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# Evaluation of Camel Milk and Urine in the Management of Diabetes Mellitus in Alloxan Induced Albino Rats

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*Keywords:* *diabetes mellitus, camel milk, urine, alloxan, rats.*

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# Evaluation of Camel Milk and Urine in the Management of Diabetes Mellitus in Alloxan Induced Albino Rats

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*Abstract-* There are many anecdotal reports on traditional use of camel milk and urine in the treatment of Diabetes Mellitus (DM). The need therefore arises to validate this claim. The objectives of the study is to; compare the effect of camel milk and urine on serum glucose of rats, compare the effect of camel milk and urine on serum lipids of rats and to Compare Different doses of the products on serum glucose and lipids. Thirty-six adult albino rats were used in 4 X 3 factorial experiment involving 4 product treatments and 3 doses. A significant decrease in the blood glucose level in the experimental groups fed camel milk when compared to diabetic untreated (control) group. In treatment group treated with camel urine singly and in combination there was a significant decrease in glucose compared to control. The result shows that there were significant decrease in TG, TC, LDL-C and VLDL-C compared with the control while the HDL was significantly increased. The results indicate that camel milk possess anti-diabetic effects on alloxan induced rats. This study recommended that awareness should be created on the therapeutic value of camel urine and its combination.

*Keywords: diabetes mellitus, camel milk, urine, alloxan, rats.*

## I. Introduction

The camel belongs to the family camelidae and divided into two genera: genus camelus (the true or old world camels) and genus lama (the new world camels). The genus camelus includes two species, the Dromedary, (Camelus dromedarius) or one-humped camel and the Bactrian camel (Camelus bactrianus) the two humped camel. The Dromedary (C. dromedarius) is adapted to hot arid environments and contributes significantly to the food security of the nomadic camel pastoral households (Schwartz & Dioli, 1992). Camelids are ruminating animals and are in proximity to ruminants but are not part of the suborder Ruminantia. Differences such as foot anatomy, stomach system and the absence of horns confirm this fact. They belong to the suborder Tylopoda (Werney, 2003).

According to FAO (2013) the total population of camel in the world is 25.89 million, of which 89% are dromedary (C. dromedarius). The remaining 11% are C. bactrianus, which are generally found in the cold deserts of Asia. While more than 60% of the dromedary camel population is concentrated in the arid areas of North East African countries like Somalia, Sudan, Ethiopia and Kenya. Ethiopia ranks third in the world by the number of camel head after Somalia and Sudan (Simeneh et al., 2015). Nigeria has a population of more than Ninety-two thousand, four hundred and ninety-four (92,494) of one humped Camel (Felsner 2002).

Camel is a good source of various vitamins and minerals and is characterized for its low cholesterol and high concentration of insulin-like factor (Agrawal, et al., 2005). Camel milk and urine are used therapeutically against hepatitis, dropsy, problems of spleen, and asthma, (Mal et al., 2000).

DM is the fourth leading cause of death in most developed countries, and its prevalence is rising in Nigeria (IDF, 2013). The conventional medications for DM such as Biguanides, Sulfon'ylureas and Thiazolidinedione are associated with undesirable side effects such as allergic reactions, nausea and vomiting, diarrhea, sexual dysfunction, haemoglobin disorders and lipodystrophy (Oliver and Tellervo 1993). For this reasons cheaper alternatives such as medicinal plants and animal products are sought. Outstanding among these alternatives to the conventional drugs are camel milk and urine, for which there are many anecdotal reports and few scientific studies. The need therefore arises to validate this claim.

The aim of the study is to investigate the anti-diabetics effects of camel milk and urine in alloxan induced diabetic rats. The objectives of the study are to; compare the effect of camel milk and urine on serum glucose of rats, compare the effect of camel milk and urine on serum lipids of rats and to compare different doses of the products on serum glucose and lipid profiles.

## II. Materials and Methods

The study was carried out at the Animal house, Department of Biochemistry, located in the Biological garden of Usmanu Danfodiyo University, Sokoto. Sokoto is located between Latitudes 12° and 13° N, and Longitudes 4° and 6° E in the Northern part of Nigeria and at an altitude of 350 m above the sea level (Mamman et al., 2000). The state falls within the Sudan savannah vegetation zone with alternating short and dry

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seasons. A hot dry spell extends from March to May and sometimes to June, in the extreme northern part of the state. A short, cool, dry period (harmattan) occurs between October and February (Mamman et al., 2000; SSMIYSC, 2007).

