

## ***Effect of Fructose Ingestion on Some Minerals Status of Diabetic Rats***

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### ***Abstract***

This study was conducted to investigate the relation between fructose consumption and some minerals status of diabetic rats. Thirty six adult male albino rat of Sprague Dawley strain, weighing ( $180 \pm 5$  gm) were divided in two groups. The first group (n=6) was kept as negative control group, the second main group (n=30) was injected with Streptozotocin (STZ) to induce diabetes, then these rats were divided into five subgroups. Subgroup one was fed on the basal diet and served as a positive control group, subgroups from 2 to 5 were fed on basal diet and supplemented with fructose at the level of 5, 10, 15 and 25%, respectively. At the end of the experimental period (6 weeks), rats were sacrificed and blood samples were collected to obtain serum. The results indicated that, STZ treated rats showed significant reduction ( $P < 0.05$ ) in serum insulin concentration and, increased glucose levels compared to normal rats. Supplementation with different levels of fructose in the diet caused significant increase in the concentration of insulin while glucose level was significantly decreased compared to the positive control one. It was also observed that the concentration of serum zinc, magnesium and iron was significantly increased in all tested groups, compared to the positive control group. In conclusions, diet supplemented with 15% fructose caused the best of the biochemical results in diabetic rats. Hence the study recommends a trial to be conducted on diabetic patients before general recommendation.

### ***Introduction***

Diabetes mellitus (DM) is a group of physiological dysfunctions characterized by hyperglycemia resulting directly from insulin resistance, inadequate insulin secretion, or excessive glucagon secretion. There are two main types of diabetes. Type 1 diabetes, is an autoimmune disorder leading to the destruction of pancreatic beta-cells. Type 2 diabetes, which is much more common, is primarily a problem of progressively impaired glucose regulation due to a combination of dysfunctional pancreatic beta cells and insulin resistance (***Lewis et al., 2014 and Ignatavicius and Workman, 2016***).

The world prevalence of diabetes among adults (aged 20–79 years) is 6.4%, affecting 285 million adults, in 2010, and is expected to increase to 7.7%, to reach to 439 million adults by 2030. It has been estimated that by the year 2030, there will be 8.6 million diabetic adults in Egypt, making it the country with the tenth largest population of diabetics in the world (***Shaw et al., 2010***).

Fructose is found in a variety of foods. In table sugar, it is bound to glucose to form the disaccharide sucrose, whereas in honey it occurs in monosaccharide form. In fruit, berries, and vegetables, fructose occurs in both monosaccharide and disaccharide forms. Measured as intake from caloric sweeteners in USA, fructose intake was rather stable throughout the fifties and sixties but increased from the seventies until the end of the nineties, after which intake has declined (***White, 2013***). Fructose was initially proposed as a natural substitute of sucrose for diabetic patients because it does not depend on insulin secretion, at least for its initial steps, and its ingestion causes a limited rise in glycemia (***Lê KA, 2007***). Some studies found no effects of moderate or even high doses (3.5 g fructose/kg fat-free mass/day) of fructose ingestion or infusion on serum levels of glucose of diabetic subjects (***Sunehag et al., 2008 and***

*Ngo et al., 2009*). However, a number of studies demonstrated that fructose ingestion (7.5gm) or fructose-enriched meals (25% of daily energy requirements as fructose) markedly reduced plasma glucose, serum fructosamine, serum glycated hemoglobin, serum glycosylated albumin and serum insulin in diabetic subjects (*Shiota, et al., 2002; Teff, et al., 2004 and Stanhope, et al., 2011*).

There is accumulating evidence that the metabolism of several trace elements is altered in (DM) and that these nutrients might have specific roles in the pathogenesis and progress of this disease (*Kazi, et al., 2008*). Insulin action on reducing blood glucose was reported to be potentiated by some trace elements as chromium, magnesium, vanadium, zinc, manganese, molybdenum and selenium (*Candlish, 2000*).

