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Antidiabetic Effects of Shrimp Shell-Derived Glucosamine on Biochemical, Immunological, and Hormonal Parameters in Diabetic Rats

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ABSTRACT:

This

study investigates the effects of two levels of glucosamine obtained from shrimp scale extract on the organ functions of diabetic rats. A total of forty-two healthy adult male albino rats were divided into six groups, each consisting of seven rats. Group 1 was the negative control and was fed the basal diet. The remaining groups received a subcutaneous injection of alloxan at a dosage of 150 mg/kg body weight to induce diabetes. Group 2 was the positive control, while Groups 3 and 4 were administered the basal diet supplemented with glucosamine at doses of 1.5 mg and 3 mg, respectively. Groups 5 and 6 were fed a natural diet with 10% and 20% shrimp scales extract. The experiment continued for 28 consecutive days. The study assessed several parameters, including serum liver enzymes, kidney function, blood glucose levels, and immunoglobulin production. The results demonstrated improvement in blood glucose levels, liver enzyme activities, kidney functions, insulin hormone concentrations, and immunoglobulin production across the treatment groups. It was noted that the higher glucosamine dosage and its source exhibited the most pronounced effects. In conclusion, the findings of this study underscore the considerable potential of glucosamine derived from shrimp scales in enhancing protective mechanisms against diabetes, thereby warranting further research and exploration in this field.

Keywords: Glucosamine, Shrimp Scales, Liver, Kidney Functions, Diabetic Rats

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1. INTRODUCTION

A group of metabolic diseases known as diabetes is distinguished by hyperglycemia that is the result of defects in insulin secretion, insulin action, or both. Chronic hyperglycemia

in diabetes is associated with long-term failure, dysfunction, and impairment of a variety of organs, including the eyes, kidneys, nerves, heart, and blood vessels¹.

In the development of diabetes, there are numerous pathogenic processes. The origin of

these deficiencies is the result of inadequate insulin secretion and/or reduced tissue responses to insulin at one or more sites in the complex pathways of hormone action. Additionally, abnormalities that lead to insulin resistance and autoimmune degradation of the β -cells of the pancreas, which results in insulin deficiency, are contributing factors. The primary cause of abnormalities in carbohydrate, lipid, and protein metabolism in diabetes is the insufficient action of insulin on target tissues. The primary cause of hyperglycemia is frequently equivocal in many cases, as the same patient may exhibit both impaired insulin secretion and defects in insulin action [2].

According to ADA [3], there are two main etiopathogenetic categories into which the vast majority of diabetes cases are classified. A clear deficiency of insulin secretion is the distinguishing characteristic of type 1 diabetes, as determined by genetic markers. The prevalence of Type 2 diabetes is considerably higher due to a combination of an insufficient compensatory insulin secretory response and resistance to insulin action. The degree of hyperglycemia (if present) may fluctuate over time due to the severity of the underlying disease process. The same disease course may result in impaired glucose tolerance (IGT) and/or impaired fasting glucose (IFG) even if the diagnostic criteria for diabetes are not met (ADA) [4].

Weight loss, which may be accompanied by polyphagia, impaired vision, polyuria, and polydipsia are symptoms of marked hyperglycemia. Chronic hyperglycemia may also result in impaired growth and increased susceptibility to specific infections. Acute, life-threatening complications of uncontrolled diabetes include hyperglycemia with ketoacidosis or the nonketotic hyperosmolar syndrome [5,6].

