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IMPACT OF CAMEL MILK AND ITS PROTEIN FRACTIONS ON DIABETES IN ALBINO MALE RATS

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ABSTRACT

In this study Forty-eight pathogen-free adult male albino rats weighing (120±10 g) aged 4-6-week-old were divided into 6 groups, T1 as negative control (normal group; T2 as positive control (diabetic group; T3 diabetic group supplemented with camel milk; T4 diabetic group supplemented with camel milk whey proteins; T5 diabetic group supplemented with Diamicron drug and T6 diabetic group supplemented with camel casein. The results revealed a significant difference ($P < 0.05$) between diabetic group (T2) and treated groups T3, T4 and T5. However, there was no significant difference between the treatments T6 and diabetic group (T2). Moreover, T3, T4 and T5 reduced blood glucose with no significant differences when compared to normal group (T1). There was an effect of treatments (T3, T4 and T5 ($P < 0.05$) on body weight gain. Liver functions (ALT and AST) and kidney function (creatinine) replenished to almost the normal status at treatment with camel milk, camel milk whey proteins and drug treated groups. The histopathological sections of pancreatic tissues from groups T3, T4 and T5 showed no presence of degeneration, inflammatory cells, and necrosis. The data obtained in this study clearly show that camel milk and camel milk whey proteins significantly reduced blood glucose level and can be used as adjunctive therapy in type 2 diabetes mellitus.

Key words: Streptozotocin, Diphenyl-2-picrylhydrazyl, Total red blood cells, Total number of platelets, Mean corpuscular volume, White blood cells, Alanine amino transferase, Aspartate amino transferase

INTRODUCTION

Diabetes is a chronic metabolic disorder characterized by high blood glucose levels resulting from either defect in insulin secretion, or insulin action, Glucose homeostasis is achieved through the coordinated actions of glucagon hormone (secreted by α-pancreatic cells), which increases blood glucose, and insulin hormone (secreted by β-pancreatic cells), which decreases blood glucose. The prevalence of diabetes is increasing worldwide, with an estimated 463 million adults living with the disease in 2019, The total number is expected to surge to 552 million by 2030 **(Whiting *et al.* 2011).** Despite advances in treatment, diabetes remains a major public health concern due to its associated complications, including cardiovascular disease, blindness, kidney failure, and amputations. Oral hypoglycemic drugs and insulin are used to treat T2DM, but they are ineffective in terms of preventing the development of macrovascular complications and frequently cause side effects such as hypoglycemia, weight gain, and gastrointestinal intolerance. Thus, implementing nutritional strategies to mitigate diabetic complications would be extremely beneficial. In consequence, compounds of natural sources such as camel milk or its derivatives are more desirable as they are thought to possess lower risk of negative side effects when compared to the synthetic drugs.

Camel milk has been traditionally used in many countries as a natural therapy for various diseases, including diabetes mellitus. Studies have shown that camel milk contains bioactive compounds that have anti-diabetic properties, such as insulin-like proteins and peptides, which may help to regulate blood sugar levels. Additionally, camel milk is also low in lactose and cholesterol, which makes it a suitable alternative for people with diabetes who are lactose intolerant or have concerns about their lipid profile. The growing body of evidence suggests that camel milk may be a promising therapy for individuals with diabetes, and further research is warranted to explore its potential as a complementary or alternative treatment option. While more research is needed to fully understand the potential benefits of camel milk as a natural therapy for diabetes, preliminary studies suggest that it may have promising therapeutic effects. The aim of current study is to investigate the direct effect of whole camel milk, camel milk whey proteins and camel milk casein on blood glucose levels and changes in histological architecture of pancreas organs in diabetic rats.

Materials.

Analytical reagents: Unless otherwise specified, all chemicals used in this study were of analytical grade. Streptozotocin (STZ) and DTNB (5,5' dithiins 2 – nitrobenzoic acid) were obtained from Sigma Chemicals Co. (St. Louis, MO, USA). STZ was dissolved in cold 50mM citrate buffer (pH) 4.5and was always freshly prepared for immediate use. Diphenyl-2-picrylhydrazyl (DPPH) from (BDH company). Camel milk samples were obtained from private farm from Halayeb & Shalateen - Aswan- Egypt and transferred in ice tank with the following composition.

Methods.

Physico-chemical analysis of camel milk:

pH, acidity, Moisture and Total solids, ash, fat, and proteins were determined as described in **AOAC, (2005)**.

Camel casein preparation. Camel casein was prepared according to method described by **Al-Saleh *et al.* (2014).**

Preparation of un-denatured (native) camel milk whey proteins.

The method was followed according to **Ebaid *et al*. (2015).**

Animals and experimental design: A total of 48 sexually mature male albino rats, weighing between 80 and 120 g and aged 4-6 weeks, were gotten from the Faculty of Pharmacy's Central Animal House at Bani sweif, derayah University, Egypt. The animal experiments were designed and performed in accordance with the ethical standards approved by the e Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA) and the National Institutes of Health (NIH). Before the experiment, the rats were given two weeks to get used to the wired cages in a well-ventilated room. During this period, they were kept in normal healthy conditions such as a temperature of 23°C, relative humidity of 60-70%, and a 12-hour light/dark cycle. They were fed with standard commercial pellets and always allowed access to water. To help them adjust, the rats were fasted for 8 hours before the experiment.

Experimental design:

Forty-eight pathogen-free adult male albino rats weighing (120±10 g) aged 4-6-week-old were divided into six groups as follow, T1 as negative control: receiving normal diet, T2 diabetic rats (untreated) as positive control, T3 diabetic rats. supplemented with camel milk (8.3ml/ kg of body weight), T4 diabetic rats supplemented with camel milk whey proteins (100 mg/kg of body weight), T5 diabetic rats supplemented with Diamicron drug, (1mg/kg body weight), T6 diabetic rats supplemented with camel milk casein (500mg/kg of body weight).

Induction of diabetes

Diabetes was induced into experimental rats (Albino type) by a single intraperitoneal injection of freshly dissolved STZ (60 mg/kg of body weight) in 50 mM/l citrate buffer (pH 4.5) according **to Ebaid, *et al*. (2015)**. Control rats (control negative) were injected with citrate buffer. Rats were screened for analyze blood glucose from a tail-vein using commercially available glucose detector (ON CALL PLUS, Auto code A-334, Germany). On the third day, the rats were considered diabetic if their blood glucose level ≥ 200mg/dl.

Body weight

Body weight was recorded at the beginning, weekly and the end of the study (8 weeks) in all the experimental groups.

Collection of blood samples

At the biggening, 30 days and at the end of the experiment period, the overnight fasted animals (the control and experimental animals) were removed from the cage then sacrificed under mild ether anesthesia. Blood samples were collected individually from the jugular vein of each rat in tubes containing EDTA (ethylenediamine- tetra acetic acid) as an anticoagulant) for CBC. Blood samples for serum were also obtained individually from each rat's jugular vein in simple tubes (non-heparinized tubes) and allowed to clot at

room temperature. The serum was then separated by centrifugation for 10 minutes at 4000 rpm. Aliquots of serum were extracted and kept at -20°C in Eppendorf tubes until use. To analyze the biochemistry of the liver, levels of Alkaline phosphatase, creatinine, AST, and ALT were measured after taking a blood sample. Sections of liver tissue were collected, with one part being preserved in 10% formalin for histology research, and the remaining parts being stored at -20°C for examining the levels of oxidative stress and antioxidant parameters.

Organ weight.

After the collection of the blood samples, the pancreas was removed and weighed and expressed as relative weight using the formula:

$$\text{Relative organ weight} = \frac{\text{absolute organ weight}}{\text{body weight at sacrifice}} \times 100\%$$

Histopathology

Pancreas organs were collected from the slaughtered rats after 4 weeks and 8 weeks of the experiment. The organs were prepared according to **Ibrahim *et al.* (2018**) using Hematoxylin and Eosin staining dye.