*a) Experimental Animals and Their Management*

Thirty Six adult albino rats of both sexes weighing between 150 -170 g, were obtained from National Veterinary Research Institute, (NVRI) Vom, and used for the study. The rats were housed in cages in a well-ventilated room with free access to feed (grower mash) and water. The rats were allowed to acclimatize under laboratory condition for a period of two weeks before the commencement of the experiment. Fresh Camel milk and urine were administered to the rats by oral intubation, in doses according to the experimental protocol.

*b) Induction of Diabetes Mellitus*

Diabetes mellitus was induced according to Szkudelski (2001), the rats were injected with a single dose of 120mg/kg bw of alloxan monohydrate, in dorsally recumbent position via penial vein. Food and water were given to the animals 30 minutes after the drug administration. A sample of the rat□s venous blood was collected 7 days after induction and DM was confirmed by measuring the serum glucose level with the aid of Accu Chek glucometer (mode: AE-350, BY ERMA INC). Rats that had serum glucose level >7.0 mmol/l were considered diabetic.

*c) Experimental layout*

The 36 diabetic albino rats were randomly allocated into four treatment groups of nine rats each. A 4 X 3 factorial design involving 4 product treatments (milk, urine and milk-urine combination) and 3 dose levels (0.5. 1.0 and 1.5 ml) were used.

*d) Blood collection*

Blood samples for monitoring of blood glucose level were taken from the tail. The tail of each of the rats was pricked with lancet and a drop of the blood was collected on the test strip and inserted into the glucometer to read glucose concentration on the screen in mg/dl. Readings were taken before, after the induction and at 28 days post treatment. The first blood collection (pre-induction) was for screening of the animals, while the second collection (post-induction) was for the confirmation of DM. The third collection was for the determination of the effect of treatments.

The serum lipids were measured 28 days after the commencement of the treatments where the animals were fasted overnight and sacrificed. The blood of the animals was collected in plain bottle, centrifuged and the serum separated and kept in labeled sample bottles at 4°C until required for lipid profile analysis.

*e) Data collection*

Serum glucose level was measured using glucometer, Total cholesterol (TC), Triglycerides (TG) and High Density Lipoprotein (HDL) were determined using Randox cholesterol kit (mode: CAT/TYP 05075548002, ROCHE INC). Low Density Lipoprotein (LDL) and Very Low Density Lipoprotein (VLDL) were calculated using Friedewald formula (Friedewald et al., 1972).

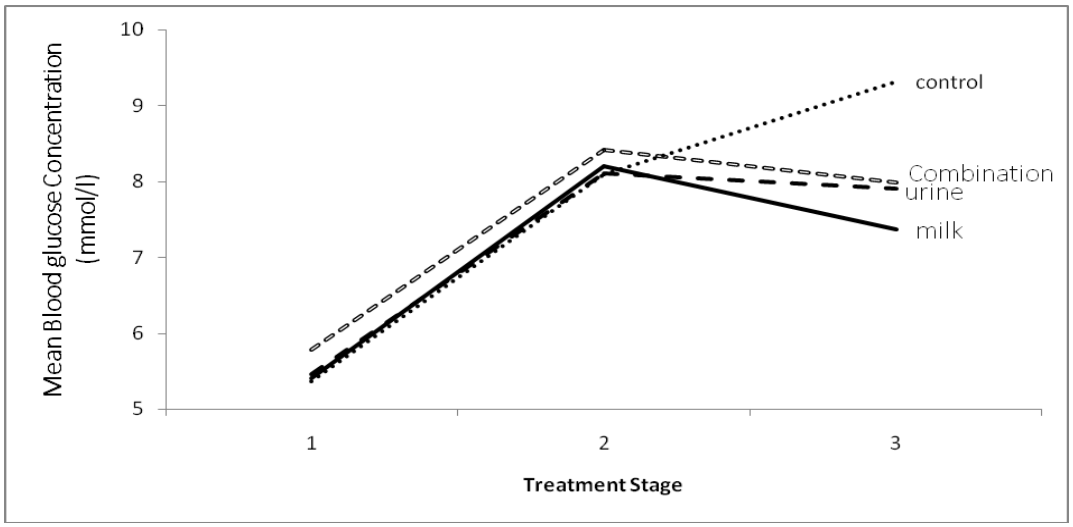
*f) Data analyses*

General Linear Model (GLM) univariate procedure was used to determine the effects of the product treatments and dose on serum glucose, TC, TG, HDL, LDL, and VLDL significant means were separated using tukey test.

## III. Results and Discussions

*a) Effect of Camel Milk and Urine on Serum Glucose*

Before induction all the animals were in non-diabetic state, however, after successful induction there was a sharp increase in blood glucose levels in all the rat groups. With the commencement of treatment there was a steady decline in serum glucose in groups except the control groups. The groups administered camel milk had the lowest concentration of serum glucose, while there was no decline in serum glucose in the control groups (Figure 1).

