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Investigation of the diabetic effects of maternal high-glucose diet on rats

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ABSTRACT

Background: Diabetes mellitus become an epidemic problem throughout the world. Relation of the diabetes with diet is known. Some evidence is reported about mother died and risk of diabetes in babies during the life related with gestational diabetes. This study was conducted to examine the effects of the exposure of high-dose sucrose to rats and pups during pregnancy and lactation.

Methods: The mother rats were categorized into four groups, during pregnancy and until the offspring were 1-month-old, as follows: Group 1, provided with normal drinking water; Group 2, provided with water containing 10%; Group 3, 20%; and Group 4, 30% table sugar. During the study, the weights and daily fluid consumption of the animals were recorded. At the end of the study, the changes in blood, urine, and pancreatic tissues of the rats were examined.

Results: The pups in the groups supplemented with sugar had more weight gain than those of the control group. Although serum glucose levels of mothers and young rats in the groups fed with sugar-containing water did not reach the diabetic limits, it was observed that these animals had statistically significantly higher blood glucose levels than those in the control group. Insulin levels were also similarly increased by an increase in the amount of sugar. Immunohistochemical studies on the mother rats showed that insulin secreted cell numbers and insulin receptors significantly decreased in some pancreatic islets in the groups supplemented with sugar. Glucagon immunoreactivity examination showed that the number of glucagon-expressing cells decreased in the rat groups supplemented with sugar. Similar and more severe findings were observed in the offspring.

Conclusion: This study has experimentally demonstrated that high daily intake of sugar in healthy pregnancies causes adverse effects on the mother and offspring.

1. Introduction

Gestational diabetes mellitus (GDM) is one of the most common complications of pregnancy, affecting up to 7–14% of all pregnancies, and a cause of concern due to the increased risk on both mother and fetus [1,2].

Although the majority of cases return to normal glycemic levels after delivery, there is strong evidence to suggest that GDM could be a major cause for chronic disease in mothers and can also predict the occurrence of diseases later in life for the newborn [3,4].

Metabolic disorders related to GDM are known to cause long-term side effects, including obesity, insulin resistance, and diabetes, in the offspring [5–7]. Pathological pregnancies, such as GDM, are accompanied by a heightened oxidative stress level [8]. Multiple biochemical pathways and mechanisms of action for glucose toxicity have been suggested [9]; all these pathways have in common the formation of reactive oxygen species (ROS), and they are associated with insulin resistance [10]. The etiology and the pathophysiological mechanisms of GDM have not yet been completely understood.

There is also strong evidence indicating that intrauterine exposure to maternal diabetes conveys a high risk for obesity and type 2 diabetes in the offspring, in addition to genetic predisposition, regardless of the maternal diabetes type [11].

Therefore, the present study was planned to elucidate possible risk for DM of offspring related mother high sucrose died during pregnancy and lactation period in rat model.

2. Material and methods

2.1. Animals

The experiments were performed in accordance with the guidelines for animal research of the National Institutes of Health and were approved by the Committee on Animal Research at Mehmet Akif Ersoy

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University, Burdur, Turkey. The animals were maintained and used in accordance with the Animal Welfare Act and the Guide for the Care and Use of Laboratory Animals (Ethic No: 275, Feb 2017/02).

The mother rats were (3 months old) categorized into four groups, during pregnancy and until the offspring were 1-month-old, as follows: Group 1 animals were fed with normal drinking water; Group 2 animals were given water containing 10% table sugar; Group 3 animals were fed with water containing 20% table sugar; and Group 4 animals were administered water added with 30% table sugar. A total of 84 rats (7 mothers and 2 young pups) were included in this study. One mother and two pups kept in a cage. The mother rats were fed with drinking water; water containing 10%, 20%, and 30% table sugar for 5 days a week; and normal drinking water for the remaining 2 days.

The weights and the daily fluid consumption of the animals were recorded during the study. The rats were checked two times a day for their movement and activation in the cages. At the end of the study period, rats were euthanized with a mixture of ketamine hydrochloride (90 mg/kg) and xylazine (10 mg/kg), which were injected intraperitoneally before sacrificing. The rats were fasted one night before euthanasia. After abdominal incision, blood samples were collected from inferior vena cava immediately after heart beat stopping. The changes in urine and, blood glucose and insulin were analyzed. In addition, pancreatic tissues were examined pathologically and immunohistochemically.

2.2. Biochemical analysis

Blood glucose levels were evaluated using a blood glucose monitor and a lancing device (Accu-Chek Active, Roche Diagnostic GmbH, Mannheim, Germany). Serum insulin levels were analyzed using an ultrasensitive rat/mouse insulin ELISA kit (Cat. #EZRNI-13 K; Millipore, Billerica, Mass) with a multiplate ELISA reader (EPOCH microplate reader; Bio-Tek, Inc., Winooski, Vt). Urine glucose levels were determined using reagent strips for urinalysis (Siemens Multiatix 10 SG-Siemens Healthcare Diagnostic Inc. NY, USA).

2.3. Histopathological examinations

The pancreas samples collected from the animals during necropsy were fixed in 10% neutral formalin. The samples were then routinely processed in an automatic tissue processor equipment (Leica ASP300S, Wetzlar, Germany) and embedded in paraffin wax. Tissue sections were cut into 5-μm-thickness by a rotary microtome (Leica RM2155, Leica Microsystems, Wetzlar, Germany). Then, they were stained with hematoxylin–eosin (HE), placed on a coverslip with mounting media, and examined under a light microscope. Histopathological changes were graded in a blinded manner. Microscopic changes were examined in 10 different areas in a rat. Scores were made according to the numbers of the degenerative cells. The histopathological changes were scored as (0) no degenerative cells; (1) 1–3 degenerated cells; (2) 4–7 degenerated cells; and (3) > 7 degenerated cells in each pancreas section totally.

