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EFFECT OF FEEDING AT DIFFERENT LEVELS OF CHROMIUM PICOLINATE AND MAGNESIUM SULFATE ON DIABETIC RATS.

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ABSTRACT

This investigation was carried out to study the hypoglycemic effects of Chromium picolinate and Magnesium sulfate on streptozotocin induced diabetic rats. Chromium at levels of 8 and 10ug/ml, Magnesium at level 10 and 12mg/ml. Furthermore, mixture of (chromium 8u/ml + Magnesium10mg) and (chromium10u/ml + Magnesium12mg/ml) administered to diabetic rats for six weeks. Blood glucose and, body weight gain of rats were determined. Also, total cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), and triglycerides were determined in the serum of the rats. As well as the activities of serum aspartate transaminase (AST) and alanine transaminase (ALT) were determined.

The results indicated that Chromium picolinate and Magnesium sulfate led to a significant reduction in weight gain and blood glucose of diabetic rats. In addition, total cholesterol, LDL-C, triglycerides, creatinine and urea in serum decreased while HDL-C increased after administration Chromium picolinate and Magnesium sulfate.

The activities of hepatic markers were significantly decreased in GPT, but GOT no change elevated in diabetic rats as compared to control rats.

Finally, it can be concluded that, using different levels of Chromium picolinate, Magnesium sulfate and mixture of them have pronounced effect for lowering blood glucose and cholesterol levels of the serum in experimental diabetic rats.

INTRODUCTION

Diabetes mellitus is a chronic disease disorder of glucose Intolerance. It is characterized by high blood glucose level and glucosuria from dysfunction of pancreatic cells and insulin resistance. The defective cells results in lack of total or partial synthesis of insulin. The resistance is caused by cell membrane where glucose is not transported to the cell for oxidation. As glucose is not metabolized. High amount of glucose is circulating in the blood (hyperglycemia).

To keep the normal level of glucose in the blood, the kidney removes the extra sugar from the blood and excretes it in the urine (glycosuria). Because glucose is not utilized by the body cells, the body is under constant impression of hunger and that is why diabetes feels increased appetite (polyphagia) and eats more frequently (Safadar *et al.,* 2006).

Diabetes mellitus is one of the most common chronic diseases in children and adolescents (Kelly *et al.,* 2006 and Ogden *et al.,* 2002).

Diabetes mellitus is considered to be a major health problem in the world. The number of diabetics has been increasing at a rate of around 6%per year.

A major goal of dietary and drug is based on management of diabetes mellitus to achieve normal control of glucose metabolism and glycemia, thereby hopefully prevent macro and micro-vascular complication. So, modification of the diets is considered the most important factor in the therapeutic plan especially for diabetic patients with type 1 (insulin Dependent Diabetes Mellitus) and for some diabetic patients with type 2 (Non- insulin Dependent Diabetes Mellitus), besides, it is the only intervention that needed to control the metabolic abnormalities associated with the disease (wolever *et al.,* 1990). Furthermore, ChandraMohan *et al.,* (2008).

Declared the importance of maintaining the level of glucose at normal level for the diabetics to avoid episodes of hyperglycemia that might contribute to the risk of several, late chronic complication.

As reported by (WHO, 2000), in Egypt there are about 5 million diabetics. The number of diabetics has been increasing at rate the incidence of diabetes can be expect to double every 15 years.

Chromium is an essential nutrient involved in the metabolism of glucose and lipids. Suboptimal dietary intake of Cr is associated with diabetes and cardiovascular diseases (Anderson, 1998).

Kobrin and Goldfarb, (1990). reported that, Magnesium plays an important role in carbohydrate metabolism. It may influence the release and activity of the hormones that helps control blood glucose levels.

The present investigation was undertaken to study the effect of chromium and magnesium on changes on blood sugar level, some organ weight, serum and liver lipid parameters of diabetic rats.

MATERIALS AND METHODS

Materials:-

Chemicals:

Magnesium Sulfate, chromium picolinate, casein, vitamins mixture, Cellulose, salts mixture and streptozotocin (STZ), were purchased from El-Gomhoria Company for Drugs and Chemical, Cairo, Egypt.

Animals:

Adult male Wistar rats weighing 200-205g were obtained from Food Technology Research Institute Agric. Research center, Giza, Egypt.

Reagant methodology kites:

Sugar, total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, urea, creatinine and transaminases (G.O.T and G.P.T) kits were obtained from Boehringer Mannheim GMBH, Germany.

Methods:

Experimental animals:

Animals were housed individually in stainless steel cages and maintained at 24±20 °C and 12 hr light dark cycle. Rats were feed on basal diet for one week to acclimate them to our facility and basal diet. Basal diet containing casein 20%, cane sugar 10%, corn starch 50%, corn oil 10%, vitamin mixture 1% ,Cellulose 5% and salt mixture 4% as reported by Helmy, (2006).

