

Effect of Monosaccharide Rich Diets on Blood Lipid Profile of Rats.

By

**Manal S. El Gendy ¹; Suzan A. Saad¹;
and Nawal A. Tphoon ².**

- 1- Nutrition and Food Sciences Department, Nawag Faculty of Home Economics, Al Azhar University, Tanta, Egypt.**
- 2- Home Economics Department, Faculty of Specific Education, Banha University, Egypt.**

Effect of Monosaccharide Rich Diets on Blood Lipid Profile of Rats.

Manal S. El Gendy¹; Suzan A. Saad¹; and Nawal A. Tahoon².

1. Nutrition and Food Sciences Department, Nawag Faculty of Home Economics, Al Azhar University, Tanta, Egypt. manalegendy@yahoo.com
2. Home Economics Department, Faculty of Specific Education, Banha University, Egypt.

ABSTRACT

The objective of this study was to investigate the effect of six types of monosaccharide feeding regimen on blood lipid profile and recommend the best monosaccharide of moderate effect on growth rate and changes in blood lipid profile. Thirty five male albino rats (sprague dawley) were segregated into 7 groups. The first served as normal control. The second received 10% glucose diet, the third received 10% fructose diet, the fourth received 20% glucose diet, the fifth received 20% fructose diet, the sixth received 5% glucose plus 5% fructose fed diet, the seventh received 10% glucose plus 10% fructose diet. Samples were taken after 3 weeks of feeding. Body weights, organs weights and blood lipid profile were recorded. The relative growth rate was enhanced with fructose feeding than others. This enhancement is moderate for 10% fructose feeding. The relative organs weights were better in 10% fructose fed group than other groups. 10 % fructose fed diet showed lowest elevation lipid fractions than other groups.

These findings clearly indicate that 10% fructose fed diet is the best choice for its moderate enhancement of relative growth rate and its lowest effect on serum lipid profile and so can be recommended for use in sweets production.

Key words: glucose; fructose; rats; lipids; body weights.

INTRODUCTION:

Added sugars represent more than 30% of carbohydrate consumption (Popkin and Nielsen, 2003). Studies showed that a small amount of fructose in the presence of high levels of glucose increased the net hepatic glucose uptake and the glycogen synthesis (Shiota *et al.*, 1998 and 2002 and McGuinness and Cherrington, 2003). It was reported that high carbohydrate diet induced elevations in both serum and liver lipids, including cholesterol, triglycerides, phospholipids and total lipids (Shalan, 1996).

Fructose feeding has been shown to induce hypertriglyceridemia, hyperinsulinemia, and hyperglycemia (Raiamani *et al.*, 2005). High fructose (21%) feeding induced diabetes in rats (Yadav *et al.*, 2006). Metabolism of dietary fructose differs from that of glucose. Hepatic glucose metabolism is acutely regulated by phosphofructokinase 1, a key regulatory step of glycolysis. In contrast, fructose enters the glycolytic pathway at the

triose level, bypassing phosphofructokinase 1. This difference in initial metabolism of fructose not only acutely affects carbohydrate metabolism by changing supply of intermediate metabolites, but also induces metabolic adaptation including changes in gene expression (Koo *et al.*, 2008). Most Egyptian sweet producers use glucose in their products.

Therefore, the objective of the present study was to determine the best monosaccharide percentage composition in the diet that has little effect on blood lipid profile to be recommended for use in sweet production.

MATERIAL AND METHODS:

Animals:

Thirty five male albino rats (sprague dawley strain) 6 weeks old, purchased from the Egyptian Organization for Immunity and Vaccine Giza, Egypt, were randomly housed in plastic cages. Animals were housed in a controlled-temperature (25 ± 2 °C) and

humidity (25 ± 2 %) environment, with a 12 hours light / dark cycle and free access to food and tap water. Body weight and food intake were determined at week intervals for 3 weeks of experiment.