2.4. Immunohistochemical examinations

The pancreas samples were then immunostained with insulin (anti-insulin antibody [7F8] (ab8302)), glucagon (anti-glucagon antibody [K79bB10] (ab10988)), and insulin receptor (anti-insulin receptor beta antibody [C18C4] (ab69508)) by the streptavidin–biotin technique. All primary sera and secondary antibodies were purchased from Abcam (Cambridge, UK). The sections were incubated with the primary antibodies for a period of 60 min, and immunohistochemistry was performed using biotinylated secondary antibody and streptavidin–alkaline phosphatase conjugate. EXPOSE Mouse and Rabbit Specific HRP/DAB Detection IHC kit (ab80436) was used as the secondary antibody. The antigens were demonstrated using diaminobenzidine (DAB) as the chromogen. For negative controls, the primary antiserum step was omitted. All examinations were performed on blinded samples. All the slides were analyzed for hormone positivity, and a semiquantitative analysis was carried out as detailed later. The overall number of positive cells in 1 high-power field and the number of cells per islet that were positive for each hormone were recorded and compared with normal pancreatic tissue counts. An attempt was made to quantify the percentage positivity of each hormone-producing cell or insulin receptor in each of the islets. All islets in at least 5 low-power fields (40×) were selected, the total number of nuclei in each islet was counted, and the average number of nuclei/islet was calculated. Results obtained from the image analyzer were subjected to statistical analyses. Morphometric analyses were performed using the Database Manual Cell Sens Life Science Imaging Software System (Olympus Co., Tokyo, Japan).

Table 1
Daily water consumptions during the study and body weights, glucose levels, and insulin values of the rats at the end of the study.

	Groups		P value
Daily water consumption (ml/day/per rat)			
Mothers	0	30.28 ± 3.09	0-10% < 0.001
(n = 28)	10%	23.42 ± 1.51	0-20% < 0.001
	20%	17.28 ± 1.25	0-30% < 0.001
	30%	11.28 ± 1.11	10%-20% < 0.001
			10%-30% < 0.001
			20%-30% < 0.001
Body weights (g)			
Mothers	0	217.14 ± 5.52	0-10% < 0.01
	10%	264.85 ± 17.31	0-20% < 0.001
	20%	265.07 ± 11.69	0-30% < 0.001
	30%	281.85 ± 22.71	10%-20% < 0.05
			10%-30% > 0.05
			20%-30% > 0.05
Pups	0	31.92 ± 3.92	0-10% < 0.001
(n = 56)	10%	50.00 ± 3.12	0-20% < 0.001
	20%	60.35 ± 8.14	0-30% < 0.001
	30%	77.75 ± 11.80	10%-20% < 0.05
			10%-30% < 0.001
			20%-30% < 0.001
Glucose levels (mg/dl)			
Mothers	0	117.71 ± 5.21	0-10% > 0.05
	10%	129.28 ± 5.15	0-20% < 0.01
	20%	141.28 ± 14.44	0-30% < 0.001
	30%	151.00 ± 9.62	10%-20% > 0.05
Pups	0	111.71 ± 6.48	0-10 % < 0.001
	10%	128.71 ± 7.02	0-20% < 0.001
	20%	147.28 ± 9.66	0-30% < 0.001
	30%	154.92 ± 9.89	10%-20% < 0.001
			10%-30% < 0.001
			20%-30% > 0.05
Insulin values (ng/ml)			
Mothers	0	0.54 ± 0.10	0-10% < 0.05
	10%	0.80 ± 0.15	0-20% < 0.001
	20%	1.12 ± 0.11	0-30% < 0.001
	30%	1.50 ± 0.25	10%-20% < 0.05
			10%-30% < 0.001
			20%-30% < 0.05
Pups	0	0.67 ± 0.12	0-10% > 0.05
	10%	1.01 ± 0.13	0-20% < 0.05
	20%	1.10 ± 0.12	0-30% < 0.001
	30%	1.53 ± 0.77	10%-20% > 0.05
			10%-30% < 0.05
			20%-30% < 0.05

Values expressed as (mean ± SD), SD: Standard deviation, P < 0.05 statistically significant. The relationships between groups and results of immunohistochemical scores are assessed by Bonnferroni-Dunn and one-way ANOVA.

2.5. Statistical analysis

One-way analysis of variance test was carried out to determine any significant differences between the groups with regard to their blood

