PREVALENCE OF DIABETES IN CAMEL-MILK CONSUMING 'RAICA' RURAL COMMUNITY OF NORTH-WEST RAJASTHAN

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ABSTRACT

Anecdotal reports suggest that camel-milk consumption is associated with low prevalence of diabetes. To determine prevalence of diabetes and impaired glucose tolerance in habitually camel-milk consuming *Raica* community in North-Western Rajasthan, we conducted a cross-sectional survey using stratified sampling of a representative *Raica* community subjects consuming camel milk, and *Raica* community and non-*Raica* community subjects not consuming camel milk. We used 75 gm oral glucose-load tolerance test to determine the glucose intolerance.

The fasting as well as post-glucose load glucose levels were significantly lower in *Raica* community subjects as compared to the non-*Raica* community subjects in the same region (fasting 89.0 ± 15.0 vs. 96.2 ± 20.3 mg/dl; post-glucose 120.2 ± 17.5 vs. 131.2 ± 30.2 mg/dl; p<0.001). In camel-milk consuming *Raica* subjects the age-adjusted prevalence of diabetes (0.0%), impaired fasting glucose (3.2%), and impaired fasting glucose (8.6%) was significantly lower than other-milk consuming *Raica* subjects (4.6%, 7.8%, and 20.6%) and non-*Raica* subjects (7.5%, 13.4% and 15.1%) respectively (p<0.01).

The prevalence of impaired glucose tolerance as well as diabetes is low in the rural *Raica* community subjects of north-west Rajasthan. The prevalence of both is the lowest in camel-milk consuming *Raica* community subjects.

KEY WORDS: Raica community; Prevalence of diabetes; Camel milk.

INTRODUCTION

Substantial variations exist in prevalence of diabetes worldwide (1-3). Studies within India have reported that the prevalence of diabetes varies from

less than 2% in rural Kashmir to more than 20% in urban areas of Hyderabad (4-9). International studies have also reported a wide variation in diabetes prevalence. The lowest prevalence, practically zero, are in rural Third World areas whereas the highest 37-50% are among the Nauru Islanders of tropical Pacific, Pima Indians in Arizona and urban Wanigela people in Papua New Guinea (3). Most of the world's broad geographic groupings of people include populations of both very low and very high prevalence. For instance, Mapuche Indians versus Arizona Pima Indians in USA, rural New Guineans versus the urban Wanigela, and rural Kashmiris versus the urban Tamilians in India. Genetic differences as well as dietary factors have been implicated to explain these regional differences.

Camel milk is part of the staple diet in parts of Africa and Asia and is considered as health promoting (10). Anecdotal reports suggest a very low prevalence of diabetes among subjects that are consuming camel milk. We previously reported improved glycemic control in type-1 diabetes subjects whose diet was supplemented with camel milk (11, 12). In the Thar Desert region of Rajasthan, camel is not only used for transportation and support but its milk is consumed by certain communities. Raica is a tribal community of North-West Rajasthan who not only habitually cares for the camel but also consumes its milk. Prevalence of diabetes in subjects that habitually consume camel milk has not been reported previously. Therefore, to determine the prevalence of diabetes as well as impaired fasting glucose we performed an epidemiological study in the camel-milk consuming Raica community of Rajasthan.

MATERIAL AND METHODS

This is a cross sectional study with participants from different places of Bikaner region in North West Rajasthan. The number of *Raica* subjects required

*Department of Medicine, SP Medical College and Associated Group of Hospitals, Bikaner 334001; **Desert Medical Research Center, Jodhpur 303001; ***Home Science Department, University of Rajasthan, Jaipur 302004 India. from every basic geographical area was calculated based on the percentage of the population living in that region. The population of the survey was similar in distribution with regards to age, sex and geographic conditions. We included most of the Raica subjects aged 20 years and more residing in North West Rajasthan. Screening camps were organized in the locality of Raica and the remaining Raica (who were not able to attend the screening camp) were evaluated by a household survey. In the same manner individuals from non-Raica Community residing in same environment were selected and screened, and served as control subjects. Written consent was taken from all participants and project was approved by the institutional ethical committee. The base line home interview with clinical and blood examinations were performed between September 2002 and December 2002.

