Original Article

Anti-schistosomal activity of colostral and mature camel milk on Schistosoma mansoni infected mice

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The aim of the present study was to investigate the anti-schistosomal activity of colostral and mature camel milk on Schistosoma mansoni infected mice. Six weeks post infection, mean percentage of protection was detected through the hepatic portal vein. Glutathione-s-transferase (GST), alanine, aspartate transaminase (ALT and AST) and immunoglobulin G (IgG) levels were detected in sera of treated mice before and after infection. Antischistosomal activity of colostral and mature camel milk on Schistosoma mansoni infected mice were 12.81% and 31.60% respectively. The results showed that GST levels in sera of mice fed on colostral and mature camel milk were increased with mean values of 0.070, 0.108, 0.128 and 0.120 in colostral milk groups and 0. 072, 0.085, 0.166 and 0.20 in mature camel milk groups compared with the mice fed on basal diet with means values of 0.070, 0.085, 0.078 and 0.069 before infection and after two, four and six weeks of infection, respectively. On the other hand, there were slight differences on ALT and AST activities. Mice treated with colostral and mature milk (200 µl/day) showed an immunostimulatory effect by inducing IgG titers against soluble worm antigen preparation (SWAP) compared with control. Nevertheless, the difference was not considered significant (0.31 ± 0.1) for colostrum (0.34 ± 0.1) and for mature milk, as compared to normal control (0.2 ± 0.04). Two, four and six weeks post infection, IgG level showed no significant change in sera from mice treated with colostral and mature milk as compared to control. In conclusion, colostral and mature camel milk showed an immunomodualatory effect in normal healthy mice by inducing IgG and GST levels before and after infection with Schistosoma mansoni. Colostral and mature camel milk have a protective response against schistosomiasis.

Key Words: colostrum, camel milk, parasites, Schistosoma mansoni, schistosomiasis, lactoferrin, GST, ALT, AST.

Introduction

Camel milk is given to the sick, the elderly and the very young because of the belief that it is not only healthier, but it works especially well in bone formation. The belief among the Bedouin of the Sinai Peninsula is that drinking camel milk can cure any internal disease.[1] Camel colostrum has total solids of 50.4%. Postpartum solids decrease to 18.4 % after the two days of lactation. This decline was due to the decrease in protein and minerals rather than its fat content.[2] Compared to cow, buffalo and ewe milk fat, camel milk fat contained less short-chained fatty acids, but the same long-chained fatty acids can be found. Compared with cow's milk, colostral and mature camel milk contain a high quantity of protective milk proteins, especially lactoferrin, which has multifunction properties in clinical nutrition.[3]

Schistosomiasis is one of the most wide spread parasitic infections of man, next to malaria. The major infectious species for human are *Schistosoma mansoni, Schistosoma haematobium* and *Schistosoma japonicum.* Five to fifteen percent of subjects infected by *Schistosoma mansoni* develop a severe hepatosplenic disease that may be fatal if left untreated.[4] Although infections can be cured by schistosomicides, chemotherapy is not appropriate for the long-term control of these infections and a major World Health Organisation objecttive is the develop-ment of a vaccine against schistosomiasis. [5]

The host is exposed to antigens produced by several stages of the parasite and its products, all of which elicit a very intense humoral response. In terms of antibodies, IgG_1, IgG_2, IgG_3, IgG_4, IgA, IgE and IgM have all been shown to be stimulated by schistsome infection.[6] Immunomodulators of natural origin can modulate the immune response by utilizing the host's endogenous substances or by using exogenous products of various sources. Immunomodulators include any agent or substance that has a stimulatory, suppressive or regulatory effect. However, the differentiation between immunoregulators and immunostimulants is not always clearly defined.[7]

Colostral and camel milk contain a very high quantity of protective and bioactive proteins that play an important role in the immune system response and in protecting the body

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against infections. Besides the stimulation of the immune system, scientific studies have revealed that lactoferrin also prevents the growth of pathogens, exerts antibacterial and antiviral properties, controls cell and tissue damage caused by oxidation, and facilitates iron transport. In addition, these bioactive proteins contribute to the primary defense system against invading pathogenic organisms, they stimulate the immune system and the growth of various cell lines, help regulate iron status in the body and also serve as natural antioxidants.[3]

The aim of the present study is to determine whether camel milk modulates the immune responses of *Schistosoma mansoni* infected mice. This study was conducted in accordance with the internationally agreed ethical principles for the conduct of medical research.

