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EFFECT OF RAW CAMEL MILK IN TYPE 2 DIABETES ANIMAL MODELS AND PATIENTS: TEN MONTHS RANDOMISED STUDY

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ABSTRACT

The efficacy of camel milk consumption as an adjunct to routine diabetes management in maintaining long-term glycaemia control in type 2 diabetes was assessed by 32 type 2 diabetes animal models and 12 patients. Thirty-two obese diabetic rats were divided into 4 groups according to method of random digits table. After 14 weeks treatment, it was observed that no significant variance compared Rosiglitazone Maleate group, Rosiglitazone Maleate+camel milk group and camel milk group with normal group. In blood glucose, blood triglycerides , blood total cholesterol, and plasma insulin there were a statistically significant change compared with diabetes group. Rosiglitazone Maleate group and Rosiglitazone Maleate +camel milk group showed significant variance in blood triglycerides (0.68±0.19 vs. 0.51±0.11 $p<0.05$), plasma insulin (43.52±18.93 vs. 26.49±5.60 $p<0.05$). Rosiglitazone Maleate+camel milk group was more similar to normal group. Rosiglitazone Maleate+camel milk group had better treatment of type 2 diabetes animal models than Rosiglitazone Maleate group.

Throughout the duration of the study, 6 randomly assigned patients underwent routine diabetes management and 6 randomly assigned patients additionally undertook daily consumption of raw camel milk (500 ml/day). In both groups, the dose of potential Rosiglitazone Maleate administration was adjusted to maintain a euglycaemic. In the group receiving camel milk, there was a significant increase in body weight (74.6±5.2 to 78.3± 3.4; $p<0.05$) and a significant reduction in mean blood sugar (123±19.8 to 94.2±14.3; $p<0.001$), plasma insulin (19.76±2.3 to 6.21±0.56; $p<0.001$), dose of drug (4.67±0.91to 1.67± 1.3; $p<0.001$), blood triglycerides (1.83±0.38 to 1.42±0.94; $p<0.001$), blood total cholesterol (7.7±1.53 to 6.1±0.91; $p<0.001$) compared to the values at the initiation of the study.

Based on the results, camel milk consumption may be considered as a useful prevention and adjunct to potential Rosiglitazone Maleate administration in the management of type 2 diabetes.

Key words: Animals models, camel milk, glycaemia control, Rosiglitazone Maleate, Type 2 diabetes

Diabetes is a syndrome characterised by disordered metabolism and abnormally high blood sugar resulting from insufficient levels of the hormone insulin (Tierney *et al*, 2002). Type 1 diabetes is also known as insulin-dependent diabetes mellitus (IDDM) or juvenile-onset diabetes. Type 2 diabetes is related to phenotypes such as obesity and insulin resistance. In 2000, according to the World Health Organisation, at least 2.8% of the population (Wild *et al*, 2004) suffers from diabetes. Diabetes especially type 2, is increasing rapidly and this number is estimated to be double that by the year 2030. Recent statistical data indicate that type 2 diabetes is a global health challenge. Studies showed that the total prevalence of diabetes in USA alone is 20.8 million (almost 7% of the total population) (Centres for Disease Control and Prevention, National Centre for Health Statistics, 2005). Despite production the genetic predisposition and the risk of developing type 2 diabetes in human beings increases with age, obesity, cardio-vascular disease (hypertension, dyslipidaemia) and a lack of physical activity, so controlling of this disease will require multi-purpose strategies, including dietary prevention as part of the solution.

It was found that one of the camel milk proteins has many characteristics similar to insulin (Beg *et al*, 1989). Radioimmunoassay tests of human milk revealed the highest concentration of insulin, i.e.

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60.23±41.05 micro unit/ml. The concentration of insulin in camel milk is also significantly higher (52 micro unit/ml) whereas, it is low in cow milk 16.32±5.98 micro unit/ml (Shehadeh *et al*, 2001). Camel milk is safe and efficacious in improving long term glycaemia control, and in the reduction of insulin requirement in type 1 diabetes patients (Agrawal *et al*, 2005). However, no data are available for the use of raw camel milk in type 2 diabetes.

The purpose of the present study were: to establish type 2 diabetes animal models; to determine the effect of blood sugar, blood lipids, and fasting blood insulin in type 2 diabetes animal models and to determine the long-term efficacy and safety of camel milk as prevention and adjunct to therapy in patients with type 2 diabetes.

