E-only Article

The Therapeutic Effects of Camel Milk: A Systematic Review of Animal and Human Trials

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Abstract

The clinical effectiveness and value of camel milk as a therapeutic agent is currently unclear. MEDLINE (1946 to March 2016), EMBASE (1974 to March 2016), and Google Scholar were searched using the following terms: milk, bodily secretions, camels, camelus, camelini, camelidae, dromedary, bactrian camel, body fluid, and bodily secretions. Articles identified were reviewed if the study was investigating the use of camel milk for the potential treatment of diseases affecting humans. Of 430 studies, 24 were included after assessment. Identified studies highlighted treatment with camel milk of diseases, including diabetes, autism, cancer, various infections, heavy metal toxicity, colitis, and alcohol-induced toxicity. Although most studies using both the human and animal model do show a clinical benefit with an intervention and camel milk, limitations of these studies must be taken into consideration before widespread use. Based on the evidence, camel milk should not replace standard therapies for any indication in humans.

Keywords

natural products, dietary supplements, systematic review

Camel milk is used extensively within a variety of populations for its proposed healing properties and disease prevention mechanisms.[1] Some of the more common indications associated with its use include diabetes, allergies, immune disorders, and cancer.[2] It is also advocated as an alternative to cow's milk for those who are allergic or intolerant to cow milk proteins.[3] Although traditionally used within Middle East, Africa, and Asia, the advent of online pharmacies and natural health product awareness has increased the availability of camel milk to nontraditional settings such as North America and Europe. Therefore, clinicians must be aware of its properties, stated claims, and clinical data when encountering patients and making therapeutic management plans.

There are 2 distinct species of camel: *Camelus dromedarius* (also known as the dromedary, Arabian camel, or one-hump camel), and the *Camelus bactrianus* (also known as the Bactrian, or two-hump camel). The Bactrian is found in the cold desert regions of Central Asia, whereas the dromedary is native to the hot deserts of North Africa and Western Asia.[2] Milk from both types of camels is composed of high mineral and vitamin content, high unsaturated fats, and protein with some differences in macronutrients that varies depending on the region.[4] The scientific rationale for the use of camel milk as a natural health product comes primarily from its purported antioxidant, immunomodulating, anti-inflammatory, insulin-like, and anti-apoptotic attributes.[4-9] These properties were largely determined through in vitro studies and therefore only provide hypothetical mechanisms of benefit.

The clinical effectiveness and value of camel milk as a therapeutic agent is currently unclear. Although studies in animal and human populations exist, they are mostly small in nature and assess a wide variety of indications and populations. However, patient beliefs may drive use of this agent as a therapeutic alternative or supplemental health product to modern medical practices.[10] Therefore, a critical review is necessary to provide clinicians with a strong background in effectiveness data of camel milk as a health product.

The objective of this systematic review was to summarize and evaluate literature regarding the therapeutic efficacy and

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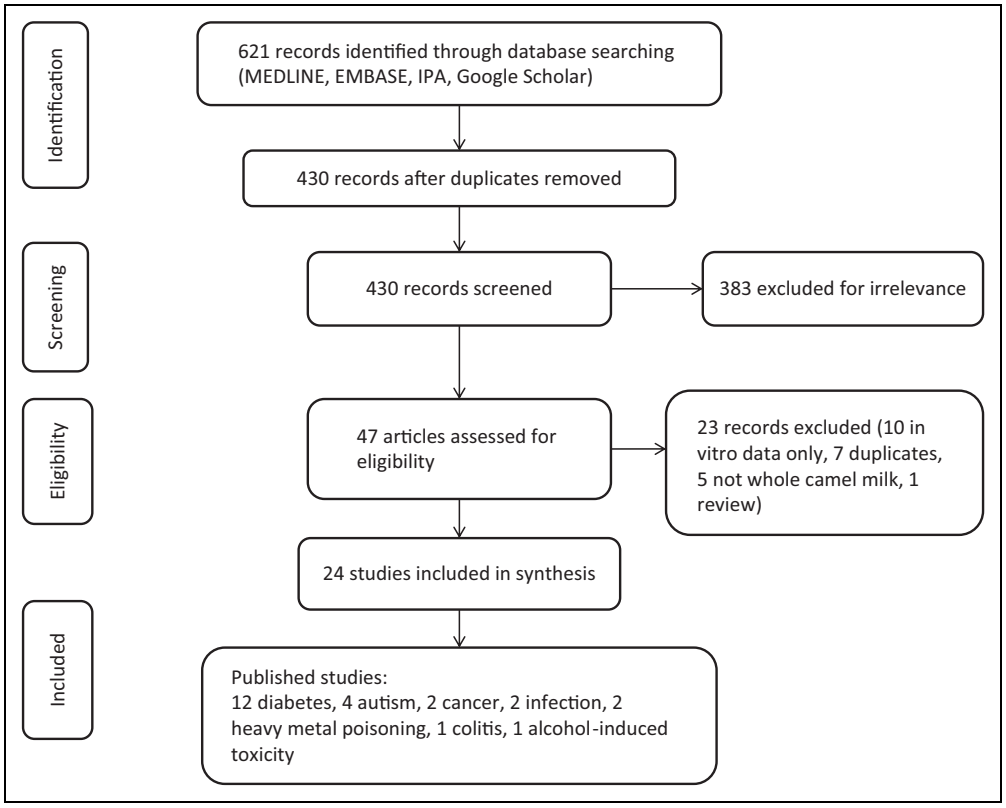

Figure 1. Search strategy flow diagram.

safety of camel milk as both a therapeutic agent and natural health product supplement.

Materials and Methods

We conducted a systematic computerized search from the inception date (1946 in MEDLINE and 1974 in EMBASE) to March 30, 2016 to identify all potentially eligible studies. We applied the following algorithm in both medical subject heading (MeSH) and free text words. In MEDLINE, the MeSH terms "milk" OR "bodily secretions" were combined with "camels." Free text words "camel" OR "camelus" OR "camelini" OR "camelidae" OR "dromedary" OR "bactrian camel" were also included in the search as these were not MeSH terms. In EMBASE, the MeSH terms "milk" OR "camel milk" OR "body fluid" OR "bodily secretions" were combined with "camel" OR "Bactrian camel." Free text words "camelus" OR "camelini" OR "camelidae" were also included in the search as these were not MeSH terms. International Pharmaceutical Abstracts (1970-October 2015) and Google Scholar were also searched, and references of relevant publications were reviewed to identify any articles not captured during initial search.