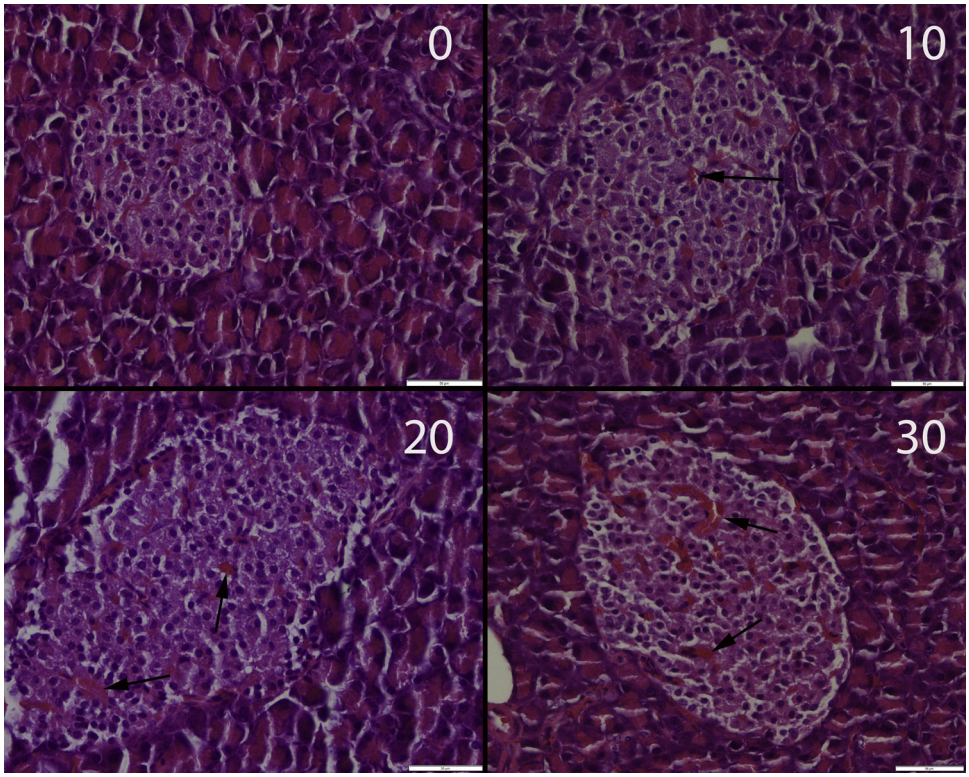

Fig. 1. Histopathological appearance of the mother rats' pancreases in the control and 10%, 20%, and 30% sugar-treated groups; hyperemia (arrows) with different severity was observed in the sugar-treated groups. HE, bars = 50 μm.

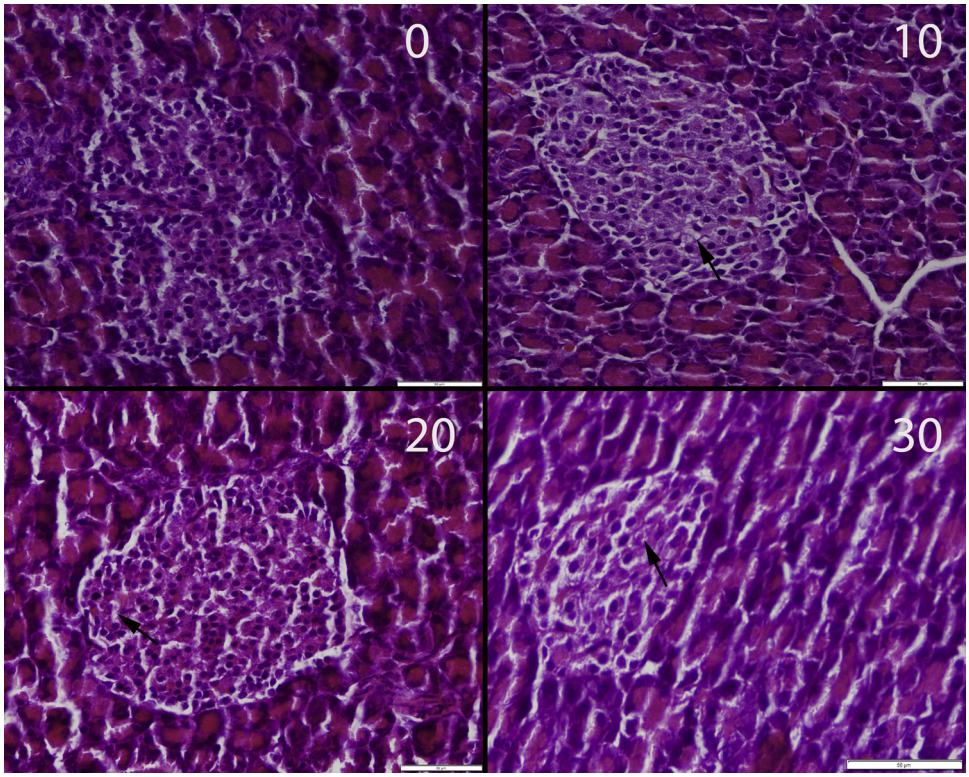

Fig. 2. Histopathology of pups' pancreases. Degenerative cells (arrows) were observed in the sugar-treated groups. HE, bars = 50 μm.

glucose, insulin levels, histopathological scores and immunohistochemical cell percentage. The Bonferroni–Dunn multiple comparison method was used for determining the differences between the groups. Calculations were made using the SPSS 13.0 program package. $p < 0.05$ was set as the level of significance.

Table 2
Statistical analysis of histopathological scores and positive cell percentage.

	Groups		P value
Histopathological scores			
Mothers	0	0.00 ± 0.00	0-10% > 0.05
(n = 28)	10%	0.15 ± 0.14	0-20% > 0.05
	20%	0.57 ± 0.29	0-30% < 0.05
	30%	1.28 ± 0.42	10%-20% > 0.05
			10%-30% > 0.05
			20%-30% > 0.05
Pups	0	0.00 ± 0.00	0-10% > 0.05
(n = 56)	10%	0.57 ± 0.20	0-20% > 0.05
	20%	0.85 ± 0.34	0-30% < 0.05
	30%	1.71 ± 0.42	10%-20% > 0.05
			10%-30% > 0.05
			20%-30% > 0.05
Insulin positive cell percentage			
Mothers	0	88.14 ± 1.06	0-10% < 0.01
	10%	84.71 ± 1.38	0-20% < 0.001
	20%	82.28 ± 0.75	0-30% < 0.001
	30%	79.42 ± 1.27	10%-20% > 0.05
			10%-30% < 0.001
			20%-30% < 0.05
Pups	0	89.00 ± 1.41	0-10 % < 0.001
	10%	84.28 ± 2.28	0-20% < 0.001
	20%	78.85 ± 1.21	0-30% < 0.001
	30%	75.71 ± 2.05	10%-20% < 0.001
			10%-30% < 0.001
			20%-30% < 0.001
Glucagon positive cell percentage			
Mothers	0	28.14 ± 0.89	0-10% < 0.01
	10%	23.57 ± 1.51	0-20% < 0.001
	20%	20.71 ± 1.11	0-30% < 0.001
	30%	17.00 ± 1.15	10%-20% > 0.05
			10%-30% < 0.001
			20%-30% < 0.05
Pups	0	33.42 ± 2.76	0-10% < 0.001
	10%	29.00 ± 1.00	0-20% < 0.01
	20%	22.00 ± 4.12	0-30% < 0.001
	30%	16.00 ± 1.15	10%-20% < 0.001
			10%-30% < 0.001
			20%-30% < 0.001
Insulin receptor positive cell percentage			
Mothers	0	85.42 ± 1.98	0-10% < 0.001
	10%	78.71 ± 0.95	0-20% < 0.01
	20%	54.00 ± 2.44	0-30% < 0.001
	30%	44.71 ± 1.11	10%-20% < 0.001
			10%-30% < 0.001
			20%-30% < 0.001
Pups	0	78.28 ± 1.79	0-10% < 0.001
	10%	67.71 ± 1.79	0-20% < 0.01
	20%	50.85 ± 2.67	0-30% < 0.001
	30%	35.00 ± 2.58	10%-20% < 0.001
			10%-30% < 0.001
			20%-30% < 0.001