Induction of rats:

Experimental rats were induced by a single intraperitoneal injection of streptozotocin to animals fasted overnight at a dose of 60 mg/kg body weight (1ml fresh solution in 0.1M citrate buffer, pH 4.5) and control rats were injected with the citrate buffer alone. The rats had free access to basal diet and water (Babu and Srinivasan 1997). After one week, diabetic rats with blood glucose concentration more than 200 mg/dl were selected for the study. The normal blood glucose level of rats ranged from 50 to135 mg/dl (Arun and Nalini, 2002)

Experimental Design:

Forty male Wistar rats were divided randomly into eight groups of five rats each (n = 5) after the induction of streptozotocin diabetes according to the following scheme:

Group1: normal control (untreated rats).

Group 2: diabetic control rats.

Group 3: Diabetic rats given basal diet + chromium picolinate (8µg/ml) in drinking water for six weeks.

Group 4: Diabetic rats given basal diet + chromium picolinate (10µg/ml) in drinking water for six weeks.

Group 5: Diabetic rats given basal died + magnesium sulfate (10g/l) in drinking water for six weeks.

Group 6: Diabetic rats given basal died + magnesium sulfate (12g/l) in drinking water for six weeks.

Group 7: Diabetic rats given basal diet + chromium picolinate (8µg/ml) + magnesium sulfate (10g/l) in drinking water.

Group 8: Diabetic rats given basal diet + chromium picolinate (10µg/ml) + magnesium sulfate (12g/l) in drinking water.

Blood sampling:

Blood samples were taken from the previously mentioned groups at the end of the experiment. The blood samples were collected after 12 hours fasting from vein plexus eye into dry clean centrifuge tubes and left to colt. The blood was centrifuged for 10 min at 300 rpm to separate the serum, which was carefully aspirated and transferred into clean quite plastic tubes and kept at frozen condition at -18 ± 2 °C until biochemical analysis (El-Khamissy, 2005).

Collection of organs:

All rats were scarified and the abdomen was offend and the organs were separated by carefully dissection then cleaned from the adhesive matter and washed with running water after that weighted and kept in the freezer at -18 ± 2 °C until biochemical analysis.

Biological analysis:

Body weight gain and relative weight of organs:

All rats were weighed weekly so as food intake. At the end of the experiment, body weight gain was calculated for each group of rats Weighted and recorded every week. Body weight gain percent (B.W.G) was determined

according to the method of Chapman *et al.,* (1959) using the following equation: $$BWG\% = \frac{\text{Final body weight - initial body weight}}{\textit{Initial} \text{ body weight}} x100 \cdot$$

Determination of blood glucose:

Blood glucose was measured according to the method described by Alles *et al.,* (1999) using blood glucose meter (free style TM).

Determination of serum enzymatic activity:

The activities of aspartate transaminase (AST) and alanine transaminase (ALT) of serum were determined according to the methods described by Reitman and Frankel (1957) on fully automated chemistry analyzer Roche/Hitachi-912 (Roche Diagnostics, Mannheim, Germany) using Roche Diagnostics GmbH kits. The values were expressed as *Iu/L* serum.

Determination of serum lipids:

Triglycerides, total cholesterol and high density lipoprotein cholesterol (HDL-C) levels were measured by enzymic-colorimetric procedures using commercial available kits. Triglycerides were carried out according to the method of Fossati and Prancipe (1982). Total cholesterol (TC) and HDL-C were carried out according to the methods of Richmond (1973). Low-density lipoprotein cholesterol (LDL-C) was calculated as the difference between total and HD-C according to the method of Friedewald *et al.,* (1972).

Determination of kidneys functions:

Urea was determined by using a commercial kit (Biomed Company, Germany). According to the method described by (Chaney and Marbach, 1962).

Creatinine concentrations in the plasma were determined using enzymatic colorimetric kit (Biolabo , Maizy, France) according to the method described by (Fabiny and Ertingshausen ,1971), based on the colorimetric reaction of creatinine with alkaline picrate measured at 490 nm.

Statistical analysis:

Most of the received data were analyzed statistically using the analysis of variance and the means were further tested using the least significant difference test (LSD) as outlined by Steel and Torrie (1980).

RESULTS AND DISCUSSION

Effect of feeding at different levels of Chromium picolinate and Magnesium sulfate on Final body weight (g) and Change body weight (g) gain in rats:

Data in Table (1) indicate that the mean values of initial body weights of all groups at the start of the experiment were approximately the same and ranged from 201.5 to 205.0 g. At the end of experiment (6 weeks), the final weight of the control diabetic rats (G2) was lower than that of the normal control (G1) and all diabetic rats, All diabetic rats gave negative Change body weight ranged from (-17g to -7 g). These results are in agreement with those reported by Krol *et al.,* (2010) and Tuzcu *et al.,* (2011).

Table (1) Effect of feeding at different levels of Chromium picolinate and Magnesium sulfate on Change body weight (g) in rats for 6 weeks.