The articles identified were reviewed if the study was investigating the use of camel milk for the potential treatment of diseases affecting humans. Therefore, studies investigating the use of camel milk for potential veterinary uses were not included. Studies including either humans or animals were included in this review. Articles were excluded if only in vitro data were reported or if published in a language other than English.

Information extracted from studies included design, species of camel, description of study population, number of participants, intervention including quantity and frequency of use, and study outcomes (differed depending on which disease state the intervention was being used for). Adverse reactions to any interventions were also recorded if available. Data were extracted by one investigator and checked for accuracy by a second investigator.

Results

Our search resulted in 430 studies after removal of duplicate studies. Manual search of reference lists did not identify additional studies. After screening and assessing for eligibility, 24 studies[11-34] were included with the remaining articles being excluded for reasons of irrelevance or containing only in vitro data. Among the published trials investigating the effects of camel milk, 9 studies in animals (4 studies for diabetes,[11-14] 2 studies for cancer,[15,16] 1 study for colitis,[17] 2 studies for various infections,[18,19] 2 studies for heavy metal toxicity,[20,21] and 1 study for alcohol-induced toxicity[22]) and 12 studies in humans (8 studies for diabetes[23-30] and 4 studies for autism[31-34]) (Figure 1). No studies identified were published in a language other than English. Study characteristics and results involving animal models are summarized in Table 1 and studies involving human participants are summarized in Table 2.

Discussion

Animal Trials

Diabetes. Presence of insulin in nanoparticles or an insulin-like substance in camel milk are theories that have been proposed to

Table 1. Outcomes for Studies Using Animal Models.

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results
Diabetes					
Khan et al, 2013	Randomized controlled trial Camel species unknown (from Saudi Arabia) collected by milking machine	n = 40 Male albino Wistar rats (200-250 g) 4 rats/cage	Group 1 (n = 8): control Group 2 (n = 8): normal rats fed skim camel milk (centrifuged to remove cream, 400 mL/cage/d) Group 3 (n = 8): STZ-induced (55 mg/kg IP) diabetic control rats Group 4 (n = 8): STZ-induced diabetic rats fed skim camel milk (400mL/cage/d) Group 5 (n = 8): STZ-induced diabetic rats treated with insulin (6 U/kg/d)	30 days Body weight SBG, ALT, AST, ALP, urea, uric acid, creatinine, cholesterol, LDL, HDL, TG	28% decrease in group 3 vs group 1 ($P < .05$) and significant increase in group 4 vs group 3 ($P < .05$) Significant increase in group 3 vs groups 1 and 5 ($P < .05$) and significant decrease in group 4 vs group 3 ($P < .05$) Significant increase in all parameters in group 3 vs group 1 ($P < .05$) and significant decrease in group 4 vs group 3 ($P < .05$)
Korish, 2013	RCT Camel species unknown (from camel farm in Riyadh, Saudi Arabia), collected manually by farmers	n = 70 Male Wistar rats (6-8 weeks old, 270-325 g)	Group 1 (n = 10): normal rats fed standard rat chow (control) Group 2 (n = 20): rats fed standard rat chow + camel milk Group 3 (n = 20): rats fed a high-cholesterol diet Group 4 (n = 20): rats fed a high-cholesterol diet + camel milk	8 weeks Body weight	Increase between group 3 vs group 1 ($P < .001$), decrease between group 4 vs group 3 ($P < .013$), and no difference between groups 2 and 4 vs group 1 ($P > .05$)
				FBG and HOMA-IR	Increase in group 3 vs group 1 ($P < .001$) and decrease in group 4 vs group 3 ($P = .004$), but no difference between groups 2 and 4 vs group 1 ($P > .05$)
				Fasting insulin	Inverse results to FBG and HOMA-IR
				Serum MDA and liver MDA	Increase in group 3 vs group 1 ($P < .011$) and decrease in group 3 vs group 4 ($P < .001$), but no difference between group 2 vs group 1 ($P > .05$)
				Liver GSH, serum CAT activity, and liver CAT activity	Inverse results to MDA
				TC, TG, HDL, LDL, VLDL	Increase in TC, TG, LDL, VLDL, and HDL in group 3 vs group 1 ($P < .001$) and decrease in group 4 vs group 3 ($P < .001$) Decrease in TC, TG, LDL, VLDL in group 2 vs group 1 ($P < .001$) but no change in HDL ($P > .05$)
				Atherosclerotic index	Increase in group 3 vs group 1 ($P < .011$), and decrease in group 4 vs group 3 ($P < .001$) and group 2 vs group 1 ($P = .003$). No difference between group 4 vs group 1 ($P > .05$).
				Liver weight and LBR	Both were higher in group 3 vs group 1 ($P < .001$) and lower in group 4 vs group 3 ($P < .001$), but no difference between group 2 vs group 1 ($P > .05$)

(continued)

Table 1. (continued)