Hematological analysis

Hematological parameters included total hemoglobin concentration (Hb) (gm/dl), Total red blood cells (RBCs) count, Total number of platelets (PLT). Hematocrit (HCT), Mean corpuscular volume (MCV), White blood cells (WBC) were measured by a machine (ABAXIS, VET Scan HM5, Union City, California, United States) that automatically determined a Varity of blood tests referred to as the total blood count (CBC).

Determination of serum ALT, AST, Alkaline phosphatase, and creatinine levels.

Glutamic – oxalacetate transaminase GOT activity (AST), Glutamic – pyruvic transaminase GPT activity (ALT) and creatinine were measured using commercial kits (Centronic, GmbH Chemical Company (Germany) according to the manufacturer's instructions.

Statistical analysis

MINITAB software (Version 19, 2020), developed by MINITAB in State College, PA, was used to conduct statistical analysis. To ensure the validity of further statistical analysis, Anderson Darling test was employed to test the normality of data, and the homogeneity of variances was also examined. Since the data was found to be normally distributed and variances were homogeneous, One-way ANOVA was performed to determine the overall effects of treatments. Tukey's Pairwise comparison test was then utilized to compare the treatments individually. The mean was used to express the data, and statistical significance was considered at $P< 0.05$.

RESULTS AND DISCUSSION:

Body Weight Changes (BW)

Increase in the rat BW, reflects the good health and well glucose metabolism that led to the storage of energy as fatty tissue whether in the control groups or the treated diabetic animals. In contrast, weight loss reflects disturbance of glucose metabolism because of diabetes and insulin deficiency, and the animals tend to the consumption of body fat and protein as sources of energy, leading to

BW loss **(Korish, *et al*., 2020).** As figure (1) shows, all study groups had comparable (P > 0.05) initial BW at week 0 of the study (193.3 - 199.30 gm). However, after 8-weeks, groups T1, T3, T4 and T5 showed a comparable (P > 0.05) increase in BW (327.0, 302.08, 362.87 and 345.00g), which were 66.58, 54.87, 87.54% and 73.11 % higher (P < 0.05) than their initial BW (196.30, 195.05, 193.49 and 199.30 gm). However, the T2 group (diabetes group) gained just 29.49% of their initial BW by the end of the study. (193.3 gm). These findings indicate that the healthy control animals and treated groups (T3, T4 and T5) gained more weight with camel milk, camel milk whey proteins and Diamicron drug. In contrast, group T6 (casein treated group) showed an approximately 21.58% weight loss after 8 weeks of the study compared with initial BW (190.0 vs. 149.0 g; P < 0.05). These findings indicated that glucose metabolism significantly improved in the diabetic groups supplemented with camel milk and whey proteins of camel milk and helped animals to resist the diabetes-induced metabolic disorder. The T6 group showed a significant decrease in final BW compared with initial BW (149. 0 vs. 190. 0 g). This may be due to the higher levels of blood glucose in this group **Table (2).** These findings showed that at the end of study, the diabetic groups treated with camel milk and whey proteins of camel milk (T3 and T4) were very close to control (T1) and to Diamicron treated group (T5).

The changes in organs weights of untreated and treated experimental animals are sensitive to evaluate the benefit or the harmful effect of test compounds either natural or synthetic **(Foster, *et al*., 2019).** However, the absolute value of organ weight, is meaningless because it is determined by the total weight of the animal. So, the relative organ weight should be used. Our data in **Table (3)** showed a significant difference in the relative weights of the kidney, liver, spleen, heart, and testis among the groups. There was a significant increase in the relative weights of the kidney and liver of the diabetic group compared with normal group. These results are in accordance with those obtained by **Habib, (2018) and Foster, *et al.* (2019).**

The main reason for the renal hypertrophy of the diabetic rats is the changes in nephropathy which increase the size of both glomerular and proximal tubular epithelial cells **(Habib, (2018).** After 30 days the relative liver weight significantly increased in diabetic group compared with that of the normal group and other treated groups (T3, T4, T5 and T6). **Ren, *et al.* (2017)** reported that the relative liver weight of diabetic rate (4.50 ± 0.23%) significantly increased compared with normal group (2.91 ± 0.48%). The same trend was confirmed by **Foster, *et al.* (2019).** They found the relative weight of liver in STZ rats increased significantly compared to that of control rats. Furthermore, the relative weight of spleen in diabetic rats significantly decreased (0.40 %). The mean weights of the spleen were significantly higher in the control and treated groups than in the diabetic group (T2). These findings agree with those obtained by **Ebaid, *et al.* (2015).** They observed a decrease in relative weight of diabetic rats compared with control and whey protein treated group. The decrease of relative weight of diabetic rats was

attributed to depletion of the white pulp, dilation in the blood vessels and increase of the collagen deposition. Concurrently, the relative weight of heart of the diabetic rats is significantly higher (0.47 %) than that in normal rats (0.34%). The same trend was reported by **Ferdous, (2016)** who stated that the relative weight of Wistar rats 10-12 weeks of age pointed out 4.19±0.48% in STZ rats compared to 3.59±0.45 in control rats. However, the absolute heart weight of control rats was higher (1.150±112 gm) than that of STZ rats (0.850±13 gm). The increase in relative heart weight of STZ rats was attributed to weight loss due to diabetes mellitus **(Ferdous, 2016).** Nevertheless, **Foster, *et al.* (2019)** reported that no significant differences between the heart weight of control rats (1.36 ± 0.06 gm) and that of STZ rats (1.30 ± 0.05 gm).

In addition, the relative weight of the pancreas was significantly higher in the control and treated groups of rats when compared to the untreated diabetic rats. However, there was no significant difference between the pancreatic relative weights of the diabetic rats and that of the rats treated with camel milk casein **Table (3).** These findings agree with those obtained by **Adeyemi, *et al.* (2010).** They attributed the significant decrease in pancreas of diabetic group to breakdown, degeneration, necrotic changes, and shrunken in the pancreatic islet of Langerhans, as well as to β-cell degranulation, decreased islet cellular density, and severe vacuolation. No significant changes in relative weight of thymus gland in control and diabetic (0.25 and 0.22% respectively) rats were observed (p values were 0.084). **Oishi, *et al.* (1979)** reported 0.23% thymus relative weight (% bw). Additionally, the relative weight of testis significantly decreased in diabetic rats (0.61 %) compared with control group (1.26%). The tests of diabetic groups supplemented with camel milk, whey proteins of camel milk and Diamicron drug restored to the normal group.

Changes in blood glucose

The diagnosis of diabetes has been based on glucose criteria, either the fasting blood glucose (FPG) ≥ 126mg/dL (7.0 mmol/L) in plasma or the 75-g oral glucose tolerance test (OGTT) ≥200 mg/dL (11.1 mmol/L).

