Effect of Some Natural and Artificial Sweeteners on The Biological and Biochemical Parameters of Normal Male Rats

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Abstract

Due to growing worries about the possible health implications of consuming large amounts of sugar and, more recently, research into new natural and synthetic sugar as alternatives to sucrose, the demand for sugar has largely decreased globally. The purpose of this study was to assess how various artificial and natural sugar affected the biological and biochemical characteristics of healthy albino mice. The findings demonstrated that, in comparison to the commercial and control cohorts, the mean values of the mice' feed intake increased when natural sugar at 5 and 10% were consume. Mice fed a baseline diet containing fructose and glucose as natural sweeteners showed a considerably higher body weight gain (BWG) than the control and commercial sugar cohorts (saccharine, and aspartame). The feed efficiency ratio (FER) of all cohorts that used commercial sugar dropped considerably when compared to the cohorts that used natural sugar, according to the data. The cohorts that used natural sugar had the highest liver relative weight, while the cohorts that used artificial sugar had the highest kidney relative weight. The findings showed that consuming natural sugar raised blood glucose levels, particularly fructose levels. The mean blood glucose value significantly decreased as a result of the commercial sugar, particularly at high levels (10%). Artificial sugar reduced RBC levels more than natural sugar did. Furthermore, macrocytic anaemia may be the cause of the significant drop in red blood cells and haemoglobin (Hb) levels seen in all experimental cohorts. The ninth cohort's white blood cell count had the highest mean value. Natural sugar were found to have less of an effect on kidney functions than artificial sugar, and the mean values of the cohorts that consumed the commercial sugar showed considerably greater liver enzymes than the natural sugar at various levels. It was evident from the results that although natural sugar reduced the level of leptin, artificial sugar up the level of the insulin hormone rapidly ,Because of the potential long- and short-term health risks, this study advises limiting the use of artificial sugar. It is better to use natural sugar that don't create metabolic disorders and have a good effect on the safety and health of the body's numerous organs.

Key word: sugar-Natural sugar- artificial sugar- glucose- Fructose- Screen- Aspartame

Introduction

It is well recognised that the most appealing flavour is sugar, which appeals to our senses and frequently influences whether a food product is accepted or rejected **(Grembecka ,2015)** .Sugar continues to be a staple in diets, valued for its unique sugar qualities and flavour as well as its ability to preserve food.(**Arshab et al.,2022)** Consuming excessive amounts of sugar is associated with an increased risk of noncommunicable diseases, including diabetes, heart disease, cancer, tooth decay, weight gain, and neurodevelopmental issues in children. With 35 million deaths each year, this rising frequency is turning into a serious public health issue that is worse than infectious diseases **(Arnone et al ., 2022)** Investigating natural sugar that improve food's palatability while reducing calories is vital given the growing need for a healthy diet **(Wang et al .,2022)**

Sugar are naturally occurring and artificially synthesised chemical substances whose sugar flavour dictates their use as sugar agents. For thousands of years (**Grembecka ,2015)** They are also useful food enhancers that give meals a sugar taste. Natural sugar and artificial or manufactured sugar are the two main categories into which sugar fall. There are two types of natural sugar **(Das & Chakraborty, 2018).**

Natural sugar is made or found in nature without the need for sophisticated machinery or additional chemicals. Only natural sugars and starches found in living vegetables, trees, seeds, nuts, and roots, as well as wild, non-hybridized, seeded fruits, are the best sources of sugars to consume **(Neacsu & Madar, 2014)**

Simple carbohydrates (glucose, fructose, and sucrose) that are metabolised to produce quick energy sources and complex carbs (starch) for long-term energy and storage are the main ingredients of foods with a sugar flavour.

However, other nitrogen-containing molecules such as glycosides, proteins, D-amino acids, peptides, coumarins, dihydrochalcones, and substituted aromatic chemicals can also contribute to the sugar flavour **(Bilal et al ., 2022).**

Table sugar can be replaced with sugar replacements, such as artificial sugar or strong sugar. They can be used to reduce weight and obesity because they are calorie-free and many times more sweetener than natural sugar. **(Khan ,2015)** artificial sugar. They are also known as non-caloric sugar and non-nutritive sugar (NNS). It could be calorie-free bulk sugar or strong sugar **(Jain & Grover ,2015).**

Six NNS (saccharine, aspartame, sucralose, neotame, acesulfame-K, and stevia) have been approved for use in humans by the US Food and Drug Administration (US-FDA) and are categorised as generally recognised as safe (GRAS) **(Sharma et al .,2016).**

Artificial sugar are generally very sugar and low in calories, which appeals to both food manufacturers and customers. **(Mooradian et al., 2017).**

There fore, this study aimed to investigate the effect of natural sweeteners (glucose and fructose) and artificial sweeteners (saccharine and aspartame) on biochemical parameters of albino rats.

Materials and methods

1. Materials

1.1.Experimental diet and tested sugar

We purchased casine, cellulose, methionine, choline chloride, salt mixture, vitamin mixture, glucose, fructose, saccharine, and aspartame from Gomhoryia Co. in Dokki, Giza, Egypt.

1.2 Kits

El Gomhoria Company in Giza, Egypt, provided the biochemical analysis kits.

1.3. Mice

In this investigation, sixty-three mature male albino mice weighing 100±5g were acquired from the animal house at the Ophthalmology Hospital in Giza.

