Evaluation of Therapeutic Efficiency of Camel Milk on Alloxan-induced Diabetic Rats

[1]Manal M. E. M. Shehata and [2]Eman A. Moussa

[1]Department of Food Science, Faculty of Agriculture, Zagazig University, Zagazig, Egypt
[2]Department of Zoology, Faculty of Science, Kafr El-Sheikh University, Egypt
Shehata_manal@yahoo.com

Abstract: Diabetes mellitus (DM) is a metabolic disorder in which the carbohydrate and lipid metabolism is improperly regulated by insulin. This study aimed to evaluate the therapeutic efficiency of camel milk with different treatment. Diabetes and hyperlipidemia was induced by the intraperitoneal injection of alloxan (150mg/ kg body weight). Seventy two male albino rats were divided into nine groups of eight rats each and treated as following: G1 was fed on normal basal diet (negative control), G2 diabetic rats (positive control),G3 diabetic + raw camel milk, G4 diabetic + pasteurized camel milk, G5 diabetic + boiled camel milk, G6 diabetic + camel milk stored in the refrigerator at 4°C for two days, G7 diabetic + camel milk stored in the refrigerator at 4°C for four days, G8 diabetic + camel milk stored in a freezer at -20 °C for one day and G9 diabetic + reconstituted freeze dried camel milk. After eight weeks of feeding, results showed significant decrease $P < 0.05$ in levels of glucose, glycosylated hemoglobin A_{1c}, and significant increase $P < 0.05$ in insulin as compared with diabetic rats. Data revealed significantly decrease $P < 0.05$ of TC, TG, LDL and VLDL as compared with diabetic rats. However, HDL-C was significantly increase $P < 0.05$ as compared with diabetic rats. ALT and AST in treated rats were decreased significantly $P < 0.05$, in contrast the reduced glutathione was increased significantly $P < 0.05$ as compared to the diabetic group. Urea and creatinine in treated rats were decreased significantly $P < 0.05$ as compared to the diabetic group. Regarding liver tissue extract, the levels of total cholesterol and triglyceride decreased significantly for treated rats with raw camel milk and camel milk stored in the refrigerator at 4°C for two days as compared to the diabetic group. Data also indicated that, therapeutic efficiency of camel milk was lost after heating camel at 100°C (boiled camel milk). Raw camel milk showed the significant highest efficiency in all parameters. These findings indicate that camel milk have a potential benefits in the treatment of diabetes and play a role in its management as well as reduces the risk of diabetic complications.

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1. Introduction

Diabetes mellitus, an epidemic with numerous devastating complications, has risen dramatically over the past two decades, with substantial variation worldwide (**King *et al.*, 1998 and Diamond, 2003**). The global prevalence of diabetes mellitus for all age groups was estimated to be 2.8% in 2000 and is projected to rise to 4.4% in 2030 (**Wild *et al.*, 2004**). Major part of this increase has been projected to occur in third world countries. Diabetes mellitus (DM) is a metabolic disorder disease in which the carbohydrate and lipid metabolism is improperly regulated by insulin (**Radhika *et al.*, 2011**). Diabetes mellitus is characterized by constant high levels of blood glucose (hyperglycemia). The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart and blood vessels (**ADA, 2008**). Diabetes mellitus is known to cause hyperlipidemia through various metabolic derangements. Among several metabolic derangements, insulin deficiency has been known to stimulate lipolysis in the adipose tissue and gives rise to hyperlipidemia and fatty liver. Thus, in diabetes hypercholesterolemia and hypertriglyceridemia often occurs (**Goodman *et al.*, 2006**). The treatment for diabetes mellitus involves either administration of exogenous insulin or oral hypoglycemic drugs. However, this approach is not completely satisfactory in a large proportion of patients and there is still a need to look for new drugs as no drug (except strict glycemic control with insulin) has been shown to modify the course of diabetic complications. Throughout the world, many types of traditional food treatments for diabetes exist (**Lee *et al.*, 2003; Fujita*et al.*, 2003 and Lee *et al.*, 2004**).