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results
				AST, ALT, AP, GGT, bilirubin	Increase in group 3 vs group 1 ($P < .001$) and decrease in group 4 vs group 3 ($P < .001$), but no difference between group 2 vs group 1 ($P > .04$)
				Serum protein and albumin	Decrease in group 3 vs group 1 ($P < .001$) and increase in group 2 vs group 1 ($P < .001$)
Korish 2014	RCT Dromedary camel from Riyadh, Saudi Arabia, collected manually by farmers	n = 80 Male Wistar rats (6-8 weeks old)	Group 1 (n = 20): Control Group 2 (n = 20): Control rats fed camel milk (~35 mL/d) Group 3 (n = 20): STZ-induced (50 mg/kg) diabetic control rats Group 4 (n = 20): STZ-induced (50 mg/kg) diabetic rats fed camel milk (~35 mL/d)	8 weeks Body weight	Decrease between group 3 vs group 1 ($P < .001$) and increase between group 4 vs group 3 ($P < .001$) but still lower than group 1 and group 2 ($P < .001$)
				FBG and HOMA-IR	Increase in group 3 vs groups 1 and 2 ($P < .001$), decrease in group 4 vs group 3 ($P < .001$) but still higher than groups 1 and 2 ($P < .05$), and no difference between group 2 and group 1 ($P = .99$)
				Fasting insulin	Inverse results to FBG and HOMA-IR
				TC, TG, HDL, LDL, VLDL	Increase in TC, TG, LDL, VLDL and decrease in HDL in group 3 vs group 1 ($P < .001$) decrease in TC, TG, LDL, VLDL and increase in HDL in group 4 vs group 3 ($P < .001$) Decrease in TC, TG, LDL, VLDL in group 2 vs group 1 ($P < .001$) but no change in HDL ($P > .05$)
				GLP-1, GIP, TNF-α, and TNF-β	Increase in group 3 vs groups 1 and 2 ($P = .001$), decrease in group 4 vs group 3 ($P = .001$) but no change in group 2 vs group 1 ($P = .52$)
Korish et al, 2015	RCT Dromedary camel from Riyadh, Saudi Arabia, collected manually by famers	n = 80 Male Wistar rats (6-8 weeks old, 245-265 g)	Group 1 (n = 20): control Group 2 (n = 20): control rats fed camel milk (~65 mL/d) Group 3 (n = 20): STZ-induced (65 mg/kg) diabetic-nephropathy control rats Group 4 (n = 20): STZ-induced (65 mg/kg) diabetic-nephropathy rats fed camel milk (~50 mL/d)	8 weeks Body weight	Decrease in group 3 vs group 1 ($P < .05$) and increase in group 4 vs group 3 ($P < .05$) but still lower than group 1 and group 2 ($P < .05$)
				FBG and HOMA-IR	Increase in group 3 vs group 1 ($P < .05$) and decrease in group 4 vs group 3 ($P < .05$) but still higher in group 4 vs groups 1 and 2 ($P < .05$)
				Fasting insulin	Inverse results to FBG and HOMA-IR
				Urine volume, serum urea, serum creatinine, proteinuria	Increase in all parameters in group 3 vs group 1 ($P < .001$) and decrease in group 4 vs group 3 ($P < .001$). Decrease in urine volume in group 2 vs group 1 ($P = .017$) but no

(continued)

Table 1. (continued)

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results
					difference in urea, creatinine, or proteinuria.
				Glomerular expression of Smad1 mRNA	Increase 2-fold in group 3 vs group 1 ($P < .05$) and decrease in group 4 vs group 3 ($P < .001$) but no difference in group 2 vs group 1
				Renal excretion of Smad1	16-fold increase in group 3 vs group 1 ($P < .001$) and decrease in group 4 vs group 3 ($P < .05$)
				Glomerular expression of Col4 mRNA	No change between any of the groups
				Renal excretion of Col4	3-fold increase between group 3 and all other groups ($P < .05$) and >2-fold decrease between group 4 vs group 3 ($P < .05$)
Cancer					
Alhaider, 2014	RCT Camel species unknown	n = 40 Swiss albino mice aged 5-6 weeks, weighing 20-30 g body weight Cannulated sponge disks implanted subcutaneously as a sponge implant angiogenesis model	Group 1 (n = 10): control (phosphate buffered saline) Group 2 (n = 10): camel milk 12.5 mg/kg daily Group 3 (n = 10): camel milk 25 mg/kg daily Group 4 (n = 10): camel milk 50 mg/kg daily All treatments given through cannula installed into the implanted disks	14 days Extent of vascularization of sponge implants by determining amount of hemoglobin detected in tissue Cytokines in disks: VEGF, TNF-α, IL-1B, IL-6, IL-10, TFG-B, and MCP-1 Neutrophil accumulation from MPO Infiltration of neutrophils from levels of NAG Total soluble collagen Morphometrics and blood vessel quantification	Significant decrease in groups 2, 3 ($P < .01$), and group 4 ($P < .001$) compared with group 1 with a trend toward greater decrease with higher dose VEGF significantly lower in group 2 ($P < .01$) and group 3 ($P < .001$) vs group 1 Other factors were also reduced
Salwa 2010	RCT *C. Hamra* dromedary camels	(n = 70) Male Swiss albino mice 8-9 weeks old, 25-30 g body weight	Group 1 (n = 7): solvent Group 2 (n = 7): Camel milk 33 ml/kg via oral intubation Group 3 (n = 7): cisplatin 0.5 mg/kg Group 4 (n = 7): subacute pretreatment camel milk 2 h before cisplatin 0.5 mg/kg Group 5 (n = 7): simultaneous treatment with camel milk and cisplatin Group 6 (n = 7): acute treatment of cisplatin 2.5 mg/kg	MnPCEs Mitotic index	Pretreatment with camel milk caused significant decrease ($P < .001$) in frequency of MnPCEs and increase in the mitotic index induced by cisplatin ($P < .001$) when compared with groups treated with cisplatin alone

(continued)

Table 1. (continued)