2. Methods

2.1. Biological experiment

2.1.1.Basal diet composition

To prevent feed from being scattered, the diet was given to the mice in special feed cups. Water was also given to them via a glass tube that passed through the wire case. In their most basic form, the powdered natural and artificial sugar were utilised at levels 5 and 10% to replace the starch calories. The following formula was used to prepare the basic diet, as shown in **(Table 1).**

Table (1): The composition of basal diet

Ingredients	Amount
Corn starch	67.5
Corn oil	10 (10%)
Casein	12 (10%)
Cellulose	5 (5%)
Salt mixture	4 (4%)
Vitamin mixture	1(1%)
Methionine	0.3 (0.3%)
Choline chloride	0.2(0.2%)

Source :Campbell(1963)

2.1.2.Experimental design and animal cohorts

The weight of sixty-three male Sprague Dawley Strain albino mice was 100 ± 5g.The animals, which included seven mice, were divided into nine cohorts.

In the animal house laboratory of the Ophthalmology Hospital in Giza, Egypt, experimental mice were used. For a week as an adaptation phase, mice were housed in wire cages under usual laboratory conditions and provided a regular food. Mice were given their food in special feed cups to prevent feed loss, and they were given water in glass tubes that were affixed to one side of the cage. The food and water were administered ad-labium and were monitored every day. All cohorts of mice were fed on the experimental diet for 28 days according to the following cohorts:

Cohort (1): Mice fed on basal diet as a control cohort.
Cohort (2): Mice fed on basal diet and 5%the glucose powder.
Cohort (3): Mice fed on basal diet and10% glucose powder.
Cohort (4): Mice fed on basal diet and 5% fructose powder.
Cohort (5): Mice fed on basal diet and 10% fructose powder.
Cohort (6): Mice fed on basal diet and 5% saccharine powder.
Cohort (7): Mice fed on basal diet and 10% saccharine powder.
Cohort (8): Mice fed on basal diet and 5% aspartame powder.
Cohort (9): Mice fed on basal diet and 10% aspartame powder.

2.2. Blood samples and organs collection

2.2.1. Blood samples

Following a 12-hour fast at the conclusion of the experiment, blood samples were taken. Blood was drawn into a dry, clean centrifugal tube using the retro-orbital technique using a micro capillary glass tube, and it was allowed to coagulate for 30 minutes at room temperature in a water bath set at 37°C. The serum was separated from the blood by centrifuging it for 10 minutes at 3000 rpm in a clean, well-stopped glass container, and it was then stored at -20°C until analysis **(Schermer,1967).**

2.2.2. Organs

The liver, kidney, and heart were taken out, cleaned in saline solution, weighed, and preserved in 10% v/v formalin solution in accordance with **(Drury and Wallington,1980).**

.2.3. Biological evaluation

The diet consumed and body weight were documented daily and weekly for each experimental portion during the trial time.

BWG, feed intake by daily meal consumption, FER, and organ weight were measured in order to evaluate the various diets (**Chapman et al.,1959)** using the following formulae:

BWG= Final weight – Initial weight

BWG = Final weight – Initial weight

$$FER = \frac{\text{Gain in body weight (g)}}{\text{Feed intake (g)}}$$

Relative organs weight (ROW)= $\frac{\text{Organ weight (g)}}{\text{Body weight(g)}} \times 100$

2.4. Biochemical analysis

2.4.1. Determination of serum glucose

Using an enzymatic colorimetric approach, blood glucose is measured in accordance with **(Kaplan,1984).**

2.4.2.Blood analysis

Hb, RBC, PLC, haematocrit, and lymphocytes are all included in that test. Highly automated electronic and pneumatic multichannel analysers use aperture-impedances and/or laser beam cell size and counting to provide the CBC results **(Mauro and Mario , 2008).**

2.4.3.Determination of lipid profile

Triglycerides were determined using an enzymatic colorimetric method in accordance with **(Fassati and Prencipe, 1982).** Total cholesterol was calculated using **(Allain et al, 1974)**.

HDL-c, or high density lipoprotein cholesterol, can be measured using **(Lopes-Virella et al .,1977)**. Very low density lipoproteins, or VLDL, and LDL were calculated using the methodology of (**Lee and Nieman ,1996)** as follows:

VLDL (mg/dl) = Triglycerides/5

LDL (mg/dl) = (Total cholesterol – HDL) – VLDL

Calculation the ratio of LDL- c/ HDL- c was calculated according to **(Kikuchi et al., 1998).**

2.4.4.Determination of kidney functions

Urea was measured using the enzymatic technique of (**Patton andCrouch, 1977**). Creatinine levels were measured using the kinetic technique of **(Henry, 1974).** The percentage of uric acid in the sample determines how intense the red colour that forms is, according to **Young ,2001).**

2.4.5. Determination of liver functions

ALT determination was done using the methodology of **(Tietz et al 1983)** and AST determination was completed using the methodology of (**Henry, 1974**). Alkaline phosphates were determined by enzymatic calorimetry in accordance with **(Belfield and Goldberg, 1971)** and g/dl of serum albumin was calculated using **(Doumas et al., 1971).**

2.4.6.Determination of some metabolic hormones

The measurement of leptin hormone was done in accordance with **(Cosidine et al.,1996)**. The insulin hormone measurement was done in accordance with **(Defronzo et al., 1979).**

2.4.9.Determination of antioxidant enzymes

Glutathione peroxidase (GPX), superoxide dismutase (SOD), catalase (CAT), glutathione s-transferases (GSTs), total antioxidant capacity (TAC) and malondialdehyde (MDA) were measured according to **(Diego, 2011)**. respectively.

