$) differences in ALT and AST enzymes were observed among rats fed on melon fractions. Ibrahim and Abd El-Maksoud (2018) reported that diabetic rats treated with *Cucumis melo* var. flexuosus leaf extract caused a significant reduction in the levels of ALP, ALT, and AST enzymes.

Although melon fractions at different levels reduced ALP enzyme levels in the diabetic hypercholesterolemic rats, their values were still higher than in negative control rats. This may be due to the short trial period. Rats fed all melon fractions restored the normal AST enzyme of negative control rats. However, rats fed 9% melon fractions restored the normal ALT enzyme of negative control rats.

Table 6: Effect of melon fractions at different levels on liver functional of normal diabetic hypercholesterolemic rats.

Melon fractions	Negative control rats	Diabetic hypercholesterolemic rats				Means[1]
		Positive control rats	3%	6%	9%	
ALP						
Melon fruit	125.18	211.72	183.44	158.35	132.59	162.27±2[b]
Peeled melon fruit	125.18	211.72	195.48	165.14	142.11	167.93±4[a]
Melon peels	125.18	211.72	165.23	141.87	129.65	154.73±5c
Means[2]	125.18±2[e]	211.72±3[a]	181.38±3[b]	155.12±4[c]	134.78±2[d]	
ALT						
Melon fruit	23.57	53.25	38.14	31.32	23.10	33.88±2[a]
Peeled melon fruit	23.57	53.25	48.09	42.08	32.89	39.98±3[a]
Melon peels	23.57	53.25	43.24	35.37	27.51	36.59±2[a]
Means[3]	23.57±2[d]	53.25±2[a]	43.16±5[b]	36.26±4[c]	27.83±3[d]	
AST						
Melon fruit	47.23	74.55	42.67	32.25	23.32	44.01±2[a]
Peeled melon fruit	47.23	74.55	48.45	37.27	26.29	46.76±3[a]
Melon peels	47.23	74.55	37.08	26.15	21.64	41.33±1[a]
Means[4]	47.23±2[b]	74.55±3[a]	42.73±2[b]	31.89±3[c]	23.75±3[d]	

[1]Means in the same column with different letters are significantly different ($p \leq 0.05$)
[2-4]Means in the same row with different letters are significantly different ($p \leq 0.05$)

Catalase and glutathione S-transferase levels of positive control rats were lower ($P \leq 0.05$) than negative control rats. However, malondialdehyde level of positive control rats was higher ($P \leq 0.05$) than negative control rats (Table 7). Similar results were reported by Ibrahim and Abd El-Maksoud (2018) for diabetic rats treated with *Cucumis melo* var. flexuosus leaf extract.

Table 7: Effect of melon fractions at different levels on catalase enzyme, glutathione S-transferase and malondialdehyde of normal and diabetic hypercholesterolemic rats

Melon fractions	Negative control rats	Diabetic hypercholesterolemic rats				Means[1]
		Positive control rats	3%	6%	9%	
Catalase enzyme						
Melon fruit	115.23	59.86	84.28	95.98	110.85	93.24±3[a]
Peeled melon fruit	115.23	59.86	92.54	105.47	115.55	97.73±3[a]
Melon peels	115.23	59.86	87.32	99.74	113.19	95.07±2[a]
Means[2]	115.23±6[a]	59.86±3[d]	89.93±2[c]	100.40±3[b]	113.20±2[a]	
Glutathione S-transferase						
Melon fruit	4.58	1.84	2.96	3.79	4.51	3.54±0.2[a]
Peeled melon fruit	4.58	1.84	2.65	3.45	4.37	3.38±0.2[a]
Melon peels	4.58	1.84	2.47	3.26	4.19	3.27±0.1[a]
Means[3]	4.58±0.3[a]	1.84±0.1[d]	2.69±0.1[c]	3.50±0.2[b]	4.36±0.1[a]	
Malondialdehyde						
Melon fruit	73.48	195.28	110.87	85.95	72.21	111.90±2[b]
Peeled melon fruit	73.48	195.28	141.34	101.42	80.86	118.48±2[a]
Melon peels	73.48	195.28	122.71	92.91	75.12	107.56±3[c]
Means[4]	73.48±3[d]	195.28±4[a]	124.97±3[b]	93.43±3[c]	76.06±2[d]	

[1]Means in the same column with different letters are significantly different ($p \leq 0.05$)
[2-4]Means in the same row with different letters are significantly different ($p \leq 0.05$)

The reduction in catalase and glutathione S-transferase activities may be attributed to the exhaustion of the enzymes because of oxidative stress induced by the

alloxan or the inactivation of the enzyme protein in the lipid peroxides (Hadrami and Al-Khayri, 2012). On the other hand, the increase in malondialdehyde value may be related to the destruction of erythrocytic membranes and tissues, as an effect of oxidative stress (Aluwong *et al.*, 2016).