An amino sugar, glucosamine ($C_6H_{13}NO_5$) is a significant precursor in the biochemical synthesis of glycosylated proteins and lipids. The fibrous tissue that cushions joints in

crustacean scales is composed of glucosamine, a natural compound. In supplement form, glucosamine is either synthesized in a laboratory or extracted from fish scales. N-acetyl glucosamine, glucosamine hydrochloride, and glucosamine sulfate are among the numerous forms of glucosamine that exist. In dietary supplements, scales of fish are the most prevalent source of glucosamine. A material known as chitin is present in the rigid exoskeletons of crustaceans, including prawns and crabs. Glucosamine is abundantly present in chitin, which serves as the foundation for numerous dietary supplements. Blood, urine, and fecal parameters were not adversely affected by oral glucosamine administration. For adults in the United States, it is among the most frequently utilized non-vitamin, non-mineral dietary supplements [7,8]. Simon et al. [9] subjects experienced a modest decrease in fasting plasma glucose levels following 66 weeks of oral glucosamine administration.

The objective of this investigation was to examine the impact of varying glucosamine levels and shrimp scales extract on the biochemical parameters of diabetic rats.

2. MATERIALS AND METHODS

2.1 Materials

This study was carried out using glucosamine which was obtained from Al-Amria Pharmaceutical Industries Company. Bolti fish scale as a source of glucosamine which were obtained from the local market, Menoufia Governorate, Egypt. The basal diet which consists of casein as a source of protein, corn oil as a source of fat, choline chloride, vitamin mixture, cellulose as a source of fiber, salt mixture and corn starch were obtained from Gomhoryia Co., Giza, Egypt. Alloxan and Biochemical kits were purchased from Kets Company for Drugs, Chemical and Medical Instruments, Cairo, Egypt. Forty-two (42) mature male albino rats (Sprague Dawley strain) each, weighing 150 ± 5 g, were obtained

from Research Institute of Ophthalmology, Medical Analysis Department, Giza, Egypt

2.2 Methods

2.2.1 Preparation of the shrimp scale

The extraction of glucosamine was carried as described previously by Anderson et al. [11]. The shrimp scales should be washed before they are weighed and diced into fine pieces. Spray 25 ml of 4M ammonium sulphate onto the fine granules. The dice should be dried in an oven at 90 °C for a period of 48 hours. To extract glucosamine, grind the desiccated dices into a fine powder and mix it with distilled water at room temperature. Collect the supernatant by centrifuging the sample at 10,000 rpm for 10 minutes. For evaporation, the supernatant was exposed to heated air at a temperature of 100 °C. The quantity of glucosamine extracted from the desiccated sample was determined using high-performance liquid chromatography (HPLC). Each 100 g dried shrimp scales contained 20% glucosamine.

2.2.2 Animals

In this investigation, forty-two white male albino rats of the Sprague Dawley Strain were utilized, each weighing 150± 5g. The rats were housed in cylindrical wire enclosures with wire bottoms, where they were maintained at an ambient temperature of 20–25°C and a relative humidity of 50–55%. They were also subjected to a 12-hour dark and light cycle. A special food cup was used to introduce the diet to prevent the dispersion of food. In addition, the rats were supplied with water through the wire enclosure by means of a glass tube projection. Daily monitoring and provision of ad libitum food and water will be implemented. During the investigation, the animals were treated and handled in accordance with the animal management guidelines. The protocol was approved by the ethics committee, approval number MUFHE/S/NFS/43/24.

2.2.3 Induction of diabetes mellitus

The study employed a pure fine chemical, Alloxan, obtained from Sigma, to induce diabetes in normal, healthy adult male rats. The rats were administered 150mg/kg body weight of alloxan intraperitoneally, following the method outlined in reference of Jelodar [12]. Fasting blood samples were collected from the tail rats six hours following the injection of alloxan to estimate fasting serum glucose. Diabetic rats were defined as those with a fasting serum glucose level exceeding 200mg/dl [13].

2.2.4 Diets

As suggested by AIN, the fundamental diet was prepared using the following formula [14]. The trial procedure was accepted in Animal House, Department of Nutrition and Food Science, Faculty of Home Economic, Menoufia University, (Egypt).

