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The protective role of camel milk on the pancreas in streptozotocin-induced diabetic rats

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Abstract

Diabetes mellitus, a common metabolic disorder characterized by dysfunction of pancreatic beta-cells and increased oxidative stress, damages the pancreas. These experiments are a promising study to determine the safety of camel milk in treating streptozotocin-induced diabetic rats. Male albino rats were divided into six groups: negative control, untreated diabetic, diabetic treated with metformin, and three diabetic rats were exposed to higher levels of freeze-dried camel milk (4, 6, and 8 mL/kg). Serum glucose, insulin, and Malondialdehyde (MDA) were measured, and insulin resistance (HOMA-IR) was determined. Pancreatic tissues were histologically investigated. When camel milk is consumed at 8 mL/kg, the glucose level was significantly decreased in the rats that were given camel milk (138.05 ± 5.74 mg/dL) compared with the diabetes groups (252.5 ± 33 mg/dL), and levels of insulin (485.3 ± 28.1 pg/mL). Conversely, MDA levels were low with a 39.43 ± 6.62 ng/mL. The improvement in islet structure on record was clear with preserved beta cells. Camel milk protects the pancreas in streptozotocin‑ induced diabetes by restoring glycemic control (including insulin secretion, oxidative stress, and pancreatic histoarchitecture), making its use as a natural adjunct to the treatment of diabetes more effective, potentially enhancing the pancreas's organ functions.

Keywords: camel milk, diabetes mellitus, oxidative stress, HOMA‑ IR.

Introduction

Diabetes mellitus is a chronic metabolic disorder in which persistent hyperglycemia results from the failure of secretion, release, or action of glucose and insulin. The World Diabetes Federation reported in 1999 that approximately 783 million people will be diagnosed with diabetes by 2045. Diabetes mellitus is commonly classified into type 1 diabetes mellitus, which involves chronic diabetes, and type 2 diabetes mellitus, which involves irreversible diabetic resistance in both blood sugar and beta cell release, and is usually considered a manifestation of diabetes[1][2].

Streptozotocin (STZ) is an established diabetogenic drug, well known to trigger diabetes in animal models, and is the most popular and commonly used in diabetes genetics research. STZ is a toxic glucose analogue, and it is carried back from pancreatic beta cells via the GLUT2 glucose transporter. When stored in beta cells, it forms glucophenylzotocin, a sugar and methylnitrosourea molecule of molecular nature. Alkylating into the glucose receptor, breakdown breaks and destruction of beta cells are essential for diabetes. Targeting mitochondrial DNA damages mitochondria and inhibits the glucophenylzotocin-dependent diabetes by the metabolism process in the beta system, and it can be explained that the gene expression in glucose and mitochondria leads to glucose-induced diabetes. The STZ-induced diabetic rat model closely resembles metabolic features of human diabetes, such as hyperglycemia, hypoinsulinemia, and oxidative stress symptoms of humans, making it the best candidate for analyzing possible antidiabetic agents[3][4].

Camel milk has been used for centuries in desert and semi-arid regions as a meal and as medicine. The traditional medicine systems have advocated for clinical use of camel milk for diabetes[1], hypertension, and gastrointestinal disorders. Camel milk has higher protective proteins, lactoferrin, immunoglobulins, and insulin-like proteins at higher concentrations than bovine milk. Camel milk is also rich in zinc as well as bioactive peptides in both antioxidant and anti-inflammatory processes, which are believed to mimic insulin action[1], [5][6].

There is growing factual evidence of possible antidiabetic benefits of camel milk. With some academic evidence of camel milk treatment of diabetes, as well as good glycemic control, increased insulin sensitivity, and reduced oxidative stress in animal models and human subjects with diabetes. Studies conducted on various animals have also found that camel milk can show better glycemic control, sensitivity of cells in animal model diabetes, and reduced oxidative stress in animal and human treatment control and diabetes. camel milk’s unique bioactivity results are the reason why camel milk leads to reduced blood sugar levels by 30% through its bioactive composition[1], [5][7] although camel milk had greater efficiency for managing diabetes in reducing blood sugar levels through the diabetes process due to its bioactive and unique characteristics of its unique nature by lowering the blood sugar by one in every single aspect and Camel milk is far superior to cow's milk in treating diabetes [1]

Recent advancements have led to a better understanding of the cellular and molecular mechanisms behind the antidiabetes effects of camel milk. It was discovered that camel milk whey protein has an optimal role in insulin receptor and associated downstream signals, such as AKT kinase[8]. Anwar et al. (2025 identified 9 synthetic peptides from camel milk hydrolysate that have partial activity around the insulin receptor and enhance glucose uptake in human cells, and are potentiated 104% to 147%. Moreover, camel milk peptides were shown to act on the dipeptidyl peptidase IV (DPP-IV) in their concentration at higher concentrations and by reducing incretin concentration[5].

Mudgil et al. 2021 studied the effect of camel milk protein hydrolysates on diabetic rats in STZ-induced diabetes, showing significant reductions of malondialdehyde (MDA) content (indicative of oxidative stress and preserved pancreatic β-cell in the liver, for example) and the lack of lipid in hepatocytes. These findings showed that camel milk protein hydrolysates in 500 mg/kg body weight have high hypoglycemic activity and enhanced antioxidant enzyme activity for the release, namely superoxide dismutase and catalase[7][9][10].