Monoaminooxidase (MAO), acetylcholinestrse (AChE), and creatine phosphokinase (CPK) were ascertained using the methods of **(Kettler et al., 1990 ; Perry et al., 2000 and Koller,1991)** respectively.

2.4.10.Determination of some sexual hormones and immunoglobin production

The levels of serum testosterone (T) were assessed using the techniques of **(Tietz ,2018)** . The method used to measure oestradiol was (Santen, 2015).The process of (**Ratcliffe ,2016)** was employed to ascertain the synthesis of immunoglobulins.

2.5. Histopathological investigation

From every experimental cohort, small liver, kidney, and heart specimens were extracted, preserved in neutral buffered formalin, dehydrated in increasing ethanol concentrations (70, 80, and 90%), cleaned in zylene, and embedded with paraffin. Haematoxylin and eosin were used to stain sections that were 4–6 µm thick, in accordance with **(Carleton et al ,1976)** for

histopathological examination

2.6.Statistical analysis

The method used for statistical analysis is **(Snedecor and Cochran ,1980)**.The mean± SD was used to express all results. The statistical program for social science for Windows (spss, version 11.0 Chicago, DL-USA) was used to perform the statistical analyses.

Results and Discussion

1- Effect of different sugar sources on FI, BWG and FER of normal mice

In terms of feed intake, it was found that mice' mean feed intake values increased when natural sugar at 5 and 10% were consumed **as shown in (Table 2)**, in contrast to the commercial and control cohorts. As the amount of natural sugar increased, so did the feed intake. Consuming fructose sugar resulted in the highest mean feed intake value, followed by glucose cohorts; nevertheless, at the same level, the differences were not statistically significant. The mean values for the glucose cohorts at the same levels were 12.98± 0.28 and 13.54± 0.25, respectively, whereas the mean values for the fructose cohorts at 5 and 10% were 13.28± 0.35 and 13.83± 0.23. Feed intake decreased when commercial sugar were ingested; the cohort that consumed saccharine and aspartame, particularly at the 10% level, experienced the largest decrease. 10.16 ± 0.61 g and 11.23 ± 0.02 g were the corresponding mean values. Between the 5% aspartame-fed cohort and the control cohort, which were 12.09± 0.77 and 12.45± 0.09, respectively, no discernible alterations were found. Additionally, there is no discernible difference between the cohorts that took 5% saccharine and 10% aspartame. Regarding body weight increase, mice fed a baseline diet containing fructose and glucose showed a considerably higher BWG

than the control and commercial sugar cohorts. Every studied cohort's mean body weight growth values that used natural sugar showed non-significant variations at the same level. With mean values of 24.37 ± 1.01 and 37.92 ± 1.46g, respectively, the final cohort (cohort 9) that drank 10% saccharine had the least effect compared to the first cohort (cohort 1). The cohorts that consumed 5% aspartame (33.97± 0.51g) and control (35.01± 1.56g) did not differ significantly from the first cohort (37.92± 1.46g), and the cohorts that consumed 5% saccharine (28.92± 0.59g) and 10% aspartame (30.67± 0.87g) did not differ significantly either.

The FER of all cohorts that used commercial sugar dropped considerably when compared to the cohorts that used natural sugar, according to the data. In contrast to the aspartame cohorts, which showed nonsignificant changes when compared to the control cohort, the commercial sugar saccharine was much more effective on FER at various levels. The high mean value of FER with nonsignificant variations was caused by the high levels of glucose and fructose ingestion. For 5% glucose and fructose, the mean values were 0.104±0.003 and 0.105±0.001, respectively, whereas for 10% natural sugar, the mean values were 0.106±0.001 and 0.109±0.002, respectively.

It was seen from the results that artificial sugar reduced BWG, FI, and FER. 5% saccharine gave the highest effect on the biological parameters.these result were disagreed with a study by**(Rosales-Gómez et al.2018)**while **(Gul et al., 2017)** agree with our results that artificial sweeteners decrease the body weight to the indirect inhibition of intestinal alkaline phosphatase (IAP) by phenylalanine (PHE), a metabolite of ASP, phenylalanine can affect appetite by releasing cholecystokinin and stimulating hypothalamic adrenoreceptors. Aspartate metabolite is taken up by the brain's ARC nucleus, which synthesizes neuropeptide Y that stimulates carbohydrate intake **(Prokic et al., 2015)**

Table (2): Effect of different sweetener sources on feed intake (FI), body weight gain percentage (BWG)and feed efficiency ratio (FER) of normal rats