Values expressed as (mean ± SD), SD: Standard deviation, P < 0.05 statistically significant. The relationships between groups and results of immunohistochemical scores are assessed by Bonnferroni-Dunn and one-way ANOVA.

3. Results

In the control and the 10% sugar-added groups, the water consumption was normal, whereas the water consumption was decreased in the groups administered water containing 20% and 30% sugar. Water consumption data of the mother rats are shown in Table 1.

Clinically, the rat groups fed with sugar-containing water were more active and their movement in the cage faster than the control rats. Pups were more active than their mothers and they were moving the cage quickly. At the end of the study, the body weights of pups were higher in the sugar-treated groups than in the control group. The weights of the mothers and the pups at the end of the study are shown in Table 1. There was no glycosuria in any of the mothers of pups in any of the groups as assessed by urinalysis before euthanasia using urine sticks.

The blood glucose levels were found to be statistically significantly higher in the sugar-treated groups than in the control group. However, all levels were postprandial blood glucose levels in all groups. The blood glucose levels of the mothers and pups are shown in table. Similarly, the insulin levels were statistically significantly higher, but the levels were in normal postprandial insulin levels in all groups. The insulin levels of the mothers and pups are shown in Table 1.

At necropsy, rats treated with water containing sugar showed marked abdominal lipidosis compared with the control rats. Except lipidosis, there were no marked differences in any of the groups. In the pancreas samples collected during necropsy, slight hyperemia was observed in the groups fed with sugar-containing water.

Histopathological examinations of the pancreases of the control groups revealed normal tissue architecture in both mothers and pups. Moderate-to-marked hyperemia was observed in the islets of Langerhans in the pancreases of some mothers and pups in the sugar-treated groups (Figs. 1 and 2). Except hyperemia, there were no pathological findings in the mothers' pancreases, but slight degenerative changes were observed in some pups in the sugar-treated groups. Statistical analysis results of histopathological scores shown in Table 2.

Immunohistochemical examination revealed the localization of insulin-secreting cells in large areas of the islets of Langerhans. Marked expression was observed in the control group rats, whereas the sugar-treated groups exhibited decreased expression in some islets in the same rats. The decreases were found to be associated with the concentration of the sugar. Similar findings were observed in the pups (Figs. 3 and 4).

Immunohistochemistry of glucagon expression showed the localization of glucagon-secreting cells at the periphery of the islets of Langerhans. Similar expressions were observed in the control and the 10% sugar-treated groups, whereas rats fed with water containing 20% and 30% sugar showed atrophy characterized by a decrease in the number of cells and the severity of expression. Similar findings were also observed in the pups (Figs. 5 and 6).

Insulin receptor expressions were observed throughout the islets of Langerhans. Marked expressions were observed in the control and the 10% sugar-treated groups, whereas there was decreased expression in some islets in the 20% and 30% sugar-treated groups. Similar findings were observed in the pups (Figs. 7 and 8). Statistical analysis results of positive cell percentage for each marker for mothers and pups shown in Table 2.

4. Discussion

Type 2 diabetes, GDM, and diabetes-related complication are increasing rapidly throughout the world. This serious increase has been attributed to lifestyle changes and eating habits. Diabetes in pregnancy, overweight baby births, congenital anomalies, and various long- or short-term complications occur frequently [12]. However, research shows that these complications can occur not only in GDM or pregestational diabetes mellitus but also in animals fed with a diet containing high sugar content without GDM. Based on these studies, this study was aimed at evaluating the effects of daily feeding of water containing sugar on weight gain, blood glucose and insulin levels, pancreas morphology, and pathology and immunohistochemical findings in rats and their infants.