Fasting blood glucose changes during the experiment are shown in **Table (4)**. The findings indicated that there were no discernible distinctions between the treatments and control group initially, with blood glucose levels ranging from 84.33 to 93.67 mg/dl. However, the study demonstrated that both the treatment (P=0.01) and time (P<0.031) had a significant impact on blood glucose over an 8-week period, as shown in Table (4). Following the injection of streptozotocin at a dose of 60 mg/1kg body weight, there was a substantial ($p < 0.05$) rise in blood glucose levels from 90.33 to 340.7 mg/dl for non-diabetic (T1) and diabetic (T2) rats at the zero time. Additionally, stable diabetes was observed after three days with average mean values 304.0, 312.7, 300.0, and 423.7 mg/dl in all other groups (T3, T4, T5 and T6 in order). After treatments, the results showed statistically significant decrease ($P< 0.05$) in serum glucose of the camel milk (T3), whey proteins (T4) supplemented groups and drug treated group (T5) as

compared with buffer-loaded untreated group (T2) and casein treated group (T6). On the other hand, there was no significant difference (P> 0.05) between camel milk (T3), whey proteins (T4) supplemented groups and drug treated group (T5). This observation is very important because it ensures that camel milk and its whey proteins can be used as alternative for medicine drugs. However, serum glucose of casein treated group increased significantly (*P*< 0.05) when compared with other treated groups. The blood glucose level of camel milk treated group decreased by about 22.21%. This is consistent with findings of **Agrawal, *et al.* (2007)** they reported that consumption of camel milk deceased insulin requirement by about 30%. Moreover, **Agrawal, *et al.* (2011)** stated that hemoglobin A1c levels decreased in patients with type one diabetes from 7.81±1.39 to 5.44±0.81% and consequently insulin doses decreased from 32.50±9.99 to 17.50±12.09 U/day. Insulin requirements reached to zero in three patients out of 12 with camel milk consumption. **Homoud, (2015) recorded** a reduction in blood glucose level by ~ 49% for camel milk, compared with 11.1 and 11.6 for buffalo and bovine groups respectively was reported. **Amjad, *et al.* (2013)** conducted a long-term study to assess the efficacy of camel milk as an adjunct to insulin therapy in type 1 diabetes. The results demonstrated that, camel milk consumption decreased the mean blood glucose, and insulin doses. Therefore, they concluded that camel milk is safe and efficacious to control glycemic mellitus, with a significant reduction in the doses of insulin in type 1 diabetic patients. Moreover, **Ejtahed, *et al.* (2015)** studied the effect of camel milk consumption in comparison with cow milk (500 ml/day for two months) on insulin levels in the blood of patients with type 2 diabetes. They found consumption of camel milk significantly increased concentration of insulin from 64.59 to 84.03 pmol/l and may contribute to glycemic control for type 2 diabetes. However, **Almohmadi, (2020)** reported that camel milk does not contain insulin and the hypoglycemic effect of camel milk could be attributed to other insulinotropic hormones in milk. The author interpreted the absence of insulin in camel milk to use different insulin ELISA kits specified to human. Concurrently, diabetic group treated with whey proteins (T4) showed significant decrease in blood glucose (37.42%). These results are in concomitant with that obtained by **Badr, (2013)** who observed blood glucose decrease in diabetic mouse treated with camel whey protein from 411 ± 37 mg/dl to 261 ± 25.5 mg/dl (by about 36.5%) after two weeks. Meanwhil**e, Chiang, *et al.* (2022)** found that the concentration of blood glucose decreased by about −2.67 and about −1.59 mmol/L after 60 and 120 min respectively in patients with type 2 diabetes mellitus supplemented with whey proteins in comparison with placebo group. The secretion of insulin was significantly higher in WP treated group than in placebo group. The investigations of **Ebaid, (2013, 2014)** have shown that the camel milk whey protein has a powerful effect in reducing blood glucose levels. The glucose level decreased from about 550 mg/dl to 150 mg/dl after 5 weeks in rats supplemented with native camel milk whey proteins. The hypoglycemic effect of camel milk

could be attributed to many factors. According to **Khaliq, *et al.* (2020)** the effects of camel milk on diabetes can be attributed to its ability to reduce inflammation, prevent cell death, and act as an antioxidant, which can potentially stimulate the secretion of insulin from pancreatic β-cells. Additionally, consuming camel milk regularly can act as an antioxidant by promoting the production of antioxidant enzymes like catalase, lactoperoxidase, and superoxide dismutase (SOD) and non-enzymatic antioxidants such as glutathione, vitamins E and C. This antioxidant activity helps to eliminate free radicals, which in turn can promote insulin secretion. **(Badr, *et al.* (2011). Kilari, *et al.* (2021)** stated significant improvements in the hypoglycemic and hypolipidemic of rats fed with 500 mg/kg body weight of camel milk protein hydrolysate. They also observed a decrease in levels of liver function enzymes (aspartate aminotransferase; AST and alanine aminotransferase; ALT) with improvement in antioxidant activity (superoxide dismutase, catalase, and reduced glutathione). **Ebaid, (2013, 2014)** found that providing diabetic rats with camel milk whey protein helped to increase the levels of antioxidant enzymes such as glutathione (GSH), superoxide dismutase (SOD), and catalase (CAT) compared to diabetic rats that did not receive the treatment. The main whey protein found in camel milk whey is α-lactalbumin, as reported by **Al-saleh, *et al.* (2014). Yamaguchi, *et al.* (2014)** stated that the glucose intolerance in Wister rat group significantly improved with the administration of α-lactalbumin consequently, the blood glucose levels decreased at 30 and 60 min compared with the control group. They concluded that consumption of α-lactalbumin is effective in reducing the risk of type 2 diabetic mellitus. Interestingly, it has been stated that the α-lactalbumin from camel whey has several motifs of Trp-containing dipeptides that can be converted into effective DPP-IV inhibitory peptides.

Additionally, it has been reported that whey proteins contain a greater proportion of branched-chain amino acids (BCAA) including leucine, isoleucine, and valine compared to casein, leucine alone or BCAA alone cause a potent increase in protein synthesis in T2DM (**Bassil, *et al.*, 2013, Olowookere, 2021). Striffler, *et al.* (2001), Badawi, and Motawee, (2016)** concluded that camel milk supplemented with chromium improved serum blood glucose via enhancement effect of insulin on glucose metabolism through the enhancement of insulin receptors activity towered glucose utilization. The mean values of 193 ng/ml chromium in camel milk were reported by **Elhardallou, and El-Naggar, (2016)** in comparison with 10 ng/ml in cow milk **(Fox, and Mcsweeney, 1998). Karunakaran, and Park, (2013)** reported that prolonged hyperglycemia leads to pancreatic beta-cell dysfunction and increases the oxidative stress therefor, generating excess of free radicals causing imbalance between oxidants (free radicals) and endogenous antioxidants. Moreover, **Power, (2013)** stated that the antioxidant enzymes in diabetic patients are lower compared with non-diabetics. Camel milk has high

levels of antioxidants either enzymes or non-enzymes that help scavenging the free radicals. It has also high contents of zinc and magnesium which act as antioxidant defense and/or required for enhancement of antioxidant enzymes activity. Absorption of vitamins C and E increases in the presence of magnesium. It has been reported that super oxide dismutase enzyme (SOD) increased significantly in animals treated with camel milk **(El-Sayed *et al.*, 2011, Ebaid, *et al.*, 2013 and 2015). Bae, (2017)** reported that Glucagon-Like Peptide-1 (GLP-1) inhibits glucagon hormone causing glucose homeostasis. Moreover, Glucose- Dependent Insulinotropic Polypeptide (GIP) promotes insulin secretion **(Szablewski, 2011).** Both GLP-1 and GIP decrease the rate of gastric emptying into the small intestine, therefor, decreases the production of glucose **(Bae, 2017 and Rojas-Henao, 2018).** The concentrations of GLP-1 and GIP in camel milk are higher compared with bovine milk. **Almohmadi, (2020)** determined GLP-1 and GIP in raw and pasteurized camel milk in comparison with other types of milk. The results indicated that the level of GIP in camel milk ranged between 54.86 and 64.15 pg/ml compared with 40.46 pg/ml in bovine milk while, GLP-1 concentration in camel milk ranged from 62.09 to 74.41 pg/ml but not detected in cow milk.

Nauck, and Meier, (2018) reported that incretin hormones like GLP-1 and GIP (gut hormones) inhibit of dipeptidyl peptidase-4 (DPP-4), in addition they reduce the appetite leading to weight loss and glucose homeostasis. **Almohmadi, (2020)** stated that camel milk reduces insulin requirement in diabetes subjects via enhancement of insulin sensitivity and other insulinogenic proteins like leptin and visfatin. Leptin is a hormone produced mainly by adipose cells in the small intestine. Its primary function is to regulate energy balance by reducing hunger and controlling blood glucose levels. On the other hand, Visfatin is a protein mainly found in visceral adipose tissue but can also be present in the brain, kidney, lung, spleen, and testis. It serves various purposes, including exhibiting a hypoglycemic effect.