Fructose affects to some extent the bioavailability of iron, zinc, and copper. It forms stable complexes with iron and promotes its absorption and also that of zinc. Compared with starch, fructose and sucrose decrease copper bioavailability in rats fed diets based on egg white and containing 60% carbohydrate (*O'Dell, 1993*).

The aim of this study was to investigate the relation between different levels of fructose consumption and mineral status of diabetic rats.

## **Materials and methods**

### **Materials:-**

Chemicals Casein, vitamins, minerals and cellulose were obtained from El-Gomhoria Company – Cairo – Egypt. Kits for blood analysis were obtained from Gama Trade Company for Chemical, Cairo, Egypt. Streptozotocin was purchased from Sigma Company. Fructose was purchased from Morgan Company for Chemicals, Cairo. Adult male albino rats (Sprague- Dawley strain) were purchased from Helwan Experimental Animals Station.

### **Methods**

#### **Induction of Diabetes mellitus in rats:**

Diabetes was induced by single intraperitoneal injection of freshly prepared Streptozotocin (STZ) (40mg/kg b.wt.) (*Sarkar, et al., 1996*). Three days after STZ administration, plasma glucose level of each rat was measured. Random blood samples were taken from retro orbital plexus by capillary tube and centrifuged to obtain serum.

#### **Diet composition and experimental animal design:**

The basal diet was formulated according to AIN-93M diet (*Reeves, et al., 1993*). Thirty six adult male albino rats were housed in well-aerated cages under hygienic conditions and were fed on the basal diet for one week for adaptation. After this period rats were divided into two main groups, the first group (6 rats) was fed on basal diet (as a negative control group). The second group (30 rats) diabetic rats, was assigned to five subgroups each subgroup consisted of (6 rats) as follows: subgroup (1) was fed on basal diet and kept as positive control group. Subgroups from 2 to 5 were fed on basal diet supplemented with fructose at the level of 5, 10, 15 and 25%, respectively.

At the end of the experimental period (6 weeks) rats were anesthetized with diethyl ether after fasting for 12h and blood samples were collected from each rat. Serum was carefully separated into vacuum tubes and kept frozen till biochemical analysis.

#### **Biochemical analysis of serum:**

Activities of liver enzymes; alanine transaminase (ALT) and aspartate transaminase (AST) were determined according to the method of *Reitman and Frankel, (1957)*. While, serum total cholesterol (TC) and triglycerides (TG) were determined according to the method of *Richmond, (1973)* and *Fossati and Praneipe, (1982)*, respectively. Determination of HDL-c level was carried out according to the method of *Richmond, (1973)*. VLDL-c and LDL-c were

calculated according to the equation of *Friedewald et al., (1972)*. While, Insulin activity was estimated using enzyme linked immunosorbent assay ELISA method as described by *Clark and Hales, (1994)*. Glucose level was determined according to *Asatoor and King, (1954)*. Serum creatinine and uric acid level was determined by the method of *Tietz, (1999) and Wills and Savory, (1981)* respectively. While serum zinc, magnesium and iron were determined according to the method of *Johnsen et al., (1987), Thomas (1998) and Kaplan and Pesce, (1984)*, respectively.

**Statistical analysis:** The obtained results were analyzed according to SPSS program version 18. ANOVA test was used to compare results among groups, P-value (  $p \leq 0.05$ ) was considered statistically significant (*Snedecor and Cochran, 1986*).

## Results

Effect of diet supplemented with different levels of fructose on serum insulin and blood glucose of diabetic rats was shown in Table (1). Injection with STZ to rats caused a significant decrease ( $P < 0.05$ ) in the concentration of insulin and significant rise in the level of glucose ( $P < 0.05$ ) compared to the negative control group. The administration of diet with different levels of fructose significantly increased ( $P < 0.05$ ) the insulin concentrations, and significantly decreased ( $P < 0.05$ ) the level of blood glucose, compared to the positive control group. It was observed that there were no significant differences in insulin and glucose levels among the rats fed either 5 or 25% fructose. Whereas, there were a significant differences in insulin and glucose levels between the three tested levels of fructose (5, 10 and 15%). The results revealed also that, the most pronounced increase of insulin and the most pronounced decrease of glucose was observed in rats fed on diet supplemented with 15% fructose.