Although there are many studies investigating the hypoglycemic effect of camel milk, few have studied its dose-dependent protective activity on the structure of pancreatic tissue from streptozotocin or diabetes. Moreover, these mechanisms can also be used to further connect the findings to hypoglycemic effects, pancreatic histopathological changes, and oxidative stress markers[1], [5]. While the data were used for chronic clinical studies, a variety of camel milk uses in the dose-response model allows us to find the optimal protective dose at one dose level, but the mechanisms for these effects did not seem to be well-understood. In fact, few studies have been conducted on raw camel milk, and little research has been done to create transformed camel milk (such as powder) to provide patients with an effective anti-diabetic and more affordable treatment experience. This study was designed to investigate the protective effect of camel milk on the pancreas in streptozotocin-induced diabetic rats. Three levels of camel milk (4, 6, and 8 mL/kg) were compared to one another: blood glucose levels, insulin resistance, or HOMA-IR and pancreatic histopathology, and the results were used to compare with those of metformin. Age distribution was also investigated, and oxidative stress was looked into by measuring malondialdehyde in serum. Our results will add to the evidence for camel milk as a natural adjunct to diabetes treatment based on animal data, which have been developed through various studies to strengthen diabetes management interventions to regain control over pancreatic function and, in particular, to prevent diabetes.

Methods

Study Design

This experiment was designed to assess the protective effect of camel milk at high blood glucose levels and pancreatic tissue in streptozotocin-induced diabetic rats. The experiment was conducted on October 1st

and December 31st, 2025, with a total of thirty-six male albino rats present.

Experimental Animals

These 36 male albino rats, aged 7-9 weeks, weighing between 200 and 250 grams, were obtained at College of Pharmacy, Al-Nahrain University as laboratory animals. The rats were kept in special cages that were shaded with wood shavings. The cages and the surrounding area were cleaned. A temperature of 22 to 25 ′ C and ventilation were kept in the same place during a clinical experiment. A 12h light-dark cycle was maintained. The rats were allowed to be prepared for the experimental work for 10 days before the work in detail started. At that time, the rats received water and were given animal feed ad libitum.

Induction of Diabetes

Before diabetes was induced Body weight was recorded, average diabetes in the tail vein was recorded using an ACCU-Chek device by pricking blood flow through a tail vein and dropping some blood on a test strip, and reading it. Streptozotocin was administered at 40 mg/kg of body weight in phosphate buffer and peritoneally at 45 degrees of angle in an attempt to minimize organ damage and disease risk. They were added 20% glucose solution for 24 hours if they were near dying from hypoglycemia or death. In the studies, the diagnosis of diabetes was confirmed by measuring blood glucose levels on the second and third days post injection; animals with blood glucose levels above 200 mg/dL were considered diabetic and included in the study.

Experimental groups

The male rats were divided into six groups, where each was composed of six rats as follows: Group I (Negative Control Group): Non-diabetic rats fed a typical diet and given tap water. Group II (Diabetic Control Group): Diabetes controlled rats induced with streptozotocin at 40 mg/kg and left untreated.Group III (Diabetic + Metformin Group): Diabetic rats treated with metformin. Group IV (diabetic + Camel Milk low concentration group): Diabetic rats given the first concentration of dried camel milk orally. Group V (Diabetic + Camel Milk Medium Concentration Group): Diabetic rats treated with a second, higher concentration of camel milk. Group VI (diabetic + Camel Milk High Concentration Group): Diabetic rats were given the third concentration of camel milk, the highest concentration among the groups treated with camel milk.

Camel Milk Collection and Preparation

The Camel milk was provided from a desert-colored Khawar female camel belonging to a breeder at Rawah; city in Anbar. This female camel was aged 7 to 9 years. The camel was milked regularly, with the animal producing 1000 ml of milk in clean glass bottles. The milk was then sent to the laboratory for analysis. The physical properties of the milk on arrival were measured: pH (6.4), acidity (0.14%), and specific gravity (1.25). The freeze-drying method was employed to convert camel milk into powdery dry milk that was dry. Lyophilizer was used at (-50 C) within 72 hours to make it do so. The powder was then placed in a tightly shut plastic cup and

stored in the refrigerator in a sealed plastic container.

Administration of Camel Milk and Metformin

Dried camel milk was given orally to rats after being diluted slightly with warm water in fixed volumes of 2 ml and at different concentrations of 4, 6, and 8 mg/kg.The mice were given camel milk every morning for four consecutive weeks, administered orally using a 10 ml syringe with a 16 mm oral feeding needle. Results showed an increase in body weight with camel milk consumption. Metformin (0.2 g/ml) was imported from a pharmaceutical manufacturer in Samarra as a white powder, and the daily dose was prepared with distilled water. Male mice were given an oral dose of 2 mL per mouse, equivalent to 100 mg/kg of body weight.

Body Weight and Blood Glucose Monitoring

Body weight and glucose monitoring were kept before and after the start with animals. A standard scale and then the rat was placed into a plastic container so that it would not move during weighing were used. Weight levels of fasting blood glucose were measured weekly using the ACCU-Chek device to check the hypoglycemic effects of camel milk and metformin.

Collection of Blood and Pancreatic Tissue Samples

Over the course of four weeks of treatment, we brought the animals into the animal house to sample blood and pancreatic tissue. The animal to be anesthetized was placed in a sealed container, a cotton swab soaked in chloroform was placed inside, the container was tightly closed, and the animal was left for a short time until it became anesthetized. Blood has been drawn directly from the heart and sent to test tubes without an anticoagulant using a 10 ml syringe. The tubes are then centrifuged at 3000rpm for 15 min to separate the serum. Blood was transferred to dry sterile (differently labeled) Eppendorf tubes and stored at -18°C for biochemical analysis.