Diets preparation:

Basal balanced diet:

Basal diet prepared from fine ingredients per 100g according to AIN (1993). The diet composed of 14% protein (derived from neutral casein); sunflower oil 10%; salt mixture 4% (hegested et al., 1941); vitamin mixture 1% (campbell 1961); DL-methionine 0.3%, choline chloride 0.2% and corn starch up to 100g.

Experimental design:

Animals were segregated into 7 groups:

- 1- Control group: A group of animals offered Basal balanced diet and tap water *ad-libitum*.
- 2- 10% glucose group: Rats received the same diet but containing 10% at expens of starch glucose and tap water *ad-libitum*.
- 3- 10% fructose group: This group received 10% at expens of starch fructose diet and tap water *ad-libitum*.
- 4- 20% glucose group: A group of animals fed 20% at expens of starch glucose diet and tap water *ad-libitum*.
- 5- 20% fructose group: Rats received 20% at expens of starch fructose diet and tap water *ad-libitum*.
- 6- 5% glucose plus 5% fructose group: This group received a diet containing 5% glucose plus 5% fructose at expens of starch and tap water *ad-libitum*.
- 7- 10% glucose plus 10% fructose group: In this group rats received a diet containing 10% glucose plus 10% fructose at expens of starch and tap water *ad-libitum*.

At the end of experiment all groups of animal were anaesthetized with diethyl ether and Blood samples were collected

from the inferior vena cova in glass centrifuge tubes. then centrifuge for 15 min at 1000 x g. Sera were separated and stored at -20°C in deep freezer till further biochemical analysis

Biochemical analysis:

Serum triglycerides were determined using kits, Germany, utilizing the method of Shephard and Whiting (1990). Serum cholesterol was measured by the method of Allain *et al.*, (1974) using Human kits, Germany. Serum total lipids were measured according to the method of Knight *et al.*, (1972) using Bio Adwic kits, Egypt. Serum high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) were estimated by the method of fridewald W.T1972

Relative growth rate:

The relative growth rate of rats was calculated using the following formula:

$$(W1-W0)/W0 \times 100\%$$

Where:

W0: The mean body weight at the experimental beginning.

W1: The mean body weight at the experimental final.

Statistical analysis:

Values were statistically analyzed by one-way analysis of variance (Anova-Tukey test) using SPSS 10.1 software package. The P values <0.05 were considered significant.

Results

The results showed that relative growth rate (RGR) is more enhanced with 10% and 20% fructose feeding than other groups. This enhancement (after 3 wk.) amounted to 14.56% and 17.35 % respectively compared to normal control (Table 4). The relative hepatic weight (Table 6) was significantly reduced only in 10% glucose feeding group amounted to 20.57% compared with normal control. On the other hand relative kidney weight was decreased significantly in 10, 20% glucose and 20% fructose feeding decrease groups amounted to 23.73,

27.12 and 18.64% respectively compared with normal control.

Results of table (5) indicated that spleen/body weight ratio was decreased significantly in 10, 20% glucose, 20% fructose and 5% glucose + 5% fructose diet groups in comparison with normal control, with magnitudes of 20.5, 28.21, 28.21 and 25.64% respectively. The relative heart weight was decreased significantly by 20.69% in 20% glucose diet group compared with normal control (Table 6).

Serum total lipids tended to be of higher level in 10% glucose + 10% fructose group, however the lowest level was recorded in 10% fructose diet group compared with normal control. Total cholesterol and triglycerides were elevated in 10% glucose + 10% fructose fed group than normal control with ratios of 27.39 and 76.07% respectively. The lowest levels compared with normal control for the two parameters were recorded in 10% fructose fed group (Table 7).

LDL cholesterol tended to be of higher level in 20% glucose fed group with magnitude of 170.62% compared with normal control, however the lowest level was recorded in 20% fructose fed group with the ratio of 68.99% compared with normal control. Finally, HDL cholesterol tended to be of lower level in 10% fructose diet group by 35.73% compared with normal control. HDL Cholesterol was approximately not affected in 10% glucose + 10% fructose fed group.