**Table (1):  
Effect of diet supplemented with fructose on serum insulin and blood glucose levels of diabetic rats.**

Parameters Groups	Insulin (IU/mL)	Glucose (mg/dL)
Control (- ve)	5.43±0.44 <sup>a</sup>	92.50±2.12 <sup>e</sup>
Control (+ ve)	0.78±0.05 <sup>e</sup>	216.33±1.85 <sup>a</sup>
Fructose (5%)	1.95±0.03 <sup>d</sup>	196.66±4.26 <sup>d</sup>
Fructose (10%)	2.91±0.07 <sup>c</sup>	156.00±4.04 <sup>c</sup>
Fructose (15%)	4.04±0.16 <sup>b</sup>	134.00±5.50 <sup>b</sup>
Fructose (25%)	1.86±0.06 <sup>d</sup>	204.66±2.84 <sup>d</sup>

Mean values are expressed as means ± SE.

Means with different superscript letters in the same column are significantly different at  $P < 0.05$ .

The results illustrated in the Table (2) shows the effect of diet supplemented with different levels of fructose on serum ALT and AST of diabetic rats. Both of serum ALT and AST concentration were significantly increased ( $P < 0.05$ ) as a result of STZ injection compared with the negative control group. Administration of fructose in the diet at the two different levels (10% & 15%) caused a significant reduction in serum ALT and ALT levels, compared to the positive control group. There were no significant difference in the serum concentration of AST among groups fed on fructose levels at 5, 10, and 25%. The level of fructose that caused the best improvement in liver function was observed at 15%.

**Table (2):  
Effect of Diet Supplemented with Fructose on serum concentration  
of ALT, AST, of Diabetic Rats.**

Groups	Parameters	ALT (U/L)	AST (U/L)
Control (- ve)		50.49±1.89 <sup>b</sup>	88.93±1.79 <sup>d</sup>
Control (+ ve)		80.63±5.23 <sup>a</sup>	144.00±3.78 <sup>a</sup>
Fructose (5%)		74.66±4.05 <sup>a</sup>	137.39±6.39 <sup>ab</sup>
Fructose (10%)		61.83±4.51 <sup>b</sup>	129.00±4.04 <sup>b</sup>
Fructose (15%)		58.36±3.50 <sup>b</sup>	103.76±2.82 <sup>c</sup>
Fructose (25%)		74.75±3.44 <sup>a</sup>	139.66±2.60 <sup>ab</sup>

Mean values are expressed as means ± SE.

Means with different superscript letters in the same column are significantly different at P < 0.05.

Effect of diet supplemented with different levels of fructose on serum uric acid and creatinine concentration of diabetic rats was shown in Table (3). Injection with STZ to rats caused a significant increase (P<0.05) in the level of uric acid and creatinine compared to negative control group. It was observed that there were no significant differences in uric acid and creatinine concentration among groups fed different levels of fructose. Moreover, there were no significant difference in serum uric acid level among the groups fed 5% or 25% of fructose and the positive control group.

**Table (3):  
Effect of Diet Supplemented with Fructose on serum concentration of Uric Acid  
and Creatinine of Diabetic Rats.**

Groups	Parameters	UricAcid (mg/dL)	Creatinine (mg/dL)
Control (- ve)		3.44±0.20 <sup>c</sup>	0.91±0.06 <sup>c</sup>
Control (+ ve)		8.43±1.00 <sup>a</sup>	1.73±0.09 <sup>a</sup>
Fructose (5%)		6.60±0.25 <sup>ab</sup>	1.41±0.04 <sup>b</sup>
Fructose (10%)		6.00±0.85 <sup>b</sup>	1.30±0.09 <sup>b</sup>
Fructose (15%)		5.73±0.70 <sup>b</sup>	1.20±0.11 <sup>b</sup>
Fructose (25%)		7.26±0.63 <sup>ab</sup>	1.43±0.03 <sup>b</sup>

Mean values are expressed as means ± SE.