2.2.5 Experimental design

Rats were randomly assigned to two primary groups. The first group of six rats was maintained on the basal diet for the duration of the experiment (negative control). The second primary group of 36 rats was injected with alloxan to induce diabetes and subsequently divided into six groups. All rats were fed for 28 days.:

Group1 : Normal rats will be fed on the basal diet as a negative control (healthy rats).

Group2 : Diabetic rats fed on the basal diet as a positive control.

Group3: Diabetic rats fed on the basal diet containing 1.5 mg glucosamine.

Group4: Diabetic rats fed on the basal diet, which contained 3 mg of glucosamine.

Group5: Diabetes rats fed on basal diet that contained 10% shrimp scale extract.

Group6: Diabetic rats fed on the basal diet containing 20% shrimp scales extract.

2.2.6 Blood sampling

Blood samples were collected in all experimental groups at the conclusion of each

experiment using the retro orbital method, using microcapillary glass heparinized vials, after a 12-hour fast. Dry, spotless centrifuge tubes were used to capture blood samples, which were then allowed to clot in water at 37°C for 20 minutes. This was accomplished by centrifuging the blood at 3000 r.p.m. for 10 minutes to separate the serum. In accordance with Schermer's guidance, the serum was meticulously aspirated into a sterile cuvette tube and stored at -20°C for analysis [15].

2.2.7 Biochemical analysis

The collected samples were analyzed to determine blood glucose, and glycated hemoglobin levels according to Trinder [16] and Nathan et al [17]. Alanin amino transferase (ALT) activity was determined calorimetrically according to the method of Tietz [18]. The activity of aspartate amino transferase (AST) was assessed calorimetrically using Henry's method [19]. ALP was determined in serum in accordance with the IFCC [20]. The serum creatinine was quantified using the kinetic method, as per Young [21]. The maximal absorbance was measured at 578 nm through photometric analysis of an albumin bromocresol green complex that was formed at pH 4.2 [22]. Uric acid was quantified through an enzymatic colorimetric assay on products that were utilized in accordance with Baraham and Trinder [23]. Serum urea was determined enzymatically in accordance with the Patton and Coruch method [24]. The methods of estimation were employed to estimate immunoglobulin productions Anna et al [25]. The determination of thyroid stimulating hormone (TSH) underwent the following procedure Uotila et al [26]. Radioimmunoassay (RIA) was employed to estimate thyroid hormones (free T4 and free T3) in serum, as indicated by Patrono and Peskar [27]. The determination of leptin hormone was conducted in accordance with Cosidine et al [28] and determination of

insulin hormone carried out according to Defronzo et al. [29].

2.2.8 Statistical Analysis

Accepted statistically in accordance with the method outlined by Steel and Torrie [30], the markers' levels were analyzed using the least significant difference (LSD). Significant differences at $p \leq 0.05$ are indicated by means with varying superscript letters for the same analysis.

3. RESULTS AND DISCUSSION

Blood glucose in diabetic rats received glucosamine and its source is illustrated in Table (1). It might be observed that the mean value in the first week was 98.51 ± 1.21 mg/dl while in the 4th week, it was 89.81 ± 0.87 mg/dl for negative control group with significant differences. In case of positive control group, the blood glucose levels changed during the experimental time, it was 269.5 ± 6.58 mg/dl in the first week while it was increased in the fourth week which being 302.11 ± 4.92 . As regards the treated groups, all groups were significantly higher than the negative control group and significantly lower than the positive control group. There is no significant difference between groups 3 and 5 in the first week while in the fourth week there were significant differences between the treated group between each other. The highest effect was recorded in the group administrated with levels of 3mg glucosamine and its source 20% shrimp scales which being 180.55 ± 4.53 and 202.6 ± 2.78 mg/dl respectively. These results are in agreement with those reported by Monauni et al. [31], who stated that glucosamine provided significant protection and reduced blood sugar levels Robertson et al. [32] suggested that, glucose lowering effects are most often associated with the high-level dose of glucosamine which effect on glucose and insulin levels. Another important report indicated that glucosamine could

reduce glucose in diabetic rats to near the normal level [33].