Measurement of Serum Glucose Concentration

To measure glucose concentration in our test samples on blood, i.e., with a test kit from Linear (based on data obtained using fluorescence) based on the approach of Dingeon et al. (1975) It is based on the process of enzymes converting glucose into hydrogen peroxide that reacts with 4-aminoantipyrine with phenol to make a coloured compound. We measured the absorbance with a diffuse spectrophotometer, and the absorbance of the sample was calculated with spectral measurement.

Measurement of Serum Insulin Concentration

The serum insulin concentration measurement in vivo (globally developed insulin measurement). Serum insulin concentration was estimated in a ready-made analytical kit from Sunlong (Chinese company) according to the manufacturer according to their specifications. A sandwich ELISA kit with 450 nm optical density was applied to the test to measure

the concentration. The concentrations in the patient's blood, along with the comparison control

Assessment of Insulin Resistance (HOMA-IR)

Assessment (HOMA-IR) of Insulin Resistance. Insulin resistance was estimated based on the Homeostatic Model Assessment indices based on fasting blood glucose and fasting serum insulin concentrations (Zhou et al., 2014), using the following equation:

HOMA-IR = Fasting insulin concentration (μU/mL) × Fasting glucose concentration (mg/dL) /22.5

Measurement of Malondialdehyde as an Oxidative Stress Marker

Analytical measurement of Malondialdehyde as an oxidative stress markers In diabetes, malondialdehyde is an oxidant, which is associated with pancreatic damage. This was measured in serum using the ready-made test kit published by the American company Elabscience. The measurement was done on a spectrophotometer at a wavelength of 532 nm.

Histological analysis of the pancreas

Pancreatic tissue Standard stain was examined using a light microscope to investigate the protective effects of camel milk on the pancreatic tissue structure, especially the islets of Langerhans, beta cells, and the surrounding acinar tissue. The histological scans were done using a compound light microscope equipped with a digital camera

Statistical Analysis

The results were statistically investigated with GraphPad Prism version 8.0 to identify that the values of the treated groups differ significantly. First, we do one-way analysis from each test (Duncan's multiple range test), and the results are described in terms of mean ± standard deviation and with probability $P \leq 0.05$.

Results

Table (1): Mean ± SD values of glucose, insulin, HOMA-insulin, and MDA in the experimental groups

Parameters / Groups	Glucose (mg/dl) (mean+SD)	Insulin (mU/L) (mean+SD)	HOMA-Insulin (mean+SD)	MDA (ng/ml) (mean+SD)
Negative control	E 99.34±4.18	A 538.7±59.1	BC 132.15±15.82	D 32.74±5.01
Daibetic (Setreptozotocine)	A 252.5±33	D 173.67±22.48	D 108.8±24.3	A 90.83±7.23
D.M + Medformin	D 125.48±5.52	B 485.3±28.1	A 150.4±11.69	D 39.43±6.62
D.M + Milk Conc.1	B 191.23±5.93	C 335.9±29.1	A 158.52±13.3	B 70.83±6.48
D.M + Milk Conc.2	C 165.05±4.31	C 350.3±26.2	AB 142.71±10.2	B 68.12±6.07
D.M + Milk Conc.3	D 138.05±5.74	C 362.6±27.8	CD 123.4±7.94	C 55.25±6.76

The mean ± SD value of glucose, insulin, HOMA-insulin, and MDA for different experimental groups in the negative control group, diabetic group, metformin-treated group, and camel milk-treated groups are indicated in the table 1 . Also in Table 1 are the letter groupings that demonstrate the statistical difference among the groups for each parameter. The means that have different letters in the same column are significantly different compared to the difference between the other groups in Table. All three studies shown here show some significant variations among the experimental groups over the parameter in the study, which could be linked to the treatment groups in the study.

Table (2): analysis variance results for insulin, MDA, glucose, and HOMA-IR in the experimental groups

Variable	Source	DF	Adj SS	Adj MS	F-Value	P-Value	R-sq	R-sq(adj)
Insulin	C1	5	490717	98143	82.89	0.000	0.9325	0.9213
MDA	C1	5	13925	2785	68.01	0.000	0.9189	0.9054
Glucose	C1	5	89355	17870.9	87.54	0.000	0.9359	0.9252
HOMA-IR	C1	5	10039	2007.8	9.12	0.000	0.6031	0.5370

Table 2 shows the results associated with the one-way analysis of variance (ANOVA) for insulin, MDA, glucose, and HOMA-IR, as well as DF, adjusted sum of squares (Adj SS), adjusted mean squares (Adj MS), F-value, P-value, and coefficients of

determination (R-sq and R-sq(adj The results show that overall, all the studied variables have P-values of 0.000, indicating the significant differences in the experiments of the respective groups. Values of F and R-squared as well depict the variance that is caused by differences among the experimental groups on each parameter, as we estimated this to be due to the group differences, which is also observed for all F-values as well.