Materials and methods

Colostral and mature camel milk

Camel milk and colostrum were obtained from a camel local farm (Egyptian Hegin Co.).

Diets and animals

The composition of basal diets used in this study are milk protein (12%), sucrose (5%), fat (10%), vitamin mixtures (1%), salt mixtures (4%), fiber (4%) and starch (64%). Thirty female Swiss albino mice (18-20 gram) were divided randomly into three-test groups (each containing 10 mice). Animals were placed in cages and were given the diets containing colostral and mature camel milk for 45 days. The first group (G_1) was fed on the basal diet + Colostral camel milk injected orally (200lµ/mouse/day). The second group (G_2) was fed on basal diet + mature camel milk injected orally (200µl/mouse/day). The third group (G_3) was fed on basal diet only.

Infection and experimental groups

After forty-five days post feeding, each mouse was infected subcutaneously (S.C.) with 100 *Schistosoma mansoni* cercariae.

Assessment of worms burden

Perfusion and recovery of adult worms were performed at 6 weeks post infection through hepatic portal vein by the perfusion method.[8] The total number of worms in the liver and intestine were determined. Protection was assessed as the percentage reduction in worm counts in liver and intestine according to the formula:

$$P = \frac{C - T}{C} \times 100$$

Where, P: percentage reduction of worms.
C: mean worm burdens in control infected animals.
T: mean worm burdens in pre-treated infected animals

Preparation of sera from feeding mice before and after infection with Schistosoma mansoni

After forty-five days post feeding the blood was collected from colostral, mature as well as basal diet feeding mice. Six weeks post-infection, the blood was allowed to coagulate at room temperature for 15 min. Sera were obtained by centrifugation at 3000 r.p.m and kept in aliquots at -80ºC until used.

Detection of IgG levels against soluble worm antigen preparation (SWAP) using enzyme-linked immunoassay (ELISA)

Enzyme linked immunosorbent assay was performed according to Hiller *et al.*[9] Plates were coated with cercarial soluble worm and soluble egg antigens SWAP. The antigens were diluted to 10 µg/ml in 50mM carbonate buffer, pH 9.6 (100 µL/well) and incubated overnight at 4°C. Plates were washed with the working buffer (PBS-T20) and blocked for sites free of antigen against non-specific binding using 200 µL/well of 1% Bovine serum albumin in the blocking buffer (PBS-T20-BSA), for one hour at room temperature. Sera from S. mansoni infected control and treated infected mice were used at dilution 1:100 in PBS (100 µL/well) and the plates were incubated at room temperature for 2 hrs then washed three times using working buffer (PBS-T20). 100µl of anti-mouse IgG and IgM peroxidase conjugate were used at dilution of 1:3000 and 1:10000 in PBS-T20-BSA respectively for one hour at room temperature. Bound antibody was detected by the addition of O-phenylene diamine dihydrochloride (OPD) as a substrate for visualization of the enzymatic reaction. The reaction was developed for 30 min. at room temperature and stopped using 50 µL/well of 4 M H_2SO_4. The change in optical densities was recorded at λ_{max} 490 nm by the aid of an automatic Titertek multiskan Reader model ELX 800 UV, INC, USA for reading ELISA plated.

Determination of GST

The enzyme activity of GST was determined according to Habig *et al.*[10] Method is based on the fact that the GST enzyme catalyzes the conjugation of glutathione with 1-chlor 2,4 dinitrobenzene (CDNB) and forms a complex which has an absorbance at UV region (340 nm). The mean decrease of absorbance per min was calculated. To 100µl serum, 100µl glutathione solution, 10 µl fresh CDNB and 1290µl phosphate buffer were added and gently mixed. The contents were incubated at room temperature for 1hour. The U.V absorbance was measured using UV spectrophotometer at 340nm. The UV absorbance of blank was obtained using assay mixture without serum. Enzyme activity was defined as the amount of enzyme catalyzing the formation of 1mol of products per min under condition of assay.
Activity = A 340 nm/(9.6) x 1000 = M/min.