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results
			Group 7 (n = 7): subacute treatment of camel milk followed by acute cisplatin 2.5 mg/kg 2 h after fifth administration of camel milk Subacute by intraperitoneal injection × 5 consecutive days and acutely by intraperitoneal injection × 1 dose		
Colitis					
Arab et al, 2014	RCT Camel species unknown (from Saudi Arabia)	n = 32 Adult Wistar rats. Weight 200 ± 20 g	Group 1 (n = 8): control (saline) Group 2 (n = 8): control + camel milk (unknown pasteurization) dose was 10 mL/kg administered by oral gavage twice daily starting 1 week before induction and continued 4 days after instillation Group 3 (n = 8): 2,4,6- TNBS-induced colitis by rectal instillation Group 4 (n = 8): TNBS-induced colitis + camel milk	5 days postinduction Body weight, colon weight/length ration, colon macroscopic damage (score 0-4; 0 normal), area of colonic lesion (cm^2) and histopathological examination Colon MPO activity, inflammatory cytokines (TNF-α and IL-10) Oxidative stress markers: MDA, NO GSH, TAC	 Group 3 significantly worse than group 1 ($P < .05$). Group 4 significantly improved compared with group 3 ($P < .05$). Group 3 significantly increased compared with group 1 ($P < .05$). IL-10 levels were significantly lower in group 4 compared with group 3, and significantly higher than group 1 ($P < .05$). TNF-α levels were significantly lower in group 4 compared with group 3 ($P < .05$) but not significantly different from group 1 ($P > .05$) Group 3 had significantly higher levels compared with group 1 ($P < .05$). Group 4 had significantly higher levels than group 1 and significantly lower levels than group 3. Group 3 had significantly lower levels compared with group 1. Group 4 had significantly higher levels than group 3 and no statistical difference compared with group 1
Infection					
De Almeida Cardoso et al 2013	Prospective observational Camel species unknown (purchased from Brazil)	n = 50 *Mus musculus* mice, white, male, age 50-60 days, average weight 40 g	Group 1 (n = 10): noninjected mice, normal ration (control) Group 2 (n = 10): injected mice with *Salmonella enterica* sp *enterica*, normal ration (control 2)	31 days Survival analysis by Kaplain-Meier	Group 1 = all survived Group 2 = all died Group 3 = all survived Group 4 = 6 survived, 4 died Group 5 = 2 survived, 8 died Group 1 statistically similar to group 3.

(continued)

Table 1. (continued)

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results
			Group 3 (n = 10): injected mice with inactivated Salmonella, normal ration (control 3) Group 4 (n = 10): injected mice with *Salmonella enterica* sp *enterica*, camel milk (pasteurization unknown) soaked ration Group 5 (n = 10): injected mice with *Salmonella enterica* sp *enterica*, cow milk soaked ration Rations soaked with milk for 2 hours, reweighed until no increase in weight showing saturation. Rations stored at −4°C until use. Rations and water supplied ad lib		Group 4 statistically improved survival compared with groups 2 and 5 but statistically lower survival compared with groups 1 and 3. Group 5 statistically lower compared with groups 1, 3, and 4 and similar to group 2
Maghraby et al 2005	RCT Camel species unknown (from Egypt)	Female, Swiss albino mice, 18-20 g Infected with 100 *Schistosoma mansoni* cercariae given SC	Run in of 45 days given camel colostrum/ milk Group 1 (n = 10): basal diet + 200 μL unpasteurized camel colostrum per day Group 2 (n = 10): Basal diet + 200 μL unpasteurized mature camel milk per day Group 3 (n = 10): basal diet only	45-day run in, infection, 42-day follow-up. Outcomes assessed at 2, 4, and 6 weeks. 1. Total worm burden 2. IgG levels against soluble worm antigen preparation by ELISA 3. GST 4. AST 5. ALT	No statistical analyses done on outcomes. Basal diet vs colostrum or mature CM: "no significant change in IgG levels at regular time intervals at 2,4 and 6 weeks" (*P* value not reported)
Other					
Dallak, 2009	Prospective observational Unknown camel species (from Saudi Arabia). Collected fresh daily	Albino rats, male, weight 230-250 g	Group 1 (n = 6): control (saline PO daily) Group 2 (n = 6): cadmium-induced anemia (10 mg/kg PO daily) Group 3 (n = 6): cadmium-induced anemia (10 mg/kg PO daily) + CM (2 mL PO daily)	21 days Hematological anemia markers: RBC, hematocrit, hemoglobin, MCV, MHC, MCHC Oxidative stress: SOD, CAT Oxidative stress: GSH	Group 2 significantly lower in all anemia markers compared with group 1 ($P < .05$). Group 3 had significantly improved anemia markers compared with group 2 ($P < .05$) Group 2 significantly lower compared with group 1 ($P < .05$). Group 3 significantly higher compared with group 2 ($P < .05$). Group 2 significantly lower compared with group 1 ($P < .05$). No difference between group 3 and group 2. No reported comparisons of group 3 with group 1
Al-Hashem, 2009	Prospective observational Unknown camel species (from Saudi Arabia). Collected fresh daily	White albino male rats, weight 230-250 g	3 groups, n = 10 each Group 1 (n = 10): Control (saline PO daily) Group 2 (n = 10): $AlCl_3$-induced anemia (0.5 mg/kg PO daily)	30 days Albumin, bilirubin, cholesterol, triglycerides, total serum protein, AST, ALT, ALP, LDH Liver measured TBARS, hydrogen peroxide, GSH, CAT, SOD	Group 2 has statistically poorer outcomes for all endpoints when compared with group 1 ($P < .05$) except for a nonsignificant difference in ALP elevation.

(continued)

Table 1. (continued)

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results
			Group 3 (n = 10): $AlCl_3$-induced anemia (0.5 mg/kg PO daily) + CM (1 mL PO daily 10 min before $AlCl_3$ administration)	Serum urea, serum creatinine	Group 3 had statistically improved outcomes for all endpoints when compared with group 2 ($P < .05$).
				Kidney measured TBARS, hydrogen peroxide, GSH, CAT, SOD	No reported comparisons of group 3 with group 1
Darwish, 2012	RCT Camel species unknown	Wistar albino male rats. Weight 100-150 g	1 week run-in period Group 1 (n = 8): control (saline) Group 2 (n = 8): EtOH 6 g/kg/d × 7 days then 8 g/kg/d × 21 days Group 3 (n = 8): EtOH + CM (2 mL PO daily) given 10 min later (prophylaxis) Group 4 (n = 8): EtOH + CM (2 mL PO daily) given during weeks 2-4 (treatment)	28 days Liver weight/body weight ratio	Group 2 had statistically higher ratio compared with group 1 ($P < .05$). Groups 3 and 4 had statistically lower ratio compared with group 2 ($P < .05$) and statistically similar ratios compared with group 1 ($P > .05$).
				ALT, AST, ALP	Group 2 had statistically higher values compared with group 1 ($P < .05$). Groups 3 and 4 had statistically similar ratios compared with group 1 ($P > .05$)
				TG	Group 2 had statistically higher TGs compared with group 1 ($P < .05$). Groups 3 and 4 had statistically higher TGs compared with group 1 but statistically lower TGs compared with group 2 ($P < .05$).
				Cholesterol	All statistically similar ($P > .05$)
				Oxidative stress markers: MDA, TAC	Group 2 had statistically worse levels compared with group 1 ($P < .05$). Group 3 had statistically worse levels compared with groups 1 and 2 ($P < .05$). Group 4 had statistically improved levels compared with group 2 ($P < .05$) but similar levels compared with group 1 ($P > .05$)
				Apoptosis marker (caspase-3) and inflammatory marker (TNF-α)	Group 2 had significantly higher levels compared with group 1 ($P < .05$). Groups 3 and 4 had significantly lower levels than group 2 ($P < .05$) and statistically similar levels compared with group 1 ($P > .05$). No comparisons were made between groups 3 and 4