Cohorts	Parameters		
	Feed intake g/day	Body weight gain g/28d	Feed efficiency ratio
Control cohort (G1)	$12.45^{c} \pm 0.09$	$35.01^{c} \pm 1.56$	$0.100^{c} \pm 0.002$
Mice fed on 5%glucose (G2)	$12.98^{b} \pm 0.28$	$37.92^{b} \pm 1.46$	$0.104^{b} \pm 0.003$
Mice fed on 10% (G3)	$13.54^{a} \pm 0.25$	$40.28^{a} \pm 0.09$	$0.106^{a} \pm 0.001$
Mice fed on 5% fructose (G4)	$13.28^{b} \pm 0.35$	$39.04^{b} \pm 0.82$	$0.105^{b} \pm 0.001$
Mice fed on 10% fructose (G5)	$13.83^{a} \pm 0.23$	$42.21^{a} \pm 1.71$	$0.109^{a} \pm 0.002$
Mice fed on 5%Aspartame (G6)	$12.09^{e} \pm 0.77$	$33.97^{c} \pm 0.51$	$0.100^{c} \pm 0.003$
Mice fed on 10% Aspartame (G7)	$11.23^{d} \pm 0.02$	$30.67^{d} \pm 0.87$	$0.097^{c} \pm 0.001$
Mice fed on 5%Saccharine (G8)	$11.01^{d} \pm 0.64$	$28.92^{d} \pm 0.59$	$0.094^{d} \pm 0.002$
Mice fed on 10% Saccharine (G9)	$10.16^{e} \pm 0.61$	$24.37^{e} \pm 1.01$	$0.086^{e} \pm 0.001$
L.S.D	0.55	1.93	0.003

Means under the same column bearing different superscript letters are different significantly (p≤0.05).

2. Effect of different sugar sources on relative organs weight of normal mice

In table (3) is clear that while there were substantial alterations when comparing cohorts 3 and 5 to the other cohorts, the relative liver and kidney weights of these cohorts did not exhibit any significant differences from one another. Nonsignificant variations were also observed between cohorts 7 and 8 for relative liver weight and between cohorts 6 and 8 for relative kidney weight in cohorts 2 and 4. The cohorts that used natural sugar had the highest liver relative weight, while the cohorts that used artificial sugar had the highest kidney relative weight.

In the same table, the relative pancreatic weights of cohorts 2, 4, and the control cohort showed both significant and non-significant variations from one another. Except for the ninth cohort, which had the highest value, the other cohorts' differences were not statistically significant.

In comparison to the other cohorts, the control cohort's relative spleen weight was considerably lower. Cohorts 2 and 4 do not differ much from one another. Additionally, cohorts 3,5, 6, and 8 all reacted in the same way. Cohort 9 showed the highest value by a large margin.

Table (3): Effect of different sweetener sources on relative organs weight of obese and normal rats

Relative organs weight (%)				
Cohorts	Liver	Kidneys	Pancreases	Spleen
Control cohort (G1)	$2.52^{c} \pm 0.23$	$1.21^{f} \pm 0.032$	$0.37^{c} \pm 0.008$	$0.35^{d} \pm 0.003$
Mice fed on 5%glucose (G2)	$3.68^{b} \pm 0.02$	$1.30^{e} \pm 0.02$	$0.37^{c} \pm 0.005$	$0.37^{c} \pm 0.01$
Mice fed on 10% (G3)	$3.82^{a} \pm 0.03$	$1.50^{d} \pm 0.06$	$0.41^{b} \pm 0.002$	$0.39^{b} \pm 0.002$
Mice fed on 5% fructose (G4)	$3.73^{b} \pm 0.04$	$1.33^{e} \pm 0.03$	$0.37^{c} \pm 0.006$	$0.38^{c} \pm 0.006$
Mice fed on 10% fructose (G5)	$3.91^{a} \pm 0.03$	$1.45^{d} \pm 0.02$	$0.42^{b} \pm 0.01$	$0.40^{b} \pm 0.01$
Mice fed on 5%Aspartame (G6)	$2.41^{c} \pm 0.02$	$1.57^{c} \pm 0.01$	$0.40^{b} \pm 0.007$	$0.39^{b} \pm 0.004$
Mice fed on 10% Aspartame (G7)	$2.33^{d} \pm 0.04$	$1.71^{b} \pm 0.03$	$0.42^{b} \pm 0.009$	$0.41^{a} \pm 0.009$
Mice fed on 5%Saccharine (G8)	$2.30^{d} \pm 0.03$	$1.62^{c} \pm 0.02$	$0.41^{b} \pm 0.02$	$0.39^{b} \pm 0.007$
Mice fed on 10% Saccharine (G9)	$2.11^{e} \pm 0.02$	$1.79^{a} \pm 0.01$	$0.45^{a} \pm 0.005$	$0.42^{a} \pm 0.005$
LSD	0.11	0.07	0.02	0.01

Means under the same column bearing different superscript letters are different significantly ($p \leq 0.05$).

3- Effect of different sugar sources on blood glucose (mg/dl) of normal mice.

According to the findings in table (4), the control cohort's mean blood glucose level was 172.4 ±1.07. Consuming natural sugar raised blood glucose levels, particularly fructose levels. The mean blood glucose value significantly decreased as a result of the commercial sugar, particularly at the high level (10%). In comparison to the control cohort, their mean values were substantially lower. With notable variations, the cohort fed 10% fructose had the greatest mean value and the cohort fed 10% saccharine the lowest.