The demographic data and medical history were recorded using a standardized questionnaire. It included working conditions, eating habits, social and cultural habits, camel milk consumption, family history of diabetes, and other chronic disorders. Diabetes was explored by history, age at diagnosis, symptoms, time since the participant was diagnosed and treatment related issues. The physical examination emphasized measurement of height, weight, waist-hip ratio (WHR) and blood pressure. Height was measured in centimetres and weight in kilograms using calibrated spring-balance. Waist girth was measured at the level of umbilicus with person breathing silently and hip girth was measured at intertrochanteric level according to the World Health Organisation (WHO) guidelines (13). Blood pressure was measured using standard mercury manometer. At least two readings at 5 minutes intervals as per WHO guidelines were recorded (14). If a high blood pressure reading of \geq 140/90 was noted a third reading was taken after 30 minutes. The average of lower two of the three readings was taken as blood pressure. Body mass index (BMI) was calculated by dividing weight in Kg by squared height in metres and expressed as Kg/m².

The participants were advised to come after at least 8 hour overnight fast and abstinence from smoking was observed during the oral glucose tolerance test. Fasting samples of venous blood were drawn. The participants who were previously diagnosed diabetics and were on oral hypoglycemic agents or insulin treatment were excluded from the 2-hour oral glucose tolerance test. All other participants were given 75g anhydrous glucose dissolved in 200ml water to be drunk within 5 minutes and advised to rest during the entire process. Another venous blood sample for 2-hour post glucose load was drawn. The drawn samples were carefully labeled and within 30 minutes they were taken to the nearby laboratory where the plasma glucose levels were estimated by glucose oxidase method. The WHO criteria were followed for diagnosis of diabetes (15).

Statistical Analysis: All the data were computerized. Numerical variables are reported as mean ±1SD and ordinal variables as percent. Prevalence rates are reported in percent. Inter-group comparisons for numerical variables were performed using t-test for two-group comparisons and analysis of variance (ANOVA) for multiple groups. For ordinal variables chi-square test or F-test were used as appropriate. P value <0.05 was considered significant.

RESULTS

Of the target sample size of 1000, we could examine 782 subjects (response rate 78.2%). 605 subjects (77.4%) belonged to the Raica community and 177 subjects (22.6%) were of non-Raica community. The demographic characteristics of the two groups are shown in Table 1. There was no significant difference in gender distribution, smoking status, and dietary factors. Subjects belonging to the Raica community were significantly younger and the BMI was lower as compared to the non-Raica community subjects. The mean blood glucose in the two communities is shown in Table 2. In the Raica community subjects as compared to non-Raica subjects fasting blood glucose (89.0+15.0 vs. 96.2+20.3 mg/dl, p<0.0001) as well as 2-hour postload blood glucose (120.2+17.5 vs. 131.2+30.2 mg/ dl) was significantly lower (p<0.0001). Prevalence of impaired fasting glucose and diabetes was significantly lower in Raica community subjects (p<0.001) while that of impaired glucose tolerance was similar.

 Table 1: Demographic and Physical Characteristics

 of Raica and Non-Raica Community Subjects (n=882)

	<i>Raica</i> Community	Non- <i>Raica</i> Community	χ2 test, P value
	(n=605)	(n=177)	
Gender			
Male (n=418)	319 (52.7)	99 (55.9)	0.44, 0.505
Female (n= 363)	286 (47.3)	78 (44.1)	
Age Groups		. ,	
20-29	285 (47.1)	43 (24.3)	48.66,
30-39	144 (23.8)	34 (19.2)	< 0.0001
40-49	60 (9.9)	35 (19.8)	
50-59	64 (10.6)	31 (17.5)	
60+	52 (8.6)	34 (19.2)	
Occupation			
Household	137 (22.6)	50 (28.2)	34.42,
Manual labour	233 (38.5)	54 (30.5)	< 0.0001
Unskilled labour	97 (16.0)	46 (26.0)	
Others	56 (9.3)	25 (14.1)	
Student	82 (13.6)	2 (1.1)	
Socioeconomic status			
Poor	560 (92.9)	155 (87.6)	4.36, 0.037
Lower middle class	43 (7.1)	22 (12.4)	
Smokers			
Only smokers	127 (21.0)	38 (21.6)	1.32, 0.250
Smokers/oral	9 (1.5)	6 (3.4)	
Family history diabetes	0	5 (2.8)	-
Dietary factors			
Vegetarian	402 (66.4)	135 (76.3)	5.69, 0.017
Non-vegetarian	203 (33.6)	42 (23.7)	
Camel milk consumption	343 (56.7)	15 (8.5)	<0.0001
Body-mass index (kg/m2)			
>15	22 (3.6)	2 (1.1)	31.27, <0.0001
15-19.9	319 (52.7)	75 (42.4)	
20-24.9	226 (37.4)	66 (37.3)	
25-29.9	30 (5.0)	29 (16.4)	
30+	8 (1.3)	5 (2.8)	