Camel's milk is differed from other ruminant milk; it is low in cholesterol, sugar and protein but high in minerals (sodium, potassium, iron, copper, zinc and magnesium), vitamins A, B_2, C and E, and contains a high concentration of insulin (**Knoess, 1979**). It also contains fat with a relatively large amount of polyunsaturated fatty acids and linoleic acids, which are essential for human nutrition (**Gorban and**

Izzeldin, 2001). Camel milk has a high biological value due to the higher contents of antimicrobial factors such as lysozyme, lactoferrin and immunoglobulins (**Elagamy *et al.*, 1992**) and it has concentration of these components higher than cow and buffalo milk (**Elagamy, 2000**). Several studies have been carried out at the Zayed Complex, UAE showed that camel milk has the IgA and IgG that have proved effective against several viral and bacterial Pathogens (**Khitam, 2003**). Camel milk is known to have medicinal properties since ancient times. Oral camel milk is well tolerated by lactase-deficient children who are allergic to cow milk (**Elagamy *et al.*, 2009**), and it shows protective effects against heavy metal toxicity (**Al-Hashem *et al.*, 2009**), chronic pulmonary tuberculosis (**Mal *et al.*, 2001**) and viral and bacterial infections (**Elagamy *et al.*, 1992**). Additionally, Indians used camel milk for the treatment of multiple acute and chronic health problems, including asthma, anemia, jaundice, and spleen problems (**Rao *et al.*, 1970**). Interestingly, the low prevalence of diabetes in the Raica community was attributed to the regular consumption of camel milk (**Agrawal *et al.*, 2007a**). This was further supported by the better glycemic control in diabetic patients and animals receiving camel milk (**Agrawal *et al.*, 2003 and Al Haj and Al Kanhal, 2010**).

Therefore, the present study was carried out to evaluate the therapeutic efficiency of camel milk on alloxan-induced diabetic rats. Effect of heat treatments, cooling, storage and drying of camel milk on its therapeutic efficiency was also a goal of the study.

2. Material and Methods

Material

Camel milk samples

Daily milk samples were collected early in the morning from camel farm in Bilbis desert area (Sharkia governorate). The samples were collected in sterile screw bottles and kept in cool boxes until transported to the laboratory.

Chemicals:

Alloxan was purchased from Sigma Chemical Company (St Louis Mo, USA). All of kits were purchased from Biodiagnostic, Dokki, Giza, Egypt. All other chemicals were of analytical grade.

Experimental animals

A total of seventy two male albino rats weighing (100-110 g) were obtained from El- Salam Farm Giza, Egypt and used in this study.

Methods

Induction of diabetes

The animals were fast overnight, and received a single intraperitoneal injection of freshly prepared alloxan using citrate buffer 0.1M (pH = 4.5) as vehicle, at a dose of 150 mg alloxan/kg body weight (**Szkudelski, 2001**). Control rats were injected with citrate buffer alone. The animals were considered as diabetic, if their blood glucose values were above 240 mg/dl on third day after alloxan injection.

Experimental design

Seventy two male albino rats were housed in stainless steel cages under standard conditions of humidity, temperature and light (12 h light/12 h dark) and give free access to food and water at all time. After one week of acclimatization, animals were divided randomly into nine groups of eight rats in each group. The rats were fed a standard diet according to (**Reeves *et al.*, 1993**). It contained 14% casein, 5% cellulose, 3.5% mineral mixture, 1% vitamin mixture, 0.25% choline, 0.3% Dl-methionine, 5% oil and 65% starch. All treatments rats were given camel milk at a dose of 50 ml /rat/day (**Korish and Arafah, 2013**), using feeding bottle, every day for eight weeks. The groups were treated as follows:

Group (G1) control rats (negative control group).

Group (G2) diabetic rats (positive control group).

Group (G3) diabetic rats fed with raw camel milk.

Group (G4) diabetic rats fed with pasteurized camel milk (at 63°C for 30 min.).

Group (G5) diabetic rats fed with boiled camel milk (at 100°C for 3min.).

Group (G6) diabetic rats fed with camel milk stored in the refrigerator at 4°C for two days.

Group (G7) diabetic rats fed with camel milk stored in the refrigerator at 4°C for four days.

Group (G8) diabetic rats fed with camel milk stored in a freezer at -20 °C for one day.

Group (G9) diabetic rats fed with reconstituted freeze dried camel milk (camel milk dried in freeze-dryer, ALPHA 1-2 LD plus).