Materials and Methods

Milk sample

Raw camel milk was collected in the village of Suoyang (Jiuquan), Gansu from 2 herds of 45 females. The milk was collected, immediately cooled and stored in refrigerator at 4°C for therapy.

Drug

Rosiglitazone Maleate (4 mg Tablets, Glaxo Smith Kline), were kept sealed after pulverisation, and precisely measured with electronic weighing scale before use. The dosage was adjusted to 2 mg/kg in accordance with the weekly rat's weight.

Animals

Sixty of the 6 week old healthy female Wister rats (Purchased from Lanzhou University Laboratory Animal Centre, LULAC were selected, with a body weight of 80-200 gm. The rats were kept in our vivarium, 6 to each cage in a room illuminated for 12 h/day with artificial light. Two groups of Wister rats were maintained on 2 different diets and given water ad-libitum. The experimental group (n=50) received a specially designed high-fat diet (Purchased from LULAC) known to induce a diabetes state, and the control group (n=10) were fed a standard normal diet (Purchased from LULAC, Total calories 16.17×10KJ/Kg), blood samples were taken by cutting the tail. The Rats was treated according to international regulation dealing with the treatment of animal and approved the institution ethical committee applied experimental procedures.

The establishment of type 2 diabetes animal models

The animals were weighed on the day and comparing weekly body weight with the experimental group and the normal group. Blood glucose level and insulin concentration in the serum on the empty stomach was detected by radio-immunoassay. Blood triglycerides (TG), and blood total cholesterol (TC) were measured by enzyme method using a fully automatic Biochemical Analyser (Hitachi 7170S). After six weeks (Table 1), diet-induced method has produced forty animal models for the study of type 2 diabetes (Magdy El-Sally, 2002), (Table 2). Thirty-two obese diabetes rats were divided into 4 groups according to method of random digits table (Table 3); diabetes group (n=8), drug group (n=8), drug+camel milk group (n=8), camel milk group (n=8).

Treatment of animal models

Intragastric administration was begun at 9 every morning. Diabetes group were received 4 ml

Table 1. The data of rat's body weight (mean ± SD).

Time	Rats body weight (g)	
	Normal group	Experiment group
After 1st week	202.5±8.41	207.4±8.51
After 2nd week	208.0±6.99	223.6±12.91
After 3rd week	221.5±9.40	228.86±31.30
After 4th week	233.50±7.52	239.80±40.31
After 5th week	244.00±10.35	256.10±10.87**
After 6th week	253.50±8.41	269.60±9.21**

** indicates significance at p<0.001 in comparison to normal group

Table 2. Characteristics of the animal model for diet-induced type 2 diabetes mellitus (all values expressed as mean ± SD).

	Normal group	Animal models group
Body Weight (g)	253.50±8.41	269.60±9.21**
Blood glucose	6.24±0.89	8.65±1.45**
Blood triglycerides	0.74±0.10	0.72±0.61*
Blood total cholesterol	1.33±0.16	1.72±0.24**
plasma insulin(FIN)	18.91±6.32	53.40±34.60**

** indicates significance at p<0.001 in comparison to normal group

* indicates no significance at p<0.05 in comparison to normal group

Table 3. The number of experimental animals.

Group	n	The number of animals
Normal group	10	10,16,21,26,33,38,41,47,49,55
Diabetes group	8	1,17,22,35,36,42,58,59
Drug group	8	11,13,28,40,46,51,53,50
Drug+camel milk group	8	7,19,23,25,29,43,52,56
Camel milk group	8	9,12,14,27,34,39,48,54

distilled water; drug group were received 2 mg/kg Rosiglitazone Maleate (resuspended in 4 ml distilled water); drug+camel milk group were received 2 mg/kg Rosiglitazone Maleate (resuspended in 4 ml camel milk); camel milk group were received 4 ml camel milk.

Patients

A total of 12 type 2 diabetes patients were randomly recruited from the outpatient diabetes clinic in the First Hospital of Lanzhou University, Lanzhou, China. All subjects approved the protocol and gave written consent before participation in this study. The patients were advised to follow a strict diet, exercise and drug treatment regime for 1 month. After a one-month period these patients were randomly divided into groups. Group 1 (n=6) received usual care, i.e. diet, exercise and drug and group 2 patients (n=6) received 500 ml of fresh camel milk daily for 10 months in addition to the usual care.