Table 2: Blood Glucose Levels, Impaired Fasting Glucose, Impaired Glucose Tolerance and Diabetes Prevalence

	Raica	Non-Raica	t-test/c2 test,
	(n=605)	(n=177)	P value
Fasting blood glucose (mg/dl, mean <u>+</u> SD)	89.0 <u>+</u> 15.0	96.2 <u>+</u> 20.3	5.15, <0.0001
Fasting glucose (mg/dl)			
<75	42 (6.9)	6 (3.4)	
75-89	321 (53.1)	73 (41.2)	
90-109	199 (32.9)	64 (36.2)	28.09, <0.0001
110-125	31 (5.1)	22 (12.4)	
<u>></u> 126	12 (2.0)	12 (6.8)	
2 hour PG blood glucose	120.2 <u>+</u> 17.5	131.2 <u>+</u> 30.2	6.12, <0.0001
(mg/dl, mean <u>+</u> SD)			
2 hr PG glucose (mg/dl)			
<90	4 (0.7)	0 (0.0)	
90-109	192 (31.7)	33 (18.6)	
110-125	178 (29.4)	54 (30.5)	
126-139	149 (24.6)	48 (27.1)	43.70, <0.0001
140-159	70 (11.6)	21 (11.9)	
160-199	8 (1.3)	9 (5.1)	
<u>></u> 200	4 (0.7)	12 (6.8)	
Impaired fasting glucose	31 (5.1)	22 (12.4)	12.29, <0.0001
Impaired glucose tolerance	78 (12.9)	45 (25.4)	1.57, 0.211
Diabetes (known or fasting			
glucose <u>></u> 126 and/or	4 (0.7)	12 (6.8)	22.10, <0.0001
post-glucose ≥200 mg/dl			

Table 3: Age-specific fasting blood glucose levels (mg/dl, Mean<u>+</u>SD)

Age Group	Ra (n=	<i>iica</i> 605)	Non- (n=						
	Camel-milk (Group I, n=343)	Camel-milk No camel-milk (Group I, (Group II, n=343) n=262)		No camel-milk (Group IV, n=162)	ANOVA F value, p				
20-29	89.2 <u>+</u> 9.4	92.8±10.6**	103.0 <u>+</u> 0.0	87.1 <u>+</u> 7.9*	22.80, <0.0001				
30-39	88.1 <u>+</u> 9.7	89.9 <u>+</u> 18.5	85.2 <u>+</u> 12.6	95.1 <u>+</u> 8.8**	11.03, <0.0001				
40-49	88.7 <u>+</u> 10.4	93.8±17.6**	108.0 <u>+</u> 0.0	98.4 <u>+</u> 15.5**	24.15, <0.0001				
50-59	85.9 <u>+</u> 8.1	83.8 <u>+</u> 9.5**	108.0 <u>+</u> 0.0	104.3 <u>+</u> 48.0**	33.32, <0.0001				
60 <u>+</u>	83.3 <u>+</u> 12.0	99.9 <u>+</u> 19.2**	75.5 <u>+</u> 8.7	114.5 <u>+</u> 32.6**	98.40, <0.0001				
Comparison of Group Land II. Comparison of Group Land IV. Uppaired t-test *p<0.01									

Comparison of Group I and II. Comparison of Group I and IV. Unpaired t-test *p<0.01, **p<0.00