Biochemical analysis

After eight weeks of feeding, the animals were fasted overnight before being sacrificed. The blood samples were divided into two tubes. Tube (1) blood was centrifuged with 3000 rpm for 10 min to separate blood serum, which is kept at –20°C until analysis. Tube (2) containing the anticoagulant ethylene diamine tetra acetic acid (EDTA) for determination of plasma glycosylated hemoglobin (HbA_{1C}). Samples were analyzed for the following biochemical parameter: Blood glucose (**Trinder, 1969),** insulin (**Temple *et al.*, 1992)** and HbA_{1C} **(Geiger and Binder, 1986)**. Lipids profile including total cholesterol (TC) (**Richmond, 1973)**, triglycerides (TG) **(Jacobs and Vandermark, 1960)** and high-density lipoprotein (HDL-C) (**Burstein *et al.*, 1970).** Calculation of LDL-C, V LDL-C, atherogenic index (AI) and HTR ratio involves an equations developed (**Friedewald *et al.*, 1972)**. Kidney functions including urea **(Patton and Crouch, 1977**) and creatinine (**Larson, 1972).** Liver functions including AST and ALT (**Reitman and Frankel,**

1957) and reduced glutathione (GSH) **(Beutler *et al.*, 1963).**

Extraction and Determination of TG and TC in Liver Tissue:

At the end of the experimental, Liver was removed, rinsed in ice chilled normal saline and blotted on filter paper and then tissues were cut into small portion and stored at - 20°C before use. Extraction of liver was analyzed for total cholesterol and triglycerides according to the method described by **Hostmark, 1987**: 1g of liver portion from each animal was homogenized in 10 ml isopropanol. The liver homogenate was allowed to stand for 48 h at 4°C. The mixture was centrifuged 15 min at 2500 rpm and the supernatant was used for analysis.

Statistical analysis

The obtained results were statistically analyzed using SPSS program (version 17.0), and expressed as mean and standard deviations (SD). Statistical significance (p <0.05) among the groups were determined by one-way ANOVA followed by Duncan's multiple range test according to the method by **Bailey, 1995**.

3. Results and Discussion

The effect of feeding camel milk of different treatments on levels of glucose and insulin in diabetic rats was shown in Table 1. Data in illustrated that a significant increase P <0.05 in level of glucose (243.35±2.21 mg/dl) of diabetic group (positive control group) compared to negative control group (99.62±2.43 mg/dl). This increase can be explained by a single dose of alloxan injected to rats was able to produce a reproducible model of diabetes mellitus that had minimal beta cell activity and elevated glucose. Similar results obtained by **Radhika *et al.*, 2011 and Akpan, 1989** who demonstrated that alloxan administration was associated with hyperglycemia. After eight weeks of administration of raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days, freezed camel milk at -20°C and reconstituted freeze-dried camel milk reduce level of glucose by 49.8, 36.8, 39.5, 33.8, 33.2 and 28.5%, respectively. Furthermore, the highest reduction in glucose level was in G3 (diabetic rate fed with raw camel milk). Also, the results indicated that therapeutic activity of camel milk was lost after heating at 100°C (boiled camel milk). These results probably due to the highest insulin/insulin like protein concentration in camel milk. Results were in agreement with **Singh, 2001** who reported that the camel milk contains a high concentration (52 units/liter) of insulin. It should be noted that camel milk does not form coagulum in the stomach or the acidic media, thereby it prevents degradation of insulin in the stomach (**Wangoh, 1993**). Amino acid sequences of some camel milk proteins are rich in half- cystine, which has superficial similarity with insulin family of peptides (**Beg *et al.*, 1986**). High mineral content (sodium, potassium, zinc, copper and magnesium) as well as a high vitamin C intake may act as antioxidant thereby removing free radicals (**Farah, 1993**). All these factors may contribute to the observed hypoglycemic effect of camel milk in the present study.

Insulin levels in diabetic rats were significantly P <0.05 decreased compared to control group. These results approved by **Mahmud *et al.*, 2004** who reported that insulin levels were decreased in diabetic rats. The treatments with raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days, freezed camel milk at -20°C and reconstituted freeze-dried camel milk showed significant increase P<0.05 in insulin levels when compared to the diabetic group. While, on feeding raw camel milk showed the highest increased of insulin levels (10.00±2.61 µU/ml) in the rats compared to the diabetic group (5.51±2.69 µU/ml). This increase may be due to insulin like protein and high amount of zinc present in camel milk.

Data of HbA_{1C} levels are shown in Table 1. Results indicated a significantly increase p< 0.05 in HbA_{1C} of diabetic group in comparison with control group. Treatments with raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days, freezed camel milk at -20°C and reconstituted freeze-dried camel milk decreased significantly p<0.05 the levels of HbA_{1C} as compared with diabetic group. These results probably due to the decrease of glucose levels in diabetic rats treated with a different treatments.