Means with different superscript letters in the same column are significantly different at P < 0.05.

Effect of diet supplemented with different levels of fructose on serum minerals Zn, Mg and Fe of diabetic rats was shown in Table (4). Serum elements (Zn, Mg and Fe) concentrations were significantly (P<0.05) decreased as a result of STZ injection compared with the negative control group. Diet supplementation with different levels of fructose significantly increased (P<0.05) Zn and Fe concentrations compared to the positive control group. Moreover, It was observed that there were no significant differences in Zn, Mg and Fe levels among the rats fed on either 5% or 25% fructose. Also, there was no significant differences in Mg levels among the rats fed 5%, 10% or 15% fructose. Moreover,

supplementation with fructose at 15% showed the better result compared to the other groups for serum Zn, Fe and Mg concentration.

**Table (4):  
Effect of Diet Supplemented with Fructose on som Serum minerals concentrations  
(Zn,Mg,Fe) of Diabetic Rats.**

Parameters Groups	Zn (µg/dL )	Mg (mg/dL)	Fe (µg/dL )
Control (- ve)	445.33 ± 14.71 <sup>a</sup>	3.70 ± 0.19 <sup>a</sup>	178.63 ± 3.78 <sup>a</sup>
Control (+ ve)	204.62 ± 2.81 <sup>e</sup>	1.82±0.08 <sup>e</sup>	119.86 ±1.84 <sup>e</sup>
Fructose (5%)	238.90± 4.45 <sup>d</sup>	2.46 ± 0.23 <sup>cd</sup>	138.36 ± 5.21 <sup>d</sup>
Fructose (10%)	329.69 ± 3.15 <sup>c</sup>	2.93 ± 0.12 <sup>bc</sup>	151.43 ± 5.11 <sup>c</sup>
Fructose (15%)	354.78 ± 5.62 <sup>b</sup>	3.16±0.12 <sup>b</sup>	163.23 ± 2.68 <sup>b</sup>
Fructose (25%)	247.86 ± 5.05 <sup>d</sup>	2.30 ± 0.17 <sup>de</sup>	136.50±3.09 <sup>d</sup>

Mean values are expressed as means ± SE.

Means with different superscript letters in the same column are significantly different at P < 0.05.

Results in Table (5) illustrated the effect of diet supplemented with different levels of fructose on serum lipid profile of diabetic rats. Injection with STZ to rats caused a significant decrease (P<0.05) in the concentration of HDL-C and significant increase in the level of TC, TG, LDL-C and VLDL-C compared to the negative control group. The supplementation of 10% and 15% fructose significantly increased the HDL-C concentrations, and significantly decreased the levels of TC, TG, LDL-C, and VLDL-C compared to the positive control group. It was observed that there were no significant differences in the TC ,TG , HDL-C , LDL-C and VLDL-C levels among the rats fed 5 or 25% fructose as well as the positive control group. Furthermore, results revealed that there was no significant differences in the levels of TG and VLDL-c compared with the rats fed either 10 % or 15% fructose. Moreover, supplementation with 15% fructose had better result compared to the other treated groups for serum TC, TG, HDL-C, LDL-C and VLDL-C concentrations.