Table 4: Camel Milk Consumption, BloodGlucose, Impaired Fasting Glucose, ImpairedGlucose Tolerance and Diabetes Prevalence

	Con	<i>Raica</i> nmunity	Non- <i>I</i> Comm	ANOVA F/ χ2 test, p value		
	Camel milk (Group I, n=343)	Other milk forms (Group II, n=262)	Camel milk (Group III, n=15)	Other milk form (Group IV, n=162)	ns	
Age (yr, mean <u>+</u> SD)	39.1 <u>+</u> 15.2	46.0 <u>+</u> 17.3**	34.3 <u>+</u> 15.0	45.4 <u>+</u> 17.7**	11.73, <0.0001	
BMI (kg/m2, mean <u>+</u> SD)	19.1 <u>+</u> 3.2	20.5 <u>+</u> 4.0	19.3 <u>+</u> 3.0	20.43.7**	9.4, <0.0001	
Blood glucose (fasting, mean <u>+</u> SD)	87.6 <u>+</u> 10.2	91.6 <u>+</u> 15.7**	92.5 <u>+</u> 14.3	99.0 <u>+</u> 26.3**	17.36, <0.0001	
Blood glucose (2 hr PG, mean <u>+</u> SD	115.3 <u>+</u> 15.7)	114.6 <u>+</u> 11.3	126.2 <u>+</u> 20.9	136.7 <u>+</u> 39.0*	25.30, <0.0001	
Impaired fasting glucose (no., %)	11 (3.2)	24 (9.2)**	1 (6.7)	24 (14.8)**	18.32, <0.001	
Impaired glucose tolerance (no., %)	30 (8.6)	48 (18.3)**	0 (0.0)	30 (18.5)**	13.38, 0.005	
Diabetes (no., %)	0	8 (3.0)**	0	15 (9.3)**	30.66, <0.001	

Comparison of Groups I and II. Comparison of Groups I and IV. Student t-test *p<0.01, **p<0.001

Figure 1: Frequency Distribution of Fasting Blood Glucose in Camel-Milk Consuming *Raica* Community (Group I), Other Milk-Forms Consuming *Raica* Community (Group II) and Non-*Raica* Community Subjects Consuming Other Milk Forms (Group IV). The Distribution shows a Significant Positive Skew with a Long-Right Tail in Groups II and IV Suggesting Higher Fasting Glucose in these Groups.



Age Group	<i>Raica</i> Camel Milk (n=343)				Raica Other Milk (n=262)		Non- <i>Raica</i> Camel Milk (n=15)			Non- <i>Raica</i> Other Milk (n=162)						
	No.	IFG	IGT	DM	No.	IFG	IGT	DM	No.	IFG	IGT	DM	No.	IFG	IGT	DM
20-29	112	2 (1.8)	7 (6.3)	-	131	10 (7.6)	18 (13.7)	-	2	-	-	-	37	-	2 (5.4)	-
30-39	60	2 (3.3)	`8´ (13.3)	-	54	6 (11.1)	4 (7.4)	2 (3.7)	5	-	-	-	25	2 (8.0)	2 (8.0)	2 (8.0)
40-49	76	5 (6.6)	9 (11.8)	-	29	-	4 (13.8)	4 (13.8)	2	-	-	-	26	8 (30.8)	5 (19.2)	`3´ (11.5)
50-59	39	-	6 (15.4)	-	10	-	2 (20.0)	-	2	-	-	-	25	4 (16.0)	6 (24.0)	2 (8.0)
60+	56	2 (3.6)	-	-	38	8 (21.0)	18 (47.4)	2 (5.3)	4	1 (25.0)	-	-	49	10 (20.4)	15 (30.6)	`8´ (16.3)
Total	343	11 (3.2)	30 (8.6)	0 (0.0)	262	24 (9.2)	48 (18.3)	8 (3.0)	15	1 (6.7)	0	0 (0.0)	162	24 (14.8)	30 (18.5)	15 (9.3)
Age-adju prevalen	sted ce	Re	ference Gr	oup		7.8%*	20.6%**	4.6%**		4.1%	-	-		13.4%**	15.1%*	7.5%**

 Table 5: Age-Specific Prevalence of Impaired Fasting Glucose, Impaired Glucose Tolerance and