Table (3): bast concentration of grouping of insulin, MDA, glucose، and HOMA-IR among the experimental groups

Variable	Group	N	Mean	Grouping
Insulin	A	6	538.7	A
Insulin	F	6	485.3	B
Insulin	E	6	362.6	C
Insulin	C	6	350.3	C
Insulin	D	6	335.9	C
Insulin	B	6	173.67	D
LSD			**33.71297**	
MDA	B	6	90.83	A
MDA	D	6	70.83	B
MDA	C	6	68.12	B
MDA	E	6	55.25	C
MDA	F	6	39.43	D
MDA	A	6	32.74	D
LSD			**6.26972**	
Glucose	B	6	252.5	A
Glucose	D	6	191.23	B
Glucose	E	6	165.05	C
Glucose	F	6	138.05	D
Glucose	C	6	125.48	D
Glucose	A	6	99.34	E
LSD			**13.99725**	
HOMA-IR	D	6	158.52	A
HOMA-IR	A	6	132.15	A
HOMA-IR	C	6	150.4	A
HOMA-IR	E	6	142.71	A
HOMA-IR	F	6	123.4	A
HOMA-IR	B	6	108.8	B
LSD			**14.53884**	

At first glance, the table summarizes the results of Fisher's Least Significant Difference test in terms of insulin, MDA, glucose, as well as HOMA-IR variables for all groups: group, number of animals, mean value, and statistical grouping letters based

on the groups. The same letter in groups is not much different, and the different letters in each group indicate a significant difference within the same set of variables with respect to each other, respectively. In addition, the table gives LSD for each studied parameter for 33.71297 for insulin, 6.26972 for MDA, 13.99725 for glucose, and 14.53884 for HOMA-IR values.

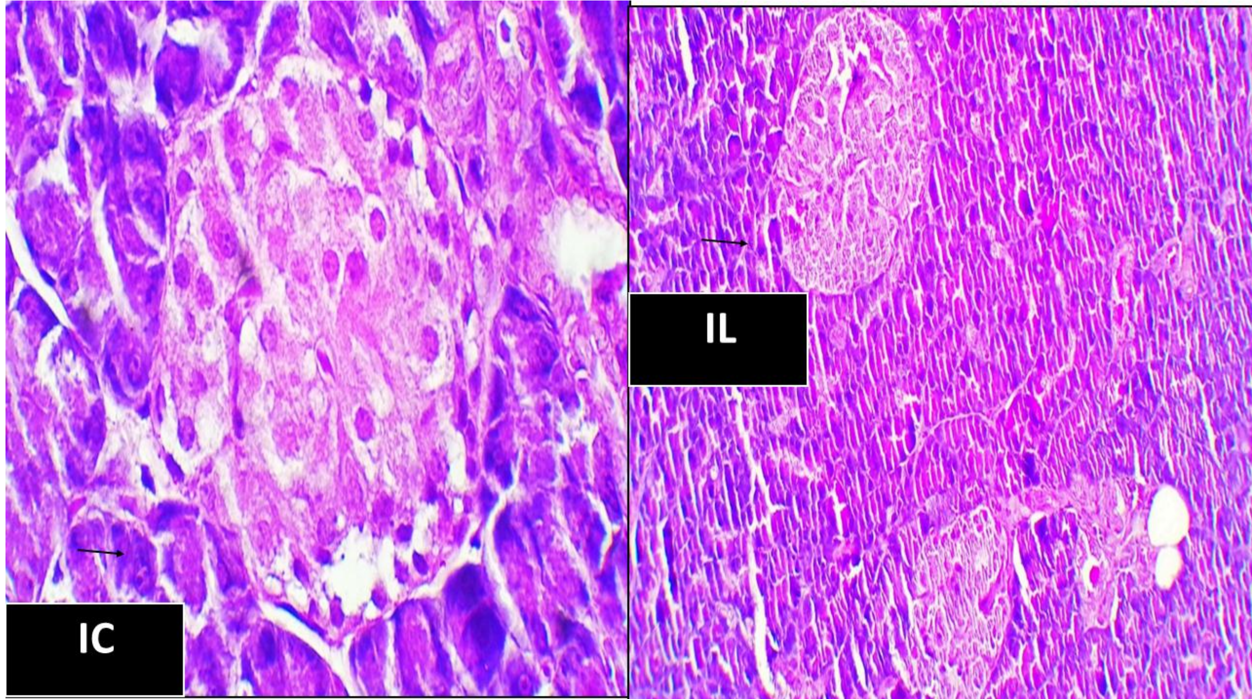

Figure 1 :Histological section of the pancreas in the control group, stained with hematoxylin and eosin at 40× magnification

Rat pancreatic histological sections stained with hematoxylin and eosin of the control group (40x magnification). Arrowheads represent the boundary of islets of Langerhans (IL), arrows represent characteristic of cells inside the islets (IC).