Treatment	Initial body weight (g)	Final body weight (g)	Change body weight (g)
G1	201.5 ± 0.50 a	237.0 ± 1.00 e	+ 35.5 ± 0.50 f
G2	202.0 ± 1.00 a	185.0 ± 1.00 a	- 17.0 ± 0.50 e
G3	202.5 ± 0.86 a	193.66 ± 0.57 c	- 8.84 ± 0.50 b
G4	204.0 ± 1.00 a	197.0 ± 1.00 d	- 7.0 ± 0.50 a
G5	203.0 ± 1.00 a	190.0 ± 1.00 b	- 13.0 ± 0.50 d
G6	204.5 ± 0.50 a	193.5 ± 0.50 c	- 11.0 ± 0.50 c
G7	203.5 ± 0.50 a	190.66 ± 0.57 b	- 12.84 ± 0.04 d
G8	205.0 ± 1.00 a	195.0 ± 1.00 c	- 10.0 ± 0.50 b

Each value is an average of five determinations.
Values followed by the same letter in column are not significantly different at P ≤ 0.05.
G1: normal control (untreated rats).
G 2: diabetic control rats.
G3: Rats fed on basal diet + chromium picolinate (8µg/ml) in drinking water
G4: Rats fed on basal diet + chromium picolinate (10µg/ml) in drinking water
G5: Rats fed on basal died + magnesium sulfate (10g/l) in drinking water
G6: Rats fed on basal died + magnesium sulfate (12g/l) in drinking water
G7: Rats fed on basal diet + chromium picolinate (8µg/ml) + magnesium sulfate (10g/l) in drinking water
G8: Rats fed on basal diet + chromium picolinate (10µg/ml) + magnesium sulfate (12g/l) in drinking water

Effect of feeding at different levels of Chromium picolinate and Magnesium sulfate on the organs weight and relative organs weight in rats:

Liver, kidney, heart and pancreas of rats fed on basal diet and other treatments, as well, were weight at the end of experimental period (6 weeks) and the ratio of each organ to final body weight of rats was calculated. The results presented in Table (2) revealed that all treatments showed no significant changes in the weight of kidney and Pancreas of all experimental rats. On the other hand, the liver of positive control group had the highest liver weight (9.33 gm) and relatively liver weight (5.043) between all groups. This may be due to high blood glucose.

Whereas negative control G1 and G6 had the lowest liver weight and relatively liver weight. This may be referring of negative control and G6 may be due to treatment. Furthermore, , On the other hand, the heart weights of rats fed with basal diet + chromium picolinate (8ug/ml) + magnesium sulfate (10g/l) in drinking water G7 was higher than that of control rats.

Table (2): Effect of feeding at different levels of Chromium picolinate and Magnesium sulfate on the organs weight and relative organs weight in rats for 6 weeks.

Dietary groups	Final body weight (g)	Liver		Kidney		Heart		Pancreas	
		g	R.O.W.* %	g	R.O.W.* %	g	R.O.W.* %	g	R.O.W.* %
G1	237.00 ± 1.00 e	5.82 ± 0.61 a	2.456	1.57 ± 0.28 a	0.662	0.66 ± 0.05 a	0.278	0.09 ± 0.05 a	0.038
G2	185.00 ± 1.00 a	9.33 ± 2.62 b	5.043	1.71 ± 0.14 a	0.924	0.90 ± 0.21 ab	0.486	0.23 ± 0.12 a	0.124
G3	193.66 ± 0.57 c	8.26 ± 0.45 b	4.267	1.98 ± 0.06 a	0.868	0.93 ± 0.10 b	0.480	0.21 ± 0.06 a	0.108
G4	197.00 ± 1.00 d	8.22 ± 0.23 b	4.173	1.94 ± 0.9 a	0.985	0.89 ± 0.11 b	0.452	0.20 ± 0.8 a	0.102
G5	190.00 ± 1.00 b	6.93 ± 0.40 ab	3.647	1.80 ± 0.52 a	0.947	0.84 ± 0.14 ab	0.442	0.24 ± 0.15 a	0.126
G6	193.50 ± 0.50 c	6.99 ± 0.34 ab	3.612	1.75 ± 0.43a	0.904	0.82 ± 0.21 ab	0.424	0.22 ± 0.12 a	0.114
G7	190.66 ± 0.57 b	9.08 ± 0.09 b	4.762	1.77 ± 0.12 a	0.928	1.00 ± 0.04 b	0.525	0.21 ± 0.12 a	0.110
G8	195.00 ± 1.00 c	9.00 ± 0.4 b	4.615	1.82 ± 0.17 a	0.933	0.98 ± 0.6 b	0.503	0.24 ± 0.14 a	0.123

Each value was an average of five determinations.
Values followed by the same letter in column are not significantly different at P ≤ 0.05.
*** Relative organ weight (R.O.W.) = organ weight ÷ Final body weight × 100**
G1, G2, G3 G4 …..etc as in Table (1).

Effect of different levels of Chromium picolinate and Magnesium sulfate on serum lipids in rats:

Although the relationship between lipids abnormalities and diabetes is complex, there is usually a specific lipid abnormality found in diabetes (Rosalyn and Bauman, 1983). Also, hypertriglyceridemia, hypercholesrolemia and reduced HDL-C levels were commonly seen in diabetes. The abnormal high level of lipids in diabetes is mainly due to the increase in the mobilization of free fatty acids from the peripheral depots, since the insulin inhibits the hormone sensitive lipase but glucagons, catecholamines and other hormones enhance lipolysis. The marked hyperlipidemia that characterizes the diabetic state may therefore be regarded as a consequence of the fat depots (Al-Shamaony *et al.,* 1994). According to the results given in Table (3), it could be concluded that treated diabetic rats had significantly lower serum total cholesterol, low density lipoprotein cholesterol and triglycerides but they had a significantly higher serum high density lipoprotein cholesterol compared to diabetic control rats.