An earlier study by Zhang et al., in 2011 showed that feeding with high content of carbohydrates and fat during pregnancy increases the risk of obesity and cardiovascular disease, insulin resistance, and the development of type 2 diabetes in children [13]. Furthermore, another study reported that the incidence of macrosomia is higher than that in pregnancies fed a high glycemic index diet; in addition, a low glycemic index diet could prevent macrosomia and obesity-related postprandial blood glucose levels and insulin resistance, diabetes mellitus, cardiovascular diseases, and hypertension that it would be in longer period [14]. Jürgens et al., showed that mice treated with 10% and 15%

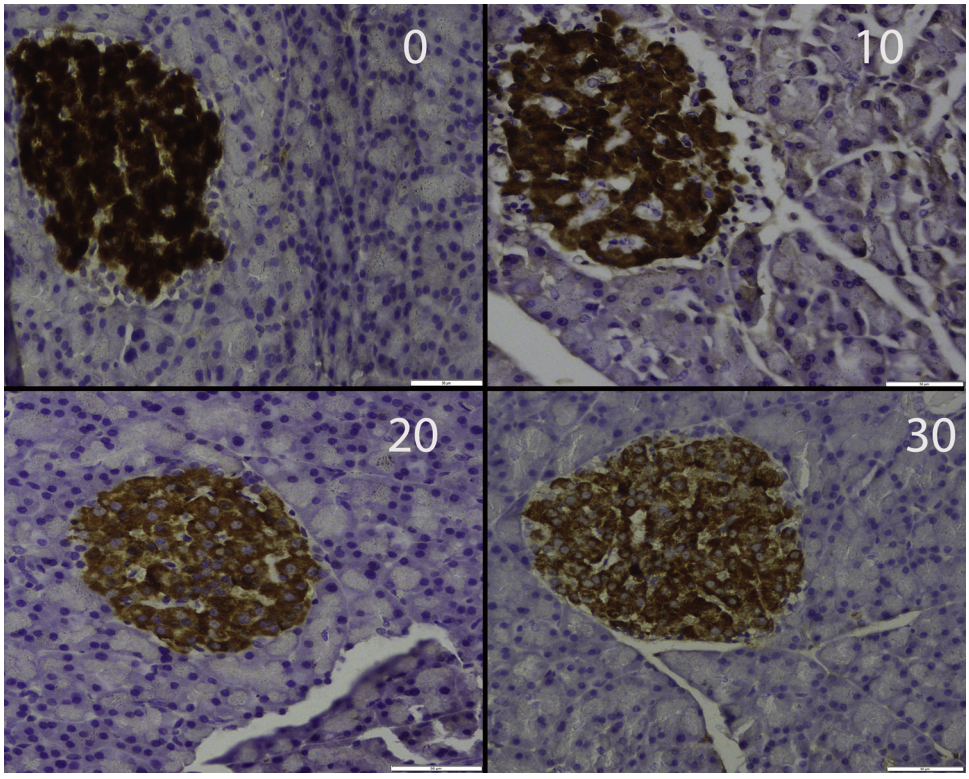

Fig. 3. Insulin expression in the mother rats, showing normal expression in the control group and marked decreased expression in the sugar-treated groups. Streptavidin–biotin peroxidase methods, bars = 50 μm.

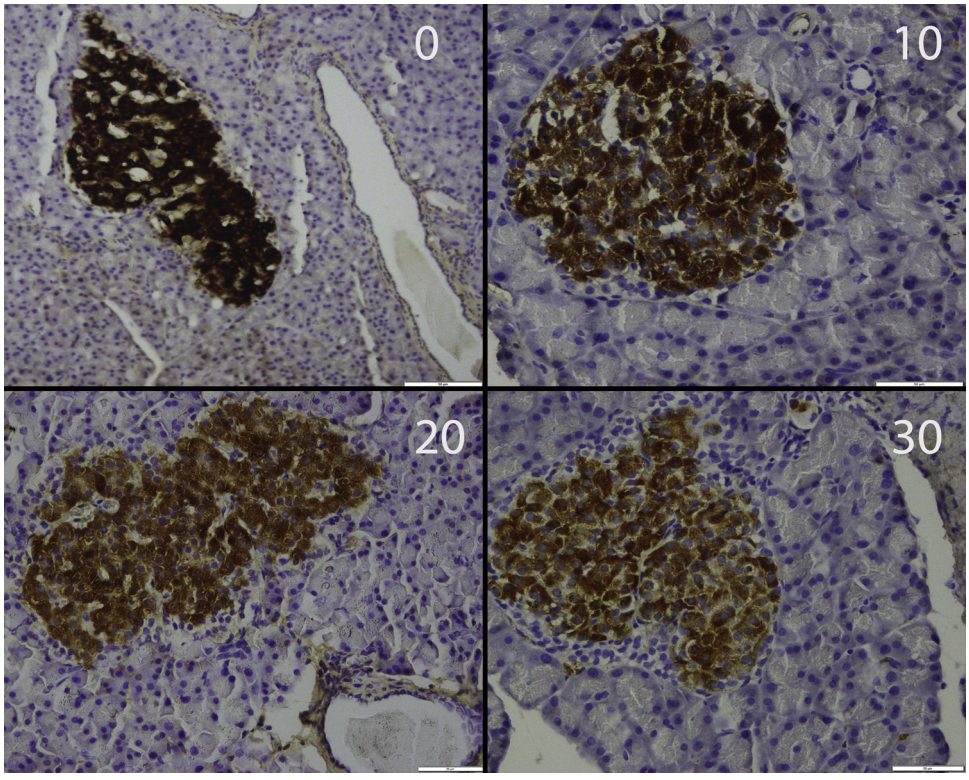

Fig. 4. Insulin expression in the pups' islets of Langerhans showing marked expression in the control rats and decreased expression in the sugar-treated groups. Streptavidin–biotin peroxidase methods, bars = 50 μm.