When the aspartame-treated cohorts were compared to the control cohort in parallel studies to assess the effect of aspartame on blood glucose levels, it was found that the aspartame-treated cohorts' blood glucose levels were significantly higher than those of the control cohort **(Lebda et al., 2017**). Because aspartame interacts with the sugar taste receptors in enteroendocrine cells, it is known to have a detrimental effect on glucose metabolism by controlling SGLT1 expression and enhancing passive active intestinal glucose absorption **(Pepino, 2015).** Parallel to this study, research on healthy and diabetic experimental animals to assess how natural sugar affect blood glucose levels demonstrates that they have an effect on glucose intolerance by reducing the duodenum's ability to absorb glucose and interfering with beta cell K+ATP channel activity and/or cAMP levels **(pehlivan and köksal , 2024)**.

Table (4): Effect of different sweetener sources on blood glucose (mg/dl) of normal rats.

Cohorts	YBlood glucose (mg/dl)
Control cohort (G1)	172.4^{e} ±1.07
Mice fed on 5%glucose (G2)	181.1^{d}±2.11
Mice fed on 10% (G3)	199.6^{b} ±3.11
Mice fed on 5% fructose (G4)	190.5^{c}±0.87
Mice fed on 10% fructose (G5)	206.3^{a}±1.15
Mice fed on 5%Aspartame (G6)	120.7^{f} ±2.57
Mice fed on 10% Aspartame (G7)	111.6^{g} ±1.07
Mice fed on 5%Saccharine (G8)	104.1^{h}±2.11
Mice fed on 10% Saccharine (G9)	95.6^{I} ±3.11
LSD	5.9

Means under the same column bearing different superscript letters are different significantly (p≤0.05).

4- Effect of different sugar sources on blood components (mg/dl) of normal mice.

Table (5) makes it evident that mice fed a diet devoid of sugars as the control cohort had a haemoglobin level of 12.94 ± 0.05 g/dl, which

was significantly higher than that of the other cohorts. The cohorts given a regular diet, 5% glucose, and 5% fructose, as well as the ones given 10% glucose, 10% fructose, and 5% aspartame, did not differ significantly. Cohorts 7, 8, and 9's mean values did not significantly differ from one another.

The data in the same table demonstrated how feeding artificial and natural sugar affected the number of red blood cells. At the evaluated levels, there are no appreciable differences between the natural sugar and the control cohort. Additionally, at the 10% level, there are no appreciable differences between the two artificial sugar. Artificial sugar reduced RBC levels more than natural sugar did.

The same table shows that the white blood cell count in the mice fed simply a baseline diet as the control cohort was 5.01 ±0.40 ×103. 8.59 ±0.06×103 was the highest mean value found in the ninth cohort.

Adding 5% glucose, 10% glucose, and 10% fructose produced values that were almost mean and did not differ significantly, and cohort 6 and cohort 8 did not alter significantly either.

It was observed that there were nonsignificant differences in the haematocrit counts of the 5% glucose cohort (34.98 ±0.07) and the normal control cohort (35.74 ±1.55). The two cohorts and the other cohorts differed significantly from one another. However, there is no discernible difference between the cohorts that took 10% saccharine and aspartame. Artificial cohorts' mean values are substantially lower than those of natural cohorts.

The artificial sugar cohorts, particularly the saccharine cohorts, had the greatest mean values in the plate count, whereas the normal control cohort had the lowest. The cohorts that drank 5% glucose and the control cohort did not differ significantly. Significant differences were between the two cohorts and the others.

Additionally, the levels of Hb and Hct were significantly lower in all experimental cohorts. Instead of a loss of cells, the increase in WBC counts is caused by the relocation of cells to wounded organs, such as the liver **(Abukhomra et al., 2022).** Furthermore, macrocytic anaemia, a haematological disorder marked by the bone marrow's abnormal production of disproportionately large RBCs, may be the cause of the significant decline in RBCs and Hb levels seen in all experimental cohorts. Despite not being a serious illness, macrocytic anaemia can cause serious health issues if left untreated. The cause of macrocytic anaemia is inadequate consumption of vitamin B12 and folate, which impairs erythropoiesis or the absorption of these vital nutrients as a result of illnesses. We had an underlying problem that resulted from

using natural and artificial sugar for four weeks, which led to the development of macrocytic anaemia in our study. This outcome was consistent with other research **(Nagao and hirokawa , 2017).**

Sugar cause the body to go through a sequence of sensory cues that help become ready for metabolic digestion and utilisation. According to the results of haematological studies, we found that the glucose cohort had a less pronounced effect on blood morphological indicators than the aspartame cohort. This outcome is consistent with prior research **(Liauw and saibil ., 2019).**

Table (5): Effect of different sweetener sources on Hemoglobin, WBC and RBC of normal rats.

Cohorts	Hemoglobin (g/dl)	WBC $\times 10^3$	RBC $\times 10^6$
Control cohort (G1)	12. 94 ^{a} ±0.05	5.01 ^{f} ±0.40	4.12 ^{a} ±0.03
Mice fed on 5%glucose (G2)	12.71 ^{a}±0.44	6.61 ^{d} ±0.14	4.12 ^{a} ±0.04
Mice fed on 10% (G3)	12.33 ^{b}±0.34	6.83 ^{d} ±0.29	4.11 ^{a} ±0.11
Mice fed on 5% fructose (G4)	12.53 ^{a} ±0.26	6.26 ^{e}±0.11	4.13 ^{a}±0.06
Mice fed on 10% fructose (G5)	12.24 ^{b} ±0.01	6.79 ^{d}±0.26	4.12 ^{a}±0.004
Mice fed on 5%Aspartame (G6)	11.81 ^{b} ±0.31	7.09 ^{c}±0.25	3.96 ^{b}±0.09
Mice fed on 10% Aspartame (G7)	10.53 ^{c} ±0.14	7.98 ^{b}±0.17	3.21 ^{d}±0.10
Mice fed on 5%Saccharine (G8)	11.04 ^{c} ±0.33	7.36 ^{c}±0.18	3.56 ^{c}±0.12
Mice fed on 10% Saccharine (G9)	10.13 ^{c} ±0.49	8.59 ^{a} ±0.06	3.13 ^{d} ±0.04
LSD	0.52	0.41	0.13

Means under the same column bearing different superscript letters are different significantly ($p \leq 0.05$).