Table (1): Effect of administration of glucosamine and shrimp scales on blood glucose of diabetic rats

Blood glucose	Animal groups						LSD
	Negative control G1	Positive control G2	Glucose amine (1.5mg) G3	Glucose amine (3mg) G4	Shrimp scales (10%) G5	Shrimp scales (20%) G6	
1st week	98.5e ±1.21	269.5a±6.58	260.5b±5.48	252.2c±34.58	263.7b±6.14	259.5c±2.58	7.98
2 rd week	95.2e±3.98	288.7a±4.52	250.7b6.1±1	240.2d±4.16	252.4c±2.33	242.5d±4.55	5.87
3rd week	90.7d±1.34	292.3a±5.78	238.7b±1.6	224.5c±1.03	240.7b±3.2	230.7c±3.4	6.32
4th week	89.8f±0.87	302.1a±4.92	210.2c±4.62	180.5e±4.53	223.9b±3.39	202.6d±2.78	5.03

Values are mean±SD. Values in the same row sharing the same superscript letters are not statistically significantly different at ($p \leq 0.05$).

Glycosylated Hb in diabetic rats received glucosamine and its source is illustrated in Table (2) . It could be observed that there were significant differences between both controls during the experimental period . In comparison to the negative control group, the treated groups demonstrated a significant increased, while demonstrated a significant decreased in the positive control group. There is no discernible difference between groups 2 and 5 in the first week, but there were significant disparities between the treated groups in the fourth week. The highest effect was recorded in the group administrated with

levels of glucosamine 3mg and its source 20% shrimp scales . Studies that evaluated the effect of glucosamine on the HbA1c level, instead of fasting plasma glucose, had a variation in this measurement making it hard to compare the HbA1c levels. In the United States the HbA1c level is already used to diagnose diabetes mellitus and it can be used in studies to evaluate the glucose status. Considering this variance, a new variable was made, in contrast with previous studies, to analyze the effect of glucosamine on obtaining a high HbA1c level and/or new-onset diabetes mellitus over 6.5 years [31,33].

Table(2):Effect of administration of glucosamine and shrimp scales on Glycosylated Hb of diabetic rats

Glycosylated Hb	Animal groups						LSD
	G1	G2	G3	G4	G5	G6	
1st week	5.40e±0.13	11.09a±1.05	10.58b±1.21	8.63d±0.05	10.68a±0.78	9.71c±0.08	1.01
2 rd week	5.21e±0.04	11.12a±0.87	10.03b±0.64	8.58d±0.73	10.33b±0.05	9.55c±0.01	0.57
3rd week	5.26e±0.67	11.34a±0.57	9.52c±0.52	8.03d±0.71	10.01b±0.15	9.34c±0.23	0.34
4th week	4.97f±0.85	11.97a±1.51	9.01c±0.57	7.42e±1.35	9.64b±1.21	8.75d±1.05	0.22

Values are mean±SD. Values in the same row sharing the same superscript letters are not statistically significantly different at ($p \leq 0.05$).

The effect of acquired different levels of glucosamine and its source on liver functions is shown in Table (3).There were significant differences between positive and negative control groups ($p \leq 0.05$) for tested liver enzymes. For AST , there were significant differences among the other groups with both control groups. However, there is no significant difference between the negative control group and groups 4 that received 3mg glucosamine. The other groups recorded

significant differences with both control groups. For alanine amino transferase (ALT), the negative control group showed a level of 30.08 ± 0.31 U/L while positive control group presented 44.09 ± 1.51 U/L with significant differences. There is no statistical difference between groups 3 and 5 also, the results in the table showed no statistical difference between group 4 and negative control group. The other groups had statistical differences with both control groups. In case of alkaline phosphates

(ALP), the negative control group showed a level of 80.12 ± 0.97 U/L while positive control group presented 112.37 ± 2.41 U/L. All treated groups showed a lower statistical difference compared to the positive control group. However, there is no statistically significant difference between groups 3 and 5, nor is there a significant difference between groups 4 and 6.