Determination of alanine and aspartate transaminase (ALT& AST) activities

Alanine and aspartate transaminase (ALT& AST) activities were determined according to the method described by Reitman and Frankel.[11]

Statistical analysis

Mean and standard error of the obtained data from each different experimental group were calculated. One-way Analysis of Variance (ANOVA) was applied to the data versus the corresponding values of the control. Differences were higher than the theoretical one at $P \leq 0.05$. Statistical analysis of variance (t-test) of glutathione s-transferase, alanine and aspartate transaminase (ALT& AST) activities within groups and between groups was conducted as described by Miller and Miller.[12]

Results

Anti-schistosomal activity of colostral and mature camel milk

Figure 1 and 2 showed that the level of protection against schistosomiasis with 12.8% and 31.6% reduction in total worm burden in Colostral Camel milk (G_1) and mature camel milk (G_2), respectively as compared to positive control (Control) infected.

Glutathione S- transferase (GST) and transaminases liver enzymes (ALT and AST) activities in sera

Table (1) shows the glutathione S-transferase (GST) levels in mice fed on the experimental diets. GST levels in mice fed on colostral and mature camel milk were the highest compared with mice fed on basal diet. The results showed that glutathione S-transferase (GST) levels in mice fed on colostral and mature camel milk were increased with means values of 0.070, 0.108, 0.128 and 0.120 in colostral milk groups and 0.072, 0.085, 0.166 and 0.20 in mature camel milk groups compared with the mice fed on basal diet with means values of 0.070, 0.085, 0.078 and 0.069 before infection and after two, four and six weeks of infection with Cercariae of S*chistosoma mansoni* respectively (Table 1).

Detection of IgG level in sera from mice treated with colostral and mature milk of camel before and after infection with Schistosoma mansoni

One-way analysis of variance (ANOVA) showed positive reactivity for IgG levels in sera from mice treated with colostral (0.32 ± 0.08) and mature milk (0.35 ± 0.08), forty five days post feeding, in comparison to sera from untreated mice (0.22 ± 0.03) (Fig.3). Post infection with *S.mansoni* cercariae, sera from colostral (0.28± 0.05, 0.27± 0.04 and 0.32± 0.09) and mature milk (0.12± 0.01, 0.36± 0.05 and 0.34± 0.05) treated infected mice showed

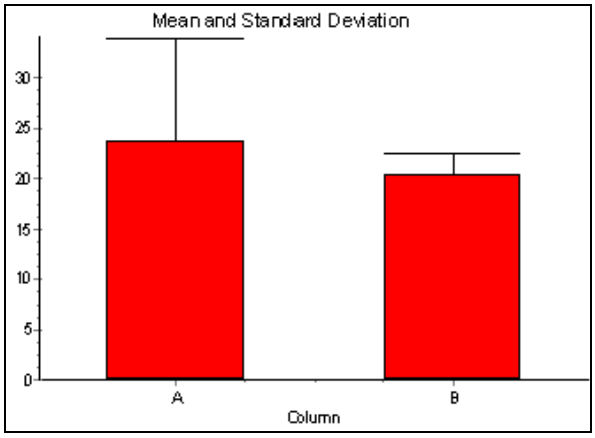

Figure 1. Mean number of worm burden in PBS and colostral camel (A and B column respectively) milk after 6 weeks post infection with Schistosoma mansoni infection.

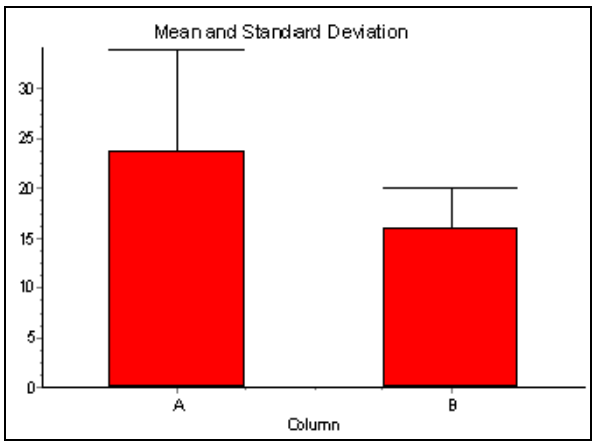

Figure 2. Mean number of worm burden in PBS and mature camel milk (A and B column respectively) after 6 weeks post infection with Schistosoma mansoni infection.