Patients with any type 2 diabetes symptoms like hyperglycaemia, high plasma insulin and high blood fat, a serious lack of insulin were not included in the study.

Blood sugar level and plasma insulin was measured twice weekly before breakfast, and drug doses were decreased weekly according to the blood sugar. Patients were also instructed to record the glucose reading and drug doses in diaries.

Statistical analysis

Data of experiment were analysed using SPSS statistical package programme (2000). The paired Student's t – test was used to study the differences between groups, data are expressed as mean±SD. If the probability (p) was below 0.05, the differences between groups were considered as significant.

Results

Animals results

After 14 weeks there were a statistically significant change between normal group and diabetes group (Table 4), in blood glucose (6.52±0.92 vs. 9.77±0.87 p<0.001), blood triglycerides (0.54±0.11 vs. 0.95±0.27 p<0.001), blood cholesterol (1.43±0.35 vs. 2.19±0.35 p<0.001) and plasma insulin (30.63±11.34 vs. 62.20±29.12 p<0.001). No significant variance were observed compared within drug group, drug+camel milk group, Camel milk group and normal group in blood glucose, blood triglycerides, blood total cholesterol, and plasma insulin. There was a statistically significant change compared with diabetes group (Table 4); drug group and drug+camel milk group had significant variance in blood triglycerides (0.68±0.19 vs. 0.51±0.11 p<0.05), plasma insulin (43.52±18.93 vs. 26.49±5.60 p<0.05). Drug+camel milk group was more similar to normal group (Fig 1-a, Fig 1-b).

Compared to drug group, drug+camel milk group, camel milk group, and diabetes group with normal group, there was a statistically significant variance in mean body weight of rats from the beginning to the third week end of treatment, but there was no significant change in each group at the end of test (Fig 2, Table 5).

Patients results

Demographic characteristics of both the groups were comparable. The group 1 (control group) and group 2 (camel milk group) were similar in age (50 years ± 2.6 vs. 49 years ± 3.2), sex (5 male and 1 female in both groups), body weight (74.3± 6.3 vs. 74.2± 5.2), FBS (125 ± 18.5 vs. 123± 19.8), plasma insulin (19.45± 2.2 vs. 19.76± 2.3), blood triglycerides (1.8 5± 0.42 vs. 1.83± 0.38) and blood total cholesterol (7.4± 1.23 vs. 7.7± 1.53) (Table 6).

After 10 months of treatment with fresh camel milk there was a statistically significant change in FBS (123± 19.8 vs. 94.2± 14.3 p<0.001), plasma insulin (19.76± 2.3 vs. 6.21± 0.56 p<0.001), body weight (74.6± 5.2 vs. 78.3± 3.4 p<0.05), blood triglyceride (1.83± 0.38 vs. 1.42± 0.94 p<0.001), blood total cholesterol (7.7± 1.53 vs. 6.1± 0.91 p<0.001) in group 2. But when

Table 4. The results of blood sugar, blood lipid, and plasma insulin by the end of the experiment (mean± SD).

Groups	Blood glucose	Blood triglycerides	Blood total cholesterol	Plasma insulin
Normal group	6.55±0.92	0.54±0.11	1.43±0.35	30.63±11.34
Diabetes group	9.77±0.87$^{\#}$	0.95±0.27$^{\#}$	2.19±0.35$^{\#}$	62.20±29.12$^{\times}$
Drug group	6.61±1.33*‡	0.68±0.19*§	1.78±0.23*	43.52±18.93*
Drug+camel milk group	6.38±1.25*‡	0.51±0.11*§	1.38±0.30*§	26.49±5.60$^{*\S\&}$
Camel milk group	7.15±1.93*§	0.71±0.20*	1.68±0.30*§	47.18±20.10*

- Compared with control group # p<0.001, * p>0.05, × p<0.05
- Compared with obese diabetes ‡ p<0.001, § p<0.05,
- Compared with drug diabetes & p<0.05

one-way ANOVA for FBS was used, no significant variance was observed (Fig 3). There were no significant change in body weight, blood triglyceride, and blood total cholesterol in group 1 (Table 6). There was a significant reduction in the mean dose of drug (4.67±0.91 vs. 1.67± 1.3; p<0.001) in patients receiving camel milk (Table 6, Fig 4). There was no significant reduction in mean doses of drug in individual patient not receiving camel milk (Table 6, Fig 5). In camel milk consuming group every patient had a significant reduction in the doses of drug. In 2 patients, there was no requirement for drug therapy after camel milk consumption (Fig 4). Severe hyperglycaemic event or DKA were not that reported in either group.