5. Effect of different sugar sources on liver enzymes (AST, ALT and ALP) of normal mice

It was evident in table (6) that the control cohort had the lowest mean values for AST, ALT, and ALP, which were 39.37 ±0.57, 38.55 ±1.61, and 74.56 ±4.03 correspondingly. The mean liver enzyme values changed as a result of sugar use, and the magnitude of the alterations varied depending on whether the sugar were commercial or natural. At varying levels, the mean values of the cohorts that used commercial sugar resulted in considerably higher liver enzymes than those who used natural sugar. The liver enzymes were markedly elevated by varying amounts of saccharine, followed by aspartame levels. However, compared to other natural sugar like glucose, fructose had a greater effect on the liver enzymes. The similar effect was seen between cohorts 2 and the control cohort, and there are no discernible changes between cohorts 3 and 4. The ingestion of both studied sugar had the greatest effect on AST enzymes, followed by ALT, while ALP enzymes had the least effect.

Natural sugar can change metabolism and lead to non-alcoholic fatty liver and metabolic syndrome **(Green and Syn, 2019).** Other negative

effects were also mentioned, such as an increase in appetite that results in higher calorie intake. Intolerance to glucose has been identified as a key detrimental effect, and other studies have confirmed this **(AL-Jaaferi et al., 2021).** Although the outcomes of the randomised control trials have been contradicting the findings, it has also been linked to weight gain. According to our research, aspartame and saccharine caused the most increase in Alanine Aminotransferase (AST). According to the results of a study done on male mice to assess the liver enzymes at various dosages of saccharine, the artificial sugar cohorts had higher levels of alkaline phosphate, aspartate transaminase, and alanine aminotransferase than the controlled and natural sugar cohorts **(Abed et al., 2020).** According to a different study, saccharine is bad for you and shouldn't be in your diet. Numerous additional research have also emphasised the comparable findings and declared saccharine to be a hepatotoxic drug that should not be used **(Helal et al., 2019)**. The same concerns regarding the sugar's hepatotoxicity are brought up by the aspartame cohort's elevated level of alanine aminotransferase. An oral dose of 80 mg/kg of aspartame was given. produced decreased enzyme antioxidant activity, increased liver enzymes, and liver fibrosis after 12 weeks **(Finamor et al.,2021).**

Table (6) Effect of different sweetener sources on liver enzymes (AST, ALT and ALP) of normal rats.

Cohorts	AST (U/L)	ALT (U/L)	ALP (U/L)
Control cohort (G1)	39.37 ^{g}±0.57	38.55 ^{g}±1.61	74.56 ^{g}±4.03
Mice fed on 5%glucose (G2)	40.42 ^{g} ±2.34	39.62 ^{g} ±3.22	77.04 ^{g} ±6.13
Mice fed on 10% (G3)	44.71 ^{f} ±3.05	42.83 ^{f} ±1.01	83.15 ^{f} ±4.57
Mice fed on 5% fructose (G4)	46.67 ^{f}±2.11	45.56 ^{f}±1.54	86.54 ^{f}±3.36
Mice fed on 10% fructose (G5)	53.09 ^{e}±2.02	49.76 ^{e}±3.28	94.86 ^{e} ±5.02
Mice fed on 5%Aspartame (G6)	60.18^{d}±1.03	57.29 ^{d}±1.64	116.11 ^{d} ±1.77
Mice fed on 10% Aspartame (G7)	66.43 ^{c}±2.16	62.15 ^{c}±1.56	133.52 ^{c}±5.03
Mice fed on 5%Saccharine (G8)	73.94 ^{b}±2.34	66.17 ^{b}±2.23	154.38 ^{b} ±4.66
Mice fed on 10% Saccharine (G9)	77.85^{a}±1.72	72.29 ^{a}±1.04	176.41 ^{a} ±5.91
LSD	3.19	3.41	6.54

Means under the same column bearing different superscript letters are different significantly (p≤0.05).

6. Effect of different sugar sources on kidney functions (mg/dl) of normal mice.

In table (7), Mice fed a baseline diet as a control cohort showed blood creatinine, urea, and uric acid levels of 0.70 ± 0.08, 23.41 ± 0.83, and 3.09 ± 0.16 mg/dl, respectively. There was a similar reaction between the cohorts that drank 10% glucose and 5% fructose, and there was no discernible difference between the control cohort and the cohort that received 5% glucose. According to the data gathered, natural sugar had a less effect on renal function than artificial sugar. While there was no significant difference between cohorts 8 and 6 that consumed 5% saccharine and 5% aspartame, levels of saccharine induced the highest

renal functions. The cohort fed a baseline diet with 5% glucose showed the least amount of effect. Both sugar had a greater effect on kidney function in terms of uric acid.