Camel milk contains high concentration of Visfatin 3961.68 to 3946.81 pg/mL compared with 325.75 pg/mL in bovine milk **(Almohmadi, 2020).** The author also found the concentrations of C-peptide (as marker for residual insulin secretion in type 1 diabetic patients) are high in camel milk (27.15 to 23.81 pg/mL) but not detected in cow milk. No significant differences were observed in glucagon concentration in camel and bovine milk. **Moreover, Ebaid, 2014 and Zhou, 2020** mentioned that expression of the protein kinase B (Akt1), Cdc42, and the co-stimulatory molecule, CD28 which are important for cell survival, actin polymerization and T lymphocytes (T cells) activation decreases in diabetic and pancreatic diseases patients. Diabetic rats fed with camel

whey proteins restored the Akt1, Cdc42 and CD28 mRNA expression to normal levels. Although interleukin (IL)-2 and interferon gamma (IFN-), which regulate lymphocyte activity, inhibit viral replication, and improve immunostimulatory and immunomodulatory systems, have been enhanced with camel milk whey

proteins. **Jakubowicz, and Froy, (2013)** attributed the antihyperglycemic effect of whey proteins to the bioactive peptides and amino acids generated during its gastrointestinal digestion. These amino acids (in particular branched amino acids) and bioactive peptides promote the secretion of gut hormones, such as GLP-1, GIP, peptide tyrosine- tyrosine (peptide YY) and inhibitors DPP-4 consequently, cause glucose homeostasis. However, no significant difference was observed between the casein treated group (T6) and control group.

According to Jonker and colleagues (2011), consuming 12 grams of casein hydrolysate can have a slight positive impact on insulin and glucose levels in individuals with type 2 diabetes. However, a lower dosage of casein hydrolysate (6 grams) did not produce any significant effects on glucose or insulin responses. These findings were contradicted with those obtained by **Cavalot, *et al.* (2006), Bonora, *et al.* (2006))** and **Koopman, *et al.* (2009),** they documented that the ingestion of relatively large amounts of casein hydrolysate ranging from 25 to 35 g enhanced insulin response and reduced serum glucose levels. Moreover**, Koopman, *et al.* (2009)** reported that the ingestion of casein hydrolysate as compared to its intact form significantly increase plasma amino acid concentrations, muscle protein synthesis rates and phenylalanine appearance rate. These effects may improve insulin secretion (**Nilsson, *et al.*, 2007).**

Complete blood count (CBC)

The Complete Blood Count (CBC) is a comprehensive test used as a general screening tool to detect various disorders, such as anemia, leukemia, and other illnesses. The CBC measures different components of blood, including red blood cells, white blood cells, hematocrit, platelets, hemoglobin, and other blood parameters.

Hemoglobin

Hemoglobin is an iron-containing protein that carries oxygen from the lung (respiratory organ) to the rest of the body and carries carbon dioxide back to the lung. Hemoglobin levels in blood are useful in diagnoses of blood disorders and other conditions. The normal level of hemoglobin differs from 13-17 and from 11.5 - 15.5 g/dL for adult male and female respectively**(Lazarova, *et al.*, 2014).** Low or high hemoglobin levels indicate blood disorder. Low hemoglobin level reflects presence of anemia with symptoms of fatigue, Pale skin (pallor), weakness., dizziness, shortness of breath and cold hands and feet. While high hemoglobin level reflect congenital heart defects, kidney and lung diseases with symptoms of headachesm blurred or double vision, dizziness, htching, and blood clots. In general hemoglobin level around or less than 10 g/indicate presence of anemia **(El‑Khateeb, *et al.*, 2014).** Data in **Table (5)** show levels of hemoglobin in control, diabetic and treated samples.

The results revealed a decrease in hemoglobin levels from 16.5 to 13.9 g/dL in rats by the injection with STZ to induce diabetes mellitus. Then, oral fed with camel milk (T3), its whey proteins (T4) and drug treated group (T5) significantly increased the hemoglobin levels to 16.0, 16.1 and 16.0 g/dL respectively compared to 13.9 g/dL in

diabetic group (T2) at the end of experiment (60 day). No significant differences were observed between treated groups (T3, T4 and T5) and control group (T1). However, the hemoglobin of casein treated group (T6) showed a lower level (14,7 g/dL) than control and other treated groups. These results are in good agreements with those obtained by **El‐Khateeb, *et al.* (2014)** who found that a significant difference in hemoglobin levels between control group (15.0±0.64 g/dL) and STZ‐induced diabetic rats (10.3±0.31 g/dL) after 30 days. Also**, Mahmoud, (2013)** reported that the hemoglobin level in diabetic rats significantly decrease to 10.17 ± 0.08 g/dl compared to 15.33 ± 0.68 g/dl in normal group. The same trend was confirmed by **Erukainure, *et al.* (2013).** However, the results of hemoglobin level obtained by **Foster, *et al.* (2019)** showed no significant difference between control group (17.23 ± 0.3 g/dL) and untreated diabetic group (16.6 ± 0.2 g/dL).

Red blood cells and related indices Parameters

Red blood cells, or erythrocytes carry oxygen from lungs to the rest of the bodies, then they take carbon dioxide back to the lungs to be breathe out. Because hemoglobin is a protein found inside red blood cells, there is a positive relationship between red blood cell levels and hemoglobin. The results in **Table** (**6)** show a significant decrease in total RBC count in diabetic group (T2) compared with the control (T1). However, no significant differences in total RBC count level among three treatments T3, T4 and T5. Casein treated group (T6) did not show significant differences in the RBC level compared with the diabetic group (T2). These results were confirmed by the above recorded results of hemoglobin **Table (5)**. These findings were partially similar with those obtained by **Mahmoud, (2013)** who reported a significant decrease ($P < 0.05$) in RBC of diabetic rats (5.75 ± 0.21 x 10^6/µl) as compared to normal control rats (7.09 ± 0.15 x 10^6/µl). **El‐Khateeb, *et al.* (2014)** also reported that the RBC decreased in diabetic group to 6.15±0.17 10^6/µl compared with 8.33±0.24 10^6/µl in control group. However, **Foster, *et al.* (2019)** found no significant differences in RBC of diabetic group (8.95 ± 0.20 10^6/µl) and normal group (9.45 ± 0.21$0^6$/µl). The same trend was reported by **Najafzaheh, *et al.* (2012)** and **Arafa, *et al.* (2020)** they stated that the RBC were 7.5±0. 1 and 7.5±0:25 (10^6/µl) in normal and 7.6 ±0:07 and 7:9±0:15 (10^6/µl) in diabetic groups respectively.

There were no significant differences observed in the levels of mean corpuscular hemoglobin (MCH), red cell distribution width (RDW), and mean corpuscular hemoglobin concentration (MCHC) between the diabetic group (T2) and the other treated groups. MCH represents the average hemoglobin level within a red blood cell, while RDW measures the variation in red blood cells' size, and MCHC indicates the average hemoglobin concentration in red blood cells. Nevertheless, the obtained values were in the range of normal values **Jacob, Filho., *et al.* (2018)** reported values 16 -20 pg for mean corpuscular hemoglobin (MCH) for rats in age one -24 month. Our findings **Table (7)** were in the range 16.1- 18.6 pg. Concerning the mean corpuscular hemoglobin concentration (MCHC) the present data showed no

significant differences among the groups under studies (p=0.092). The reported values ranged from 30 -33.5 g/l. these results are in good agreement with those obtained by **Mahmoud. (2013**) who reported value 30.14 ± 0.57 and**, Jacob, Filho, *et al.*, 2018 and Foster, *et al.*, 2019)** they reported (values ranged from 30 – 35 g/dL. The obtained values for RDW showed instability and ranged from 12.4 to 16.1 %.