Catalase and glutathione S-transferase activities were significantly ($P \leq 0.05$) increased by increasing the melon fraction levels in the diabetic hypercholesterolemic rat diets. However, the malondialdehyde value was significantly ($P \leq 0.05$) decreased by increasing the melon fraction levels. Ibrahim and Abd El-Maksoud (2018) reported that diabetic rats treated with *Cucumis melo* var. flexuosus leaf extract significantly raised catalase enzyme activity and lowered malondialdehyde value.

Non-significant ($P > 0.05$) differences in catalase and glutathione S-transferase activities were observed among rats fed melon fractions. However, malondialdehyde values were affected ($P \leq 0.05$) by feeding melon fractions. The highest reduction in malondialdehyde value was observed in the rats fed melon peels diets. The lowest reduction in malondialdehyde value was observed in the rats fed peeled melon fruit. Rats fed 9% melon fractions restored the normal catalase enzyme, glutathione S-transferase enzyme and malondialdehyde value of negative control rats.

Conclusion

From the above results, it could be concluded that feeding rats with different levels of melon fractions reduced body weight gain, organ weight, blood glucose, total cholesterol, LDL, triglyceride, kidney functions, liver functions and malondialdehyde as well as increased insulin, catalase enzyme and glutathione S-transferase of diabetic hypercholesterolemic rats.

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ARABIC SUMMARY

تأثير مكونات شمام كوز العسل على الفئران المصابة بالسكرى وإرتفاع الكوليسترول

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هدفت الدراسة إلى معرفة تأثير استبدال نشا الذرة في الوجبة القياسية للفئران المصابة بالسكرى وارتفاع مستوى الكوليسترول بمستويات 3 و 6 و 9٪ من مسحوق فاكهة شمام كوز العسل، مسحوق شمام كوز العسل المقشر، ومسحوق قشور شمام كوز العسل (مكونات الشمام) على وزن الفئران، مستوى الأنسولين، وزن الأعضاء، مستوى الكوليسترول الكلى، والكوليسترول منخفض الكثافة LDL، الجلسريدات الثلاثية، وظائف الكبد والكلى، إنزيم الكتاليز والجلوتاثيون، والمالونالدهيد. أوضحت النتائج زيادة معنوية فى وزن الأعضاء، مستوى الجلوكوز، مستوى الكوليسترول الكلى، الكوليسترول منخفض الكثافة، الجلسريدات الثلاثية، وظائف الكبد والكلى، والمالونالدهيد. بينما حدث إنخفاض معنوى فى مستوى الأنسولين، وإنزيمات الكتاليز والجلوتاثيون للفئران. أدت التغذية بأى مستوى من (مكونات الشمام) إلى إستعادة الوزن الطبيعى للكبد وكذلك مستوى إنزيم AST فى الفئران المصابة بالسكرى وإرتفاع مستوى الكوليسترول إلى القيم الطبيعية بالمقارنة بمجموعة الكنترول السالبة (-). أيضاً أدت التغذية بمستوى إستبدال 9٪ من (مكونات الشمام) إلى إستعادة الوزن الطبيعى للجسم، مستوى الأنسولين، وزن الأعضاء (القلب - الكلى)، اليوريا، الكرياتينين، وإنزيمات (ALT - الكتاليز - الجلوتاثيون)، والمالونالدهيد إلى القيم الطبيعية بالمقارنة بمجموعة الكنترول السالبة. وبالرغم من إنخفاض مستوى الجلوكوز، الكوليسترول الكلى، الكوليسترول منخفض الكثافة LDL، الجلسريدات الثلاثية، وإنزيم ALP فى مجموعة الفئران المصابة بالسكرى وارتفاع الكوليسترول، إلا أنها لم تعد إلى القيمة الطبيعية عند مقارنتها بمجموعة الكنترول السالبة.