These results were consistent with those reported by (Anderson, 1992; Fox *et. al.,* 2001 and El-Sayed, 2013). The ratio of TC/HDL-C was significantly higher in case of diabetic control rats than that of other groups. These results may be due to the treatment of diabetic rats with streptozotocin helped to increase of TC/HDL-C ratio. These results are in a harmony with those reported by Katan *et al.,* (1994) they reported that total cholesterol is not as useful a predictor of coronary heart diseases risk as the relative distribution of cholesterol among lipoprotein e.g.

TC/HDL-C and LDL-C/HDL-C ratios. The increasing of triglycerides in streptozotocin diabetes rats that observed in this study may be due to lack of insulin, which normally activates the enzyme lipoprotein lipase. (Baur 1995) stated that the TC/HDL-C ratio should be ranged between 4 and 6 and when it increased above 6 is high risk on heart. The TC/HDL-C ratio is important as an indicator of the coronary artery disease. Hypertriglycermia is one of the risk factors in coronary artery disease and diabetes mellitus is always associated with raised triglycerides.

The same table, showed that, supplementation of drinking water with all type of chromium picolinate led to improvement the TC/HDL-C and TC/LDL-C ratios. Chromium picolinate at a level (8 µg/ml and 10 µg/ml) also recorded the best and nearest of TC/HDL-C, TC/LDL-C and LDL-C/HDL-C to the negative control and comparing with other group, the mean value were (1.75 and 1.67), (3.67 and 3.99) and (0.48 and 0.42) respectively. These values were significantly different comparing with that recorded in positive control. Sayed-Ahmed (2002) supports our findings.

Table (3): Effect of feeding with different levels of Chromium picolinate and Magnesium sulfate on serum lipid parameters in rats.

Dietary groups	Total cholesterol mg/dl	HDL-C mg/dl	LDL-C mg/dl	TC/HDL-C ratio	TC/LDL-C ratio	LDL-C/HDL-C ratio	Total triglyceride mg/dl
G1	131.66 ± 2.13 c	59.66 ± 0.57 b	54.00 ± 1.73 f	2.21	2.44	0.90	90.00 ± 1.00 a
G2	250.53 ± 1.00 e	40.00 ± 1.00 a	181.00 ± 1.00 g	6.26	1.38	4.53	147.66± 0.57 d
G3	121.26 ± 0.64 a	69.33 ± 0.57 c	33.00 ± 0.00 b	1.75	3.67	0.48	94.66 ± 0.57 c
G4	122.46 ± 0.51 a	73.33± 0.57 d	30.66 ± 0.57 a	1.67	3.99	0.42	92.33 ± 0.57 b
G5	132.00 ± 2.07 c	70.33 ± 1.52 c	42.66 ± 0.57 d	1.88	3.09	0.61	95.00 ± 1.73 c
G6	128.93 ± 0.98 b	72.00 ± 1.00 d	38.66 ± 0.57 c	1.79	3.33	0.54	91.33 ± 0.57 ab
G7	134.53 ± 0.50 d	70.00 ± 1.00 c	45.66 ± 0.57 e	1.92	2.95	0.65	94.33 ± 0.57c
G8	131.86 ± 0.48 c	74.00 ± 1.00 e	39.33 ± 0.57 c	1.78	3.35	0.53	92.66 ± 0.57 b

Each value was an average of five determinations ± standard error.
Values followed b the same letter in column are not significantly different at P ≤ 0.05.
G1, G2, G3 G4 …..etc as in Table (1).
Normal values in human should be in the range of:
Total cholesterol (below 200 mg/dl) HDL-C (above 45 mg/dl)
Total triglyceride (50 – 250 mg/dl) LDL-C (< 160 mg/dl) (Baur, 1995)

Effect of feeding with different levels of chromium picolinate and magnesium sulfate on serum alanine aminotransferase and aspertate aminotransferase activities in rats.

Elevated activities of serum transaminase enzymes are a common sing of hepatic dysfunction, and are more frequently observed among people with diabetes than in the general population. Furthermore, diabetic complications

such as limited joint mobility, retinopathy and neuropathy are associated with liver enzyme activities, independent of alcohol consumption, body mass index and metabolic control of diabetes (Brownlee, 2001). Table (4) represents the effect of different levels of Chromium picolinate and Magnesium sulfate on changes in the activities of serum aspertate transaminase and alanine transaminase.

The activities of hepatic markers were significantly elevated in diabetic rats compared with control rats. The treatment of diabetic rats Chromium and Magnesium reversed the above changes in a significant manner compared with untreated diabetic rats. These results are in the same trend of those reported elsewhere. (Naveen and Farhath, 2012) .Several investigators reported increase in aspertate and alanine transaminase in the liver and serum of streptozotocin diabetic rats (Brownlee, 2001). The changes in levels of serum enzymes are directly related to the changes in metabolism of its involved enzymes. Murugan and Pari (2007) suggested that liver and kidney functions are highly altered in diabetic state. Treatment with Chromium picolinate and Magnesium sulfate reversed these changes in diabetic rats, which indicates that these substrates protect the hepatic and renal function in the diabetic condition.