Abbreviations: ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate transaminase; AUC, area under the curve; BMI, body mass index; CAT, catalase; CM, camel milk; Col4, collagen type 4; DBP, diastolic blood pressure; DM, diabetes mellitus; ELISA, enzyme-linked immunosorbent assay; EtOH, ethanol; FBG, fasting blood glucose; GGT, gamma-glutamyl transpeptidase; GIP, gastric inhibitory peptide; GLP-1, glucagon-like peptide 1; GSH, reduced glutathione, GST, glutathione-*S*-transferase; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; HOMA-IR, homeostatic model assessment insulin resistance; HP, haptoglobin; IFG, impaired fasting glucose; IgG, immunoglobulin G; IGT, impaired glucose tolerance; IL, interleukin; IP, intraperitoneally; LBR, liver-to-body weight ratio; LDH, lactate dehydrogenase; LDL, low-density lipoprotein; MCP-1, monocyte chemoattractant protein 1; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; MDA, malondialdehyde; McPCE, micronucleated polychromatid erythrocyte; MPO, myeloperoxidase; NAG, *N*-acetylglucosaminidase; NO, nitric oxide; OGTT, oral glucose tolerance test; mRNA, messenger ribonucleic acid; SBG, serum blood glucose; SBP, systolic blood pressure; SC, subcutaneously; SOD, superoxide dismutase; STZ, streptozocin; SWAP, soluble worm antigen preparation; TAC, total antioxidant capacity; TBARS, thiobarbituric acid reactive substances; TC, total cholesterol; TFG, transforming growth factor; TG, triglycerides; TNBS, trinitrobenzene sulfonic acid; TNF, tumor necrosis factor; VEGF, vascular endothelial growth factor; VLDL, very low density lipoprotein; WHR, waist-to-hip ratio.

Table 2. Outcomes for Studies Using Human Models.

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results	
Diabetes						
Ejtahed et al, 2015	Single-blinded, parallel, randomized pilot clinical trial (Nov 2012 to March 2013) (IIb)	20 patients between 20 and 70 years old with type 2 diabetes from the Endocrine Clinic of Taleghani Hospital, Tehran, Iran	Group 1 (n = 11): 250 mL pasteurized camel milk PO twice daily × 2 months	(Mean ± SD)	Group 1	Group 1
				FBG (mmol/L)	9.38 ± 2.83	9.44 ± 2.55
				Insulin (pmol/L)	64.59 ± 79.17	84.03 ± 79.87*
				HOMA-IR	3.4 ± 2.9	4.7 ± 3.6*
				TC (mmol/L)	4.82 ± 1.01	4.71 ± 1.11
				TG (mmol/L)	1.58 ± 0.58	1.58 ± 0.59
				HDL (mmol/L)	1.22 ± 0.26	1.30 ± 0.31
				LDL (mmol/L)	2.75 ± 0.75	2.67 ± 0.83
	Camel species unknown		Group 2 (n = 9): 250 mL pasteurized cow milk PO twice daily × 2 months	SBP (mmHg)	135 ± 13	132 ± 20
				DBP (mm Hg)	86 ± 14	84 ± 15
					Group 2	Group 2
				FBG (mmol/L)	8.05 ± 2.39	8.94 ± 3.22
				Insulin (pmol/L)	61.81 ± 39.59	70.14 ± 37.50
				HOMA-IR	3.0 ± 2.4	4.0 ± 2.3*
				TC (mmol/L)	4.90 ± 0.83	5.23 ± 1.40
				TG (mmol/L)	1.73 ± 0.64	1.97 ± 0.98
				HDL (mmol/L)	1.27 ± 0.31	1.30 ± 0.28
				LDL (mmol/L)	2.69 ± 0.49	3.03 ± 0.83
				SBP (mm Hg)	132 ± 17	122 ± 19
				DBP (mm Hg)	83 ± 7	79 ± 10
Mohamad et al, 2009	Randomized, controlled trial (IIb)	54 patients (17-20 years old) with type 1 diabetes from outpatient diabetic clinics at the Faculty of Medicine, Menofia University Hospital and National Cancer Institute and 10 patients without diabetes	Group 1 (n = 27): usual care × 16 weeks	(mean ± SD)	Group 1	Group 2
				At 16 weeks		
				BMI (kg/m^2)	18.43 ± 3.59	24.3 ± 2.95*
				HbA1c (%)	9.59 ± 2.05	7.16 ± 1.84*
				Insulin dose (U/d)	48.1 ± 6.95	23.0 ± 4.05*
	Camel species unknown		Group 2 (n = 27): usual care + 500 mL/d camel milk × 16 weeks	FBG (mg/dL)	227.2 ± 17.7	98.9 ± 16.2*
				C-peptide (pmol/mL)	0.28 ± 0.6	2.30 ± 0.51*
				TC (mg/dL)	266.2 ± 18.2	192.08 ± 11.04
				TG (mg/dL)	171.4 ± 21.6	157.2 ± 18.2
				VLDL (mg/dL)	14.4 ± 4.7	11.5 ± 3.9
			Group 3: control group (healthy patients)	HDL (mg/dL)	54.6 ± 12.5	50.7 ± 11.3
				LDL (mg/dL)	82.42 ± 28.64	99.6 ± 9.78
				Serum insulin (μIU/mL)	1.8 ± 0.45	5.0 ± 1.32
				Anti-insulin antibodies (μU/mL)	26.2 ± 7.69	20.92 ± 5.45
				CrCl (mL/min)	85.75 ± 30.5	99.08 ± 5.18
Agrawal et al, 2005	Randomized, single center, controlled study (IIb)	24 patients with type 1 diabetes	Group 1 (n = 12): usual care alone	1 year	Before	After
				(mean ± SE)	Group 1	Group 1
				BMI (kg/m^2)	17 ± 5.2	18.2 ± 3.8
				HbA1c (%)	7.54 ± 1.38	7.63 ± 1.03
	Camel species unknown		Group 2 (n = 12): 500 mL camel milk + usual care	Dose of insulin (U/d)	33 ± 11	30.16 ± 8.54
				Blood glucose (mg/dL)	121 ± 17.3	105.25 ± 14.50*
				Plasma insulin (μIU/mL)	7.73 ± 2.42	19.54 ± 0.43*
				C-peptide (ng/mL)	0.22 ± 0.03	0.22 ± 0.03
					Group 2	Group 2
				BMI (kg/m^2)	17 ± 4.4	19.2 ± 2.97*