Table 1. Glutathione S-transferase levels in sera of mice fed on colostral and mature camel milk

Experimental groups	Glutathione-S- transferase				
	Min	Max	Mean	S.D.	A/9.6*1000
Basal diet befor infection	0.0678	0.0721	0.0702	0.0022	7.316
Colostral camel milk before infection	0.070	0.0702	0.0702	0.0001	7.302
Camel milk before infection	0.071	0.072	0.0725	0.0007	7.444
Basal diet after two weeks of infection	0.0778	0.0986	0.0851	0.0117	8.861
Colostral camel milk after two weeks of infection	0.1085	0.1097	0.1089	0.0007	11.344
Camel milk after two weeks of infection	0.080	0.091	0.0855	0.0078	8.906
Basal diet after four weeks of infection	0.0738	0.0837	0.0787	0.00625	8.301
Colostral camel milk after four weeks of infection	0.1260	0.1338	0.1282	0.0049	13.351
Camel milk after four weeks of infection	0.133	0.20	0.1665	0.0474	17.344
Basal diet after six weeks of infection	0.0688	0.070	0.0691	0.0008	7.201
Colostral camel milk after six weeks of infection	0.120	0.121	0.120	0.0007	12.490
Camel milk after six weeks of infection	0.199	0.21	0.200	0.0007	20.781

Min: Minimum concentration; Max: Maximum concentration ; SD: Standard Divination; C.V.: Coefficient of Variation

Table 2. ALT and AST (U/ml) levels in mice fed on colostral and mature camel milk

Experimental groups	sAST (U/L)				sALT (U/L)			
	Min	Max	Mean	S.D.	Min	Max	Mean	S.D.
Basal diet before infection	25.1	29.50	26.07	2.506	16.90	20.22	18.40	1.68
Colostral camel milk before infection	26.10	28.22	27.10	1.064	16.1	18.21	17.15	1.490
Camel milk before infection	23.90	28.90	26.41	3.521	17.90	17.92	17.91	0.0071
Basal diet after two weeks of infection	24.10	27.20	26.06	1.704	16.89	19.88	18.22	1.521
Colostral camel after two weeks of infection	24.50	25.90	25.37	0.761	17.00	18.29	17.79	0.696
Camel milk after two weeks of infection	25.70	25.90	25.81	0.127	16.11	18.22	17.17	1.492
Basal diet after four weeks of infection	24.50	25.90	26.32	2.931	18.72	20	19.36	1.501
Colostral camel after four weeks of infection	23.92	28.90	25.77	2.723	16.50	17.91	17.43	0.8112
Camel milk after four weeks of infection	25.70	25.90	25.81	0.127	18.10	18.30	18.19	0.1334
Basal diet after six weeks of infection	28.10	29.22	28.66	4.176	17.50	18.21	17.93	0.382
Colostral camel milk after six weeks of infection	19.32	28.99	24.93	5.021	17.11	17.22	17.44	0.485
Camel milk after six weeks of infection	24.10	26.90	25.49	1.965	18.10	19.90	19.01	1.258

Min: Minimum concentration; Max: Maximum concentration; SD: Standard Divination; .V.: Coefficient of Variation

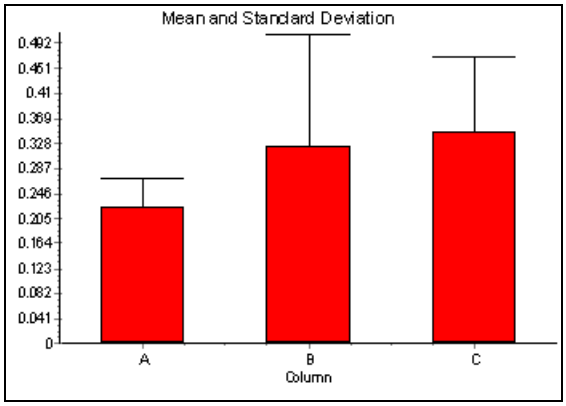

Figure 3. Detection of IgG level in sera from mice injected orally by PBS, colostral and mature camel milk (A, B, C column) respectively.