Table (4): Effect of feeding with different levels of chromium picolinate and magnesium sulfate on serum alanine aminotransferase (ALT) and aspertate aminotransferase (AST) activities in rats.

Dietary groups	ALT (IU/L)	AST (IU/L)	AST/ALT ratio
G1	14.00 ± 1.00 d	23.33 ± 0.57 a	1.67
G2	16.33 ± 1.15 e	39.66 ± 0.57 d	2.43
G3	12.33 ± 0.57 c	27.66 ± 0.57 c	2.24
G4	10.00 ± 1.00 a	24.33 ± 0.57 a	2.43
G5	12.00 ± 1.00 bc	28.33 ± 0.57 c	2.36
G6	11.33±0.57 abc	28.00 ± 1.00 c	2.47
G7	12.66 ± 0.57 cd	27.66 ± 0.57 c	2.18
G8	10.66 ± 0.57 ab	25.66 ± 0.57 b	2.41

Each value was an average of five determinations ± standard error.
Values followed b the same letter in column are not significantly different at P ≤ 0.05.
G1, G2, G3 G4 …..etc as in Table (1).

Effect of feeding with different levels of Chromium picolinate and Magnesium sulfate on blood glucose in rats.

Table (5) illustrated the mean blood glucose level of normal control and diabetic groups through the experimental periods. Blood glucose levels of diabetic groups were markedly higher than the normal control (G1), data dealing with this case that given in the same table clarified that, also different levels of Chromium picolinate and Magnesium sulfate led to cause a significant decreased in blood glucose level of the diabetic groups (G3, G4, G5, G6, G7 and G8), comparing with the diabetic group fed on control diets (G2). Reduction was observed after three week of feeding till the end of experiment periods; also, the reduction was increased with increasing the feeding period. Apparent also from the same table that, rats fed on basal diet

+ chromium picolinate (10ug/ml) + magnesium sulfate (12g/l) in drinking water, (G8) led to a more reduction of blood glucose level comparing with diabetic rats.

The results were in a good agreement with those many authors Cefalu and Hu (2004) revealed that, low serum chromium concentrations can predict the development of type 2 diabetes mellitus . Furthermore, Albarracin *et al*., (2008), they reported that,, supplementation of relatively high dosages of Cr picolinate (Cr Pic) decreased postprandial blood glucose.

Alyssa *et al.*, (2009) suggested that chromium enhances insulin internalization, insulin receptor number, and β-cell sensitivity.

However, Olatunji *et al.,* (2008) have recently reported that dietary magnesium supplementation significantly improved the impaired glucose tolerance.

Table (5): Effect of feeding with different levels of Chromium picolinate and Magnesium sulfate on blood glucose (mg/dl) in rats.

Dietary groups	Blood glucose (mg/dl)		
	Initial time	After 3 weeks	After 6 weeks
G1	88.00 ± 1.0 A a	90.00 ± 1.0 A a	88.00 ± 1.00 A a
G2	217.00 ± 2.0 BCD a	223.0 ± 1.73 F b	230.00 ± 1.0 Fc
G3	220.00 ± 1.0 EF c	168.0 ± 1.00 D b	140.00 ± 1.0 Ca
G4	221.00 ± 1.0 F c	166.0 ± 1.0 CD b	135.00 ± 1.0 Ba
G5	218 ± 1.73 CDE c	175.0 ± 1.73 E b	148.00 ±1.0 Ea
G6	219.0 ± 1.0 DEF c	174.00 ± 1.00 Eb	145.00 ± 1.0 Da
G7	216.0 ± 1.0 BC c	164.0 ± 1.00 C b	141.00 ± 1.0 Ca
G8	215.00 ± 1.00 B c	160.0 ± 1.0 Bb	134.00 ± 1.0 Ba

Each value is an average of five determinations ± s.d
Means with different superscript capital letters (between groups at the same period "column") and small letters (within groups at different period "row ") are significantly different at p <0.05
G1, G2, G3 G4 …..etc as in Table (1).

Effect of feeding on different levels of Chromium picolinate and Magnesium sulfate on serum creatinine (mg/dl) in rats:

The change in serum creatinine level during experimental period is shown in Table (6). At the beginning of the experimental period, serum creatinine recorded values of all groups were ranged between 1.0 to 2.3 mg/dl. And this range above of level of serum creatinine in normal value (0.6 to 1.4- mg/dl). But negative control had the lowest significant level of serum creatinine (1.0 mg/dl) and positive Control recorded the highest (2.4mg/dl). At the end of experimental period , from this table it was observed that diabetic control had the highest amount of creatinine contents (2.90 mg/dl),while the lowest creatinine contents from hypoglycemic diets G4 and G8 were 0.41and 0.55 mg/dl, respectively.

The results agree with (Alam, 2001) found that Streptozotocin injection caused a highly significant increase in serum uric acid, blood urea and creatinine relative to normal control. Serum uric acid was 6.0 mg/dl for diabetic control 1.6 mg/dl for normal control and urea was more than average values of the normal control 68.5 and 58.55 mg/dl, respectively.