Discussion

The present study was performed to observe the role of camel milk in prevention and adjunct to therapy in type 2 diabetes. We successfully established type 2 diabetes animal models. Through the therapeutic test of animal models, the conclusions were drawn that camel milk can effectively control blood sugar, and reduce blood lipids, fasting blood insulin in type 2 diabetes Wister rats. There was no significant change in body weight of rats in either group at the end of test. A significant improvement in mean body weight (74.6± 5.2 vs. 78.3± 3.4, p< 0.001) after ten months of camel milk treatment was obseved. Agrawal *et al* (2005) found that there was a significant improvement in mean BMI after 1 year of camel milk treatment, and explained that the positive effects in weight gain may be because of good nutritional value of camel milk.

The important observation of this study was the significant reduction in drug doses to obtain glycaemia control at the end of 10 months in patient taking camel

Table 5. The data of rat's body weight (mean ± SD).

Time	Rats body weight(g)				
	Normal group	Diabetes group	Drug group	Drug+camel milk group	Camel milk group
7 weekend	241.80±75.21	280.88±8.23*	278.75±13.82*	285.38±11.50*	280.63±14.21*
8 weekend	276.50±11.79	288.38±12.78*	291.88±13.87*	294.38±7.49*	292.25±13.82*
9 weekend	293.00±9.94	297.75±8.91	305.38±13.21*	304.88±4.68*	302.25±11.26*
10 weekend	298.50±11.05	308.25±7.10	312.75±11.26*	315.38±3.10*	311.00±12.05
11 weekend	308.60±18.02	312.13±9.03	321.25±8.76	322.25±5.17	316.50±12.82
12 weekend	316.50±11.07	323.38±12.48	320.00±11.95	325.88±5.30*	321.15±10.16
13 weekend	330.00±7.09	329.13±9.41	325.25±9.16	324.75±10.17	314.25±9.01
14 weekend	331.00±8.03	325.00±14.12	329.13±9.61	336.88±8.84	331.88±11.63

* indicates significance at p<0.05 in comparison to normal group

Table 6. Effect of camel milk on glycaemia , fat control and insulin regulation in type 2 diabetes patient (all values expressed as x±S).

Group 1 : Control group			
Variables	**Before Treatment**	**After Treatment**	**P value**
Mean Blood Sugar (mg/dl)	125±18.5	103 ±16.7	p<0.05
Plasma Insulin (mU/l)	19.45±2.2	7.89±0.67	p<0.05
Body Weight (kg)	74.3±6.3	73.2±7.4	NS
Dose of Drug (mg/day)	5±0.9	4.51±1.6	NS
Blood triglycerides (mmol/l)	1.8 5±0.42	1.81±0.39	NS
Blood total cholesterol (mmol/l)	7.4±1.23	7.2±0.82	NS
Group 2 : Camel milk group			
Mean Blood Sugar (mg/dl)	123±19.8	94.2±14.3	p<0.001
Plasma Insulin (mU/l)	19.76±2.3	6.21±0.56	p<0.001
Body Weight (kg)	74.6±5.2	78.3±3.4	P<0.05
Dose of Drug (mg/day)	4.67±0.91	1.67±1.3	P<0.001
Blood triglycerides (mmol/l)	1.83±0.38	1.42±0.94	P<0.001
Blood total cholesterol (mmol/l)	7.7±1.53	6.1±0.91	P<0.001