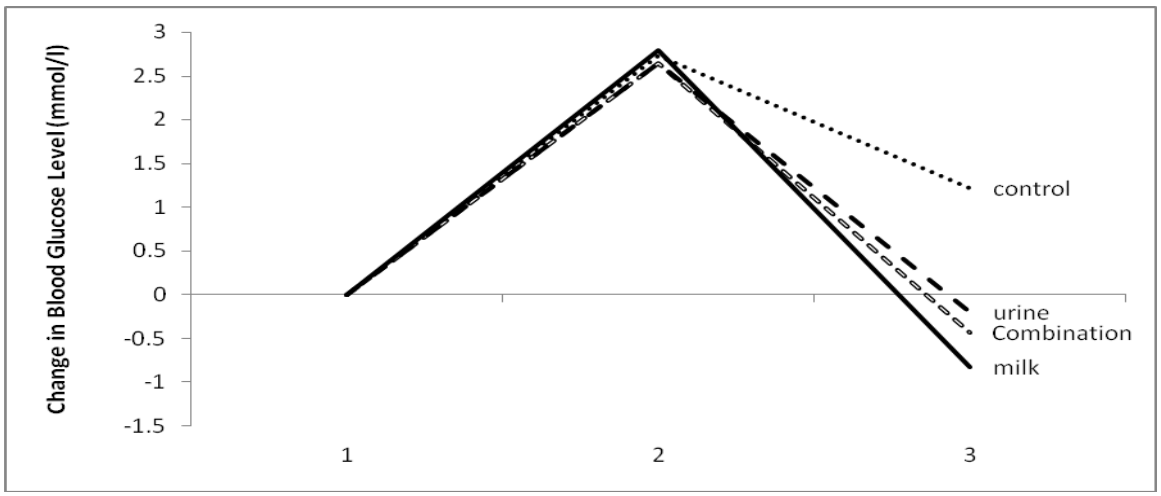



*1= Pre- induction, 2= Post- induction before treatment 3= 28 days Post-treatment.*

*Figure 1:* Change in blood glucose level among the various treatments groups

The trend indicated that the camel milk and urine had some hypoglycemic effects that might be due some hypoglycemic factors they contain.

The rate of decline in serum glucose of the control group (Figure 2) was greater in treated groups than in the control group. In all the treated groups, serum glucose fell below pre-induction stage. At the post-treatment stage, the order of decline in rate of serum glucose follow milk>combination>urine.




*1= Pre- induction, 2= Post- induction before treatment 3= 28 days Post-treatment.*

*Figure 2:* Rate of change in serum glucose across the treatment

The trend indicated that the drop in serum glucose in treated group is suggestive of the presence in the products of hypoglycemic factors, which was in fact reported for camel milk by Singh (2001). It is conceivable that this same factor might be present in the urine, in lower concentration. This may explain the greater decline in milk and milk-urine combination than urine.

There was significant difference in serum glucose between control group and groups administered with camel milk and urine. Significant differences also exist among treated groups (Table 1.). Milk recorded the lowest value followed by urine and milk-urine combination between which there was no difference (P>0.05). Furthermore, the treatments appear to be dose dependent, where significant reduction in serum glucose was recorded with increasing doses of the products.

*Table 1:* Blood glucose levels (mmol/l) of albino rats according to treatments and doses

| Factor | Serum glucose |
|---|---|
| Treatment | |
| Milk | 7.37[c] |
| Urine | 7.91[b] |
| Combination | 7.98[b] |
| Control | 9.32[a] |
| SE | 0.16 |
| Dose (ml) | |
| 1.5 | 7.44[c] |
| 1.0 | 7.88[b] |
| 0.5 | 7.96[b] |
| 0.00 | 9.32[a] |
| SE | 0.16 |
| Interaction | NS |

abc, means bearing different superscript along the same column within a subset differ (P<0.05);

*NS not significant*

The significant lower serum glucose level in rats administered with camel milk might be due to the high insulin-like protein concentration in camel milk. As reported by Singh (2001) who reported that the camel milk contains a high concentration about 52 units/l of insulin-like protein. This insulin-like protein was reported to have hypoglycemic effects (Sboui et al., 2010) by either increasing the release of insulin from the pancreatic beta-cells or by increasing its activity.

This insulin-like factor was reported (Wangoh, 1993) to be resistant to stomach acid degradation as it is encapsulated by casein micelles; this is evident in the fact that camel milk does not form coagulum in the stomach or in acidic medium.

Another possible explanation for the hypoglycemic effect of camel milk is the finding of Kamal (2012) that it has regenerative effects on damaged cells of the pancreas. All these factors may contribute to the observed hypoglycemic effect of camel milk in the study.