It could be noticed that monosaccharides feeding approximately not affected the food consumption of rats significantly (Table 8).

Discussion:

Table(4) results represents the relative growth rate for the various study groups. The results indicated that fructose fed groups enhanced relative growth rate than other groups. Decreased food intake (Table 8) may be responsible for the difference in weight gain rather than a decrease in utilization of the diet. Liver weights were not significantly different among

the groups, however significant difference ($P < 0.05$) for lower relative liver weights for the 10% glucose fed group recorded in (Table 6) comparison with normal control. These observation due to fructose feeding increases liver weight due to excess of fat accumulation (Poulsom 1986). Kidney, spleen and heart relative weights (Table 6) were decreased in glucose feeding than in fructose feeding but, more oriented with glucose + fructose diet. The liver is the most important organ for regulating glucose metabolism by assimilating increased blood glucose level in the form of glycogen and/or regulating the new synthesis of glucose through gluconeogenesis (Yoon *et al.*, 2001). The liver plays the major role in fructose metabolism (Koo *et al.*, 2008). Fructose enters glycolysis or gluconeogenesis at triphosphate level, bypassing the need for insulin and the action of phosphofructokinase.

After fructokinase catalyzes phosphorylation of fructose to fructose 1-phosphate, the resulting compound is split by hepatic aldolase B into glyceraldehydes and dihydroxyacetone phosphate. The activities of fructokinase and hepatic aldolase B are increased when the amount of fructose in the diet increased leading to enhanced hepatic lipogenesis (Reiser and Hallfrisch, 1987).

Varying the type of dietary carbohydrate could cause changes in the metabolism of lipids. Michaelis *et al.*, (1975) described an increase in total liver lipids in rats when glucose was isocalorically substituted with either sucrose or fructose. This effect was attributed to the induction of various lipogenic enzymes in liver by fructose (Anitha Nandhini *et al.*, 2002). The results showed that 10% fructose feeding induced lower effect on blood lipids and that glucose feeding elevated serum triglycerides concentration than fructose feeding and mixing both in diet elevated more triglyceride levels. Our results were in agreement with those reported by Hallfrisch *et al.*, (1983). Who reported that fructose feeding may lead to hypertriglyceridemia by

increasing the formation of glycerol-3-phosphate, a precursor of lipid synthesis (Zavaroni *et al.*, 1980). Adverse effects of glucose and fructose feeding on serum cholesterol have been previously reported in rats (Fields *et al.*, 1996 and Lingelbach and McDonald, 2000). Hypertriglyceridemia may be due to a defect in removal of VLDL from plasma or increased secretion of VLDL. Anitha Nandhini *et al.*, (2002) showed a significant reduction in the activity of lipoprotein lipase which can cause hypertriglyceridemia and accumulation of VLDL in plasma of the fructose fed rats.

The results indicated decreased serum HDL cholesterol concentration in both glucose and fructose groups and approached normal in 10% glucose + 10% fructose fed group. Anitha Nandhini *et al.*, (2002) attributed the lowered HDL cholesterol concentration in fructose fed rats to the decreased lipoprotein lipase (LPL) and lecithin cholesterol acyl transferase (LCAT) activities in plasma and in the liver, where LCAT catalyses esterification of cholesterol with fatty acids, along with LPL which is responsible for HDL cholesterol synthesis and plays an important role in cholesterol and triglyceride transport and metabolism. Results showed increased LDL cholesterol concentrations in all groups especially in glucose fed groups. It was shown that extra sugars may produce high levels of LDL cholesterol that may contribute to the induction of diabetic dyslipidemia (Mayers, 1993).

Esterbauer *et al.*, (1992) showed that fructose feeding enhanced plasma vitamin E depletion which predispose VLDL and LDL enriched with triacylglycerol to subsequent oxidative stress, which is one of the critical mechanisms involved in the progression of atherosclerosis (Girard *et al.*, 2005).