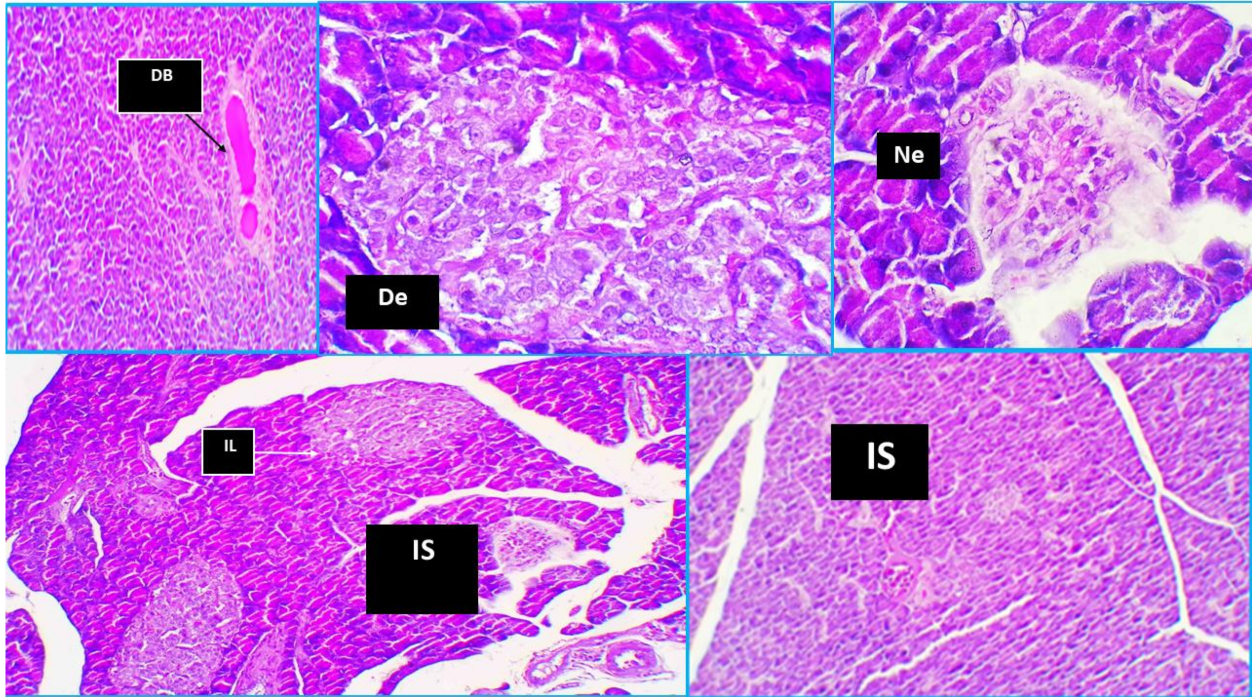

Figure 2: Histological section of the pancreas in the diabetic control group stained with hematoxylin and eosin.

Diabetic control rat pancreas showing shrinkage of islets of Langerhans (IS) with degeneration (Dg) and necrosis (Ne) of component cells, where the nucleus appeared densely basophilic (DB) and hemorrhage (He).

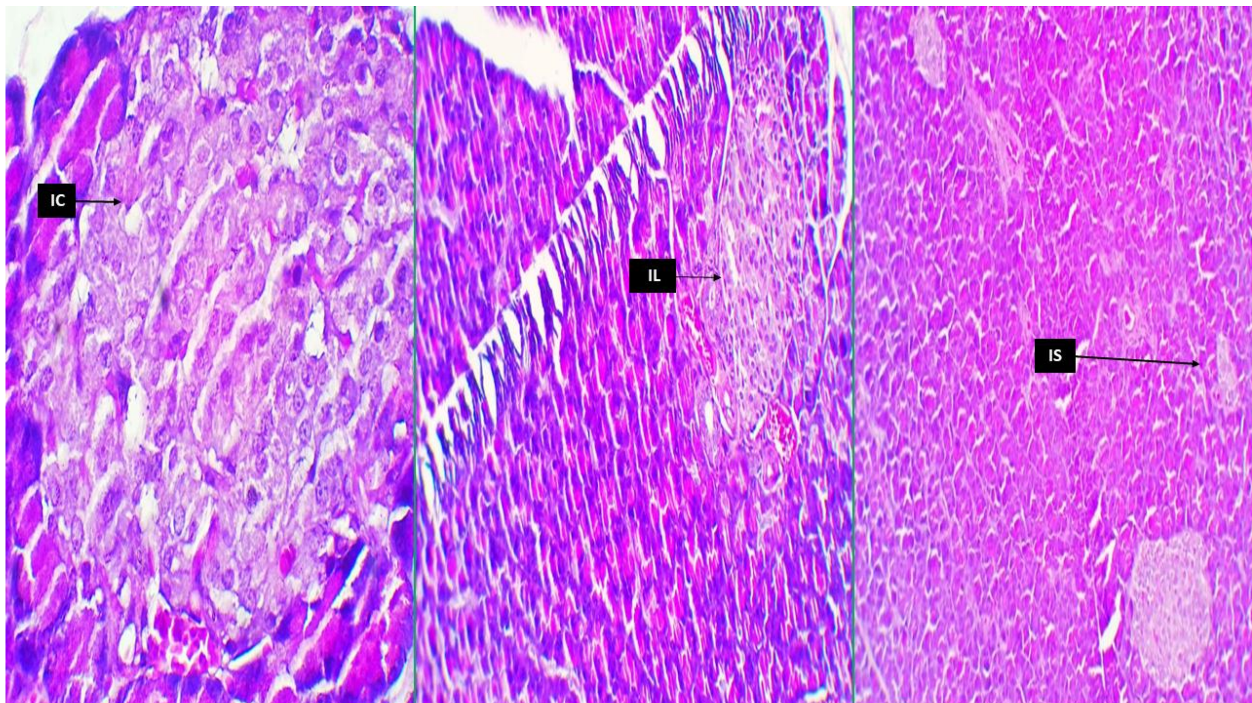

Figure 3 :Histological section of the pancreas in the standard drug group, stained with hematoxylin and eosin at 40× magnification

Rat pancreatic histological sections stained with hematoxylin and eosin of the Standard drug group (40x magnification). Arrowheads represent the boundary of islets of Langerhans (IL), shrinkage of islets of Langerhans (IS), and arrows represent characteristic of cells inside the islets (IC).