Hematocrit or HCT is the ratio of the volume of red blood cells to the total volume of blood sample. The mean normal values for adult male and female ranged from 38.3 to 48.6 percent and 35.5 to 44.9 percent respectively. The lower values indicate presence of anemia, many white blood cells and deficiencies in vitamin B_{12} or mineral. Results in **Table (10)** show the values of hematocrit for treated and untreated samples. No significant differences in), RDWC; p = 0.292), MCH; 0.060, (MCHC); p = 0.092), and (plt); p = 0.162), However, there are significant differences in red blood cell (RBC; p = 0.005); hemoglobin (HGB; p = 0.000); hematocrit (HCT; p = 0.005), and mean corpuscular volume (MCV; p = 0.020),

The following are different parameters used to evaluate various characteristics of blood cells: red cell distribution width (RDW), hemoglobin distribution width (HDW), white blood cell (WBC), red blood cell (RBC), hemoglobin (HGB), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), cell hemoglobin concentration mean (CHCM), mean platelet volume (MPV), and platelet count (PLT)..

Liver functions: Alanine amino transferase (ALT) and aspartate amino transferase (AST) activities

Alanine amino transferase (ALT) and aspartate amino transferase (AST) activities are enzymes that are released into the bloodstream because of hepatic cell necrosis. Therefore, they are used as indicators for hepatic damage **(El‑Khateeb, *et al.*, 2014).** ALT and AST enzymes are found mainly in the cytosol and mitochondrion of all cells of the hepatocyte and found in much smaller quantities in kidney, heart, and skeletal muscle cells **Kim, *et al.*, 2008 and Lin, *et al.* (2010).** The normal concentration values for ALT and AST in blood are 5 to 35 U /L and 5 to 40 U /L-respectively **(Liu, *et al.*, 2006).**

The activities of ALT and AST in serum of non‑diabetic (T1); diabetic (T2) and STZ‑treated diabetic rats (T3 – T6) were determined. The results clearly showed that the ALT and AST increased significantly from 28.55and 23.56 IU/L in non‑diabetic rats to 65.64 and 50.68 IU/L, respectively in diabetic group **Table (12).** The higher activities of ALT and AST in diabetic group suggest the presence of hepatocellular necrosis or liver cell injury.

Kim, *et al.* (2008) reported that the activity of ALT enzyme in the liver is about 3000 times higher than that of serum. As a result of hepatocellular necrosis or death, ALT is released from damaged liver cells, resulting in a dramatic increase in ALT. No significance differences were observed between the normal group (T1) and the STZ‑diabetic treated groups (T3 – T5).

The ALT values for T3, T4, and T5 were 36.86, 27.02, and 37.50 IU/L, respectively, and 33.69, 27.67, and 31.42 IU/L for AST. These results suggest that treatment with camel milk (T3) and whey protein of camel milk (T4) were effective for significantly reducing ALT and AST levels compared with diabetic group (T2) and were similar to group treated with Diamicron drug (T5). The restoration of AST and ALT activities to normal levels after supplementation of the STZ‐treated diabetic groups for 8 weeks with camel milk and whey protein of camel milk may be due to the prevention of the leakage of intracellular enzymes by their activities against liver cell necrosis. The reported values for ALT and AST in this study (28.55 and 23.56 IU/L in non‐diabetic and 65.64 and 50.68 IU/L in diabetic group) are partially similar with those obtained by **Al-Asmari, *et al.* (2014),** they got 29.15 ± 1.85 ALT (U/L) and 77.08 ± 2.5 AST (U/L) in control group. The authors also reported that the camel milk administration. restored these parameters to almost their normal range (31.65 ± 0.90 and 68.37 ± 3.53 U/L respectively), They attributed the amelioration effect of camel milk to the restoration of antioxidant enzymes activity (super oxide dismutase (SOD) and glutathione s- transferase (GST) and inhibition of lipid peroxidation. Also, **Hamad, *et al.* (2011)** reported that camel milk administration significantly improved the liver function (ALT and AST activities) of diabetic rats. The improvement reached to 41% for ALT and 38% for AST. Similar conclusion was submerged by **Khan, *et al.* (2012); Ebaid, *et al.* (2011), Kilari, *et al.* (2021).** However**, Cornejo-Garrido, *et al.* (2014)** reported higher values (59.0±2.9 U/L for ALT and 141.3±10.2 U/L for AST) in control rats. The corresponding values were 289.1±74.3 and 354.9±78.7 U/L respectively in diabetic rats. The high activities of these enzymes are also correlated with liver cirrhosis and cardiovascular diseases **(Kim, *et al.*, 2008).** The recorded values for ALT and AST significantly improved in STZ treated diabetic groups after 8 weeks. However, no significant changes were observed in diabetic group (T2).

Kidney function

Determination of serum creatinine was used as indicator for kidney functions. The results illustrated in **Table (13)** noticed that a significant increase in serum creatinine level of diabetic group (T2; 1.85 mg/dL) compared with 1.00 mg/dL in non-diabetic group (T1). At the end of experiment (8 weeks), the corresponding values were 2.12 and 0.97 mg/dL respectively. The increase in creatinine level of diabetic rats may be attributed to the toxic effect of streptozotocin on the kidney and to the metabolic changes that accompany diabetes. **Habib, (2018)** reported that long-standing diabetes/hyperglycemia may lead to major changes in renal structure associated with the deterioration of infiltration as a result of loss of nephrons or abnormal nephron function. The obtained results corroborate well with previous studies reported by **El‐Khateeb, *et al.* (2014).** They reported values 1.3±0.05 and 1.6±0.03 mg/dL in non-diabetic and diabetic group respectively at the beginning which reached to 1.2±0.01 and 2.3±0.08 mg/dL in order at the end of experiment (8 weeks). It could also be noticed from Table (??) that no

significance differences between normal group (T1) and treated rats with camel milk (T3); camel milk whey proteins (T4) and Diamicron treated group (T5) at the end of experiment period. On the other hand, serum creatinine value in casein treated group (T6) was comparable with that of diabetic group (1.90 against 1.85 mg/dL). Results also showed that the creatinine level gradually decreased during experiment period in normal and treated samples except for casein treated group.

Table (1): Chemical composition of camel milk used in the study.

PH	Acidity	Moisture	Total solids	Ash	Protein
6.5	0.155%	90.88%	9.2%	0.75%	3.55%

Table (2): Changes in body weight (in grams) in control and experimental STZ treated diabetic rats.

Weeks	Groups					
	T1	T2	T3	T4	T5	T6
0	196.3 a	193.3 a	195.05a	193.49a	199.30a	190.0 a
1	221.7 a	222.3 a	199.07b	205.31ab	210.11ab	188.7 b
2	244.7 a	226.3 a	225.38a	236.4a	243.80a	180.0 b
3	260.0 a	229.7 b	238.36b	271.86b	266.46a	168.0 c
4	286.7 a	234.0 b	249.81b	301.76b	284.05a	162.7 c
5	292.0 a	238.7 b	256.41b	314.77a	289.46a	159.3 c
6	300.7 ab	240.7 d	272.58c	320.32a	290.95bc	157.7 e
7	319.3 ab	244.3 d	292.64c	344.79a	312.46dbc	155.0 e
8	327.0 bc	250.3 d	302.08c	362.87a	345.00ab	149.0 e

Data indicate rat weights (grams) throughout the study; T1 control, T2 diabetic group, T3 diabetic group supplemented with camel milk, T4 diabetic group supplemented with camel milk whey proteins, T5 diabetic group supplemented with Diamicron drug and T6 diabetic group supplemented with camel milk casein. In the same raw values followed with the same letter means no significant differences between them at ($p<0.05$).