Conclusion:

It could be noticed that 10% fructose feeding enhanced the lowest elevation in serum lipid profile accompanied by moderate enhancement in relative growth rate and organs weights. From

this view, it is assumed to be the better choice for using in sweet production instead of other percentages of glucose and fructose.

REFERENCES:

- AIN, (1993): American Institute of Nutrition Purified diet for laboratory rodent, final report, J. Nutrition. PP, 123, 1939, and ocompactum Benth. J. Essential Oil. Res., PP., 657-664.
- Allain CC, Poon LS, Chan CSG. (1974): Enzymatic determination of total serum cholesterol. Clin Chem, 20: 470-475.
- Anitha Nandhini AT, Blakrishnan SD, Anuradha CV. (2002): Taurine improves lipid profile in rats fed a high fructose-diet. Nutrition Research, 22: 343-354.
- Campbell, M.K. (1999): Biochemistry 3rd ed, F.C.B, New York, London, Tokyo, p., 606.
- Esterbauer H, Gebichi J, Puhl H, Jurgens G. (1992): The role of lipid peroxidation and antioxidants in oxidative modification of LDL. Free Radic Biol Med, 13: 341-390
- .Fridewald W.T(1972): determination of lipoprotein in serum.clin.chem. 18:49
- Fields M, Lure MD, Lewis CG. (1996): Effect of saturated versus unsaturated fat on the pathogenesis of copper deficiency in rats. J Nutr Biochem, 7: 246-251.
- Girard A, Madani S., El Boustani ES, Belleville J, Prost J. (2005): Changes in lipid metabolism and antioxidant defense status in spontaneously hypertensive rats and wistar rats fed a diet enriched with fructose and saturated fatty acids. Nutrition, 21: 240-248.
- Hallfrisch J, Reiser S, Prather ES. (1983): Blood lipid distribution of hyperinsulinemic men consuming three level of fructose. Am J Clin Nutr, 37: 740-748.
- Hegsted, *et al.*, (1941): Salt mixture. J. Biochem., 438-459.

- Izawa S, Okada M, Matsui H, Horita Y. (1997): A new direct method for measuring HDL cholesterol which does not produce any biased values. *J Med Pharm Sci.*, 37:1385–1388.
- Knight JA, Anderson S, James MR. (1972): Chemical, basis of the sulphophosphovanillin reaction for estimating total serum lipids. *J. Clin. Chem.*, 18: 199-202.
- Koo HY, Wallig MA, Chung BH, Nara TY, Cho BHS, Nakamura MT. (2008): Dietary fructose induces a wide range of genes with distinct shift in carbohydrate and lipid metabolism in fed and fasted rat liver. *Biochimica et Biophysica Acta*, In press.
- Lingelbach LB, McDonald RB. (2000): Description of the long term lipogenic effects of dietary carbohydrates in male fischer 344 rats. *J Nutr.*, 130: 3077-3084.
- Mayers PA. (1993): Intermediary metabolism of fructose. *Am J Clin Nutr*, 58: 754S-765S.
- McGuinness OP, Cherrington AD. (2003). Effects of fructose on hepatic glucose metabolism. *Curr Opin Clin Nutr Metab Care*, 6: 441-448.
- Michaelis DC, Nace CS, Szepesi B. (1975): Demonstration of a specific metabolic effect of dietary disaccharides in the rat. *J Nutr*, 105: 1186-1191.
- Popkin BM, Nielsen SJ. (2003): The sweetening of the world's diet, *Obes Res*, 11:1325–1332.
- Poulsom, R. (1986): Morphological changes of organs after sucrose or fructose feeding. *Prog Bioch Pharm*, 21: 104-134.
- Raiamani S, Suganthi R, Ravichandran MK, Anuradha CV. (2005): Food seasoning spices mixture improves glucose metabolism and lipid profile in fructose fed hyperinsulinemic rats. *J Med Food*, 8(4): 502-507.
- Reiser S, Hallfrisch J. (1987): Lipogenesis and blood lipids. In: S Reiser, Hallfrisch J. editors. *Metabolic effects of dietary fructose*. Boca Raton, FL: CRC press, pp. 83-111.
- Shalan MG. (1996): Biochemical studies of effects of alcohol consumption on fat and carbohydrate metabolism in rats fed different levels of proteins. M.Sc. thesis, Faculty of Science, Menoufiya University, Egypt.
- Shephard MD, Whiting MJ. (1990): Falsely low estimation of triglycerides in lipemic plasma by the enzymatic triglyceride method with modified Trinders's chromogen. *Clin Chem*, 36: 325-329.
- Shiota M, Galassetti P, Monohan M, Neal DW, Cherrington AD. (1998): Small amounts of fructose markedly augment net hepatic glucose uptake in the conscious dog. *Diabetes*, 47: 867-873.
- Shiota M, Moore MC, Galassetti P, Monohan M, Neal DW, Shulman GI, Cherrington AD. (2002): Inclusion of low amounts of fructose with an intraduodenal glucose load markedly reduces postprandial hyperglycemia and hyperinsulinemia in the conscious dog. *Diabetes*, 51: 469-478.
- Yadav H, Jain S, Sinha, PR. (2006): Effect of skim milk and dahi (yogurt) on blood glucose, insulin, and lipid profile in rats fed with high fructose diet. *J Med Food*, 9(3): 328-335.
- Yoon JC, Puigserver P, Chen G, Donovan J, Wu Z, Rhee J. (2001): Control of hepatic gluconeogenesis through the transcriptional coactivator PGC-1. *Nature*, 413: 131-138.
- Zavaroni I, Sander S, Scott S, Reaven GM. (1980): Effect of fructose feeding on insulin secretion and insulin action in the rat. *Metabolism*, 10: 970-973.