**Table (5):  
Effect of diet supplemented with fructose on serum lipid profile of diabetic rats.**

Groups	Parameters				
	TC (mg/dL)	TG (mg/dL)	HDL-c (mg/dL)	LDL-c(mg/dL)	VLDL-c (mg/dL)
Control (- ve)	102.48±1.61 <sup>d</sup>	87.00±2.51 <sup>c</sup>	59.00 ±4.35 <sup>a</sup>	26.08±2.31 <sup>d</sup>	17.40 ±0.50 <sup>c</sup>
Control (+ ve)	150.33±6.22 <sup>a</sup>	131.66±4.63 <sup>a</sup>	35.66±3.96 <sup>d</sup>	88.33±5.91 <sup>a</sup>	26.33±0.92 <sup>a</sup>
Fructose (5%)	141.66±3.28 <sup>a</sup>	125.33±2.60 <sup>a</sup>	39.06±1.67 <sup>cd</sup>	77.53±4.66 <sup>a</sup>	25.06±0.52 <sup>a</sup>
Fructose (10%)	128.00±5.29 <sup>b</sup>	107.00±4.35 <sup>b</sup>	46.70±2.98 <sup>bc</sup>	59.90±3.95 <sup>b</sup>	21.40±0.87 <sup>b</sup>
Fructose (15%)	115.00±3.46 <sup>c</sup>	97.00±4.04 <sup>bc</sup>	53.93±1.79 <sup>ab</sup>	41.67±5.03 <sup>c</sup>	19.40±0.80 <sup>bc</sup>
Fructose (25%)	146.90±2.17 <sup>a</sup>	127.33±4.33 <sup>a</sup>	36.69±2.37 <sup>d</sup>	84.74±0.93 <sup>a</sup>	25.46±0.86 <sup>a</sup>

Mean values are expressed as means ± SE.

Means with different superscript letters in the same column are significantly different at P < 0.05.

## Discussion

Fructose has a lower glycaemic index compared with other natural sweeteners **Schved and Hassidov, (2008)**. The American Diabetes Association guidelines, however, acknowledge that fructose produces a lower glycemic response in people with diabetes when it replaces sucrose and starch in the diet (**Bantle , et al ., (2008)**). In 2002, **Vozzo et al**, studied the comparative effects of glucose and fructose on blood glucose, insulin, and acute food intake. When subjects drank equienergetic preloads of glucose or fructose before an ad libitum buffet lunch, glucose concentrations were lower in the fructose group compared to glucose, and insulin concentrations were 50% higher in the fructose group in type 2 diabetics than in non-diabetics. The authors concluded that fructose may be a suitable replacement for glucose in diabetic patients – although it was found that satiating efficiencies of fructose certainly offered no advantages. However, scientific data is still inconclusive as to the benefits of fructose as a sucrose substitute for the long term management of diabetes **Havel , et al ., (2005)**.

The obtained results demonstrated a significant increase in serum insulin and a significant decrease in serum glucose for rats feeding different fructose levels, compared with positive control group. These results are agreement with **Cozma , et al., (2012) and Sievenpiper, et al ., (2012) and Sievenpiper, et al ., (2014)** who reported that, moderate amounts of fructose have been shown to have positive effects on glycemic control .

In a preliminary communication in which diets matched for trace element composition and adequate in magnesium content were used, it was noted that a 66% fructose diet did not cause changes in plasma glucose or insulin concentrations in rats **Brands et al., (1993)**. The study of **Thomas et al., (1994)** concurs with this report because magnesium supplementation prevented any clear effect of fructose on insulin responsiveness.

There is evidence that a high intake of fructose can cause insulin resistance in animals **Dekker, et al., (2010)**, but several human studies have failed to demonstrate such an association **Sunehag, et al., 2002 and Stanhope, et al., (2011)**. However, more long-term studies in which the daily intake of fructose is moderate are needed **Astrid and Birger, (2015)**.

Short-term clinical studies (<10 weeks) showed that feeding of high fructose doses (16%-20% calories from total fructose) in obese adults were associated with an increased insulin resistance, dyslipidemia, liver function enzymes and intrahepatic lipid content (**Perez-Pozo, et al., 2010 and Angelopoulos, et al., (2009)**). There is accumulating evidence that the metabolism of several trace elements is altered in DM and that these nutrients might have specific roles in the pathogenesis and progress of this disease (**Kazi , et al ., 2008**). The study by **Kaur and Singh, (2015)** found the extent of deficiencies of zinc and magnesium in relation to duration of the disease. It may be utilized to optimize the control of type-2 diabetes mellitus by supplementation of these two trace elements. By doing so the metabolic complications of diabetes mellitus can be delayed.