This rise in serum ALP activity is predominantly the result of the enzymes' leakage from the liver cytosol into the circulation, which is a sign of the hepatotoxic effect of alloxan treatments. It was observed that the activity of aspartate aminotransferase (AST) and alanine transaminase (ALT) was

significantly elevated in diabetic rats, resulting in a 72.31% increase in serum ALP activity compared to normal levels. streptozotocin^{34,35&36}. These results are investigated by Russell and McCarty³⁷ who mentioned that, glucosamine affected of the activities of aspartame amino transferase (AST), alanine amino transferase (ALT), alkaline phosphates (ALP), and they were significantly decreased in both plasma and liver. Also, Ta-Liang³⁸ showed that glucosamine can improve the functions and kept them near to the normal levels. While, Jawed et al. ³³ stated that there is no effect of glucosamine on the liver activity.

Table (3). Effect of administration of glucosamine and shrimp scales on liver functions of diabetic rats (U/L).

Parameter	Animal groups						LSD
	G1	G2	G3	G4	G5	G6	
AST(U/L)	27.91e±0.27	42.2a±0.11	35.75c±0.15	28.13e±0.56	38.52b±0.21	33.13d±0.50	2.31
ALT(U/L)	30.08d±0.31	44.09a±1.51	39.57b±4.25	31.87d±2.52	40.54b±2.5	34.24c±2.51	1.99
ALP (U/L)	80.12d±0.97	112.37a±2.41	101.55b±1.35	93.07c±0.16	103.67b±2.16	95.01c±2.01	3.75

Values are mean±SD. Values in the same row sharing the same superscript letters are not statistically significantly different at ($p \leq 0.05$).

The effect of admitted different levels of glucosamine and its source on kidney function is illustrated in Table (4). For creatinine, the negative control group exhibited a level of 0.69 ± 0.01 mg/dl, while the positive control group exhibited a level of 1.96 ± 0.01 mg/dl. Groups 3 and 5 did not exhibit any statistical differences. All groups significantly showed higher values than negative control group and lower than positive control group. The group supplemented with 3 mg glucosamine followed by 20% shrimp scales significantly had high effect on creatinine levels as compared with the other treatments. to the negative control group, with the exception of the third and fifth groups, which were administered 1.5mg glucosamine and its source. These groups did not exhibit any significant differences. The negative control group exhibited a level of 3.84 ± 0.04 mg/dl for albumin, while the positive control group exhibited a level of 2.81 ± 0.08 mg/dl. The

statistical difference between all categories was less pronounced. Between groups 3 and 5, there is no statistically significant difference in uric acid. Similarly, there is no significant difference between groups 4 and 6. However, all tested groups exhibited a statistically significant decrease when compared to the negative control group. Negative control group exhibited a level of 25.2 ± 2.05 mg/dl in response to urea nitrogen, whereas positive control group exhibited a level of 48.46 ± 1.51 mg/dl. Between Groups 3 and 5, no significant disparities were found. In comparison to the positive control and tested groups, the value of group 4 that received 3mg glucosamine was lower; however, it was still higher than that of the negative control group. Mentioned results in the presents study showed that, the treatment of glucosamine decreased blood glucose and blood urea levels and had no effect on the serum proteins and albumin levels^{39&40}. Also, in the study of 41 showed

that glucosamine can reduce serum urea and creatinine levels and confer a protective effect on the kidney.