(continued)

Table 2. (continued)

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results			
				HbA1c (%)	7.8 ± 1.38		6.0 ± 0.96*	
				Dose of insulin (U/d)		32 ± 12	17.83 ± 12.4*	
				Blood glucose (mg/dL)	119 ± 19		95.42 ± 15.70*	
				Plasma insulin (μIU/mL)	6.91 ± 2.13		18.17 ± 7.12*	
				C-peptide (ng/mL)	0.18 ± 0.04		0.24 ± 0.07	
Agrawal et al, 2011	Randomized, open clinical, parallel design, single-center (IIb)	24 type 1 diabetic patients from PBM Hospital, Bikaner, India	Group 1 (n = 12): usual care	24 months (mean ± SD)	Before Group 1		After Group 1	
				BMI (kg/m^2)	15.85 ± 2.97		18.45 ± 2.75	
				HbA1c (%)	7.54 ± 1.38		6.83 ± 1.46	
				Insulin dose (U/d)	32.75 ± 11.79		34.00 ± 10.92*	
				FBG (mg/dL)	120.75 ± 17.29		122.00 ± 25.35	
				Plasma insulin (pmol/L)	113.76 ± 18.11		125.01 ± 22.16	
	Came species unknown		Group 2 (n = 12): usual care + 500 mL/d camel milk	C-peptide (ng/mL)	0.13 ± 0.06		0.14 ± 0.09	
				Anti-insulin antibody (%)	19.84 ± 3.21		18.96 ± 2.11	
					Group 2		Group 2	
				BMI (kg/m^2)	15.78 ± 2.36		18.78 ± 1.94*	
				HbA1c (%)	7.81 ± 1.39		5.44 ± 0.81	
				Insulin dose (U/d)	32.50 ± 9.99		17.50 ± 12.09*	
				FBG (mg/dL)	118.58 ± 19.00		93.16 ± 17.06	
				Plasma insulin (pmol/L)	111.98 ± 16.88		114.31 ± 15.32	
				C-peptide (ng/mL)	0.16 ± 0.08		0.21 ± 0.10*	
				Anti-insulin antibody (%)	19.48 ± 4.22		18.76 ± 2.89*	
Agrawal et al, 2007	Cohort study (IV)	50 newly diagnosed type 1 diabetic patients	Group 1 (n = 25): intensive insulin therapy	12 months (mean ± SD)	Before Group 1		After Group 1	
				BMI (kg/m^2)	16.00 ± 2.57		17.00 ± 2.59	
				HbA1c (%)	7.75 ± 1.31		7.30 ± 0.87	
	Camel species unknown		Group 2 (n = 25): intensive insulin therapy + 500 mL/d unpasteurized camel milk	Insulin dose (U/d)	30.20 ± 12.60		32.80 ± 12.62	
				Blood glucose (mg/dL)	114.40 ± 17.70		104.00 ± 15.87	
				Plasma insulin (μIU/mL)	18.78 ± 2.52		19.62 ± 3.17	
				C-peptide (ng/mL)	0.14 ± 0.07		0.13 ± 0.06	
				Anti-insulin antibody (%)	19.45 ± 3.21		19.60 ± 2.46	
					Group 2		Group 2	
				BMI (kg/m^2)	16.30 ± 3.52		17.40 ± 3.14	
				HbA1c (%)	8.20 ± 1.44		7.51 ± 0.98	
				Insulin dose (U/d)	30.40 ± 11.97		19.12 ± 13.39*	
				Blood glucose (mg/dL)	115.16 ± 14.50		100.20 ± 17.40*	
				Plasma insulin (μIU/mL)	18.64 ± 2.49		19.06 ± 2.40	
				C-peptide (ng/mL)	0.14 ± 0.10		0.16 ± 0.10	
				Anti-insulin antibody (%)	19.40 ± 3.20		19.10 ± 2.01	
Agrawal et al, 2011	Crossover study (IIb)	14 nondiabetic males and 14 males with type 2 diabetes from outpatient diabetic clinic in PBM Hospital, Bikaner, India	Group 1 (diabetic subjects, n = 14): 500 mL/d unpasteurized camel milk × 3 months, washout 1 month, 500 mL/d boiled cow milk × 3 months		Baseline	3 months	Washout	7 months
				(Mean ± SE)	Group 1	Group 1	Group 1	Group 1
				FBG (mg/dL)	184 ± 19	116 ± 11*	197 ± 24	216 ± 28
				HbA1c (%)	8.4 ± 0.6	7.3 ± 0.7*	7.3 ± 0.6	8.3 ± 0.6
				Fasting insulin (μU/mL)	13 ± 1	11 ± 1	18 ± 3	21 ± 2
				OGTT 2 hours (mg/dL)	269 ± 29	214 ± 16*	284 ± 34	330 ± 35
				AUC glucose	30672 ± 2563	30724 ± 3676*	29275 ± 2797	34120 ± 3183
				AUC insulin	3714 ± 928	3301 ± 629*	3953 ± 563	5396 ± 725
				HOMA-IR	6 ± 1	5 ± 1*	8 ± 2	10 ± 1