Insulin is believed to be stored in an inactive form of zinc crystals. Zinc ions in the secretory granules of cells are known to glue insulin  $\beta$  molecules, creating somatically stable hexamers **Olaniyan, et al., (2012)**. When the secretory granules open to the surface, the zinc ions pressure decreases rapidly and pH levels change from acid to physiological levels, which results in free insulin monomers and zinc ions will be released from the pancreas. Thus, zinc is required for insulin synthesis and storage **Sondergaard, et al., (2003) and Olaniyan, et al., (2012)**.

**Al-Marroof and Al-Sharbatti (2006)**. Reported that serum zinc levels in diabetic patients were significantly lower in comparison to healthy subjects ( $P < 0.01$ ), which is similar to our observations. In another study, the serum zinc level in the diabetic group was also lower than in the control group, and the difference was statistically significant **Anetor et al. (2002)**. In addition, it is reported that the Zn level in plasma, leukocytes and erythrocytes in diabetic patients is

significantly lower than in non-diabetics (*Zargar, et al., 2002*). In the study by *Masood, et al., (2009)* serum zinc level was reported to be significantly lower in type-2 diabetics, whereas no significant difference was found in serum magnesium level when compared with control subjects.

One mechanism through which magnesium may be acting within peripheral tissue is via its effect on tyrosine kinase, a component of the beta subunit of the insulin receptor for which magnesium is a co-factor. Activation of tyrosine kinase produces a signaling cascade that ultimately translocates GLUT4 (the major insulin-regulated glucose transporter expressed in muscle and other insulin-responsive tissues) to the membrane, and allows the cell to take up glucose (*Hruby, et al., (2013)*). Lower serum levels of these elements have been reported in the diabetic state. Observational studies and clinical trials have shown that dietary magnesium has relatively consistent beneficial associations with type 2 diabetes and related traits *Song, et al., (2006) and Dong, et al., (2011)* or sequelae, from insulin resistance and metabolic syndrome *Volpe, (2008)*.

Another study by *Walti, et al., (2003)* showed that the serum levels of some minerals are lower in patients with DM than those in healthy subjects, especially in poorly controlled diabetes. Increased urinary excretion of Zn and Mg due to hyperglycemia and osmotic diuresis may contribute to reduced levels of these elements. In the current study, there was an overall increase in serum concentration of Zn, Mg and Fe among treated groups with different levels of fructose. Similar observations are reported by *O'Dell, (1993)*, who observed that, fructose readily forms complexes with metal ions and hence may modulate the intestinal absorption and bioavailability of minerals. Fructose increases iron bioavailability in human intestinal Caco-2 and HepG2 cells *Christides and Sharp, (2013)*. In addition, a study by *Tsuchiya, et al., (2013)* it was found that a high-fructose/high-fat diet increased iron levels in livers of mice.

Fructose affects to some extent the bioavailability of iron, zinc, and copper.. Fructose forms stable complexes with iron and promotes its absorption and also that of zinc *O'Dell, (1993)*. High dietary fructose at 20% of energy significantly ( $p < 0.01$ ) increased magnesium balance during both low and high dietary magnesium intakes. Ultra filterable and ionized serum magnesium also apparently were related to magnesium and fructose intakes; they were higher when fructose was fed and when Mg intakes were high (*David, et al., 2000*).

The increase in magnesium balance when fructose was fed is consistent with observations *Holbrook et al., (1989)* who found increased absorption and retention of magnesium and other minerals when fructose was fed as compared with starch. The mechanisms by which fructose affects magnesium absorption and balance are largely unknown. It has been hypothesized that fructose may form complexes with mineral elements, such as magnesium, in the gut and these complexes may facilitate mineral absorption. Thus a magnesium-fructose complex may be more readily absorbed. This hypothesis is consistent with observations of *Van Der Heijden et al., (1994)* who suggested that, fructose enhanced the ileal solubility of magnesium, which in turn elevates the amount of magnesium that can cross the epithelium.