 Diabetes in Various Groups

IFG impaired fasting glucose, IGT impaired glucose tolerance, DM diabetes mellitus. Comparison of Groups I and II. Comparison of Groups I and IV. Student t-test *p<0.01, **p<0.001

To determine the influence of habitual camelmilk consumption on blood glucose levels, we classified subjects in Raica as well as non-Raica communities in groups consuming camel milk and other milk forms. Among Raica community there were 343 subjects who habitually consumed camel milk (Group I) and 262 subjects who consumed other milk forms (Group II) while in non-Raica community there were 15 subjects who consumed camel milk (Group III) and 162 subjects who consumed other milk forms (Group IV). Agespecific fasting blood glucose levels in different groups are shown in Table 3. Fasting blood glucose levels were significantly lower in all age-groups in Group I as compared to Groups II and Group IV (p<0.01). Group III subjects also had higher fasting blood glucose although the number of subjects in this group was small. Mean fasting blood glucose as well as 2-hour post-glucose load blood glucose levels were significantly lower in Group I as compared to Groups II, III and IV (p<0.01, Table 4). Distribution of the mean fasting glucose levels in Group I, Group II and Group IV are shown in Figure 1. There is a clear rightward shift in levels of fasting glucose in Groups II and IV as compared to Group I suggesting higher fasting blood glucose levels in these two groups.

In camel-milk consuming Group I *Raica* community subjects the prevalence of impaired fasting glucose (3.2%), impaired glucose tolerance (8.6%), as well as diabetes (no case) was significantly lower than other groups (Table 4). Age-adjusted prevalence of

impaired fasting glucose, impaired glucose tolerance and diabetes was significantly greater in non-camel milk consuming *Raica* community Group II subjects (7.8%, 20.6%, 4.6%) as well as non-*Raica* community Group IV subjects (13.4%, 15.1%, 7.5%) as compared to camel-milk consuming *Raica* community Group I subjects (p<0.01) (Table 5).

DISCUSSION

This study shows that there is a very low prevalence of diabetes in the rural *Raica* community subjects of north-west Rajasthan. The age-adjusted prevalence in camel-milk consuming *Raica* subjects shows impaired fasting glucose in 3.2%, impaired glucose tolerance in 8.6% and diabetes is zero and is lower than *Raica* subjects consuming other milk forms as well as non-*Raica* community subjects.

A low prevalence of impaired glucose tolerance has been reported in various Caucasian groups (3). In a study among European subjects in inner London, the age-standardized prevalence of impaired glucose tolerance was 3% in a population aged 40-69 (16). In the Islington Diabetes Survey the prevalence of impaired glucose tolerance in subjects aged \geq 40 years was 4.1% (17). However, in the British Hertfordshire study the prevalence of impaired glucose tolerance in a population of men not previously known to have diabetes aged 59-70 it was 18% (18). A greater prevalence of impaired glucose tolerance and diabetes has been noted among Indian emigrants to Britain (8). In South India serial studies have reported impaired glucose tolerance in 7.8% adults in 1989 which increased to 9.1% in 1995 and 16.8% in 2000 (7). There was no significant difference in urban and rural subjects in prevalence of impaired glucose tolerance. In the present study the prevalence of impaired glucose tolerance is 8.6% in camel milk consuming *Raica* subjects, 20.6% in other *Raica* subjects and 15.1% in non-*Raica* subjects. This shows a low prevalence of impaired glucose tolerance in *Raica* subjects consuming camel-milk while in other groups the prevalence is similar to the South Indian studies.

Early reports from the Indian subcontinent suggested that diabetes was not a common disorder. Gupta et al reported an overall prevalence rate of 2.1% in India in 1979 (4). In the population aged over 15 years of age Ahuja found the prevalence of diabetes (both known and new cases) to be 2.1% in urban areas and 1.8% in rural areas (5). On the other hand, studies by Verma et al (19) and Ramchandran et al (20) have reported higher prevalence rates in urban subjects (known diabetes 3% in Delhi and 5% in Chennai). In rural populations, diabetes prevalence rates in adults vary from 1.6% (Andhra Pradesh) to 2.2% (Bangalore), 2.4% (Tamilnadu) and 2.5% (Kerala) (7). Among urban subjects a very high prevalence of diabetes has been reported in various Indian studies and a recent review reported diabetes prevalence of 11.6% in urban locations, 5.9% in semiurban locations and 2.4% in rural locations (7). Prevalence of diabetes in the present study varies from 4.6% among Raica subjects consuming other milk forms to 7.5% in non-Raica subjects while it is not observed among camel-milk consuming Raica subjects. Thus the prevalence rates in non-camel milk consuming rural subjects are similar to other studies from India.