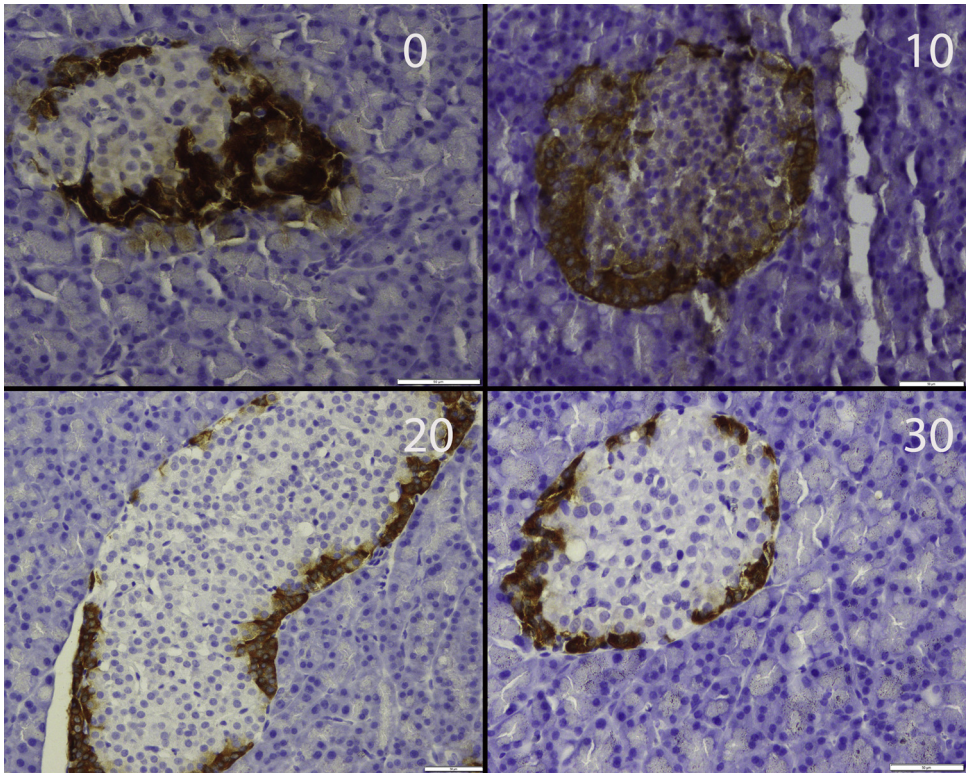

Fig. 5. Glucagon immunoreaction in the mother rats. Similar expressions were observed in the control and the 10% sugar-treated groups. Decreased expression was observed in the 20% and 30% sugar-treated groups. Streptavidin–biotin peroxidase methods, bars = 50 µm.

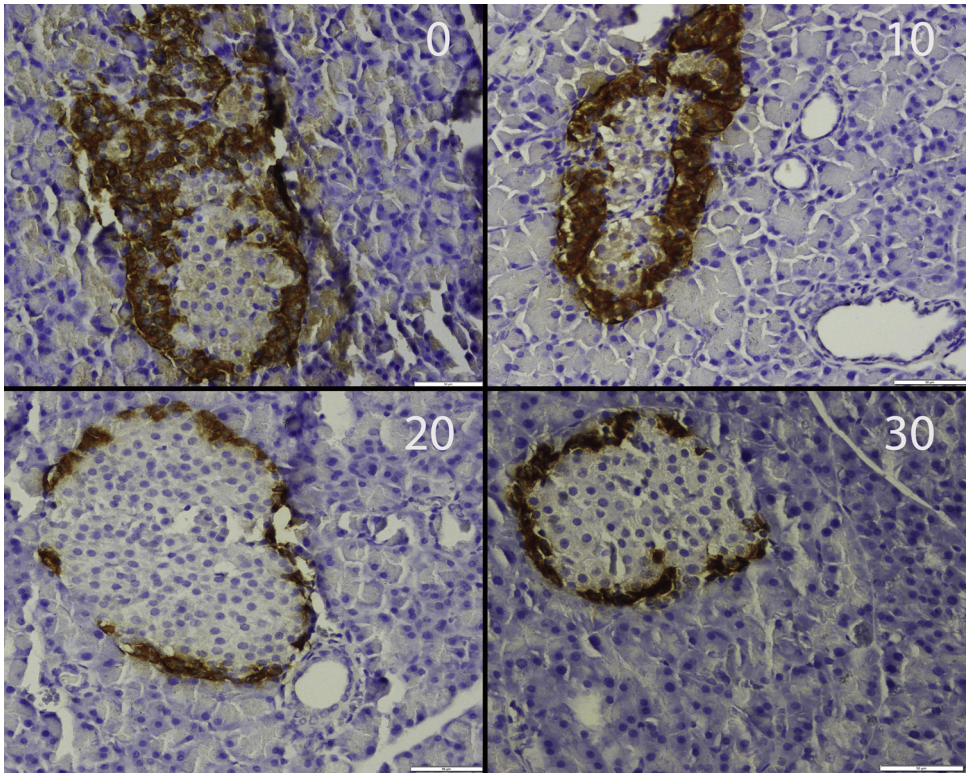

Fig. 6. Glucagon immunoreaction in the islets of Langerhans of pups. Decreased expression was observed in the sugar-treated groups compared to that in the control group. Streptavidin–biotin peroxidase methods, bars = 50 µm.