The low concentration of serum glucose in rats administered with camel urine, suggest the likely presence of the insulin-like protein, since it has been established to be present in milk (Singh, 2001), its presence in urine is therefore highly probable.

The lower concentration of serum glucose in treated rats may also be related to the report of Yadav et al. (2015) that some plants materials consumed by camel have anti-diabetic effects and the active ingredients are present in the body fluids such as urine and milk.

*b) Effect of Camel milk and urine on Serum Lipids*

Rats administered camel milk and urine separately and in combination had significantly lower TG, TC, LDL and VLDL than the control groups. HDL was however higher (P<0.05) in the treated groups. Dose had no effect (P>0.05) on all the lipid parameters among the treated groups. Treatment x Dose interaction was also not significant (Table 2).

*Table 2:* Serum lipids (mg/dl) in alloxan induced diabetic rats according to treatments and doses

| Factor | Serum Lipids (mg/dl) | | | | |
|---|---|---|---|---|---|
| Treatment | TC | TG | HDL | LDL | VLDL |
| | | | | | |
| Control | 248.33[a] | 237.11[a] | 15.33[d] | 296.11[a] | 54.44[a] |
| Camel milk | 164.44[b] | 134.89[b] | 43.44[a] | 146.44[c] | 27.67[b] |
| Camel urine | 168.78[b] | 133.0[b] | 29.36[c] | 168.79[bc] | 27.22[b] |
| Milk- Urine combination | 185.67[b] | 143.44[b] | 37.22[b] | 191.78[b] | 28.78[b] |
| S.E | 6.68 | 10.22 | 2.04 | 10.50 | 2.00 |
| Dose (ml) | | | | | |
| 1.5 | 182.33[b] | 141.22[b] | 177.33[b] | 33.67[a] | 28.22[b] |
| 1.0 | 163.89[b] | 132.89[b] | 160.67[b] | 39.78[a] | 27.44[b] |
| 5.0 | 172.67[b] | 137.22[b] | 169.00[b] | 36.78[a] | 28.00[b] |
| 0.0 | 248.33[a] | 273.11[a] | 296.11[a] | 15.33[b] | 54.44[a] |
| S.E | 7.45 | 4.85 | 9.03 | 1.90 | 0.90 |
| Treatment x Dose Interaction | NS | NS | NS | NS | NS |

abcd, means bearing different superscripts along the same column within a subset differ (P<0.05)
NS = Not significant
Key: Total cholesterol (TC), Triglycerides (TG), High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL) and Very Low Density Lipoprotein (VLDL)

Hyperlipidemia is a recognized consequence of diabetes mellitus (Sherma et al., 2003). Thus the higher TC, LDL, VLDL and TG in the control group than in treated groups. The higher lipid value of control group is due to DM, which led to abnormalities in lipid metabolism (Arkkila et al., 2001). The increase in lipids in the control group may be attributed to excess mobilization of fat from the adipose tissue due to the under utilization of the glucose (Krishna kumar et al., 2000). It appears that camel milk and urine have hypolipidaemic effects because the treated groups showed significantly lower levels of these lipids (Table 3).

Since insulin has been reported to activate lipoprotein lipase (Arkkila et al., 2001), an enzyme that hydrolyses triglyceride leading to low serum lipids. The presence of insulin-like protein (Singh, 2001) in camel milk will lower lipid components in camel milk treated rats. This supposition is supported by Hull, 2004 and Agrawal et al., (2007b) showing that a high insulin-like factor concentration of camel milk can cause the activation of lipoprotein lipase enzyme.

The HDL level in the treated groups is higher compared to the lower group. This may probably due the presence of some enzymes in camel milk and urine that enhance the reverse cholesterol transport system Al-Numair (2010). In addition the mechanisms by which HDL decreases in diabetes may be due to the impaired metabolism of triglycerides rich lipoprotein with decreased activity of lipoprotein lipase and impaired transfer of materials to the HDL components, in addition to the high level of hepatic lipase among diabetics (Balkis 2009). Finally, insulin resistance may be a direct cause of decrease of HDL concentration (Van Linthout et al., 2010).

A significant increase in LDL and VLDL levels may lead to a significant decrease in HDL levels. The inverse relationship between VLDL and HDL (Boizel 2000) might also explain lower levels of the HDL in the control groups.

The decrease in TC and TG in the treated groups and the increase in HDL in the present study are in agreement with Hassan and Emam (2012), who reported similar findings.

## IV. Conclusions

The following conclusions were drawn from the study.

1. Camel milk and urine reduced serum glucose in rats
2. Rate and extent of serum glucose reduction was highest in milk
3. The hypoglycemic and hyperlipidemic effects of the products are not dose dependent.

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