• NS= Not Significant

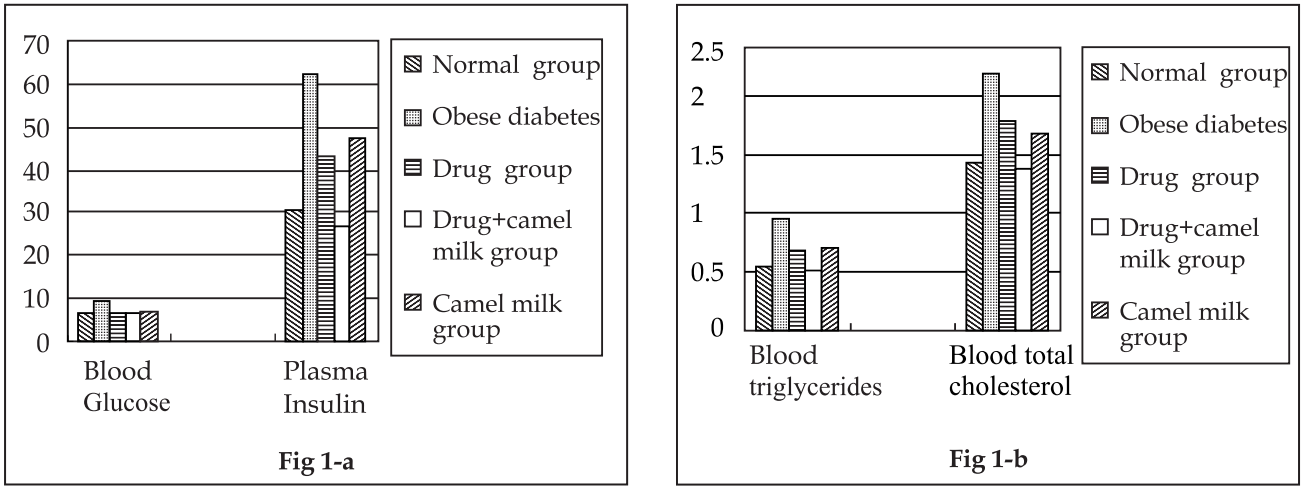

Fig 1. Comparison of normal group with treatment groups after 7 weeks therapy

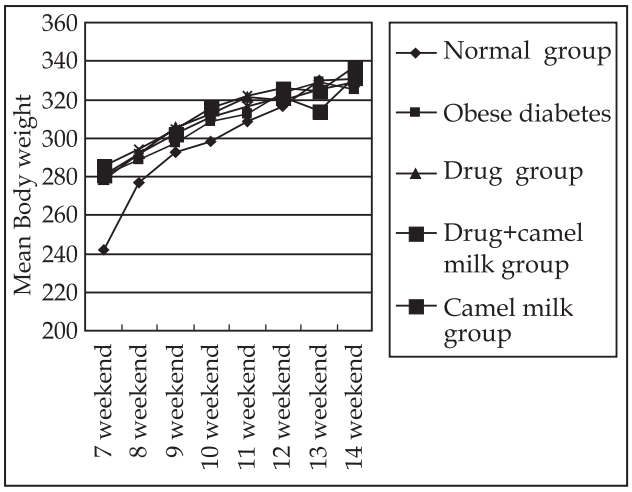

Fig 2. Body weight of rats measured over 8 weeks in normal group, obese diabetes, and treatment groups.

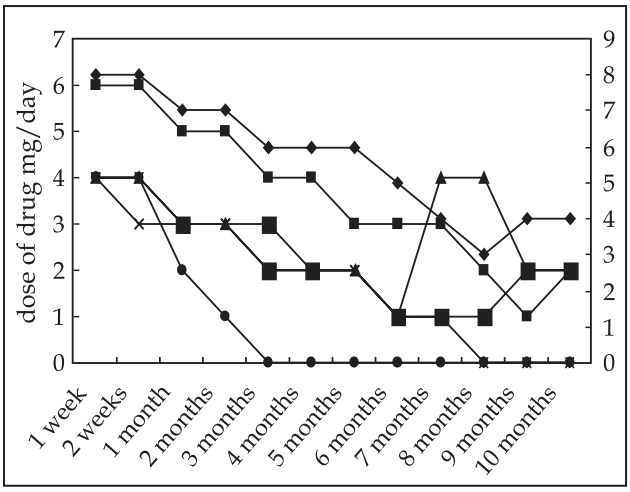

Fig 4. Mean drug doses per day individual patient of camel milk consuming in group 2 (n=6).

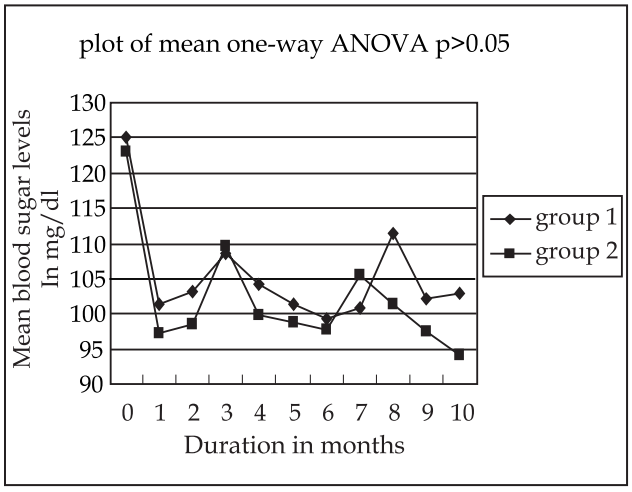

Fig 3. Mean blood sugar levels measured during the months in patient given fresh camel milk (group 2) and no camel milk (group 1).