From the obtained results heat treatments, cooled and freezed storage and drying of camel milk may be leads to decrease the insulin concentration of camel milk, which leads to decrease the efficiency of camel milk to lower blood glucose level. These results were in accordance with findings of **Agrawal *et al.*, 2005** who reported that insulin/insulin like protein activity decreases on boiled camel milk. Diabetic control rats treated with boiled camel milk did not show any significant effect on glucose, insulin and HbA_{1C} levels. These results are accordance with data reported by **Elagamy, 2000** who indicated that heating camel milk at 100°C decreased α-lactalbumin and IgG at level higher than that of cow milk. He also reported that heat treatments showed completely denaturation of camel lactoferrine. Additionally, these results are accordance with data reported by **Attia, *et al.*, 2000a and Attia, *et al.*, 2000b** who demonstrated that heat treatment of camel milk allowed to a demineralization of the caseins micelle and to an increase of soluble Ca concentrations.

The results in Table 2 showed that a significant increase P <0.05 in total cholesterol (TC), total triacylglycerol (TG), low density lipoprotein (LDL) and very low density lipoprotein (VLDL) of diabetic

group compared to control group. While, HDL-C decreased significantly P <0.05 as compare to control group. A significant increase in TG (G2) may be due to the lack of insulin under diabetic condition. This result agreed with that of **Arkkila *et al.* 2001** who found that the abnormalities in the lipid metabolism may be due to insulin deficiency. While, the groups fed with raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days, freezed camel milk at -20°C and reconstituted freeze-dried camel milk decreased significantly $p < 0.05$ the levels of (TC), (TG), (LDL-c) and (VLDL-c) as compared with diabetic group and this was associated with a significant increase $p < 0.05$ in HDL-C in these groups. Diabetic control rats treated with boiled camel milk did not show any significant effect on lipids profile. TG in G3 (120.55±2.83 mg/dl) was significantly lowest $p <$ 0.05 as compare with diabetic group. These results are supported with those of **Hull, 2004 and Agrawal *et al.*, 2007b** showing that a high insulin concentration of camel milk can cause the activation of lipoprotein lipase enzyme. Furthermore, there are decreases in total cholesterol; rate of decrease was 33.4, 19.8, 21.2,18.3, 17.6 and 16.5%, respectively of raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days, freezed camel milk at -20°C and reconstituted freeze-dried camel milk as compared to the diabetic group.

In our study, the diabetic rats treated with camel milk showed an elevation in HDL-C and reduction in LDL-C and VLDL-C. Thus, raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days, freezed camel milk at -20°C and reconstituted freeze-dried camel milk could alleviate the risk of cardiovascular diseases.

In Table 3, the results showed the effect of different heat treatments, stored temperature and drying of camel milk on AI, LDL-C/HDL-C Ratio and HTR of the diabetic rats. The atherogenic index markedly decreased causing a significant reduction in LDL/HDL ratio in groups fed with raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days, freezed camel milk at -20°C and reconstituted freeze-dried camel milk. Also, the results indicated that a significant increase p <0.05 in HTR ratio as compare with diabetic group. These results agreed with those **Makni *et al.*, 2008** who stated that the increase in HDL-C or HTR ratio is one of the most important criteria of anti-hypercholesterolemic agent.

A comparison of the liver functions parameters data for the raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days, freezed camel milk at -20°C and reconstituted freeze-dried camel milk administration is shown in Table 4. A significant increase $p < 0.05$ in the levels of liver enzymes (ALT and AST) appeared in diabetic rats. These results are accordance with data reported by **Sunil *et al.*, 2011** who indicated that the liver enzymes ALT and AST levels were increased in alloxan diabetic rats. This elevation reflected the generally recognized detrimental effect of hepatocyte damage, which represented in the leakage of ALT and AST from damaged hepatic cells. The results showed that a significant p <0.05 overall improvement in liver functions parameters appeared within diabetic rat groups of raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days, freezed camel milk at -20°C and reconstituted freeze-dried camel milk feeding, with a particular respect to the highest refinement effect in raw camel milk group. Accordingly, it is interested to note that giving raw camel milk led to an improvements in both ALT and AST activities by 47.3 and 19.2%, respectively, as compared to diabetic rats. This finding is consistent with the observation of **Magjeed, 2005 and Khan and Alzohairy, 2011** who found that giving camel milk improved the levels of ALT and AST activities in intoxicated rats.