Minor study limitations include sampling bias, lack of dietary assessment of study subjects and nonmeasurement of glycated hemoglobin levels. This study cohort examined was not selected on the basis of a random sample of an enumerated population but was constructed by community-wide screening in households and public places. However, because the number of subjects screened represented a high proportion of the estimated adult population of *Raica* and it is unlikely that the method of screening disproportionately excluded any segment of the population.

Factors associated with the development of type-2 diabetes in susceptible populations include obesity, reduced physical activity and alteration of dietary habits (3). Diet and physical activity are two major factors which influence glucose metabolism. Recent studies also highlight abnormal lipid metabolism in diabetes and a remarkable excess accumulation of intracellular triglyceride in both muscle and liver has been demonstrated (21). This lipid accumulation contributes to insulin resistance and has its origin in a common polymorphism of peroxisome-proliferatoractivated receptor gamma coactivator 1 (PGC-1) gene (22). Other genetic mechanisms have also been implicated. Type 2 diabetes is now considered a disease of glucose metabolism as well as of lipid metabolism.

Raica community is a nomadic tribal population scattered over the area of Thar Desert in India and Pakistan. These subjects are possibly descendents of ancient migrants to India from Central Asia. Raica community subjects lead an active life and an important dietary constituent is the camel milk. Both these factors might influence blood glucose levels. Although dietary factors that we have not evaluated may be responsible, the possibility appears remote as the control group is from the same area and there should not be major dietary differences in the two groups. Previous studies from north India have reported on homogeneity of diet in rural areas among different communities (23). The most possible explanation for the low prevalence of glucose intolerance and diabetes in Raica Community may therefore be that camel milk has some protective influence.

Camel milk has a high pH and when left to stand the acidity increases rapidly. The water content varies from 80-90%. In Indian camel, the fat content varies from 2.9-4.1%, proteins 2.0-4.0%, lactose 4.7-5.4% and non-fat solids from 9.6-10.1% (10). The principal fatty acids are oleic acid (38.9%), palmitic acid (29.3%), and stearic acid (11.1%) while others are myristic acid (7.3%), lauric acid (4.6%), linoleic acid (3.8%) and butyric acid (2.1%) (24). Camel milk is very similar to goat milk and compares favourably with human milk. Camel herders living on camel milk only in Kenya and Sahara region of Africa are healthy and vigorous. No study of influence of camel milk on diabetes prevalence has been previously reported. Agrawal et al reported that camel milk supplementation to standard therapy improves glycemic control in type-1 diabetes in randomized studies (11, 12). Beg et al reported that camel milk whey protein is rich in half-cystine and there are few superficial similarities with the insulin family of peptides (25, 26). It has been speculated that improved glycemic control in diabetics consuming camel milk may be due to faster gastric emptying induced by high pH of the camel milk in association with availability of insulin-like substances in proximal intestine (12). This speculation needs confirmation by further studies. Genetic influences and geneenvironment interactions to explain low prevalence of diabetes in *Raica* subjects also need further studies.