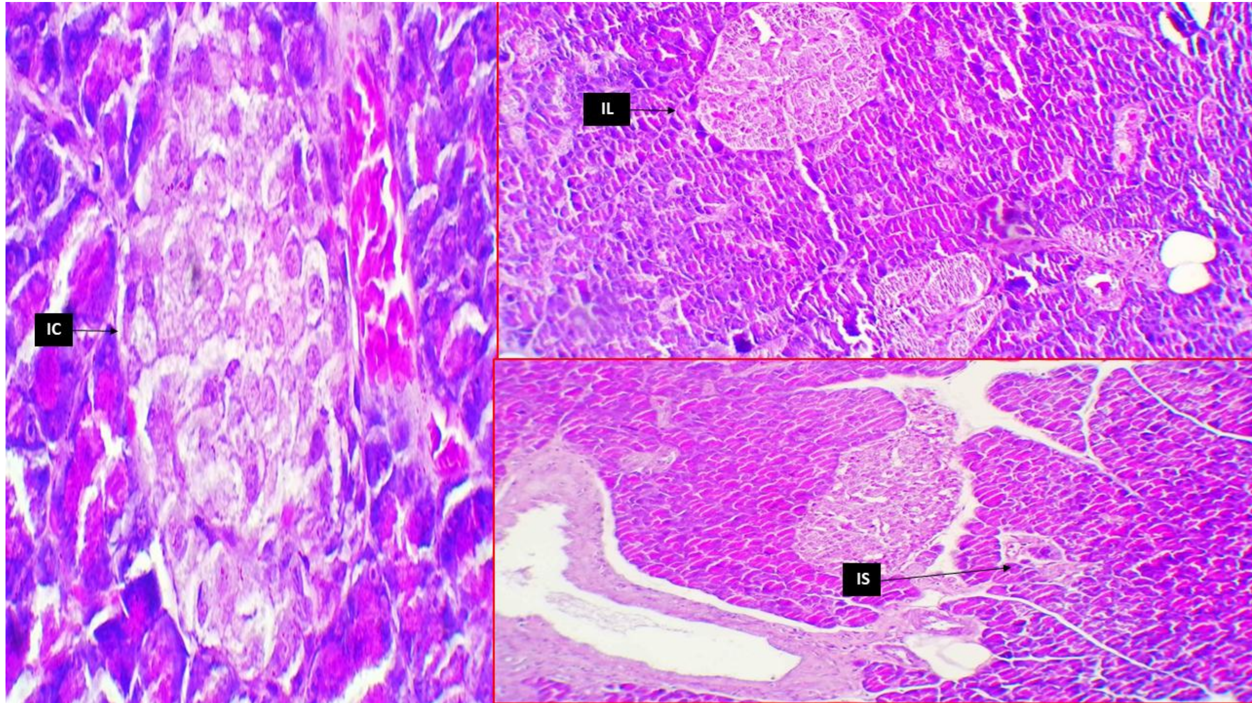

Figure 4: Histological section of the pancreas in the group treated with Camel Milk low concentration, stained with hematoxylin and eosin at 40× magnification.

Rat pancreatic histological sections stained with hematoxylin and eosin of Con.1 (Camel Milk low concentration) (the 40x magnification). Arrowheads represent the boundary of islets of Langerhans (IL), shrinkage of islets of Langerhans (IS), and arrows represent characteristic of cells inside the islets (IC).

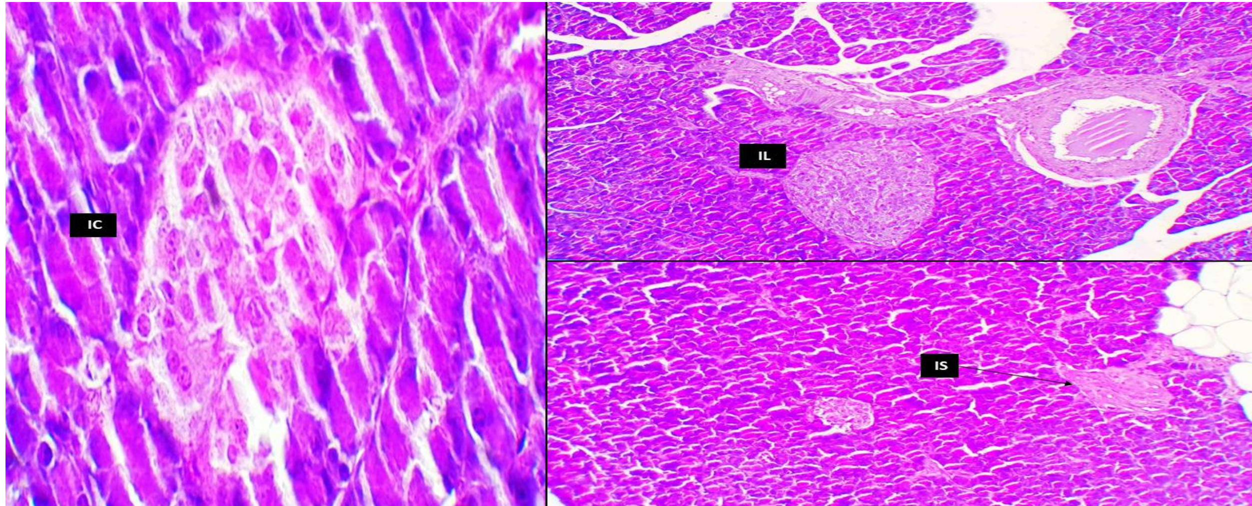

Figure (5): Histological section of the pancreas in a group of rats treated with Camel Milk Medium Concentration, and stained with hematoxylin and eosin at 40× magnification.