Data of reduced glutathione in Table 4 show decrease in glutathione (GSH) in diabetic rats (17.53±1.24 mg/dl) compared to control group (25.23±1.41 mg/dl). Our results are in accordance with the results obtained by **Annamalai *et al.*, 2003** who observed a decreased in level of GSH in streptozotocin diabetic rats. Treated with raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days and freezed camel milk at -20°C were increased significantly p <0.05 of glutathione levels as compare with diabetic group. The present findings are in accordance with data reported by **Wohaieb and Godin, 1987** who demonstrated that hepatic GSH content was lower in diabetic rats which was restored by insulin treatment. This decrease may be due to a decline in its formation which requires $NADPH+H^+$ and glutathione reductase (**Garg *et al.*, 1996)**.

Data in Table 5 illustrated that kidney functions of diabetic rats fed with raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days and freezed camel milk at -20°C and reconstituted freeze-dried camel milk. It could be noticed that significantly increase $p < 0.05$ in urea (53.40±2.23mg/dl) and creatinine (2.25±2.65 mg/dl) in diabetic group compared to control group (33.26±1.31 and 0.78±3.42 mg/dl, respectively). A similar finding was recorded by **Niewoehner *et al.*, 1986** who reported that urea were elevated in diabetic subjects. Also, this result is in agreement with that of **Sunil *et al.*, 2011** who reported that urea and creatinine levels were increased in alloxan diabetic rats. Treatment with raw camel milk, pasteurized camel milk, stored camel milk at 4°C for two and four days and freezed camel milk at -20°C and reconstituted freeze-dried camel milk

decreased significantly $p < 0.05$ levels of urea and creatinine as compared with diabetic rats. The raw camel milk was the most effective in this respect. Diabetic rats treated with boiled camel milk did not show any significant effect on liver and kidney functions.

Total cholesterol and triglycerides levels in the liver tissues of diabetic rats are given in Table 6. The diabetic rats had significantly increase $p < 0.05$ in levels of total cholesterol and triglycerides in diabetic group compared to control group. Treatment with raw camel milk, pasteurized camel milk and stored camel milk at 4°C for two days decreased significantly $p < 0.05$ levels of TG as compare with diabetic group. While, treatment with raw camel milk and stored camel milk at 4°C for two days decreased significantly $p < 0.05$ levels of TC in comparison with the diabetic group.

Table 1: Effect of feeding camel milk of different treatments on Glucose, insulin and HbA_{1C} of diabetic rats (Mean±SD).

Experimental Groups	**Glucose** (mg/dl)	**Insulin** (µU/ml)	**HbA_{1C}** (%)
(G1) Control (negative control group)	99.62±2.43^{g}	15.30 ±1.28^{a}	5.10±2.01^{f}
(G2) Diabetic (positive control group)	243.35±2.21^{a}	5.51±2.69^{c}	11.83±2.31^{a}
(G3) Diabetic + raw camel milk	122.16±1.82^{f}	10.00±2.61^{b}	7.78±2.18^{d}
(G4) Diabetic + pasteurized camel milk	153.82±1.51^{d}	9.28±1.85^{b}	9.18± 1.28^{c}
(G5) Diabetic +boiled camel milk	240.87±1.41^{a}	5.63±2.83^{c}	11.21±2.69^{a}
(G6) Diabetic + stored camel milk at 4°C for 2 days	147.23±1.26^{e}	9.45±2.16^{b}	9.01±2.11^{c}
(G7) Diabetic + stored camel milk at 4°C for 4 days	161.10±2.28^{c}	9.28±1.41^{b}	9.75±2.16^{bc}
(G8) Diabetic + freezed camel milk at -20°C for one day	162.56±1.31^{c}	9.21±2.61^{b}	9.84±1.41^{bc}
(G9) Diabetic + reconstituted freeze dried camel milk	174.10±1.21^{b}	8.89±1.22^{b}	10.41±1.69^{b}

HbA_{1C}: Glycosylated hemoglobin.
Means with different superscript letters in each column are significantly different ($P < 0.05$).

Table 2: Effect of feeding camel milk of different treatments on lipids profile (TG, TC, HDL-C, LDL-C and VLDL-C) of diabetic rats mg/dl (Mean ±SD).