Table (6): Effect of feeding on different levels of Chromium picolinate and Magnesium sulfate on serum creatinine (mg/dl) in rats:

Dietary groups	serum creatinine (mg/dl)		
	Initial time	After 3 weeks	After 6 weeks
G1	1.00 ± 0.10 Aa	1.20 ± 0.10 Da	1.10 ± 0.10 Da
G2	2.40 ± 0.10 Fa	2.60 ± 0.10 Eb	2.90 ± 0.10 Ec
G3	2.30 ± 0.10 EFc	0.90 ± 0.10 Cb	0.63 ± 0.05 BCa
G4	1.90 ± 0.10 Bc	0.64 ± 0.01 Ab	0.41± 0.01 Aa
G5	2.20 ± 0.10 Dec	0.80 ± 0.10 BCab	0.66 ± 0.05 Ca
G6	2.00 ± 0.10 BCc	0.71 ± 0.01 ABb	0.59 ± 0.01 BCa
G7	2.10 ± 0.11 CDc	0.80 ± 0.00 BCb	0.6 ± 0.00 BCa
G8	2.30 ± 0.01 EFc	0.78 ± 0.01 BCb	0.55 ± 0.01 Ba

Each value is an average of five determinations ± s.d
Means with different superscript capital letters (between groups at the same period "column") and small letters (within groups at different period "row ") are significantly different at p <0.05
G1, G2, G3 G4 …..etc as in Table (1).

Effect of feeding on different levels of Chromium picolinate and Magnesium sulfate on serum urea (mg/dl) in rats:

The results of urea, in plasma of normal control and diabetic rats, at the experimental period after feeding for 6 weeks are reported in Table (7) .The obtained results illustrated that at the end of experimental period for the control was 41.00mg/dl. The same table presented that urea contents of diabetic control showed a value of 63.00mg/dl in plasma, while the diabetic rats fed on different levels of Chromium picolinate and Magnesium sulfate had significantly lower serum urea compared to diabetic control fed on basal diet. Mean while, normal group fed on basal diet had a significantly lower mean value for urea. The obtained results are in agreed with those reported by Mita *et al.,* (2004).

Table (7): Effect of feeding on different levels of Chromium picolinate and Magnesium sulfate on serum urea (mg/dl) in rats:

Dietary groups	serum urea (mg/dl)		
	Initial time	After 3 weeks	After 6 weeks
G1	42.00 ± 1.00Aa	40.33 ± 0.57Aa	41.00 ± 1.00Aa
G2	60.00 ±1.0CDa	62.00 ± 1.00Db	63.00 ± 1.00Ec
G3	61.30 ± 0.10Dc	52.33 ± 0.57Cb	43.33 ± 0.57Da
G4	60.33 ±1.52CDc	52.00 ± 1.00BCb	41.66 ± 0.57ABa
G5	61.00 ± 1.00Dc	51.33. ± 0.57BCb	42.40 ±0.10BCDa
G6	59.66 ± 0.57CDc	51.00 ± 1.00BCb	42.5 ± 0.10BCDa
G7	58.00 ± 1.00Bc	51.66 ± 0.57BCb	43.00 ± 1.00CDa
G8	59.20 ± 0.10BCc	50.70 ± 0.10Bb	41.80 ± 0.1ABCa

Each value is an average of five determinations ± s.d
Means with different superscript capital letters (between groups at the same period "column") and small letters (within groups at different period "row ") are significantly different at p <0.05
G1, G2, G3 G4 …..etc as in Table (1).

REFERANCES

Alam, S.O. (2001). Biochemical studies on chickpea (Cicer *arictinum* L.) ultized in African food product. ph. D. Thesis, Fac, Agric., Cairo Univ., Egypt.

Albarracin, C.; Fuqua, B. and Evans, J. L. (2008). Chromium Picolinate and biotin combination improves glucose metabolism in treated, uncontrolled overweight to obese patients with type 2 diabetes. Diab Metab Res Rev. 24:41–51.

Alles, S. M.; Ross, M. N.; Blax, C. j.; Lisdonk, E.; Zock, L. P. and Hautvast, G. A. J. (1999). Composition of fructo lipids Oligosaccharides have favorably affect blood glucose and serum Concentration in patients with type-2diabetes. Am. J. Clin.

Al-Shamaony, L., Al-Khazraji, S. M., and Twaij, H. A. (1994). Hypoglycemic effect of Artemisia herba Alba II. Effect of a Valuable extract on some blood parameters in diabetic animals. J. thnopharmacol, 43: 167-171.

Alyssa, E. M.; Bahjri, S. M.; Ahmed W. H. and Al-Ama N. and Ferns, G. A. (2009). Chromium status and glucose tolerance in Saudi men with and without coronary artery disease. Biol Trace Elem Res.131:215– 228.

Anderson, R. A. (1992). Chromium, glucose tolerance, and diabetes, *Biol. Trace Element Res.* 32, 19–24.

Anderson, R. A. (1998). Chromium, glucose intolerance and diabetes, *J. Am. Coll. Nutr* . 17, 548–555.

Arun, N. and Nalini, N. (2002). Efficiency of turmeric on blood sugar and polyol path way in diabetic albino rats. Plant Foods for Human Nutr.57:41-52.