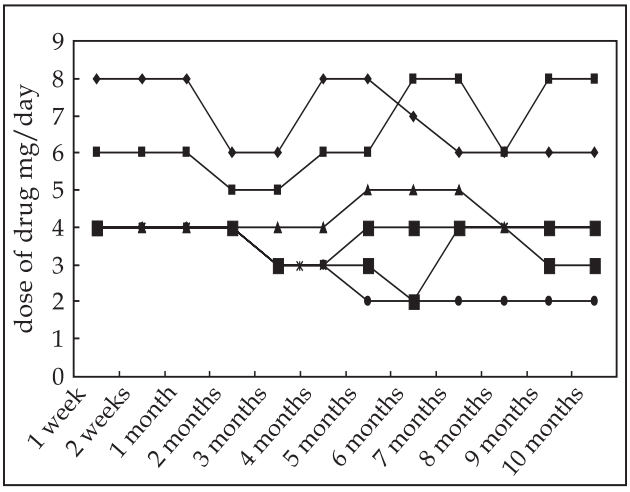

Fig 5. Mean drug doses per day in individual patients of control group (n=6)

milk. The requirement for mean dose of drug/day before treatment in patient of group 2 was 5±0.91. It came down rapidly initially and then gradually to a mean level of 2.01±1.3 (P<0.001). Six patients required the reduction dose of drug to maintain euglycaemic blood level. In 2 patients there were no requirement for Rosiglitazone Maleate therapy respectively, after 4 and 9 months of camel milk consumption.

Timmons *et al* (2001) specified that the vitamin C content of camel milk was approximately 5 times higher that vitamin C content of cow milk. There is good evidence for the efficacy of vitamin C in aqueous systems to protect cellular and liquid biological systems against oxidative damages. Yi *et al* (2006) showed that there was a certain degree of prevention and treatment of chronic complications in diabetes patient taking antioxidant treatment.

The fatty acid composition of milk is one of the important aspects linked to the discussion on the health effect of milk and milk products (Wahle and Heyes, 2002). Results of Narmuratova *et al* (2006) obtained on the fatty acid composition of camel milk fat from Kazakhstan were comparable to results of the literature, in special, the highest content of unsaturated fatty acids of these milk compared to cow milk. Stahl *et al* (2006) reported that the fraction of unsaturated fatty acids in the fatty acid pattern of camel milk was higher (49.5%) than findings by Gorban and Lzzeldin (2001) (30.2%) and Farah *et al* (1998) (35%). They also found that unsaturated fatty acids in the fatty acid pattern of camel milk was higher (49.5%) than that in the fatty acid (42.1%) pattern of cow milk, in particular higher content of C18:3n3 of camel milk (0.7%) comparable to cow milk (0.3%, P<0.012). Unsaturated fatty acids (30-35g/100g total milk fatty acids) are inversely associated to diabetes risk (Mann, 2002). Milk also contains beneficial minor components such as conjugated linoleic acid which plays an important role in prevention and treatment of diabetes (Schrezenmeir and Jagla, 2002) .

In recent years, studies have shown that polyunsaturated fatty acids are intrinsic activator of peroxisome proliferator-activated receptors (PPARs). Peroxisome proliferator-activated receptors (PPARs) belong to the steroid and thyroid hormone superfamily. PPARs are ligand-activated nuclear transcription factor receptor, and are closely related to glycolipids metabolism. There are 3 known subtypes, that is, PPARα,-β/δ and-c. These were closely related to the metabolic syndrome, especially with insulin resistance and type 2 diabetes. It can be combined with DNA and stimulate the transcription of certain genes. Forman *et al* (1997) reported that PPARs are nuclear receptors (NRs) that control many cellular and metabolic processes. Activation of peroxisome proliferator-activated receptor can enhance insulin expression in the transcription level and affect energy homeostasis and inflammation. It supports the explanation that camel milk has a good effect on the treatment of type 2 diabetes in rats and patients, and with Rosiglitazone Maleate similar treatment effectiveness is seen.