Experimental Groups	TG	TC	HDL-C	LDL-C	VLDL-C
(G1) Control (negative control group)	85.82± 2.61^{g}	70.32±2.28^{f}	36.84±2.61^{a}	16.32±3.16^{g}	17.16±3.16^{e}
(G2) Diabetic (positive control group)	183.21±2.83^{a}	114.30±3.69^{a}	28.95±3.16^{e}	48.71±2.28^{a}	36.64±2.28^{a}
(G3) Diabetic + raw camel milk	120.55±2.83^{f}	76.12±3.41^{e}	35.43±2.99^{b}	16.58±2.98^{g}	24.11±2.98^{d}
(G4) Diabetic + pasteurized camel milk	146.57±3.16^{d}	91.67±2.61^{cd}	33.47± 3.42^{cd}	28.89± 2.28^{e}	29.31± 2.28^{bc}
(G5) Diabetic + boiled camel milk	181.22± 1.21^{a}	112.88±1.31^{a}	29.92±2.61^{e}	46.72±2.28^{b}	36.24±2.28^{a}
(G6) Diabetic + stored camel milk at 4°C for 2 days	143.82±2.83^{e}	90.10±3.41^{d}	33.93±3.16^{c}	27.41±2.83^{f}	28.76±2.83^{c}
(G7) Diabetic + stored camel milk at 4°C for 4 days	150.23±2.61^{c}	93.38±2.28^{bc}	33.38±2.18^{cd}	29.95±2.98^{de}	30.05±2.98^{bc}
(G8) Diabetic + freezed camel milk at -20°C for one day	150.60±2.83^{bc}	94.18±3.69^{b}	32.86±3.32^{cd}	31.20±2.00^{cd}	30.12±2.00^{bc}
(G9) Diabetic + reconstituted freeze dried camel milk	152.80±2.83^{b}	95.44±3.41^{b}	32.40±2.83^{d}	32.48±3.25^{c}	30.56±3.25^{b}

TG: Triacylglycerol, **TC**: Total Cholesterol, **HDL-C**: High density lipoprotein Cholesterol, **LDL-C**: Low density lipoprotein Cholesterol, **VLDL-C:** Very Low density lipoprotein Cholesterol.
Means with different superscript letters in each column are significantly different ($P < 0.05$).

Table 3: Effect of feeding camel milk of different treatments on AI, LDL-C/HDL-C and HTR of diabetic rats (Mean ±SD).

Experimental Groups	AI*	LDL/HDL Ratio	HTR Ratio
(G1) Control (negative control group)	0.91±1.27 ^{c}	0.44±1.28 ^{c}	52.39±2.13 ^{a}
(G2) Diabetic (positive control group)	2.95±1.14 ^{a}	1.68±1.14 ^{a}	25.33±2.33 ^{f}
(G3) Diabetic + raw camel milk	1.15±1.58^{c}	0.47±1.25^{c}	46.54±2.92^{b}
(G4) Diabetic + pasteurized camel milk	1.74±1.79^{abc}	0.86±2.16^{b}	36.51±2.19^{cd}
(G5) Diabetic +boiled camel milk	2.77±1.14^{ab}	1.56±1.62^{a}	26.51±2.42^{f}
(G6) Diabetic + stored camel milk at 4°C for 2 days	1.66±1.04^{bc}	0.81±1.72^{b}	37.66±2.24^{c}
(G7) Diabetic + stored camel milk at 4°C for 4 days	1.80±1.36^{abc}	0.90±1.54^{b}	35.75±2.83^{d}
(G8) Diabetic + freezed camel milk at -20°C for one day	1.87±2.31^{abc}	0.95±1.21^{b}	34.89±2.83^{de}
(G9) Diabetic + reconstituted freeze dried camel milk	1.95±1.49^{abc}	1.00±1.54^{b}	33.95 ±1.53^{e}

* Atherogenic index= (TC- HDL-C) /HDL-C HTR ratio = HDL-C / TC x 100
Means with different superscript letters in each column are significantly different ($P < 0.05$).

Table 4: Effect of feeding camel milk of different treatments on liver functions (AST, ALT and GSH) of diabetic rats (Mean ±SD).