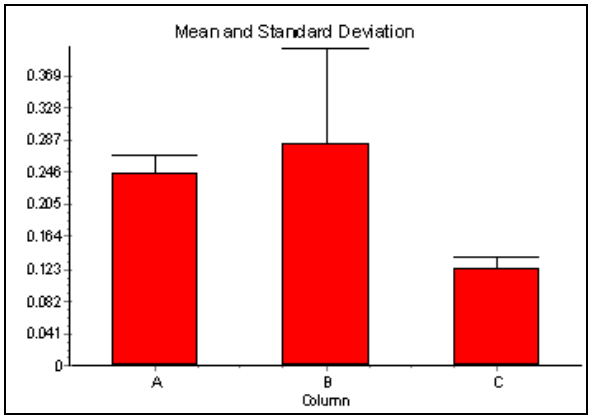

Figure 4. Detection of IgG level in sera from mice injected orally by PBS, colostral and mature camel milk (A, B, C column) respectively at 2 weeks post infection with Schistosoma mansoni cercariae.

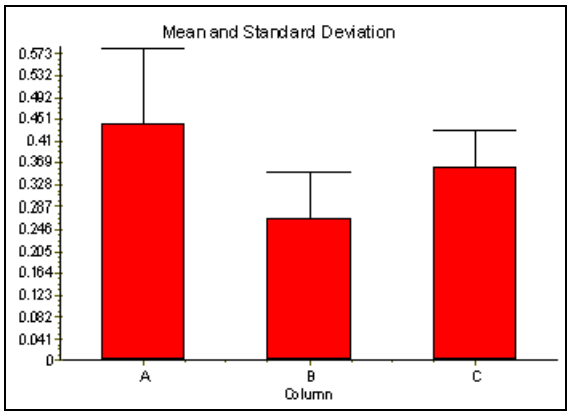

Figure 5. Detection of IgG level in sera from mice injected orally by colostral and mature camel milk (A, B, C column) respectively at 4 weeks post infection with Schistosoma mansoni cercariae.

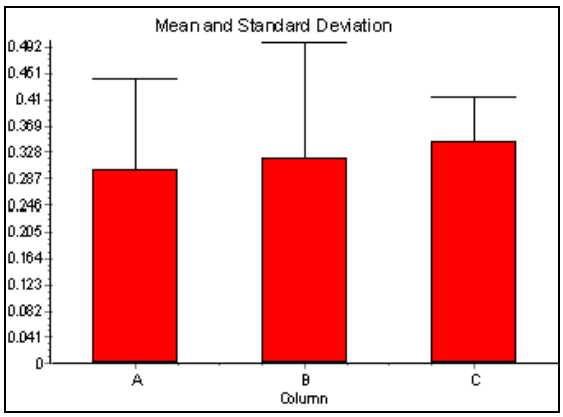

Figure 6. Detection of IgG reactivity in sera of mice fed on mature and colostral milk of camel and infected six weeks with Schistosoma manson (A, B & C column).

no significant change in IgG levels at regular time interals 2, 4, and 6 weeks respectively when compared with that in sera of infected untreated groups (0.25 ± 0.01, 0.44 0.08 and 0.30 ± 0.1) respectively (Fig.4-6).

Discussion

Our results showed that anti-schistosomal activity of colostral and mature camel milk on Schistosoma mansoni infected mice were 12.8 % and 31.60 % respectively. Rey *et al.*,[13] used milk with oltipraz in the urinary schistosomiasis patients. They were treated with a single dose of 35mg/kg oltipraz, which was given under surveillance together with either whole milk or herrings in oil. The tolerance of the product was very good as only 3% of the patients reported vomiting and 3% paresthesias of the fingers. Sixty-six percent of the patients examined on day 30 and/or on day 90 were egg-negative and egg excretion was reduced by at least 90% in 22 other subjects, bringing the percentage of good results to 74%. On day 90, the mean egg excretion was reduced by 82.5% in the overall population and by 80.9% in the 5 to 14 year-olds.

Our data are in agreement with data from a large number of epidemiological studies which have assessed the influence of milk intake on infectious diseases.[13] Studies have shown that milk proteins such as casein, whey proteins and membrane structures, might all exert a stimulatory effect on the immune system and a preventive effect on many diseases such as cancer and infectious diseases. Also, whey proteins were found to be protective relative to other protein sources,[14] this being associated with an increase in the intracellular levels of glutathione (GSH), where whey is a prime source of precursors. When liver glutathione levels rise, the liver is able to more effectively detoxify the body also; undenatured whey protein optimizes serum and liver glutathione levels.[15]