Table (3): Organs relative weights of un-diabetic and diabetic rats treated with camel milk and its derivatives or Diamicron drug.

Treat.	Kidney %	Liver %	Pancrease %	Spleen %	Heart %	Thymous %	Tests %	Lung %
One month								
T1	0.71c	3.80a	0.64a	0.61bc	0.34c	0.25ab	1.26a	0.66 bc
T2	0.94b	4.61a	0.42b	0.40c	0.47a	0.22ab	0.61b	0.72abc
T3	0.85b	4.018a	0.63a	0.43a	0.49a	0.26ab	1.27a	0.79ab
T4	0.86b	3.66a	0.61a	0.52bc	0.40b	0.27ab	1.22a	0.60 c
T5	0.95b	3.91a	0.626a	0.56bc	0.48a	0.33a	1.32a	0.71abc
T6	1.00 a	3.85a	0.44ab	0.77 b	0.45ab	0.268ab	1.22a	0.84ba
P value	**0.001**	**0.018**	**0.077**	**0.000**	**0.000**	**0.084**	**0.000**	0.0002

Table (4): Levels of fasting blood glucose (mg/dL) during 8 weeks in studied groups.

Weeks	Groups					
	T1	**T2**	**T3**	**T4**	**T5**	**T6**
Before.	84.33 a	88.33a	85.67a	84.33a	93.67a	90.33a
0	90.33c	340.7b	304.0b	312.7b	300.0b	423.7a
1	86.33d	356.0b	350.0bc	295.0c	301.7 bc	600.0a
2	99.33e	380.3b	380.3b	280.0c	117.7e	599.0a
3	113.0e	372.0b	290.7c	191.3d	287.0c	530.7a
4	87.00e	370.0b	301.0c	272.7c	188.0d	498.0d
5	92.33e	408.3b	297.3c	236.3d	270.3cd	492.7a
6	85.67d	450.7b	270.7c	301.3c	264.0c	543.3 a
7	89.33c	475.3a	251.3b	220.3b	240.0b	500.0a
8	88.33c	477.0a	236.3b	195.7b	230.3b	461.0a

Data are shown as mean. The mean values in a raw with different superscript letters are significantly different at $p< 0.05$, as assessed by Tukey's pairwise comparison test. T1, normal control; T2, diabetic untreated group; T3, diabetic group treated with camel milk; T4, diabetic group treated with camel milk whey proteins; T5, diabetic group treated with Diamicron drug and T6, diabetic group treated with camel milk casein.

Table (5): Changes in hemoglobin levels (g/dL) of treated and untreated groups during experimental studies (60 days).

		GROUPS				
DAYS	T1	T2	T3	T4	T5	T6
15	16.45a	13.85b	15.35a	15.85a	15.1a	13.7b
30	14.85a	14.20b	15.25a	16.1a	15.85a	13.86b
45	16.01a	13.9b	15.2a	16.0a	15.38a	14.67b
60	16.15a	13.8b	16.0a	16.10a	16.05a	14.75b

Table (6): Changes in Red blood cells levels (RBC) (106/µl) of treated and untreated groups during experimental studies (60 days).

	GROUPS					
DAYS	T1	T2	T3	T4	T5	T6
15	8.81a	7.73b	9.4a	8,0a	8.28a	7.3b
30	8.84a	7.57b	8.3a	9.0a	8.29a	7.06b
45	8.0a	7.78b	8.26a	8.65a	8.6a	7.47b
60	9.28a	7.9b	8.83a	9.49a	8.99a	7.79b

Data are shown as mean (n = 8) (p = p = 0.005), The mean values in a row with different superscript letters are significantly different at p< 0.05, as assessed by Tukey's pairwise comparison test. p =; 0.005).

Table (7): Effect of different treatments on MCHC mean corpuscular hemoglobin concentration.

	GROUPS					
DAYS	**T1**	**T2**	**T3**	**T4**	**T5**	**T6**
15	31.7	31.2	31.5	31.6	31.3	31.0
30	31.5	32.9	33.1	17.6	33.3	30.1
45	30.0	33.1	31.5	33.0	32.7	32.4
60	33.5	33.0	33.2	32.6	32.2	29.9

No significant differences were observed (p=0.092) among different groups for MCH, and MCHC.

Table (8): Effect of different treatments on MCH.

	GROUPS					
DAYS	**T1**	**T2**	**T3**	**T4**	**T5**	**T6**
15	18.05	17.95	18.55	17.4	18.3	18.5
30	18.05	17.55	18.3	17.8	19.1	17
45	17.1	17.75	17.25	18.4	18.2	17.95
60	17.5	17.2	17.9	16.8	17.8	16.1

No significant differences were observed (MCH p =; 0.060).

Table (9). Effect of different treatments on red blood cell distribution width (RDWC).

	GROUPS					
DAYS	T1	T2	T3	T4	T5	T6
15	13.4	14.5	13.8	15.6	14	15.2
30	14.3	12.4	12.3	12.9	12.8	15.2
45	11.4	12.7	13.3	11.9	13.1	12.8
60	14.8	12.7	13.9	13.1	14.2	16.1

No significant differences were observed (RDWC p = 0.292).

Table (10): Effect of different treatments on HCT

	GROUPS					
DAYS	T1	T2	T3	T4	T5	T6
15	52.48a	44.4b	55.65ab	43.95ab	48.35ab	48.5a
30	47.28a	43.05b	46.15ab	50.35ab	46.35ab	51.7a
45	54.35a	41.9b	41.85ab	48.5ab	43.3ab	45.3a
60	48.85a	47.4b	47.4ab	49.05ab	48.1ab	52.75a

Data are shown as mean (n = 8) p = 0.005), The mean values in a row with different superscript letters are significantly different at p< 0.05, as assessed by Tukey's pairwise comparison test.

Table (11). Effect of different treatments on platelet count

	GROUPS					
DAYS	**T1**	**T2**	**T3**	**T4**	**T5**	**T6**
15	**471**	524.5	573.3	537.5	654	**593.5**
30	**588.5**	551.5	611.5	638.5	585.5	**633**
45	**408**	674.5	674.5	491.5	468.5	**479.5**
60	**676.5**	871	871	751.5	1006	**659.5**

No significant differences were observed (p = p = 0.162)

Table (12): Liver functions (ALT and AST) in rat groups after 30 and 60 days.

Treat.	**One month**		**Two months**	
	ASTA	**ALTA**	**ASTA**	**ALTA**
T1	23.56b	28.55b	25.32c	18.30c
T2	50.68a	65.64a	59.92a	68.15a
T3	33.69b	36.86b	23.17c	14.80c
T4	27.67b	27.02b	20.20c	17.45c
T5	31.42b	37.50b	22.08c	20.95c
T6	55.71a	66.42a	39.54b	43.62b

Data are shown as mean. The mean values in a column with different superscript letters are significantly different at p< 0.05, as assessed by Duncan's multiple range test. T1, normal control; T2, diabetic untreated group; T3, diabetic group treated with camel milk; T4, diabetic group treated with camel milk whey proteins; T5, diabetic group treated with Diamicron drug and T6, diabetic group treated with camel milk casein.

Table (13): Changes in serum creatinine levels in control, diabetic and diabetic treated rats.

Treat.	One month	45days	Two months
	Creatinine levels		
T1	1.000b	1.01b	0.898b
T2	1.850a	2.03a	2.120a
T3	1.16b	1.11b	0.9000b
T4	1.18b	0.98b	0.7700b
T5	1.200b	1.08b	0.9500b
T6	1.900a	1.81a	1.700a

Data are shown as mean. The mean values in a column with different superscript letters are significantly different at $p< 0.05$, as assessed by Tukey's pairwise comparison test. T1, normal control; T2, diabetic untreated group; T3, diabetic group treated with camel milk; T4, diabetic group treated with camel milk whey proteins; T5, diabetic group treated with Diamicron drug and T6, diabetic group treated with camel milk casein.