Table (4). Effect of administration of glucosamine and shrimp scales on kidney functions of diabetic rats (mg / dl)

Parameters	Animal groups						LSD
	G1	G2	G3	G4	G5	G6	
Creatinine (mg/dL)	0.69e±0.01	1.96a±0.01	1.77b±0.09	1.43d±0.04	1.82b±0.08	1.67c±0.02	0.11
Albumin (mg/dL)	3.84a±0.04	2.81e±0.08	2.98d±0.02	3.22b±0.01	2.91d±0.05	3.09c±0.06	0.09
Uric Acid (mg/dL)	2.35d±0.05	2.95a±0.02	2.75b±0.05	2.56c±0.03	2.82b±0.09	2.65c±0.11	0.12
Urea Nitrogen (mg/dL)	25.2e±2.05	48.46a±1.51	40.77b±2.07	30.18d±1.26	42.76b±1.34	34.21c±1.17	2.11

Values are mean±SD. Values in the same row sharing the same superscript letters are not statistically significantly different at ($p \leq 0.05$).

Data in table (5) shows the effect of different levels of glucosamine and its source on insulin hormone, insulin resistance and leptin hormone. Serum insulin hormone level in negative control group was significantly higher when compared with diabetic rats. Meanwhile, the effect of different levels of glucosamine and its source on hormone level for diabetic rats were improved compared to control (+) group. There were significant differences among tested groups with both control groups. Yeonhee et al. 42 discovered that glucosamine tended to elevate insulin secretion and/or induce insulin resistance in peripheral tissues, resulting in a decrease in blood glucose levels. Additionally, another study Cornelio et al., 43 Glucosamine, which is a result of interference with glucose utilization in pancreatic cells, was found to decrease insulin secretion. Therefore, it can decrease the impact of an HFD on the metabolism of carbohydrates and lipids, thereby postponing the induction of IR.

According to the same table, the level of insulin resistance was increased in positive control group when compared to negative one, while their level decreased by increasing

the levels of treatment glucosamine. The decreasing was significant as compared to negative control group. Yeonhee et al. 42 showed that glucosamine is a natural component involved in the synthesis of glycosaminoglycans, which are important for cartilage health. Some researchers hypothesize that glucosamine might alter carbohydrate metabolism through its effects on cellular mechanisms, such as the hexosamine biosynthetic pathway

As for the level of serum leptin hormone for positive control showed significant differences as compared with the other groups. Statistically significant differences were observed in the progressive decrease of hormone levels as the supplement level increased. Glucosamine appears to assist in the prevention of bone deterioration by promoting the growth of healthy bones. Hyaluronic acid, chondroitin sulfate, and keratan sulfate are the most significant components of the extracellular matrix of the articular cartilage and the synovial fluid, in addition to collagen fibers. Consequently, it is indispensable for their production³³.

Table (5): Effect of administration of glucosamine and shrimp scales on insulin hormone, insulin resistance and leptin hormone of diabetic rats

Parameters	Animal groups						LSD
	G1	G2	G3	G4	G5	G6	
Insulin hormones (IU/ml)	11.5±1.23a	6.11±0.07f	6.65±0.97d	7.05±1.11b	6.37±1.08e	6.94±2.03c	0.03
Insulin resistance (HOMA-IR)	2.55±0.21f	4.56±0.55a	3.20±0.12 d	3.14±0.05e	3.52±0.09b	3.47±0.11c	0.02
Leptin hormones (ng/mL)	22.89±3.81f	43.54±0.86a	35.86±0.07c	28.34±0.07e	38.65±0.07b	31.87±0.07d	2.11

Values are mean±SD. Values in the same row sharing the same superscript letters are not statistically significantly different at ($p \leq 0.05$).