Babu, P. and Srinivasan K. (1997). Hypolipidemic action of curcumin, the Active princible of turmeric *Curcuma longa* in streptozotocin induced Diabetic rats. Mol. Cell Biochem. 166: 169-175.

Baur, F. J.(1995). Nutrional aspects of oils and fats. In; Food oils and fats, Technology, Utilization and nutrition. Lawson, H.(ed)., chapman and Hall, New York, Pp. 203-280.

Brownlee, M. (2001). Biochemistry and molecular cell biology diabetic complications. Nature, 414: 813-820.

Cefalu, W. T.; Hu, F. B. (2004). Role of Chromium in Human Health And in Diabetes Care.11 2741–2751.

Chandramohan, G.; Ignacimnthu, S. and Pugalendi, K. V. (2008). Anovel Compound from Casearia esculenta (Roxb.) root and its effect on Carbohydrate metabolism in streptozotocin diabetic rats. European Journal of Pharmacology, 590:437-443.

Chapman, D.G.; Castilla, R. and Campbell, J.A. (1959). Evaluation of protein in food determination of protein and Feed efficiency ratio. Can. J. Biochem. And Physiol., 378: 679-686.

Chaney, A. L. and Marbach, C. P. (1962). Enzymatic colorimetric Method. Reagent for quantitative determination of urea In Serum or plasma. Clin Chem. 8:130 – 136.

El-khamissy, A. (2005). Studies on biological Effects of some Diabetes food. Ph.D. Thesis, Dept. Hom. Economics Fac. of Specific Education, Tanta Univ., Egypt.

El-sayed, M. E. A. (2013). Effect of Feeding on Some Dietary Fibers for Reducing Blood Cholesterol. J. Food and Dairy Sci., Mansora Univ., Vol. 4 (1): 13-25.

Fabiny, D. L. and Ertingshausen, G. (1971) .Automated reaction- rate Method for determination of serum creatinine with the Centrifichem. Clin Chem. 17 (8):696-700. Food. Ph.D. Thesis, Dept. Hom. Economics Fac. of Specific.

Fossati, P. and Prencipe, L. (1982). Serum triglycerides Determined colorimetrically with an enzyme that produces Hydrogen peroxide. Clin Chem. 28(10):2077–2080.

Fox, C.; Ramsoomair, D. and Carter, C. (2001). Magnesium: its Proven and potential clinical significance. South Med J. 94:1195–1201.

Friedewald, W. T.; Levy, R. I. and Fredrickson, D. S. (1972). Estimation of the concentration of low density lipoprotein Separation by three different methods. Clin. Chem., 18:499-502.

Helmy, H. M. (2006). Chemical and biological studies on prickly pear Seed oil and fiber. J. Agric. Sci. Mansoura Univ., 31(10):6517-6528.

Katan. M. B.; Zock. P. L. and Mensink, R. P. (1994). Effect of fats and fatty acids on blood lipids in human: An Overview. AM. J. Clin. Nutr, 60: 1017-1022.

Kelly, J.; Rodriguez, B. and Ruiz, R. (2006). National Diabetes Education Program: A new partner for CCPH (a joint Initiative of CDC & NIH). Bethesda, MD: National Diabetes Information Clearinghouse (NDIC).

Kobrin, S. M. and Goldfarb, S. (1990). Magnesium deficiency. Semin Nephrol 10:525–535.

Krol, E.; Krejpcio, Z.; Byks, H.; Bogdanski, P. and Pupek-Musialik, D. (2010). Effects of chromium brewer's Yeast supplementation on body mass, blood Carbohydrates, and lipids and minerals in type 2 Diabetic patients. Biol Trace Elem Res 143(2):726–737.

MITA, Y.; ISHIHARA, K.; FUKUCHI, Y.; FUKUYA, Y. and YASUMOTO, K. (2004). Supplementation with Chromium Picolinate Recovers Renal Cr Concentration and Improves Carbohydrate Metabolism and Renal Function In Type 2 Diabetic Mice. *Biological Trace Element Research*. Biol Trace Elem Res, 105:229-248.

Murugan, P. and Pari, L. (2007). Influence of tetrahydrocurcumin on Hepatic and renal functional markers and protein levels in Experimental type 2 diabetic rats. Basic and Clinical Pharmacol. And Toxicol, 101: 241-245.

Naveen, S. and Farhath, K. (2012) .Anti-diabetic.anti-oxidant,anti-dyslipidemic and hepatoprotective properties of Coriander seeds extract in Streptozotocin induced diabetic rats .J. of Herbal Medicine and Toxicology, 6 (2): 61– 67.

Ogden, C. L.; Flegal, K. M. and Carroll, M. D. (2002). Prevalence and trends in overweight among US children and adolescents. JAMA, 288 (14), 1728–1732.

Olatunji, L. A.; Oyeyipo, I. P.; Micheal, O. S. and Soladoye, A. O. (2008). Effect of dietary magnesium on glucose tolerance and plasma lipid during oral contraceptive administration in female rats. Afr J Med Sci, 37:135–139.

Reitman, S. and Frankel, S. (1957). Acoloremetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvic transaminase. Am. J.Clin. Path, 28:56-66.

Richmond, N. (1973). Calorimetric method of determination of total Cholesterol and high density lipoprotein cholesterol. Clin. Chem.