Table (3): Body weights (in gram) of rats fed varying levels of dietary glucose and fructose.

	Control	10% glucose	10% fructose	20% glucose	20% fructose	5% glucose + 5% fructose	10% glucose + 10% fructose
0 time	151.5 ± 8.8	191.6^a ± 16.04	179.8^a ± 11.14	185.8^a ± 14.39	186^a ±14.75	186.2^a ± 25.26	180.6^a ± 18.32
1 week	178.5 ± 18.89	219^a ± 12.59	207.2 ± 8.76	203.8 ± 25.59	214^a ± 13.62	206.4 ± 23.84	200 ± 14.97
2 weeks	202.3 ± 23.30	247.2^a ± 14.38	238.8^a ± 23.29	232 ± 25.99	247^a ± 18.63	223.6 ± 37.92	225.8 ± 21.9
3 weeks	215.5 ± 26.07	270^a ± 17.71	266.8^a ± 26.75	272.5^a ± 10.7	278.2^a ± 28.55	261.8^a ± 29.89	249.2 ± 22.91

Table (4): Effect of varying levels of monosaccharides on relative growth rate of rats.

	Control	10% glucose	10% fructose	20% glucose	20% fructose	5% glucose + 5% fructose	10% glucose + 10% fructose
		a,d,f,g	d,f,g	a,b,c,e	a,f,g	b,c,e	a,b,c,e
1 week	17.82 ± 1.71	14.3 ± 1.52	15.24 ± 1.44	9.69 ± 0.91	15.05 ± 1.61	10.85 ± 1.11	10.74 ± 1.18
2 weeks	33.53 ± 3.43	29.02 ± 3.12	32.81 ± 3.14	24.87 ± 2.61	32.79 ± 3.32	30.09 ± 1.96	25.03 ± 2.66
3 weeks	42.24 ± 4.16	40.92 ± 4.18	48.39 ± 5.12	46.66 ± 4.73	49.57 ± 5.52	40.6 ±3.37	37.98 ± 4.16

Table (5): Effect of glucose and fructose feeding for 3 weeks on liver, kidney, spleen and heart weights (in gram) of rats.