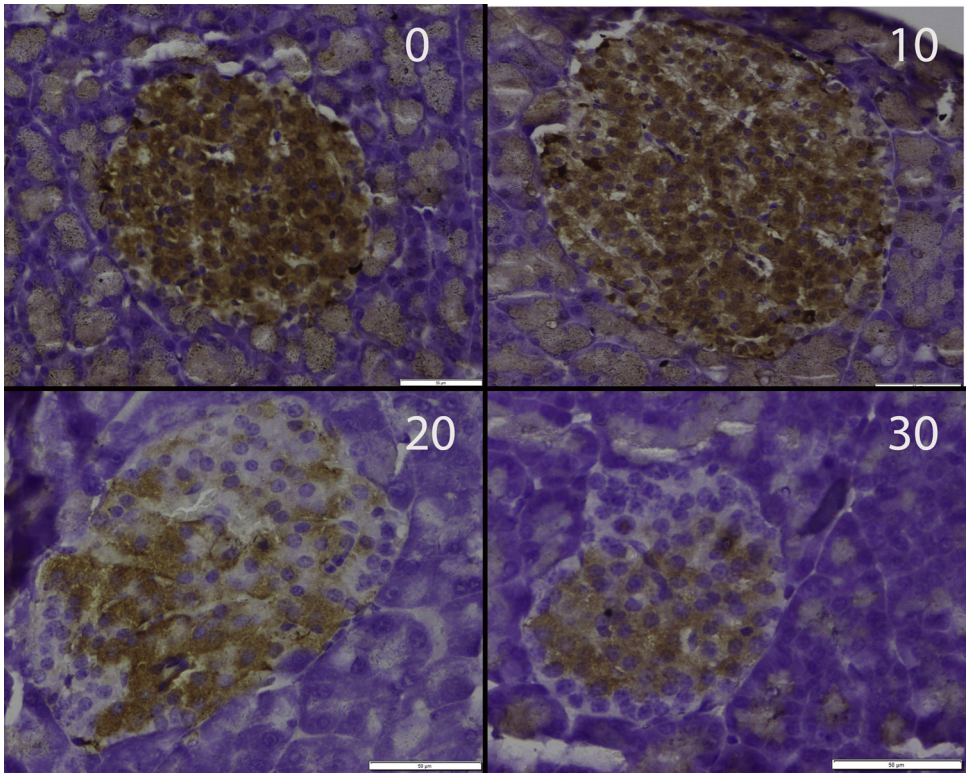

Fig. 7. Insulin receptor immunohistochemistry findings in the mother rats. There was marked increase in the control group, slight decrease in the 10% sugar-treated group, whereas marked decrease in the 20% and 30% sugar-treated groups. Streptavidin–biotin peroxidase methods, bars = 50 μm.

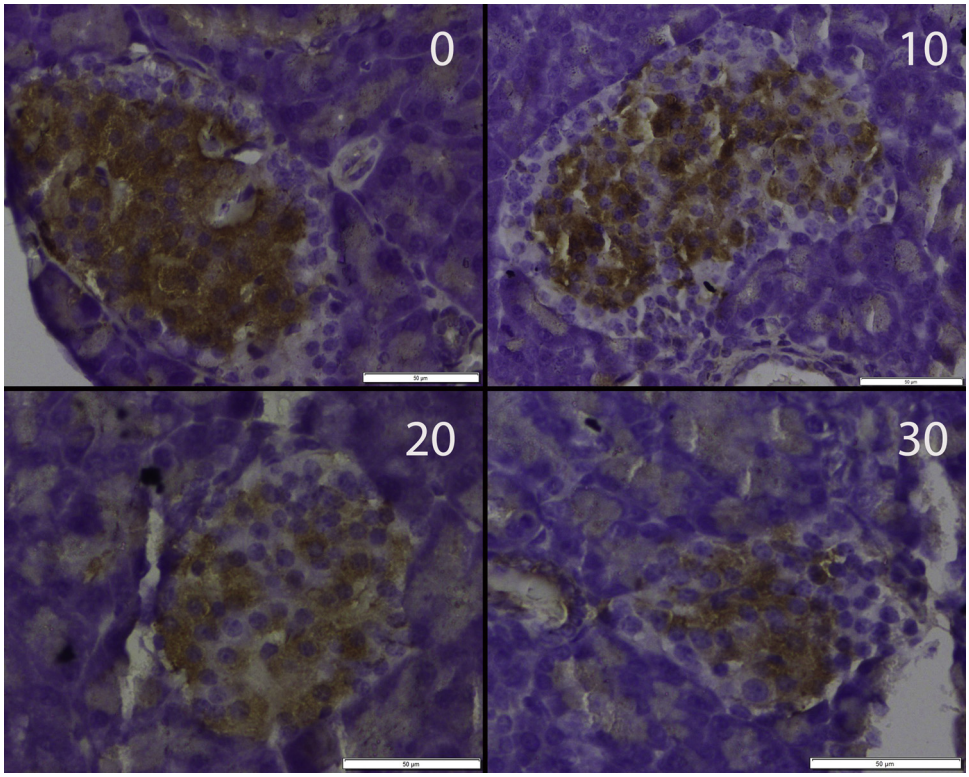

Fig. 8. Insulin receptor immunohistochemistry findings of pups. There was marked expression in the control group, slight decrease in the 10% sugar-treated group, and marked decrease in expression in the 20% and 30% sugar-treated groups. Streptavidin–biotin peroxidase methods, bars = 50 μm.

fructose and sucrose solutions decreased their feed consumption, which was interpreted as a reduction in the amount of energy the mice received from the diet to balance the total energy intake [15].

In our study, we examined the metabolic changes that may occur during the pregnancy and breastfeeding period in rats and their offspring fed with water containing different proportions of sugar. The mother rats were divided into four groups, during pregnancy and until the offspring were 1-month-old. Group 1 was the control administered normal drinking water, and Groups 2, 3, and 4 were fed with water containing 10%, 20%, and 30% sugar, respectively. Before starting the study, 10 female rats were included in each group, but 3 rats in each group were excluded, which were mothers that were not pregnant or had excessive offspring number that would affect the results of the study. Thus, the study was conducted with a total of 84 animals, consisting of 7 mothers and 2 offspring from them in each group. The weights and the daily water consumption of the animals were recorded during the study. At the end of the study, the changes in blood, urine, and pancreas tissues of the rats were examined.