Histological examination of pancreas tissue sections.

Histopathology of pancreas tissue of non-diabetic and diabetic rats stained with hematoxylin and eosin (H and E).

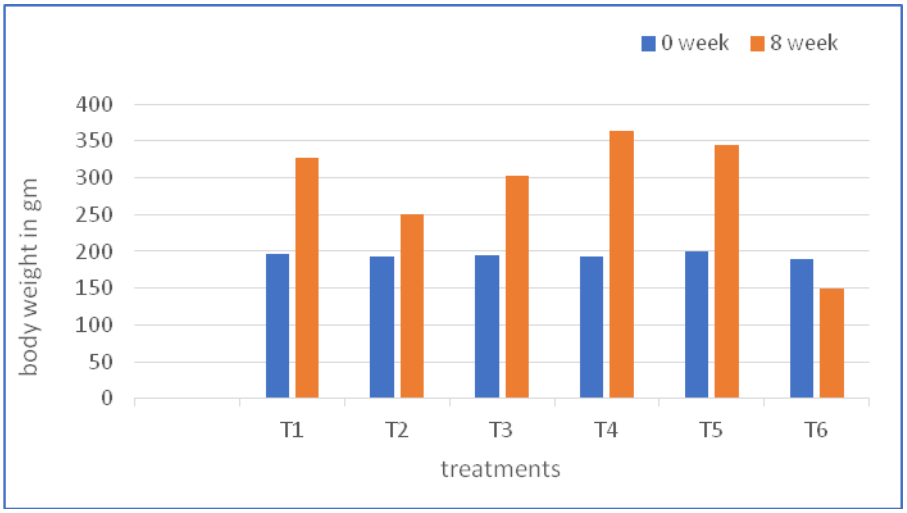

Figure (1). Changes in rat body weighs at zero and 8 weeks of study.

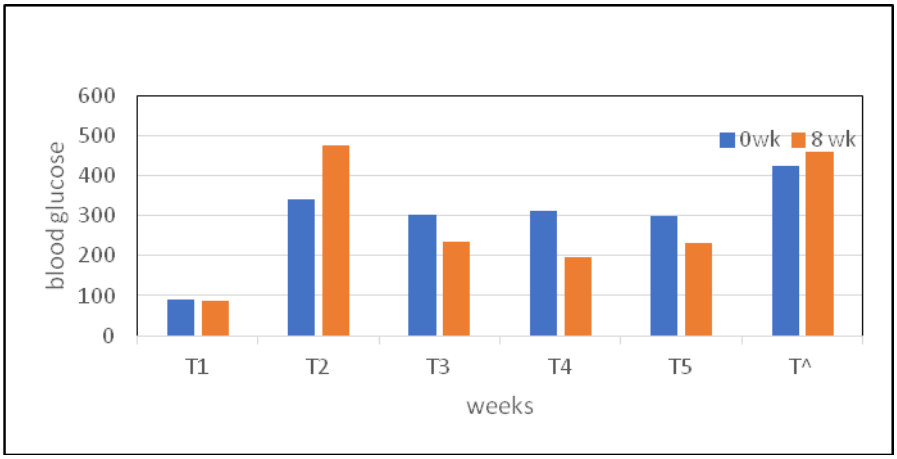

Figure (2). Levels of fasting blood glucose (mg/dL) after zero and 8 weeks in studied groups.

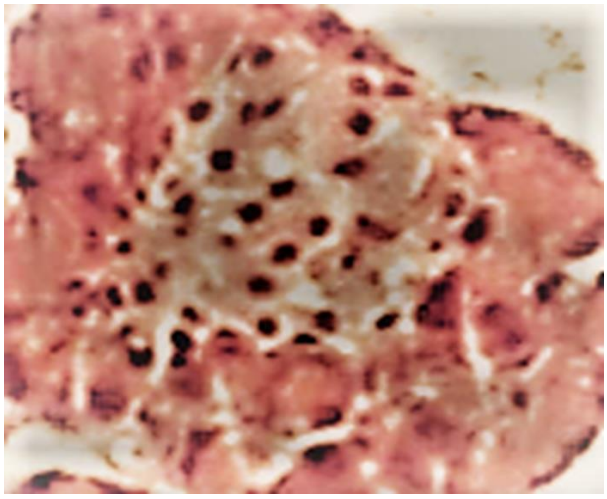

Figure (3): Histopathology of pancreas tissue of non-diabetic control group (T1). One month.

Histopathology of pancreas tissue of non-diabetic control group showed the normal histological morphology, normal Langerhans islet.

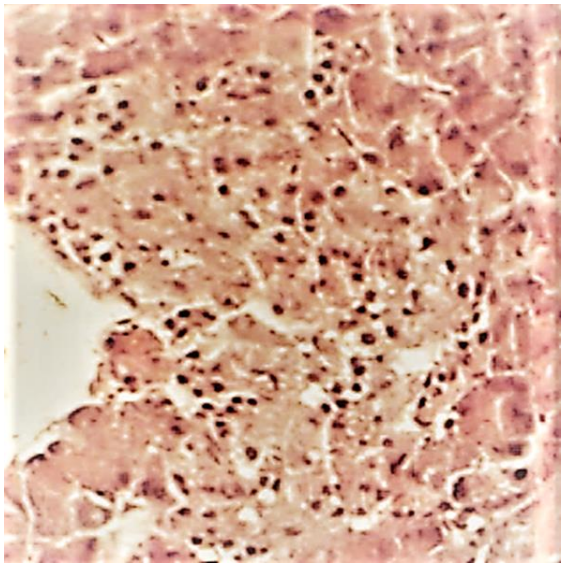

Figure (4): Histopathology of pancreas tissue of non-diabetic control group (T1). Two months.

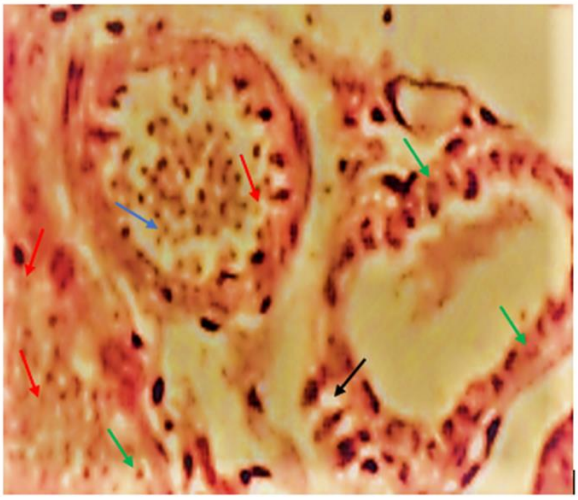

Figure (5): Histopathology of pancreas tissue of -diabetic group (T2). One month.

It has been noticed that a severe degeneration (green arrow) and necrosis (blue arrow) were observed in pancreatic tissue and shrinkage in the islets of Langerhans, karyolysis (red arrow), cytoplasmic vacuolations (black arrow) and atrophy of β cells were also observed.

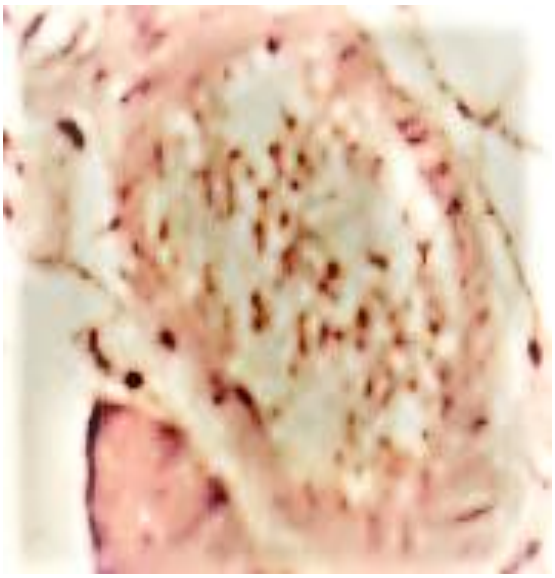

Figure (6): Histopathology of pancreas tissue of -diabetic group (T2). two months.