Rosalyn, Y. and Bauman, W. A. (1983). Plasma insulin in heaith and disease, in: diabetes mellitus theory and practice, M. Ellenbery and H. Rifkin ed., (New York: Excerpta Medica: 119-150).

Safadar, M.; Khan, A. and Habibiullah, Z. (2006). Effect of jaman fruit extract on serum glucose and lipid profile in type 2 diabetic individuals. Pakistan. J. Nutr, 5(6): 573-576.

Sayed-Ahmed, E. F. (2002). Studies on Some Foods for Cardiac Disease and Atherosclerosis. Ph. D. Thesis. Dept. Food Technol., Fac. of Agric., Moshtohor, Zagazig Univ. (Benha Branch), Egypt.

Steel, R. G. and Torrie, J. H. (1980). In: "Principles and Procedures of Statistics." 2nd Ed. Mc-Graw-Hill, New York, USA, pp. 120-150.

Tuzcu, M.; Sahin, N.; Orhan, C.; Agca, C. A.; Akdemir, F.; Tuzcu, Z.; Komorowski, J. and Sahin, k. (2011). Impact of Chromium histidinate on high fat diet induced obesity in rats. Nutr. Metab, 8:28.

(WHO)World Health Organization. (2000). Diabetes Mellitus. WHO Technical Report Series No.797, pp209, Geneva, Swizerland.

Wolever, T. M. S; Jenkins, D. J. A; Vuskan, V. and Wong, G. s. (1990). Glycemic index of foods in individual subjects. Diabetes Care. 13:126-132.

تأثير التغذية على مستويات مختلفة من الكروميوم بيكولیناتي وكبريتات الماغنسيوم على الفئران المصابة بداء السكري

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أجريت هذه الدراسة لمعرفة مدى كفاءة تأثير إضافه كلا من الكروميوم و الماغنسيوم بنسب مختلفة (٨ ، ١٠ ميكروجرام بيكولينات الكروميوم /مل) و (١٠ ، ١٢ جرام كبريتات ماغنسيوم/ لتر) على التوالي على مياه شرب الفئران وذلك على خفض مستوى سكر الدم و الليبيدات المختلفة في الدم و كذلك مدى التأثير على وظائف كلا من إنزيمات الكبد و الكلى وكذلك وزن الأعضاء الداخلية في الفئران المصابة بمرض البول السكري. ويمكن تلخيص النتائج المتحصل عليها في الأتي:

إنخفاض معدل فقد الوزن في الفئران المصابة بالبول السكري والتي تغذت على الوجبة الأساسية ومياه شرب محتوية على بيكولينات الكروميوم وكبريتات ماغنسيوم بالمقارنة بالفئران المصابة بالبول السكري و تغذت على الوجبة القياسية و مياه شرب عاديه .

وجد أن محتوى السيرم من الكوليستيرول الكلى و الليبوبروتين منخفض الكثافة كان منخفضاً بينما ارتفع محتوى السيرم من الليبوبروتين مرتفع الكثافة و ذلك في الفئران المغذاة على المواد موضع الدراسة مقارنة بالفئران المصابة بمرض البول السكري و المغذاة على الوجبة القياسية فقط.

فى بداية التجربة كان مستوى الجليسريدات الثلاثية مرتفع لكل المجموعات حيث كان فى أواخر المعدل الطبيعى (٦٥- ١٦٥ مجم/دل). وفى نهاية فترة التجربة انخفض مستوى الجليسريدات الثلاثية ليصبح فى منتصف المعدل الطبيعى بالنسبة للفئران المصابة بمرض السكرى و التى تناولت مياه تحتوى على نسب مختلفة من الكروميوم و الماغنسيوم .

بالنسبة ALT وصل إلى (١٠- ١٢.٦٦) و ذلك بالنسبة للفئران التى تناولت مياه تحتوى على نسب مختلفة من الكروميوم و الماغنسيوم. و من ناحية أخرى سجلت المجموعة الضابطة الموجبة أعلى القيم بالنسبة AST .

انخفاض مستوى سكر الدم بصورة ملحوظة و اقترابه من المستوى الطبيعي وكان اكبر معدل في خفض مستوى السكر هو تناول مياه تحتوى على ١٠ ميكروجرام من بيكولينات الكروميوم /مل مقارنة بالانخفاض الذي حدث نتيجة الإضافات الأخرى.

فى بداية التجربة كان معدل الكرياتنيين مرتفع لكل المجموعات حيث كان أعلى من المعدل الطبيعى (٠.٦-١.٤ مجم/دل) و لكن فى نهاية فترة التجربة انخفضت نسبة الكرياتنيين لتصل إلى المعدل الطبيعى.

في بداية التجربة كان معدل اليوريا مرتفع لكل المجموعات حيث كان أعلى من المعدل الطبيعى (١٠-٥٠ مجم/دل) لكن فى نهاية فترة التجربة انخفضت نسبة اليوريا لتصل إلى المعدل الطبيعى.

وبناء على ما سبق يمكن التوصية بأن استخدام كل من الكروميوم و الماغنسيوم له تأثير واضح في خفض جلوكوز وكوليسترول الدم