So, there is a relationship between Schistosome GST and immune response. Whereas, GST is one from multiepitope Schistosome vaccine candidates tested for protective immunogenicity in mice. Several promising candidate vaccine antigens including the glycolytic enzymes triose-phosphate isomerase (Sm TPI), a 28 kDa glutathione-S-transferase (Sm 28), the myofibrilar protein paramyosine (Sm 97), an integral membrane protein (Sm 23) and Calpain (Sm calpain) have been characterized by their primary sequences derived for Schistosoma mansoni.[16]

In numerous animal models, vaccination with GST of 28 kDa has been shown to generate an immune response strongly limiting the worm fecundity, in addition to the reduction of the parasite burden.[17] Mice and baboons were injected with two of Sm 28 GST in the presence of aluminum hydroxide and Bordetella pertussis. This reduced female Schistosome fecundity by 33% with more pronounced effects (66%) on faecal egg output and a decrease of the mean granuloma surface in the liver after experimental infection with Schistosoma mansoni.[18]

Immunization of mice with Sm 28 GST/liposome complex followed by challenge with Schistosoma mansoni showed that 28 GST given orally bore protective activity. This result opens the possibility of mucosal vaccination against Schistosomes.[19] Costa *et al.*, [20] showed that egg-hatching inhibition in mice immunized with recombinant Schistosome bovis 28 kD GST. Mice immunized with 14-3-3GST led to protection ranging from 25-46% as determined by reduction of adult worm burden after challenging with Schistosoma mansoni.[21]

The cellular and humoral acquired immune responses to Schistosoma haemtobium 28 GST (Sh28 GST) antigen were evaluated in a Senegalese population chronically infected with Schistosoma haemtobium parasite.[22] Intradermal injection of Sm 28 GST showed a significant reduced parasitemia and a decreased egg-induced inflammatory response in the liver and intestine.[23] Rezende *et al.*,[24] showed that GM-CSF and TNF-alfa synergize to increase in vitro granuloma size of peripheral blood induced by mononuclear cells (PBMC) from human intestinal Schistosomiasis recombinant 28-kDa GST. There is a persistence of the protective immunity (30%) to Schistosoma japonicum in yellow cattle induced be recombinant 26 kDa GST (resjc 26 GST).[25]

There was a slightly insignificant difference on transaminases liver enzymes (ALT and AST) activities (Table 2). The activities of aspartate transferase (AST) and alanine transferase (ALT) are considerably increased following the administration of various hepatotoxic compounds that lead to acute hepatocellular damage and/or extrahepatic obstructions.[26] Therefore, feeding with colostral and mature milk of camel stimulates a specific immune response that protects against Schistosoma mansoni infection. The immuno-protective response results in an increased level of GST which is able to more effectively detoxify the body.

In our study we detected anti-SWAP IgG in sera from mice treated with colostral and mature milk before and after infection with Schistosoma mansoni. Our results showed that colostral and mature milk have immunostimulatory effects by increasing the levels of IgG in mice fed with two types of milk. After infection with S. mansoni (2, 4, and 6 weeks) the levels of IgG in sera from mice treated with colostral or mature milks showed no significant change as compared with untreated infected mice.

The data indicated that colostral and mature milk of camel play a role in enhancing the immune system and increasing resistant factors against Schistosoma mansoni. Also, our results are in agreement with Nassr *et al.*,[27] who showed that anti-SWAP IgG1 and IgG4 are useful in diagnosis and cure. Anti-SWAP IgG1 and IgG4 can be used as parameters for evaluating cure. Follow-up of antischistosomal IgG1 and IgG4 is useful for assess-ment of treatment. In addition, Feng *et al.*,[28] showed that no obvious changes were found in the AWA-IgG4 positive rates of 27 schistosomiasis cases before and after treatment, whereas the antibody level of specific IgG4 was decreased. Nessim and Demerdash[29] showed that praziquantel achieved better cure rates in mice with heavy infection (infected with 120 Schistosoma mansoni cercariae) than in less intensely infected animals (infected with 60 Schistosoma mansoni cercariae). Finally, colostral and mature milk of camel have an immuno-protective response against Schistosoma mansoni. This study suggested that camel milk can be used with antischistomal drugs in Schistosomiasis patients.