Data in table (6) revealed the effect of levels of glucosamine and shrimp scales on immunoglobulin productions . For IgE, IgM and IgA levels, in contrast to the negative control group, the effect of varying levels of glucosamine and its source was significantly reduced, while it was significantly increased in the positive control group. There was no significant difference between groups 3 and 5, and the same effect was observed between groups 4 and 6. The negative control group exhibited a high level of IgG, while the positive control group recorded the lowest level. The means of glucosamine and its source at 3mg and 20%, respectively, are not statistically significant. Glucosamine has

immunosuppressive properties and may be advantageous as an immunosuppressive agent⁴³ the anti-inflammatory effects of glucosamine may be attributed to an inhibitory effect on the activation of the transcription factor nuclear factor kappa B (NFκB). NFκB is responsible for the activation of genes that encode inflammatory molecules, including inducible nitric-oxide synthase, IL, and immunoglobulins. Their findings demonstrated that GlcN can inhibit the bioactivity of unbound TNF-α, thereby demonstrating an alternative mechanism by which GlcN exerts its anti-inflammatory effects⁴⁴.

Table (6): Effect of administration of glucosamine and shrimp scales on immunoglobulin productions of diabetic rats

Parameters	Animal groups						LSD
	G1	G2	G3	G4	G5	G6	
IgE IU/mL	64.17a ±0.05	52.5±0.2d	54.76±0.05c	62.99±1.11b	55.87±1.56c	60.54±1.05b	2.33
IgM mg/d	108.2a±0.005	61.65d±0.65	75.66c ±0.96	89.66 b ±1.07	77.33 c ±2.09	87.33b±10.96	2.12
IgA mg/d	111.1a±0.1	75.5d ±0.5	83.5c±1.5	91.66 b ±6.02	80.5c±1.5	88.33b±2.08	3.05
IgG mg/d	1100.0a±0.05	748.2e±2.76	875.9c±3.85	908.6b ±6 0.27	776.6d±5.16	901.3b±2.85	7.87

Values are mean±SD. Values in the same row sharing the same superscript letters are not statistically significantly different at ($p \leq 0.05$).

4. CONCLUSION

From the obtained results, it was concluded that shrimp scales as waste products may play a role in reduction the blood glucose , improve the insulin level and reduce the insulin resistance for may patients as diabetic people and this led to contain glucosamine at the level 20 mg/100 shrimp scale extract , also, it can promote the liver, kidney and immunity functions .

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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تأثير الجلوكوزامين ومصدره (قشور الجمبري) على بعض القياسات البيوكيميائية للفئران المصابة بالداء السكري

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الملخص العربي:

هذه الدراسة قامت بتقييم تأثير مستويين من الجلوكوزامين ومصدره على صورة مستخلص قشور الجمبري على بعض وظائف أعضاء الفئران المصابة بالسكري. اثنان واربعون فار البيينو بالغ تم تقسيمهم الى ستة مجموعات (كل مجموعة تحتوى سبع فئران) كاتالى : مجمزة(1) وقد تغذت على الوجبة الضابطة كمجموعة ضابطة سالبة . المجموعات الاخرى تم حقنها بالالوكسان (150 ملجم لكل كجم من وزن الجسم) لاحداث الاصابة بالداء السكري. احدهما تم الاحتفاظ بها كمجموعة ضابطة موجبة بينما المجموعتان 4و3 حصلت على الوجبة الضابطة مع 1.5 و 3 مجم جلوكوزامين والمجموعات 5 و6 تغذت على الوجبة الضابطة تحتوى على 10 و20% مستخلص قشور الجمبري على التوالى لمدة 28 يوم. تم تقدير جلوكوز الدم، انزيمات الكبد، وظائف الكلية ، والدلائل المناعية . وقد اشارت النتائج الى تحسن جلوكوز الدم ، انزيمات الكبد ، وظائف الكلية ، هرمون الانسولين والمواد المناعية . وقد وجد ان المستوى العالى من الجلوكوز ومصدره ادى الى تأثير اعلى . ولذلك تؤكد الدراسة ان مصادر جلوكوزامين لها تأثير عالى للوقاية ضد مرض السكر.

الكلمات الكاشفة: الجلوكوزامين، قشور الجمبري، الكبد، وظائف الكلية، الفئران المصابة بالسكري

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