(continued)

Table 2. (continued)

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results			
				C-peptide (ng/mL)	3.1 ± 0.3	3.0 ± 0.3	2.7 ± 0.2	3.2 ± 0.2
			Group 2 (nondiabetic subjects, n = 14): 500 mL/d boiled cow milk × 3 months, washout × 1 month, 500 mL/d boiled camel milk × 3 months	BMI (kg/m^2)	27.1 ± 0.9	26.8 ± 0.8	27.0 ± 0.7	26.5 ± 0.8
				WHR	0.95 ± 0.02	0.96 ± 0.02	0.96 ± 0.02	0.96 ± 0.02
				SBP (mm Hg)	138.8 ± 3.7	135.7 ± 4.6	136 ± 3.7	133.0 ± 3.1
				DBP (mm Hg)	83.1 ± 2.4	79.6 ± 2.5	81.1 ± 2.2	83.7 ± 1.9
				TG (mg/dL)	151.6 ± 14.3	137.5 ± 20.8	174.6 ± 23.2	165.5 ± 34.5
				TC (mg/dL)	180.0 ± 8.7	179.0 ± 8.9	175.6 ± 23.2	185.0 ± 9.0
				HDL (mg/dL)	39.5 ± 1.9	39.2 ± 2.1	38.6 ± 1.5	39.1 ± 2.3
				LDL (mg/dL)	110.0 ± 6.7	112.2 ± 6.8	102.1 ± 6.9	112.8 ± 7.1
					Group 2	Group 2	Group 2	Group 2
				FBG (mg/dL)	86 ± 2	100 ± 3	102 ± 3	98 ± 3
				HbA1c (%)	4.9 ± 0.2	4.6 ± 0.2	4.5 ± 0.2	4.1 ± 0.2*
				Fasting insulin (μU/mL)	7 ± 2	10 ± 4	9 ± 1	14 ± 3
				OGTT 2 hours (mg/dL)	106 ± 8	97 ± 4	114 ± 8	100 ± 7
				AUC glucose	13398 ± 1102	13390 ± 760	13118 ± 997	14220 ± 599
				AUC insulin	3914 ± 871	2858 ± 727	3230 ± 560	4519 ± 569
				HOMA-IR	1.7 ± 0.5	2.5 ± 1.1	2.0 ± 0.3	3.1 ± 0.6
				C-peptide (ng/mL)	1.8 ± 0.2	2.5 ± 0.3	1.8 ± 0.2	2.1 ± 0.3
				BMI (kg/m^2)	23.1 ± 1.2	23.1 ± 1.1	23.4 ± 1.2	23.2 ± 1.2
				WHR	0.9 ± 0.02	0.9 ± 0.02	0.9 ± 0.02	0.9 ± 0.02
				SBP (mm Hg)	123.2 ± 5.2	119 ± 4.2	120 ± 3.4	121.8 ± 4.3
				DBP (mm Hg)	78.6 ± 2.8	78.2 ± 2.9	80.8 ± 2.4	84.7 ± 7.0
				TG (mg/dL)	137.9 ± 12.6	103.7 ± 12.1	154.7 ± 25.4	104.2 ± 16.0*
				TC (mg/dL)	188.2 ± 9.4	180.1 ± 9.1	176.3 ± 8.5	168.6 ± 9.2
				HDL (mg/dL)	40.8 ± 2.2	41.5 ± 2.0	38.6 ± 1.7	38.8 ± 2.3
				LDL (mg/dL)	119.7 ± 7.6	117.8 ± 8.6	106.8 ± 7.5	18.5 ± 6.8
Agrawal et al, 2009	Before-and-after study (IV)	24 type 1 diabetic patients from outpatient diabetic clinic in PBM Hospital, Bikaner, India	500 mL/d camel milk (unclear if pasteurized) + usual care	6 months (Mean ± SE)				
	Camel species unknown			Microalbuminuria (mg/dL)	119.48 ± 1.68		22.52 ± 2.68*	
				Plasma glucose (mg/dL)	128.7 ± 1.17		125.46 ± 1.24	
				Plasma insulin (pmol/L)	127.08 ± 2.86		130.63 ± 3.86	
				Plasma C-peptide (nmol/L)	0.18 ± 0.14		0.24 ± 0.17	
				HbA1c (%)	9.54 ± 0.44		8.65 ± 0.38	
				Dose of insulin (U/d)	41.61 ± 3.08		28.32 ± 2.66*	
				TC (mg/dL)	77.22 ± 0.03		76.32 ± 0.04	
				TG (mg/dL)	92.76 ± 0.18		31.5 ± 0.17	
				VLDL (mg/dL)	6.84 ± 0.02		6.3 ± 0.02	
				HDL (mg/dL)	26.82 ± 0.02		26.28 ± 0.03	
				LDL (mg/dL)	65.18 ± 0.14		45.54 ± 0.10*	
				BMI (kg/m^2)	18.52 ± 0.73		19.43 ± 0.81	
Agrawal et al, 2007	Cross-sectional study (IV)	2099 people from northwest Rajasthan (1055 Raica and 1044 non-Raica)	Group 1 (n = 501): Raica consuming camel milk		Group 1	Group 2	Group 3	Group 4
	Camel species unknown			IFG, n (%)	16 (3.2)	70 (12.65)	35 (6.8)	74 (14)
				IGT, n (%)	4.0 (0.8)	37 (6.7)	13 (2.5)	83 (15.7)
				DM, n (%)	0 (0)	4 (0.7)	2 (0.4)	29 (5.5)

(continued)

Table 2. (continued)