In the current study, there was an overall increase in total cholesterol, TG, LDL and VLDL in the group 5 (25%fructose). These results agreed with that of *Paško, et al., (2011)* who found that effect of hypertriglyceridemia after addition of 31% of fructose showed an increase in triglycerides level. In one study on young healthy women showed more resistance to fructose-induced hypertriglyceridemia, whereas obese or hyperinsulinemic women *Stanhope, et al., (2007) and Swarbrick, et al., (2008)* or men *Reungjui, et al., (2007)* are much more sensitive. *Busserolles, et al., (2003)* found that a fructose rich diet (34%) induced hypertriglyceridemia in rats after 2 weeks. In another study, when fructose at 30–60 g (~12% of energy) was added to the diet in the free-living state, induce no significant effects on lipid or glucose biomarkers *Ernst, et al., (2009)*.

In humans, in acute as well as in chronic studies, high fructose feeding (>15% Energy, more than 50g/day), has been found to elevate daylong serum triglycerides in healthy subjects *Teff, et al., (2004)* and overweight/obese subjects

**Stanhope , et al., (2009).** Results of the study by **Bellamkonda , et al., (2017)** showed that , high fructose (66%) feeding to rats for 60 days leads to fasting hyperglycemia, hypertriglyceridemia, hyperinsulinemia, glucose intolerance and impaired antioxidant potential leading to the development of insulin resistance.

## **Conclusion**

Our results showed that feeding diabetic rats 15% of their calories as fructose for 4 wk caused an increase in serum insulin level and decrease in serum glucose concentration. Furthermore, ingestion of fructose at 15% also increased serum iron, zinc and magnesium in the diabetic rats. This study recommends the trial of the study on diabetic patients using 15% of energy as fructose.

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## Hoda S. Ibrahim, et al

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## Hoda S. Ibrahim, et al

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## تأثير تناول الفركتوز على حالة بعض المعادن في الجرزان المصابة بالسكري

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### الملخص العربى

تقوم هذه الدراسة لبحث العلاقة بين تأثير تناول الفركتوز على حالة بعض المعادن في الفئران المصابة بداء السكري . تم تقسيم ستة وثلاثين فأر بالغ من سلالة (Sprague dawley) ، وزنها (180 ± 5 جم ) الى مجموعتين رئيسيتين .المجموعة الأولى (6 فئران) تركت كمجموعة ضابطة سلبية . المجموعة الرئيسية الثانية (30 فئر) تم حقنها بمادة الإستريبتوزوتوسين STZ لإحداث مرض السكري ، ثم تم تقسيم هذه الفئران الى خمس مجموعات فرعية . تم تغذية المجموعة الفرعية الأولى على نظام غذائى متوازن وأخذت كمجموعة ضابطة ايجابية ، ثم تغذت المجموعات الفرعية من (2-5) على النظام الغذائى المتوازن مع التدعيم بالفركتوز بنسب 5% ، 10% ، 15% ، 25% على التوالى . فى نهاية مدة التجربة (6 أسابيع ) تم تخدير الفئران وتجميع عينات الدم للحصول على مصل الدم . أظهرت النتائج الى أن الفئران المعالجة بـ STZ أصابها نقص معنوى (P<0.05) فى الأنسولين ، وزيادة فى مستويات الجلوكوز مقارنة بالفئران السليمة . وعند الإمداد بمستويات مختلفة من الفركتوز فى النظام الغذائى أظهرت زيادة كبيرة فى تركيز الإنسولين فى حين انخفض مستوى الجلوكوز بشكل ملحوظ مقارنة بالمجموعة الضابطة الاولى ؛ ولوحظ أيضا أن تركيز الزنك و المغنسيوم والحديد فى مصل الدم قد زاد بشكل ملحوظ لجميع المجموعات المدعمة بالفركتوز مقارنة بالمجموعة الضابطة الإيجابية .وخلاصة الدراسة أن النظام الغذائى المدعم بنسبة 15% من الفركتوز أدى الى تحسن فى النتائج البيوكيميائية الناتجة عن مرض السكري فى الفئران . نوصى بتجربة ذلك على مرضى السكري قبل التوصية بالتعميم.

الكلمات المفتاحية : السكري ، الفركتوز ، الزنك ، المغنسيوم ، الحديد ، الإنسولين ، الجلوكوز .