	Control	10% glucose	10% fructose	20% glucose	20% fructose	5% glucose + 5% fructose	10% glucose + 10% fructose
Liver	6.07 ± 0.72	6.06 ± 1.23	6.58 ± 1.07	6.37 ± 1.05	6.80 ±1.13	6.42 ± 0.41	5.87 ± 0.72
Kidney	1.27 ± 0.18	1.22 ± 0.22	1.35 ± 0.19	1.17 ± 0.12	1.34 ± 0.13	1.33 ± 0.14	1.25 ± 0.17
Spleen	0.84 ± 0.20	0.83 ± 0.18	0.90 ± 0.15	0.77 ± 0.10	0.78 ± 0.09	0.77 ± 0.05	1.91 ± 0.10
Heart	0.64 ± 0.07	0.75 ± 0.11	0.78 ± 0.10	0.63 ± 0.07	0.81 ± 0.09	0.70 ± 0.10	0.73 ± 0.08

Table (6): Effect of glucose and fructose feeding for 3 weeks on relative organs weights (organ weight (g)/body weight (g) X 100) of rats.

	Control	10% glucose	10% fructose	20% glucose	20% fructose	5% glucose + 5% fructose	10% glucose + 10% fructose
Liver	2.82 ± 0.23	^a 2.24 ± 0.24	^d 2.46 ± 0.25	^a 2.34 ± 0.24	^a 2.44 ± 0.26	^d 2.46 ± 0.27	^d 2.36 ± 0.22
Kidney	0.59 ± 0.08	^a 0.45 ± 0.05	^d 0.51 ± 0.06	^a 0.43 ± 0.04	^a 0.48 ± 0.05	^d 0.51 ± 0.05	^d 0.50 ± 0.04
Spleen	0.39 ± 0.04	^a 0.31 ± 0.03	^{d,e} 0.34 ± 0.04	^{a,g} 0.28 ± 0.03	^{a,g} 0.28 ± 0.02	^{a,g} 0.29 ± 0.03	^{b,d,e,f} 0.37 ± 0.04
Heart	0.29 ± 0.03	^e 0.28 ± 0.03	^d 0.29 ± 0.03	^{a,c,e,g} 0.23 ± 0.03	^d 0.29 ± 0.03	^d 0.27 ± 0.03	^d 0.29 ± 0.03

Table (7): Effect of varying levels of monosaccharides on serum lipid profile of rats.

	Control	10% glucose	10% fructose	20% glucose	20% fructose	5% glucose + 5% fructose	10% glucose + 10% fructose
Total cholesterol (mg/dl)	66.48 ± 7.81	^{a,c} 78.98 ± 8.45	^{d,g} 66.94 ± 5.48	^{a,c,e} 83.76 ± 7.28	^{d,g} 67.77 ± 6.58	^{a,c,e} 79.98 ± 11.45	^{a,c,e} 84.69 ± 7.36
Triglycerides (mg/dl)	57.75 ± 4.30	^{a,c} 85.04 ± 8.11	^{a,d,f,g} 69.87 ± 8.52	^{a,c,e} 97.66 ± 10.15	^{a,d,f,g} 78.24 ± 9.31	^{a,c,e} 98.4 ± 10.19	^{a,b,c,e} 101.68 ± 8.04
HDL-C (mg/dl)	42.09 ± 4.25	^{a,c,g} 32.53 ± 3.46	^{a,d,g} 27.05 ± 2.53	^{a,c} 34.51 ± 3.71	^{a,g} 30.32 ± 4.11	^{a,g} 30.05 ± 5.43	^{b,c,e,f} 40.24 ± 6.13
LDL-C (mg/dl)	12.90 ± 1.12	^{a,e,f,g} 29.44 ± 3.15	^{a,d,e} 25.92 ± 2.74	^{a,b,c,e,f,g} 34.91 ± 3.55	^{a,b,d} 21.8 ± 2.22	^{a,b,d} 22.25 ± 2.03	^{a,b,d} 24.11 ± 2.36
Total lipids (mg/dl)	147.14 ± 15.2	^{a,c} 220.64 ± 21.13	^{a,d,f,g} 181.56 ± 20.63	^{a,c,e} 257.21 ± 24.19	^{a,d,f,g} 205.29 ± 20.12	^{a,c,e} 251.5 ± 29.71	^{a,b,e} 263.27 ± 17.44

Table (8): Effect of varying levels of monosaccharides on daily food intake (in gram) of rats.