More deterioration in β cells was observed

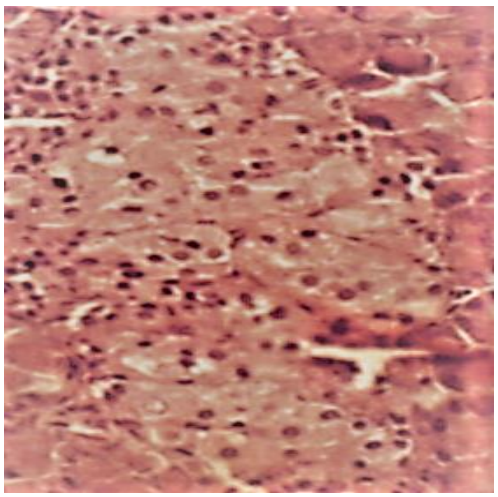

Figure (7): Histopathology of pancreas tissue of -diabetic group supplemented with camel milk (T3). One month.

Pancreatic of diabetic rats supplemented with camel milk (30 days) showed amelioration and restored the pancreas to normal architecture, regeneration of β-cell and increased the number of β- cells in the islets of Langerhans.

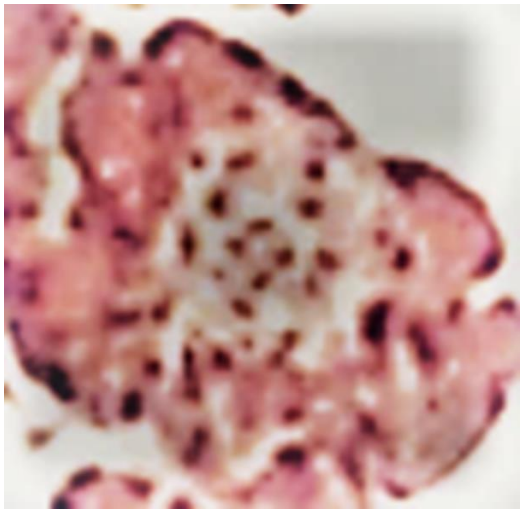

Figure (8): Histopathology of pancreas tissue of -diabetic group supplemented with camel milk whey proteins (T4). One month.

Pancreatic of diabetic rats supplemented with camel milk whey proteins (30 days) showed amelioration and restored the pancreas to normal architecture, regeneration of β-cell and increased the number of β- cells in the islets of Langerhans.

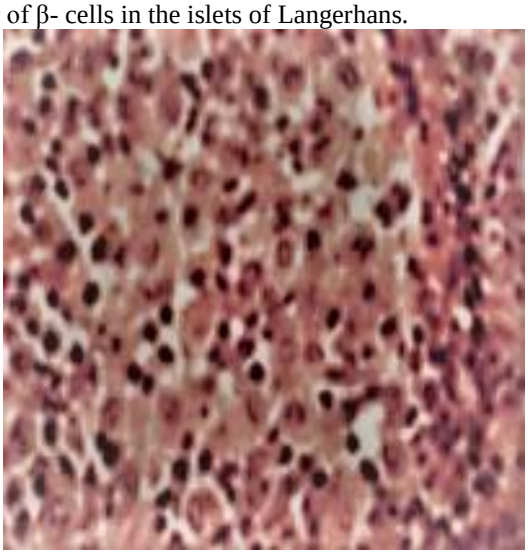

Figure (9): Histopathology of pancreas tissue of -diabetic group supplemented with Diamicron drug (T5). One month

Pancreatic of diabetic rats supplemented with Diamicron drug (30 days) showed restored the pancreas to normal architecture, regeneration of β-cell and increased the number of β-cells in the islets of Langerhans.

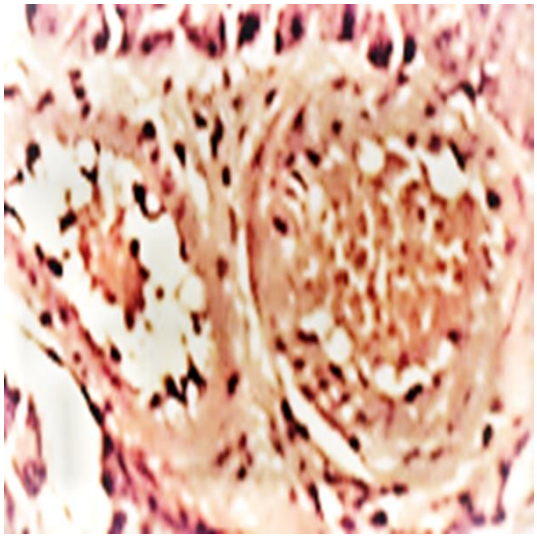

Figure (10): Histopathology of pancreas tissue of -diabetic group supplemented with camel milk casein (T6). One month

Pancreatic of diabetic rats supplemented with camel milk casein (30 days) showed some amelioration of the pancreas architecture, regeneration of β-cell and increased the number of β- cells in the islets of Langerhans.

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الملخص العربي

تأثير لبن النوق وشقوق بروتين لبن النوق على مرض السكرى فى ذكور فئران الالبينو

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فى هذه الدراسة تم تقسيم 48 فأر من الذكور البالغين والخالية من الامراض والتى يبلغ متوسط وزنها 120±10 جرام بعمر 4 الى 6 اسابيع لستة مجاميع . المجموعة الاولى(T1) هى الغير مصابة ،المجموعة الثانية(T2) المجموعة المصابة بمرض السكري ، المجموعة الثالثة (T3) مصابة بمرض السكرى ومعاملة بلبن الابل الكامل ، المجموعة الرابعة (T4) مجموعة مصابة بمرض السكرى ومعاملة ببروتينات شرش لبن الابل ، المجموعة الخامسة (T5) مجموعة مصابة بالسكرى ومعاملة بعقارديناميكرون والمجموعة السادسة (T6) مجموعة مصابة بمرض السكرى ومعاملة ببروتينات الكازين للبن الابل . اظهرت النتائج فروق معنوية $P\leq0.05$ بين المجموعة المصابة بالسكرى والمجموعات المعاملة تشمل المجموعة الثالثة والمجموعة الرابعة والمجموعة الخامسة . بالرغم من ذلك لم يكن هناك فرق معنوى بين المجموعة السادسة والمجموعة المصابة . كما انخفض مستوى السكر فى الدم فى المجموعة الثالثة والمجموعة الرابعة والمجموعة الخامسة مع عدم وجود فرق معنوى عند مقارنتها بالمجموعة الاولى (الغير مصابة) كان هناك تأثير للمعاملات الثالثة والرابعة والخامسة على زيادة وزن الجسم $P\leq0.05$ وعلى العكس بالمجموعة السادسة لم يحدث تأثير على زيادة وزن الجسم . تم تحسين وظائف الكلى والكبد الى المستويات الطبيعية فى المجموعات الثالثة والرابعة والخامسة بالعكس فى المجموعة السادسة . كما اظهرت المقاطع النسيجية للبنكرياس فى المجموعات الثالثة والرابعة والخامسة عدم وجود خلايا التهابية او تحلل بالنسيج . كما اظهرت النتائج والبيانات التى تم الحصول عليها فى هذه الدراسة ان لبن الابل وبروتينات شرش اللبن الابل خفض بشكل كبير من مستوى الجلوكوز فى الدم ويمكن استخدامة كعلاج مساعد فى مرض السكرى من النوع الثانى .