An earlier study by Shankar et al., reported that weight gain was significantly higher in rats fed with a high-carbohydrate diet than that in the control group [16]. Furthermore, Yang et al., showed that mice fed with high fat and high-sucrose exhibited obesity, metabolic syndrome, and insulin signaling disorders [17]. In addition to previous studies, our study results showed that there is a correlation between the increase in sugar water concentrations and the weight gain in the mother rats. We observed that consumption of water with high sugar concentrations reduced the intake of water in the rats. There was a significant increase in body weights of mother rats fed with water containing sugar compared to that in the control group 1 month after the birth. The difference in body weight between the other groups (10% vs 20%, 10% vs 30%, and 20% vs 30%) was statistically significant ($p < 0.05$).

Nivoit et al., reported that weight gain in the postpartum weeks was found to be significantly higher in the offspring of rats fed with a high fat and carbohydrate diet during pregnancy and lactation than that in the control group [18]. In the pups, a significant difference was observed in the weight of the mothers fed with sugar-treated water compared to that in the control group at the end of the first month. In the control group, the mean pup weight was 31.92 ± 3.92 g, whereas the mean pup weights in the remaining three groups were 50.00 ± 3.12, 60.35 ± 8.14, and 77.75 ± 11.80 g, respectively.

In our study, the blood glucose levels of mothers and young rats were found to be within normal limits. Glucose was not detected in the urine. None of the groups in our study developed GDM, but there was a modest increase in blood glucose levels. The difference in blood glucose levels between the control and the 10% sugar-treated groups was not significant, but it was significant in the rats fed with 20% and 30% sugared water. Although the blood glucose levels measured in the rats were in the normal range, there was a significant difference between the groups related to sugar concentrations. When insulin values were compared in the same manner, it was observed that the insulin levels were increased according to sugar concentrations and blood glucose levels in both mothers and offspring. Significant differences were found between the control group and the groups fed with 20% and 30% sugared water. This suggests that high-concentration diets may induce insulin resistance in their offspring as well as in their mothers. Moreover, the fact that both glucose and insulin levels were significantly higher than the diabetic limit showed that the rats were prone to prediabetes due to a high-sugar diet. Based on these results, it is believed that obesity and insulin resistance resulting from gestation and breastfeeding during pregnancy in mothers may lead to obesogenic effects in hyperinsulinemic pups and may increase in parallel with the sugar concentration.

In the study by Yang et al. [17], metabolic syndrome and insulin signaling disorders were observed in obese rats fed with high fat and high-sucrose, blood insulin levels were increased, and there was development of hyperinsulinemia. Another study by Burgeiro et al., demonstrated hyperinsulinemia, insulin resistance, hyperlipidemia, and impaired lipid and glucose metabolism in rats fed with a high-sucrose diet [19]. Furthermore, studies have also demonstrated insulin resistance, leptin levels, and weight gain in the offspring of mothers fed with high carbohydrate diets through intragastric infusion [16,20], which supports our results.

The necropsy findings of our study showed an increase in macroscopic abdominal fat. This is consistent with the transcriptomic changes in fat tissue after feeding with the high carbohydrate diet, as demonstrated by Shankar et al., suggesting a relationship between nutrition and obesity [16]. A study on maternal insulin resistance and overweight women has also shown that maternal hyperglycemia and insulin resistance may have important effects on maternal and infant adiposity [21]. Pancreatic specimens were suspected to be associated with pancreatic inflammations due to increased proinflammatory cytokine production based on macroscopically observed hyperemia, hyperglycemia, and hyperinsulinemia. Immunohistochemical staining of the pancreatic specimens revealed a decrease in the amount of staining in the islet cells, related to the increasing sugar concentrations in the results of insulin, glucagon, and insulin receptor expressions. There was also increased vacuolization of the islet cells related to increased sugar concentrations. This suggests that the high-sucrose diet causes the inflammation of the pancreatic islets due to the damage in the islet cells.

Another study showed that rats fed with 40% sucrose exhibited pancreatic inflammation and islet cell damage [20]. Zhang and colloquies examined the metabolic disorders and pancreatic damage in the offspring of diabetic and nondiabetic rats fed with 15% sucrose-treated diets. They reported that high-sucrose diet can increase pancreatic damage in nondiabetic rats [13]. In our study, the immunohistochemical findings of the pups' pancreases were similar to those of their mothers. Degeneration and decreased insulin secretion and insulin receptor expression were observed in the islets of Langerhans of the pups' pancreases. These results confirmed the possible relationship with mothers' hyperglycemia and hyperinsulinemia, which can cause degeneration and pancreatic damage in the pups.

The inadequate expression of glucagon in the pancreas of both the mother and the offspring is believed to be a secondary event in the adaptive decrease of glucagon release due to long-term high glucose concentrations. These findings indicate that feeding with high sugar during pregnancy and lactation may lead to damaging effects in the juvenile pancreatic Langerhans cells. Findings of similar pancreatic damage were observed in the offspring pancreas in a study in which a high-sugar diet was administered to nondiabetic pregnant rats by Zhang and colleagues [13]. In addition, another study showed that feeding with sugar-sweetened foods had effects on the development of metabolic syndrome and type 2 diabetes in the offspring [22]. In parallel with previous studies, we observed that in addition to the previous administration, high amounts of dietary sugar intake in the mother and offspring resulted in pancreatic damage, as well as increased pancreatic damage associated with the concentration of sugar taken.

5. Conclusion

In summary, this study showed that high daily intake of table sugar in normal healthy pregnancies has adverse effects on the metabolic balance of both mother and offspring. These effects increase in relation to the amount of sugar taken and cause damage of the islet of Langerhans in the offspring's pancreases. Consequently, insulin resistance and type 2 diabetes may lead to the risk of obesity in the long term. Further investigations, including long-term studies, are necessary to obtain more comprehensive data on this issue.

Conflict of interest

The authors have declared that there is no conflict of interest that

regarding the publication of this article.

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