REFERENCES

- 1. King H, Rewers M, Global estimates for prevalence of glucose intolerance. Diabetes Care 1993; 16:1-21.
- 2. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025. Diabetes Care 1998; 21:1414-31
- Diamond J. The double puzzle of diabetes. Nature 2003; 423:599-602
- Gupta OP, Joshi MH, Dave SK. Prevalence of diabetes in India *In*: Miller M, Bennett PH. *Eds*. Advances in Metabolic Disorders. Vol 9. New York, Academic Press 1978; pp147-165.
- 5. Ahuja MMS. *Ed.* Epidemiology of diabetes in developing countries. New Delhi, Interprint. 1979; 29-38.
- Ramachandran A, Snehalatha C, Kapur A, Vijay V, Mohan V, Das AK, et al. High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. Diabetologia 2001; 44:1094-101
- Ramachandran A, Snehalatha C, Viswanathan V. Burden of type 2 diabetes and its complications: the Indian scenario. Curr Science 2002; 83:1471-6
- Misra A, Vikram NK. Insulin resistance syndrome (metabolic syndrome) and Asian Indians. Curr Science 2002; 83:1483-96
- Gupta A, Gupta R, Sarna M, Rastogi S, Gupta VP, Kothari K. Prevalence of diabetes, impaired fasting glucose and insulin resistance syndrome in an urban Indian population. Diab Res Clin Pract 2003; 61:69-76
- Anonymous. Composition of camel milk. At: www.fao.org. Accessed 24 Dec 2003.
- Agrawal RP, Swami SC, Beniwal R, Kochar DK, Kothari RP. Effect of camel milk on glycemic control, risk factors and diabetes quality of life in type-1 diabetes: A randomized prospective controlled study. Int J Diab Developing Countries 2002; 22:70-4.
- Agrawal RP, Swami SC, Beniwal R, Kochar DK, Sahani MS, Tuteja FC, et al. Effect of camel milk on glycemic control, lipid profile and diabetes quality of life in type 1 diabetes: a randomized prospective controlled cross-over study. Indian J Animal Sci 2003; 73:1105-10.
- 13. WHO Expert Committee. Physical status: the use and interpretation of anthropometry. WHO Tech Rep Series

1995; 854:424-38.

- Rose GA, Blackburn H, Gillum RF, Prineas RJ. Cardiovascular Survey Methods. 2nd Ed. WHO Monograph Series No. 56. Geneva. World Health Organisation. 1982.
- WHO Consultation, Alberti KGMM, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. 1. Diagnosis and classification of diabetes mellitus. Diabetic Med 1998; 15:539-53.
- McKeigue PM, Pierpoint T, Ferrie JE, Marmot MG. Relationship of glucose intolerance and hyperinsulinaemia to body fat pattern in South Asians and Europeans. Diabetologia 1992; 35:783-90.
- 17. Forrest RD, Jackson CA, Yudkon IS. Glucose intolerance and hypertension in North London: The Islington Diabetes Survey. Diab Med 1986; 3:338-42.
- Hales CN, Barker DJP, Clark PMS, et. Fetal and infant growth and impaired glucose tolerance at age 64, BMJ 1991; 303:1019-22.
- 19. Verma NPS, Mehta SP, Madhu SV, Mather HM, Keen H. Prevalence of known diabetes in an urban Indian environment: the Daryaganj Diabetes Survey. BMJ 1986; 293:423-4.
- Ramchandran A, Jali MV, Mohan V, Snehalatha C, Viswanathan M. High prevalence of diabetes in an urban population in south India. BMJ 1988; 297:587-90.
- 21. Taylor R. Causation of type-2 diabetes: the Gordian knot unravels. N Engl J Med 2004; 350:639-41
- Petersen KF, Dufour S, Befroy D, Garcia R, Shulman GI. Impaired mitochondrial activity in the insulin-resistant offspring of patients with type-2 diabetes. N Engl J Med 2004; 350:664-71
- 23. Chadha SL, Gopinath N, Katyal I, Shekhawat S. Dietary profile of adults in an urban and rural community. Ind J Med Res 1995; 101:258-67.
- 24. Gorban AM, Izzeldin OM. Fatty acids and lipids of camel milk and colostrums. Int J Food Sci Nutr 2001; 52:283-7
- 25. Beg OU, Von Bahr-Lindstrom H, Zaidi ZH, Jornvall H. A camel milk whey protein rich in half cystine. Primary structure, assessment of variations, internal repeat patterns, and relationships with neurophysin and other active polypeptides. Eur J Biochem 1986; 159:195-201.
- 26. Girardet JM, Saulnier F, Gaillard JL, Ramet JP, Humbert G. Camel milk PP3: evidence ofr an insertion in the aminoterminal sequence of the camel milk whey protein. Biochem Cell Biol 2000; 78:19-26.