Kumar et al., (2014) found that giving mice a diet low in folate 40 mg/kg/day aspartame for 30 days raised the kidneys' oxidative stress and blood Cr and BUN levels, which is in line with our findings.

Significantly increased serum creatinine and urea with both high and low doses of saccharin may be caused by the toxic effects of saccharin on the kidney, particularly at high doses, which can result in renal function disorders and a decreased glomerular filtration rate, which is followed by blood retention of urea and creatinine **(Turley and Dietschy,2003).** When saccharin was administered, the renal buildup of p-aminohippurate and tetraethylammonium was reduced. At 60 days of age, there was also an increase in potassium excretion, a decrease in urine osmolality, and an increase in urine volume **(Amin et al., 2016).**

Table (7) : Effect of different sweetener sources on kidney functions (mg/dl) of normal rats.

Cohorts	Creatinine	Urea	Uric acid
Control cohort (G1)	0.70 ^{f} ±0.08	23.41 ^{f}±0.83	3.09 ^{f}±0.16
Mice fed on 5%glucose (G2)	0.71 ^{f} ±0.31	24.46 ^{f}±2.21	3.11 ^{f}±0.25
Mice fed on 10% (G3)	0.85 ^{e}±0.13	27.98 ^{e}±1.21	3.61 ^{e}±0.01
Mice fed on 5% fructose (G4)	0.87 ^{e} ±0.11	28.17 ^{e}±1.03	3.59 ^{e}±0.32
Mice fed on 10% fructose (G5)	1. 01 ^{d} ±0.03	30.54 ^{d} ±0.56	4.08 ^{d}±0.23
Mice fed on 5%Aspartame (G6)	1.14 ^{c} ±0.21	33.06 ^{c}±1.15	4.97 ^{c} ±0.37
Mice fed on 10% Aspartame (G7)	1.71 ^{b}±0.11	37.34 ^{b}±1.46	5.96 ^{b}±0.40
Mice fed on 5%Saccharine (G8)	1.16 ^{c} ±0.03	34.41 ^{c} ±0.07	5.08 ^{c} ±0.39
Mice fed on 10% Saccharine (G9)	1.94 ^{a} ±0.21	41.05 ^{a}±2.05	6.11 ^{a}±0.06
LSD	0.13	2.32	0.43

Means under the same column bearing different superscript letters are different significantly (p≤0.05).

7. Effect of different sugar sources on insulin and leptin hormones of normal mice.

as shown in (Table 8),Insulin and leptin hormone levels in the negative control were 16.13 ±1.11 µU/mL and 12.41 ±0.13 ng/mL, respectively. While the cohort that received 10% saccharine had the lowest influence on leptin hormone, it had the greatest mean value of insulin hormone when compared to the other cohorts with significant differences. On the other hand, fructose produced negative outcomes. For the examined hormones, there is no discernible difference between cohorts 2, 3, 4, and the control cohort. The cohorts 6 and 8, which were fed 5% aspartame and saccharine, did not differ much.

It was evident from the results that although natural sugar reduced the level of leptin, artificial sugar up the level of the insulin hormone rapidly. Compared to the 5% level, the largest effect was obtained with a high concentration (10%) of both sugar. The leptin hormone was reduced more by the artificial sugar than by the control and natural sugar cohorts.

When natural sugars and artificial sugar interact with the pancreatic β-cells' sugar-taste receptors, insulin production is stimulated. Through a process that depends on Ca2+ and cAMP, this contact sets off a signal transduction cascade. This finding suggests that compared to artificial sugars, natural sugar may cause a decrease in insulin secretion **(Pang et al., 2021).** Our research revealed that natural sugar have the ability to reduce insulin secretion. This result is in line with an earlier investigation carried out by **Gupta et al.(2014)** which showed that using natural sugar has been linked to the onset of prediabetes. According to the study's findings, using natural sugar causes the pancreas to suffer significant damage, losing its islets and β cells as well as its structural integrity. Due to the depletion of insulin secretory granules, prolonged exposure may cause an initial rise in insulin secretion followed by a subsequent decrease in insulin output. In the end, this results in less first-phase insulin secretion. One early sign of type 2 diabetes mellitus is a reduction in insulin secretion in its more advanced phases **(Alshafei, et al., 2023).**

The results of the study show that aspartame, an artificial sugar, reduces GIP secretion without changing insulin levels. Furthermore, it was found that these sugar reduced body weight even when feed intake was reduced. Given that the artificial sugar increased the production of GLP-1, this effect is probably due to a decrease in leptin secretion. These findings contradict those of earlier research by Alshafei et al. (2023). Taste receptor type 1 member 2 (T1R2) and 3 (T1R3) are the two subunits that make up sugar taste receptors, which are heterotrimeric G-protein coupled receptors (GPRs) that detect gustatory information after consuming either natural sugars or artificial sugar **(Fernstrom et al., 2012).**

Table (8): Effect of different sweetener sources on insulin and leptin hormones of normal rats