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References

1. Gast M, Mauboisj L, Adda J. Le lait et les produits laitiers en Ahaggar. Centre Recherche Anthropologie Prehistoric Ethnologie. 1969.
2. Yagil R, Etzion Z. Milk yields of camels (Camelus dromedarius) in drought areas. Comp Bioch Physiol 1980; 67: 207–209.
3. Abdel-Salam AM. Studies on lactoferrin of colo-strum and milk from different species of animals. MSc. Thesis in Dairy Science, Faculty of Agri-culture, Cairo University, 1996.
4. WHO. The control of schistosomiasis. Second report of a WHO expert. Committee. Geneva, World Health Organization, Technical report series no.830, 1993.
5. Berquist WE. Biofeedback therapy for anorectal disorders in children. Semin Pediatr Surg 1995; 4 (1): 48-53.
6. Dessaint JP, Capron M, Bout D, Capron A. Quantitative determination of specific IgE antibodies to Schistosome antigens and serum IgE levels in patients with Schistosoma haematobium. Clin. Exp Immunol 1975; 20: 427-435.
7. Chang CC, Naiki M, Halpern GM, Gershwin ME. Pharmacological regulation of the immune system.. J Investig Allergol Clin Immunol 1993; 3 (1): 8-18.
8. Smithers SR, Terry RJ. The infection of laboratory hosts with cercariea of *S. mansoni* and the recovery of adult worms. Parasitol 1965; 55: 695-700.
9. Hillyer GV, Gomez DRI. The enzyme linked immunosorbent assay (ELISA) for the immuno-diagnosis of Schistosomiasis. Am J Trop Med Hyg 1979; 28: 237-241.
10. Habig WH, Pabst MJ, Jakoby WB. Glutathione-S-transferase. J Biol Chem 1974; 249: 7130-7139.
11. Reitman S, Frankel S. A colormetric determination of serum Glutamic Oxaloacetic and Glutamic pyro-vic tranaminase. Am Pathol 1957; 28: 56-63.
12. Miller JC, Miller JN. Statistics for analytical che-mistry, 2nd ed. New York, London, Tokyo: Ellis Horweed, 1992.
13. Rey JL, Sellin B, Mouchet F, Sellin E, Simonkovich E, Charmot G. [Urinary schistosomiasis. 1st trial of mass treatment in the field (Niger)]. Sem Hop. 1983; 10; 59 (10): 707-10.
14. McIntosh GH. Colon cancer: dietary modifications required for a balanced protective diet. Prev Med 1993; 22: 767-774.
15. McIntosh GH, Regester GO, Le LR, Royle PJ, Smithers GW. Dairy proteins protect against dime-thylhydrazine-induced intestinal cancers in rats. J. Nutr 1995; 125: 809-816.
16. Yang W, Jackson DC, Zeng Q, McManus DP. Multi-epitope schistosome vaccine candidates tested for protective immunogenicity in mice. Vaccine 2000; Aug 15; 19 (1):103-13.
17. Riveau G, Poulain-Godefroy OP, Dupre L, Remoue F, Mielcarek N, Locht C, Capron A. Glutathione S-transferases of 28kDa as major vaccine candidates against schistosomiasis. Mem Inst Oswaldo Cruz 1998; 93 Suppl 1:87-94.
18. Boulanger D, Reid GD, Sturrock RF, Wolowczuk I, Balloul JM, Grezel D, Pierce RJ, Otieno MF, Guerret S,Grimaud JA . Immunization of mice and baboons with the recombinant Sm28 ST affects both worm viability and fecundity after experimental infection with Schistosoma mansoni. Parasite Immunol 1991; Sep 13 (5): 473-90.
19. Ivanoff N, Phillips N, Schacht AM, Heydari C, Capron A, Riveau G. Mucosal vaccination against schistosomiasis using liposome-associated Sm 28 kDa glutathione S-transferase. Vaccine. 1996 Aug; 14 (12): 1123-31.
20. Da Costa AV, Gaubert S, Lafitte S, Fontaine J, Capron A, Grzych JM. Egg-hatching inhibition in mice immunized with recombinant Schistosoma bovis 28 kDa glutathione S-transferase. Parasite Immunol 1999; Jul 21 (7): 341-50.
21. Schechtman D, Tarrab-Hazdai R, Arnon R. The 14-3-3 protein as a vaccine candidate against schisto-somiasis. Parasite Immunol 2001; Apr 23 (4): 213-7.
22. Remoue F, To Van D, Schacht AM, Picquet M, Garraud O, Vercruysse J, Ly A, Capron A, Riveau G. Gender-dependent specific immune response during chronic human Schistosomiasis haematobia. Clin Exp Immunol 2001; Apr 124 (1): 62-8.
23. Herve M, Angeli V, Pinzar E, Wintjens R, Faveeuw C, Narumiya S, Capron A, Urade Y, Capron M, Riveau G, Trottein F. Pivotal roles of the parasite PGD2 synthase and of the host D prostanoid receptor 1 in schistosome immune evasion. Eur J Immunol 2003; Oct 33 (10): 2764-72.
24. Rezende CM, Goes TS, Goes VS, Azevedo V, Leite MF, Goes AM. GM-CSF and TNF-alpha synergize to increase in vitro granuloma size of PBMC from humans induced by Schistosoma mansoni recom-inant 28-kDa GST. Immunol Lett 2004; Sep 15, 95 (2): 221-8.
25. Wu Z, Liu S, Zhang S, Tong H, Gao Z, Liu Y, Lin D, Liu Z, Wu G, Yi H, Song G, Xu Y. Persistence of the protective immunity to Schistosoma japonicum in Chinese yellow cattle induced by recombinant 26kDa glutathione-S-transferase (reSjc26GST). Vet Parasitol 2004; Sep 2, 123 (3-4):167-77.
26. Hatoff DE, Hardison WG. Hepatic bile acid content controls alkaline phosphatase during cholestasis. Gasteroenterology 1980; 78: 1307.
27. Nassr AKh, Hassan MM, Abdel Salam FM, Lashin AH, Shahin WA, Amin H. IgG isotypes in schistosomiasis patients before and after prazi-quantel. J Egypt Soc Parasitol 2002; 32 (3): 931-52.
28. Feng Z, Qiu LS, Zhang YH, Li H. [Observation on specific IgG4 antibody of schistosomiasis patients before and after treatment] Zhongguo Ji Sheng Chong Xue Yu Ji Sheng Chong Bing Za Zhi 2000; 18 (5): 291-2.
29. Nessim NG, Demerdash Z. Correlation between infection intensity, serum immunoglobulin profile, cellular immunity and the efficacy of treatment with praziquantel in murine schistosomiasis mansoni. Arzneimittelforschung 2000; 50 (2): 173-7.