Rat pancreatic histological sections stained with hematoxylin and eosin of Con.2 Camel Milk Medium Concentration (the 40x magnification). Arrowheads represent the boundary of islets of Langerhans (IL), shrinkage of islets of Langerhans (IS), and arrows represent characteristic of cells inside the islets (IC).

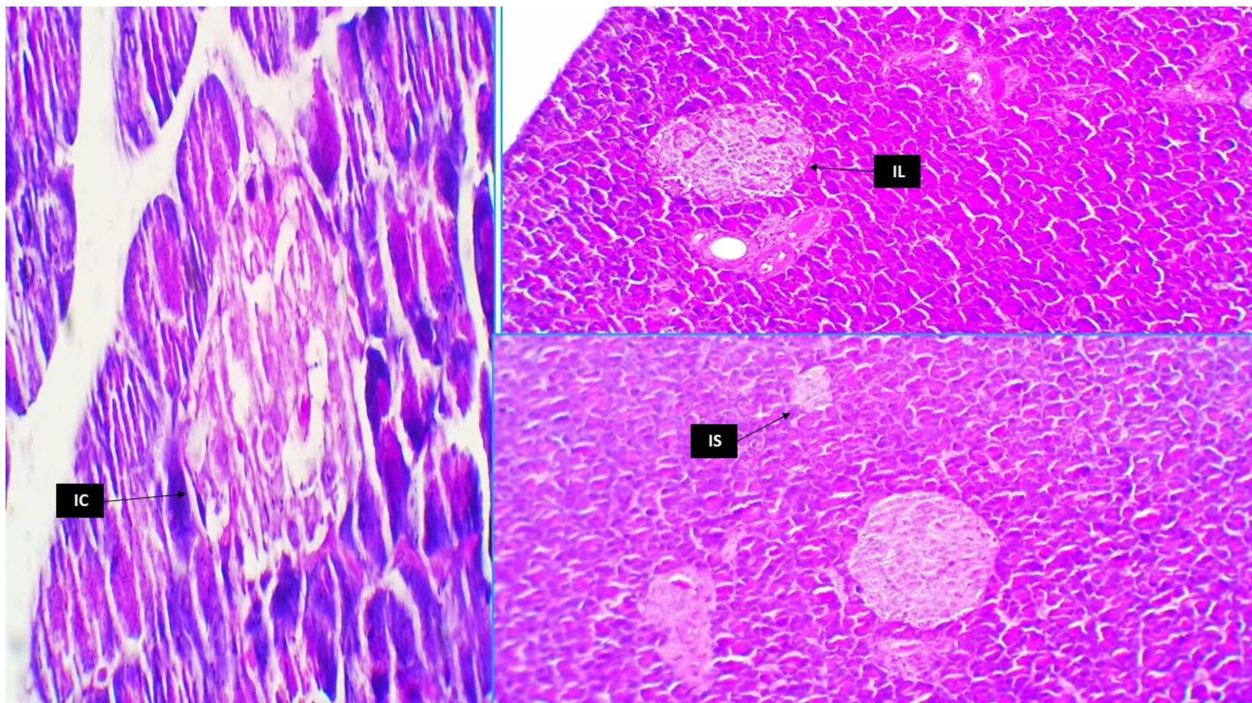

Figure (6): Histological section of the pancreas in a group of rats treated with Camel Milk high Concentration, stained with hematoxylin and eosin at 40× magnification.

Rat pancreatic histological sections stained with hematoxylin and eosin of Con.3 Camel Milk high Concentration (the 40x magnification). Arrowheads represent the boundary of islets of Langerhans (IL), shrinkage of islets of Langerhans (IS), and arrows represent characteristic of cells inside the islets (IC).

Discussion

The study showed that administering dried camel milk to rats resulted in a clear improvement in blood sugar levels, insulin secretion, reduced oxidative stress, and regeneration of pancreatic tissue structure. These results demonstrate the effective role of camel milk in managing diabetes and establish it as a safe and natural alternative treatment.

In this study, diabetic rats (Group B) have significantly elevated fasting blood glucose levels (252.5 ± 33.0 mg/dL) when compared with the control group (99.34 ± 4.18 mg/dL). The camel milk, in its highest concentration (Group F, 8 mL/kg), reduced blood glucose levels to 138.05 ± 5.74 mg/dL, which were comparable to those of metformin (125.48 ± 5.52 mg/dL). Insulin levels in group F are also significantly higher than in the diabetic control group (173.67 ± 22.48 pg/ml). The results of that study are in line with Mbye et al. (2025), who also found that milk in camel also has insulin-like proteins, lactoferrin, and bioactive peptides, some of which possess hypoglycemic aspects that contribute to glycemic control, along with more insulin-producing pancreatic β-cell activity and diabetes-based signalling mechanisms[1]. Additionally, Khan et al. (2025) reported that camel milk protein hydrolysates also significantly increase the number of β-cells and insulin secretion in streptozotocin-induced diabetic rats and support the insulin responsiveness of camel milk according to this study. The involvement of insulin-like proteins in camel's milk has been attributed to its unique physicochemical properties and its resistance to gastrointestinal degradation, providing opportunities to absorb and utilize it effectively[11].