	Control	10% glucose	10% fructose	20% glucose	20% fructose	5% glucose + 5% fructose	10% glucose + 10% fructose
0 time	61 ± 5.6	60 ± 6.04	61 ± 6.23	55 ± 5.66	58 ± 5.64	61 ± 6.19	60 ± 5.9
1st week	63 ± 6.4	61 ± 6.15	62 ± 6.51	57 ± 6.11	62 ± 6.21	63 ± 6.42	62 ± 6.51
2nd week	70 ± 7.2	68 ± 6.94	69 ± 7.17	64 ± 6.48	69 ± 6.13	68 ± 6.91	65 ± 6.67
3rd week	77 ± 7.4	75 ± 7.34	79 ± 8.23	75 ± 7.76	78 ± 6.9	75 ± 7.8	72 ± 7.41

تأثير الأغذية الغنية بأحاديات السكر علي دهون الدم في الجرذان.

منال صلاح عباس الجندي^١، سوزان عبد الرحمن سعد^١، نوال عباس طاحون^٢
١- قسم التغذية وعلوم الأطعمة، كلية الاقتصاد المنزلي بنواج، جامعة الأزهر، جمهورية مصر العربية.
٢- قسم الاقتصاد المنزلي، كلية التربية النوعية، جامعة بنها، جمهورية مصر العربية.

الملخص العربي

اختيار لتأثيره المتوسط على معدل النمو النسبي، ولتأثيره المنخفض على دهون الدم، ولذلك يمكن التوصية به لاستخدامه في إنتاج الحلوى

الغرض من هذه الدراسة هو إيضاح تأثير ستة أنواع من الأغذية المحتوية على نسب مختلفة من السكريات الاحادية على دهون الدم، و التوصية بأفضلها لاستخدامه في إنتاج الحلوي. قسم خمس وثلاثين من ذكور الجرذان البيضاء إلي سبعة مجموعات. استخدمت المجموعة الأولى كمجموعة ضابطة. أعطيت المجموعة الثانية وجبة غذائية تحتوي على ١٠% جلوكوز، أما المجموعة الثالثة فقد أعطيت غذاء يحتوي على ١٠% فراكتوز، بينما أعطيت المجموعة الرابعة غذاء يحتوي على ٢٠% جلوكوز. هذا وقد أعطيت المجموعة الخامسة غذاء يحتوي على ٢٠% فراكتوز، وأعطيت المجموعة السادسة غذاء يحتوي على ٥% جلوكوز بالإضافة إلي ٥% فراكتوز، أما المجموعة السابعة فقد أعطيت غذاء يحتوي على ١٠% جلوكوز، ١٠% فراكتوز.

هذا وقد تم أخذ العينات بعد ثلاثة أسابيع من بدء التغذية، تم تسجيل وزن الجسم والأعضاء، ودهون الدم. لوحظ أن الغذاء المحتوي على الفراكتوز قد حفز زيادة معدل النمو النسبي عن باقي المجموعات التجريبية، وقد كانت هذه الزيادة متوسطة مع التغذية على ١٠% فركتوز. هذا وقد كان وزن الأعضاء النسبي أفضل في المجموعة المغذاة ب ١٠% فركتوز عن باقي المجموعات التجريبية. كما لوحظ أن الغذاء المحتوي على ١٠% فركتوز احدث اقل تأثير على دهون الدم. تشير هذه النتائج بوضوح إلي أن الغذاء المحتوي على ١٠% فركتوز هو أفضل