Beg *et al* (1986) indicated that amino acid sequence of some of the camel milk protein is rich in half cysteine, which has some similarities with the insulin family of peptides. Camel milk was found to contain approximately 52 micro unit/ml insulin (Shehadeh *et al*, 2001). The insulin content is not much high, but test in rabbits and rats indicated that the insulin is not destroyed in the stomach. It passes into the intestines causing a reduction in blood sugar (Wernery *et al*, 2006). It may explain that a better improvement occurs in diabetes patients receiving camel milk. In the current study, the therapeutic efficacy of camel milk is consistent with earlier clinical trials in this area (camel milk + insulin therapy) (Agrawal *et al*, 2003a, b). Breitling (2002) believed that camel milk had an anti-diabetes activity possibly because of insulin-like activity. Oral insulin therapy has been known for many years, but the important drawback of oral insulin therapy is that its coagulum formation in acidic environment of stomach, neutralises its potency. Even if some of it is destroyed in the passage, the lack of coagulum formation of camel milk may act as an effective vehicle to take the milk insulin unchanged to the intestine and can be absorbed. Beg *et al* (1986) indicated that amino acid sequence of some of the camel milk protein is rich in half cysteine, which has some similarities with the insulin family of peptides.

Rosiglitazone Maleate, that is an insulin sensitiser belongs to Thiazolidinediones drug (TZD). It is a single agonist of peroxisome proliferator-activated receptor-g (PPAR-γ), by activating PPAR-γ receptor. Rosiglitazone Maleate and PUFA of camel milk in a similar mechanism have a synergistic effect. It may explain the reason that Rosiglitazone Maleate+camel milk group has better treatment of type 2 diabetes animal models than Rosiglitazone Maleate alone group.

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References

Agrawal RP, Beniwal R, Sharma S, Kochar DK, Tuteja FC, Ghorui SK and Sahani SM (2005). Effect of raw camel milk in type 2 diabetes patients: 1 year randomised study. Journal of Camel Practice and Research 12:27-31.

Agrawal RP, Swami SC, Beniwal R, Kochar DK, Sahani MS, Tuteja FC and Ghorui SK (2003a). Effect of camel milk on glycaemia control, lipid profile and diabetes quality of life in type-1 diabetes: a randomised prospective controlled cross over study. Indian Journal of Animal Sciences 73:1105-1110.

Agrawal RP, Swami SC, Beniwal R, Kochar DK, Sahani MS, Tuteja FC and Ghorui SK (2003b). Effect of camel milk on glycaemia control risk factors and diabetes quality of life in type-1 diabetes: a randomised prospective controlled study. Journal of Camel Practice and Research 10:45-50.

Beg OU, Von Bahr-Lind Strom H, Zaidi ZH and Jornvall H (1989). Characteristic of camel milk protein identifies a new beta casein fragment. Regulatory Peptides 15:5-61.

Breitling L (2002). Insulin and anti diabetes activity of camel milk. Journal of Camel Practice and Research 9:43-45.

Centres for Disease Control and Prevention, National Center for Health Statistics (2005). Division of Health Interview Statistics, data from National Health Interview Survey. U.S. Bureau of the Census, ceusu of the population and population estimates (www. cdc.gov/diabetes/statistics/).

Farah Z, Streiff T and Bachmann MR (1998). Manufacture and characterisation of camel milk butter. Milchwissenschaft 44(7):412-414.

Forman BM, Chen J and Evans RM (1997). Hypolipidemic drugs, polyunsaturated fatty acids, and eicosanoids are ligands for peroxisome proliferator-activated receptors alpha and delta. Journal Immanuel 170:6033.

Gorban AM and Lzzeldin OM (2001). Fatty acids and lipids of camel milk and colostrums. International Journal of Food Science and Nutrition 52(3):283-287.

Magdy El-Sally (2002). Gastrointestinal Transit in an Animal Model of Human Diabetes Type 2: Relationship to gut neuroendocrine peptide contents. Upsala Journal of Medical Science 107:101–110.

Mann J (2002). Diet and risk of coronary heart disease and type 2 diabetes. Lancet 360:783-789.

Narmuratova M, Konuspayeva G, Loiseau, Serikbaeva A, Barouh Nathalie, Montet Didier and Faye B (2006). Fatty acids composition of dromedary and bactrian camel milk in Kazakhstan. Journal of Camel Practice and Research 13:45-50.