Homeostatic model assessment of insulin resistance (HOMA-IR) was used to assess insulin sensitivity. While diabetic rats (Group B) had very low HOMA-IR (108.8 ± 24.3) compared to the other groups, the HOMA-IR in camel milk (Groups D, E, and F) was comparable to the control group (132.15 ± 15.82). This is similar to Korish's (2014) results that camel milk treatment in streptozotocin-induced diabetic rats decreased HOMA-IR and glucose-stimulated insulin secretion. Camel milk has also been shown to change the secretion of the incretin hormones GLP-1 and GIP to develop a higher insulin sensitivity[12].

In this study, we were able to determine that serum malondialdehyde (MDA) levels are sensitive to lipid peroxidation rates in diabetic rats in comparison and were higher (90.83 ± 7.23 ng/ml) than in the control of 32.74 ± 5.01 ng/ml. We were able to show that camel milk decreased the MDA by a factor of 5. The highest concentration (Group F) of MDA reduced the concentration of MDA to 39.43 ± 6.62 ng/ml. Mudgil et al. (2021) further demonstrated that camel milk protein hydrolysates were associated with the

reduction of malondialdehyde content and enhanced antioxidant enzymes (superoxide dismutase and catalase) in STZ-induced diabetic rats[13]. Indeed, Mohamed (2018) also reports significant improvement in the total antioxidant status (TAS) and lower MDA levels in diabetic rats treated with camel milk, demonstrating the antioxidant properties of camel milk[14].

The pancreas, histologically observed significant changes in diabetic rats, and the number of islets shrank, β cells degenerated, and necrosis and hemorrhage occurred. The treatment of camel milk with a high concentration of camel milk resulted in excellent preservation of islet shape. But these histological results are in line with Hati et al. (2025), where camel milk powder formulations led to an overall improved pancreatic histoarchitecture in diabetic rats, with a protected β-cell composition with decreased pathological changes compared to diabetic rats[15]. Mansour et al. (2017) also showed that camel milk treatment restored insulin immunostaining production in the pancreas of diabetic rats and confirmed the pancreatic protective properties in our study[16]. Pancreatic β-cells treated with camel milk are more resistant to oxidation and more protective against diabetes, due to the effect of camel milk, as Khan et al. concluded (2025) and further reported that camel milk proteins promote β-cell development and anti-streptozotocin cytotoxicity[11].

In this study, metformin (Group C) treatment reduced blood glucose levels greatly for 125.48 ± 5.52 mg/dl and restored insulin levels with the highest concentration of camel milk (Group F). Glycemic control also improved significantly in camel milk (138.05 ± 5.74 mg/dl) and oxidative stress control (group F). We observed that camel milk and metformin combination therapy have been shown by one study in the 2022 study to have a significant effect in reducing hyperglycemia in diabetic rats, with the combination effect of camel milk over not necessarily both measures proving to have greater success than metformin alone[17]. This may indicate that it is hoped that camel milk could contribute to the metformin by its own or another synergistic action, and in combination with certain kinds of therapy, is very positive or negative, but different. Further, Mansour et al. (2017) showed that camel milk supplementation in diabetic rats showed up-regulated expression of IRS-2, PK, and FASN genes, while metformin did not decrease CPT-1 expression the same way. Moreover, camel milk might offer certain additional benefits over metformin alone[16].

Another interesting finding of this survey was its dose-dependent response towards camel milk therapy. Concentration of 8 mL/kg, Group F appeared to be consistently favourable and showed the highest response in results throughout. Mudgil et al. (2021) found that camel milk protein hydrolysates at 500 mg/kg body weight exhibited potent hypoglycemic activity, with higher doses producing greater effects.[13] Similarly, Khan et al. (2025) observed dose-dependent improved glycemic control and insulin sensitivity with increased concentrations of camel milk protein hydrolysates[11]. For the present study, we employed freeze-dried camel milk powder to precisely deliver on

our dosing as well as solve the practical challenges of fresh camel milk, as highlighted by Mbye et al. (2025)[1].

A few limitations of the positive outcome are to be discussed. The sample size was so small, it was only sized out to 6 for each group. The treatment time for this period was 4 weeks. Secondly, as MDA indicates oxidative stress, additional markers such as superoxide dismutase and catalase should be tested in future studies. The bioactive components responsible for the observed effects may also be found via fractionation and proteomic investigation and should also be further focused on in upcoming work. There is also great potential to extend the use of camel milk alongside antidiabetic drugs by introducing a clinical application.

Conclusion

In conclusion, our findings from the Arab camel milk study are quite encouraging that Camel milk has significant protective effects on the pancreas in diabetic rats having streptozotocin in their diet. The highest concentration of camel milk (8 mL/kg) effectively decreased blood glucose levels, normalized insulin secretion, diminished oxidative stress, and preserved pancreatic islet structure, it has a similar effect to metformin, but less potent. The findings confirm the significance of camel milk for diabetes management, and it is an important adjunct medicine.

conflicts of Interest

The authors have no conflicts of interest. There are no commercial and financial relationships in the study, which could potentially violate any of those limitations. In any way, it was not the design of this study; data collection and analyses or interpretation were completed, publication of this study or any of these was made with no commercial or financial benefit.

Acknowledgments

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Authors' contributions

The student collected the samples, performed the diagnostic tests, and studied the histological sections by Eslam Abd Thabt. The experiment was designed and supervised by Eqbal Awadh Gatea

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