Cohorts	Parameters	
	Insulin µU/mL Mean ±SD	Leptin ng/mL Mean ±SD
Control cohort (G1)	16.13^{d} ±1.11	12.41^{b}±0.13
Mice fed on 5%glucose (G2)	16.08^{d}±0.68	12.98^{b} ±0.36
Mice fed on 10% (G3)	15.80^{d}±0.94	13.18^{b} ± 0.62
Mice fed on 5% fructose (G4)	16.17^{d}±1.21	12.79^{b} ± 0.05
Mice fed on 10% fructose (G5)	12.75^{e}±0.93	16.40^{a} ± 0.45
Mice fed on 5%Aspartame (G6)	18.67^{c}±1.04	12.01^{c}±0.23
Mice fed on 10% Aspartame (G7)	23.87^{b}±1.21	11.50^{d} ± 0.57
Mice fed on 5%Saccharine (G8)	19.05^{e}±0.93	12.40^{c} ± 0.28
Mice fed on 10% Saccharine (G9)	27.12^{a}±1.04	10.51^{e}±0.08
LSD	1.24	0.77

Means under the same column bearing different superscript letters are different significantly (p≤0.05).

Conclution

Sweeteners are commonly used as food additives in our daily life, which, however, have been causing a number of undesirable diseases since the last century. Therefore, the study carried out to detect the effect of glucose and fructose as natural sweeteners and artificial sweeteners as aspartame and saccharine on the biological and biochemical parameters of rats . Although, artifical sweetener intake remains a better strategy for combating overweight and obesity than use of natural sweeteners, the natural sweetening agents are preferred over synthetic sweetening agents due to their positive effects on the health and safety of the various organs of the body and do not cause any metabolic disorder as the artificial one.

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"تأثير بعض المحليات الطبيعية والصناعية علي القياسات البيولوجية والبيوكيميائية لذكور الفئران الطبيعية"

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الملخص العربي :

انخفض الطلب العالمي على السكر بشكل كبير بسبب المخاوف المتزايدة حول التأثيرات الصحية المحتملة الناجمة عن تناول كميات كبيرة من السكر، ومؤخراً أجريت دراسات لاستكشاف المحليات الطبيعية والاصطناعية الجديدة كبدائل للسكروز، لذلك هدفت هذه الدراسة إلى تقييم تأثير بعض المحليات الطبيعية والصناعية على المعايير البيولوجية والكيميائية الحيوية للفئران البيضاء الطبيعية، وأظهرت النتائج أن استهلاك المحليات الطبيعية بنسبة 5 و 10٪ أدى إلى زيادة متوسط قيم تناول الفئران للعلف مقارنة بالتجارية والمجموعة الضابطة. أما في حالة زيادة وزن الجسم، فقد كانت زيادة وزن الجسم (BWG)في الفئران التي تتغذى على غذاء أساسي يحتوي على الفركتوز والجلوكوز أعلى بشكل ملحوظ من تلك الموجودة في مجموعتي التحكم والمحليات التجارية(الاسبرتام والسكارين) وفيما يتعلق بنسبة كفاءه الأعلاف (FER)فقد أشارت النتائج إلى أن نسبة كفاءة التغذية لجميع المجموعات التي استهلكت المحليات التجارية انخفضت بشكل ملحوظ مقارنة بالمجموعات التي استهلكت المحليات الطبيعية، وكانت أعلى قيمة لوزن الكبد النسبي في مجموعات المحليات الطبيعية بينما كانت أعلى قيمة لوزن الكلى النسبي في المجموعات التي استهلكت المحليات الصناعية، وأشارت النتائج إلى أن نسبة الجلوكوز في الدم ارتفعت باستهلاك المحليات الطبيعية وخاصة مستويات الفركتوز. أدت المحليات التجارية إلى انخفاض كبير في القيمة المتوسطة لجلوكوز الدم وخاصة عند المستوى العالي (10٪). وانخفض مستوى خلايا الدم الحمراء في المحليات الصناعية أكثر من المحليات الطبيعية. بالإضافة إلى ذلك، يمكن أن يعزى الانخفاض الملحوظ في خلايا الدم الحمراء وانخفاض مستويات الهيموجلوبين (Hb) الملحوظ في جميع المجموعات التجريبية إلى فقر الدم كبير الخلايا، وقد تم الكشف عن أعلى قيمة متوسطة في عدد خلايا الدم البيضاء في

المجموعة التاسعة، تسببت القيم المتوسطة للمجموعات التي استهلكت المحليات التجارية بشكل ملحوظ في ارتفاع إنزيمات الكبد مقارنة بالمحليات الطبيعية بمستويات مختلفة، ولوحظ أن المحليات الطبيعية كان لها تأثير أقل على وظائف الكلى مقارنة بالمحليات الصناعية، ومن النتائج التي تم الحصول عليها، يمكن ملاحظة أن المحليات الصناعية زادت من مستوى هرمون الأنسولين، بينما خفضت المحليات الطبيعية مستوى اللبتين.

توصي هذه الدراسة الحد من استهلاك أيا من المحليات الصناعية لما لها من اضرار صحية قد تظهر علي المدي الطويل والقصير ويفضل استخدام المحليات الطبيعية و التي لها تأثيرات ايجابيه علي صحة وسلامه أعضاء الجسم المختلفة والتي لا تسبب أي خلل في التمثيل الغذائي .

الكلمات المفتاحية: السكر– المحليات الطبيعية– المحليات الصناعية– الجلوكوز– الفركتوز– السكرين– الأسبارتام.