Anti-schistosomal activity of colostral and mature camel milk on Schistosoma mansoni infected mice
骆驼初乳和成熟乳对感染曼氏血吸虫的鼠具有抗血吸虫的能力

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本研究的目的是调查骆驼初乳和成熟乳对感染曼氏血吸虫的鼠具有的抗血吸虫能力。 感染后 6 星期，通过肝门静脉检测了保护作用的平均百分率。在鼠感染前后测定了血清中谷胱甘肽-s-转移酶(GST)、丙氨酸转氨酶(ALT)、天冬酰胺转氨酶(AST)和免疫球蛋白 G(IgG)的水平。骆驼初乳和成熟乳对于感染曼氏血吸虫的鼠，其抗血吸虫的能力分别为 12.81％和 31.60％。结果表明，在感染前和感染后 2、4 及 6 个星期，对于鼠血清中 GST 的平均增加值，初乳组、成熟乳组和基础饮食组分别为 0.070、0.108、0.128、0.120，0.072、0.085、0.166、0.20，0.070、0.085、0.078、0.069。此外，ALT 和 AST 的活性有轻微但不显著的变化。和正常健康鼠相比，饲喂初乳和成熟乳(200 μl/day)具有免疫刺激作用，可诱导抗可溶性虫抗原制剂的 IgG 效价。然而，与正常控制组(0.2±0.04)相比，初乳组(0.31±0.1)和成熟乳组(0.34±0.1)的差别不显著。与控制组相比，在感染 2、4、6 星期后，初乳组和成熟乳组血清 IgG 的水平没有显著变化。总之，通过在曼氏血吸虫感染前后诱导 IgG 和 GST 的水平，骆驼初乳和成熟乳在正常健康鼠中具有免疫调节作用。骆驼初乳和成熟乳具有抗血吸虫病的作用。

关键词：初乳， 骆驼乳，寄生虫，曼氏血吸虫，血吸虫病，乳铁传递蛋白，谷胱甘肽-s-转移酶，丙氨酸转氨酶，天冬酰胺转氨酶