Shehadeh N Gelertner L, Blazer S, Perlman R Solovachik L and Etzioni A (2001). Importance of insulin content in infant diet: suggestion for a new infant formula. Acta Paediaty 90:93-95.

Stahl T, Sallmann HP, Duehlmeier R and Wernery U (2006). Selected vitamins and fatty acid patterns in dromedary milk and colostrums. Journal of Camel Practice and Research 13:53-57.

Tierney L M, McPhee S J and Papadakis M A (2002). Current Medical Diagnosis & Treatment. International edition. New York: Lange Medical Books, McGraw-Hill, 1203-1215. ISBN 0-07-137688-7.

Timmons JS, Weiss WP, Palmquist DL and Harper WJ (2001). Relationships among dietary roasted soyabeans, milk components, and spontaneous oxidised flavour of milk. Journal of Dairy Science 84:2440-2449.

Wahle KW and Heyes SD (2002). Cell signal mechanisms conjugated linoleum acids (CLAs) and anti-tumour genesis. Prostaglandins Leukot Essent Fatty Acids 67:183-186.

Wernery U, Nagy P, Bhai I, Schiele B and Johnson B (2006). The effect of heat treatment, pasteurisation and different temperatures on insulin concentration in camel milk. Milchwissenschaft 61:25-28.

Wild S, Roglic G, Green A, Sicree R and King H (2004). Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care 27:1047–53.

Yi Shu, Li-Yong Zhong and Shi Li (2006). Comparative analysis of effects of melatonin, vitamin E and glutathione on oxidative stress in alloxan-induced diabetes rats. Journal of Southeast University (Med Sci Edi) 25:88-92.

SWEETS FROM CAMEL MILK IN INDIA

National Research Centre on Camels located at Bikaner, India has made "Gulab Jamun" from camel milk. It is a popular sweet which is usually made up of cattle or buffalo milk and is available in market as choicest sweet through out India. NRCC is laying its efforts to popularise the camel milk and its products. This sweet was launched by Chairman and members of Research Advisory Committee of NRCC on 15 April 2009. Director, NRCC claimed this sweet as first of its kind made out of camel milk and said to train the sweet makers in local market to prepare it from camel milk.

(Source:Rajasthan Patrika 16 April 2009)

INJAZ, THE ONLY CLONED CAMEL, IS ANOTHER WORLD FIRST

The world's first cloned camel a female calf named Injaz was born at Dubai's Camel Reproduction Centre (CRC). Injaz, the only cloned camel, is another world first and the method used to clone camels was developed at the Central Veterinary Research Laboratory in Dubai between 2003 and 2007 by Dr Nisar Wani, formerly an Assistant Professor of Animal Reproduction at the Sheri-Kashmir University of Agricultural Sciences and Technology of Kashmir, in Srinagar, India. Once perfected, the technique was put into practice at the Emirate's Camel Reproduction Centre. The cells from which Injaz was cloned were taken from the tissue of a camel selected at random and slaughtered for meat in 2005. These were put in an incubator, operating at a temperature of 38 degrees Celsius, where the cells continued to multiply. Once they had enough material, the cells were then frozen in liquid nitrogen until needed. Some of these cells were later "brought back to life" so their DNA-bearing nuclei could be extracted and inserted into egg cells taken from Injaz's surrogate mother, from which the nuclei had been removed. Once the new nucleus was in a host egg, electric pulses were used to fuse the two together. The next challenge was to "jump-start" the division process by which the cells multiply and grow into an embryo. The main difficulty to be overcome at this stage is that while the egg cell is programmed to multiply rapidly, the new nucleus has been taken from an adult somatic cell, which has different properties. A cocktail of chemicals was used to assist the natural "reprogramming" of the imported nucleus by the host.Once division is taking place successfully, the next challenge is to insert the cells into the surrogate mother. In smaller animals, these can be inserted into the uterus via surgery. In camels, however, this procedure is complicated and risky. Over a period of about a year, Dr Wani developed a method to culture the embryo, allowing it to grow in an incubator that simulated the environment of a camel's uterus. Once an embryo was deemed suitable, it was inserted into the surrogate mother's uterus through the same process used in human *in-vitro* fertilisation. Even this stage of the process is difficult; Injaz was the only calf born from seven induced pregnancies. In future, the programme will examine the possibilities of using cloning to perpetuate the genes of valued racing and milk-producing camels. The team of JCPR congratulates Dr.Wani and his team for this achievement.