Experimental Groups	AST (U/L)	ALT (U/L)	GSH (mg/dl)
(G1) Control (negative control group)	93.70±2.61 ^{f}	43.3±3.11 ^{h}	25.23±1.41 ^{a}
(G2) Diabetic (positive control group)	150.86±2.28 ^{a}	132.07±2.61^{a}	17.53±1.24^{d}
(G3) Diabetic + raw camel milk	121.89±1.16^{e}	69.60±3.41^{g}	20.60±1.43^{b}
(G4) Diabetic + pasteurized camel milk	128.23±2.28^{d}	86.77±2.28^{e}	19.77±1.67^{bc}
(G5) Diabetic +boiled camel milk	148.90±3.22^{a}	131.24±1.41^{a}	18.00±1.45^{cd}
(G6) Diabetic + stored camel milk at 4°C for 2 days	126.72±2.61^{cd}	82.94±2.16^{f}	19.97±1.52^{b}
(G7) Diabetic + stored camel milk at 4°C for 4 days	130.95±2.23^{cd}	90.47±2.61^{d}	19.56±2.02^{bc}
(G8) Diabetic + freezed camel milk at -20°C for one day	132.91±2.43^{c}	93.64±2.16^{c}	19.46±2.02^{bc}
(G9) Diabetic + reconstituted freeze dried camel milk	138.19±2.18^{b}	99.98±2.28^{b}	19.12±1.71^{bcd}

AST: aspartate aminotransferase, **ALT**: alanine aminotransferase, **GSH**: Reduced Glutathione.
Means with different superscript letters in each column are significantly different ($P < 0.05$).

Table 5: Effect of feeding camel milk of different treatments on kidney functions (urea and creatinine) of diabetic rats mg/dl (Mean ±SD).

Experimental Groups	Urea	Creatinine
(G1) Control (negative control group)	33.26±1.31 ^{g}	0.78±3.42 ^{d}
(G2) Diabetic (positive control group)	53.40±2.23 ^{a}	2.25±2.65^{a}
(G3) Diabetic + raw camel milk	39.78±1.12^{f}	1.40±2.41^{c}
(G4) Diabetic + pasteurized camel milk	45.92±2.56^{de}	1.70±1.29^{b}
(G5) Diabetic +boiled camel milk	52.80±3.21^{a}	2.18±1.44^{a}
(G6) Diabetic + stored camel milk at 4°C for 2 days	45.07±2.81^{e}	1.69±2.46^{b}
(G7) Diabetic + stored camel milk at 4°C for 4 days	47.10±2.24^{cd}	1.80±2.67^{b}
(G8) Diabetic + freezed camel milk at -20°C for one day	47.53±1.41^{c}	1.82±2.46^{b}
(G9) Diabetic + reconstituted freeze dried camel milk	49.29±1.15^{b}	1.92±1.21^{b}

Means with different superscript letters in each column are significantly different ($P < 0.05$).

Table 6: Effect of feeding camel milk of different treatments on TC and TG in liver tissues of the diabetic rats (mg/g wet liver) (Mean± SD).

Experimental Groups	TG	TC
(G1) Control (negative control group)	12.02±1.32 ^{d}	2.54±2.12 ^{d}
(G2) Diabetic (positive control group)	16.83±2.35 ^{a}	3.74±1.45 ^{a}
(G3) Diabetic + raw camel milk	13.95±3.11^{c}	3.34±1.53^{c}
(G4) Diabetic + pasteurized camel milk	14.42±1.51^{c}	3.43±2.34^{abc}
(G5) Diabetic +boiled camel milk	16.02±2.13^{ab}	3.70±2.52^{ab}
(G6) Diabetic + stored camel milk at 4°C for 2 days	14.17±2.41^{c}	3.40±2.61^{bc}
(G7) Diabetic + stored camel milk at 4°C for 4 days	14.59±2.24^{bc}	3.47±3.25^{abc}
(G8) Diabetic + freezed camel milk at -20°C for one day	14.74±2.56^{bc}	3.49±2.33^{abc}
(G9) Diabetic + reconstituted freeze dried camel milk	15.15±2.21^{bc}	3.57±2.42^{abc}

TG: Triglycerides, **TC**: Total Cholesterol.
Means with different superscript letters in each column are significantly different ($P < 0.05$).

Conclusions

This study has demonstrated the therapeutic efficiency of camel milk for diabetic rats. Also, results indicated that camel milk with different treatments possesses anti-diabetic, hepato-renal protective and hypolipidemic effect in alloxan-induced diabetic rats. These results may have important implication for the clinical management of diabetes mellitus in humans.

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