Reference	Design/Camel Species	Population	Intervention (Quantity, Pasteurization)	Outcomes	Results	
			Group 2 (n = 554): Raica not consuming camel milk Group 3 (n = 515): non-Raica consuming camel milk Group 4 (n = 529): non-Raica not consuming camel milk			
Autism						
Al-Ayadhi et al, 2013	Double blind, randomized, placebo controlled trial Camel species unknown	60 children (2-12 years old) with ASD	Group 1 (n = 24): raw milk, 500 mL daily Group 2 (n = 25): boiled milk, 500 mL daily Group 3 (n = 11): cow milk, 500 mL daily for 2 weeks	2 weeks CARS Superoxide dismutase (ng/mL) Glutathione (ng/mL) Myeloperoxidase (ng/mL) CARS Superoxide dismutase (ng/mL) Glutathione (ng/mL) Myeloperoxidase (ng/mL) CARS Superoxide dismutase (ng/mL) Glutathione (ng/mL) Myeloperoxidase (ng/mL)	Before Group 1 37.63 0.54 (0.03) 0.37 (0.03) 2.65 (0.17) Group 2 36.82 (3.27) 0.49 (0.02) 0.34 (0.03) 2.44 (0.13) Group 3 34.18 (3.25) 0.52 (0.03) 0.36 (0.02) 2.11 (0.37)	After Group 1 34.54* 0.59 (0.02)* 0.41 (0.01)* 3.22 (0.24)* Group 2 33.80 (4.91)* 0.57 (0.02)* 0.45 (0.02) 3.08 (0.19)* Group 3 34.41 (3.25) 0.54 (0.03) 0.35 (0.04) 2.62 (0.16)
Bashir et al, 2014	Double blind, randomized, placebo control trial Camel species unknown	45 children (2-12 years old) with ASD	Group 1 (n = 15): raw camel milk, 500 mL Group 2 (n = 15): boiled camel milk, 500 mL daily Group 3 (n = 15): cow milk, 500 mL daily	2 weeks CARS TARC CARS TARC CARS TARC	Group 1 37 (3.8) 45.52 (11) Group 2 38 (5.4) 40.09 (5.4) Group 3 36 (3) 44.46 (10.11)	Group 1 32 (2.6)* 22.86 (59)* Group 2 35 (2.7) 25.25 (3.08)* Group 3 33 (3.4) 47.8 (11)
Shabo et al, 2005	Case reports	Age 4-21 years Number of participants not specified	Not specified	Subjective account of clinical status	Improvement found after use of camel milk	
Adams, 2013	Case report	9-year-old Raw milk	4 oz daily	Subjective account of clinical status	Improvement in behavior and motor planning	

Abbreviations: ASD, autism spectrum disorder; AUC, area under the curve; BMI, body mass index; CARS, Childhood Autism Rating Scale; DBP, diastolic blood pressure; DM, diabetes mellitus; FBG, fasting blood glucose; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; HOMA-IR, homeostatic model assessment insulin resistance; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; LDL, low-density lipoprotein; OGTT, oral glucose tolerance test; SBP, systolic blood pressure; TARC, thymus and activation regulated chemokine; TC, total cholesterol; TG, triglycerides; VLDL, very low density lipoprotein; WHR, waist-to-hip ratio.
*$p < 0.05$.

explain the traditional belief that consumption of camel milk helps prevent and control diabetes.[5] The therapeutic effect of camel milk given to animals with artificially induced diabetes was investigated in 4 studies[11-14] (Table 1). Khan et al[20] conducted a randomized controlled trial to assess the effect of camel milk on rats with streptozocin-induced diabetes. After 30 days, they found that rats with diabetes had a 24.8% decrease in weight compared with healthy controls, but camel milk increased body weight to near normal. They also observed a significant increase in fasting plasma glucose (FPG) in diabetic rats compared with controls and lower FPG in diabetic rats given camel milk. However, FPG was still significantly higher in the camel milk group than the healthy control group and the group treated with insulin. Likewise, they found a statistically significant increase in liver enzymes, markers of renal function, and lipid panel in the diabetic group compared with the control group and a decrease in all these parameters with the administration of camel milk.

Similar parameters were assessed in a series of studies by Korish and colleagues.[12,13] Two of the studies were randomized controlled trials looking at rats with streptozocin-induced diabetes.[12,13] They found similar trends in body weight, FBG, and fasting insulin as the study by Khan et al.[20] One of the trials also looked at lipid levels and found significantly higher total cholesterol, triglycerides, low-density lipoprotein cholesterol, and very low density lipoprotein cholesterol, and significantly lower high-density lipoprotein in the streptozocin-induced diabetes group.[12] The levels of randomized controlled trial, triglycerides, low-density lipoprotein cholesterol, and very low density lipoprotein cholesterol decreased when camel milk was given to diabetic and healthy rats, and high-density lipoprotein increased in diabetic rats. This study also assessed the effect of diabetes and camel milk on glucagon-like peptide-1, glucose-dependent insulinotropic peptide, and tumor necrosis factor (TNF)-α and TNF-β. They found a significant increase in all 4 markers in diabetic rats compared with controls, and a decrease in diabetic rats given camel milk compared with those not receiving any. The administration of camel milk to healthy rats did not show any change in these markers compared with controls. The second trial also assessed the effect of camel milk on diabetic nephropathy.[13] The authors found that rats with diabetic nephropathy had a significant increase in urine volume, serum urea, serum creatinine, and proteinuria, which were lower in the group treated with camel milk. When camel milk was given to healthy rats, it decreased urine volume compared with the control group but it did not alter any of the other parameters.

The third randomized controlled trial by Korish et al[14] assessed the effect of camel milk in rats fed either a standard diet or a high-cholesterol diet. Body weight in this study showed the opposite trend compared to the results from Khan et al,[11] highlighting the pathological differences between insulin-dependent diabetes and obesity. The lipid profile and liver enzymes showed a similar trend to the studies by Khan and Korish.[11-13] Investigators also measured serum and liver concentrations of malondialdehyde, catalase, and reduced glutathione as measures of oxidative stress on the liver. They found a statistically significant increase in malondialdehyde and decrease in catalase and reduced glutathione in rats fed a high-fat diet and a return to near-normal levels in those given camel milk.

Summary. The studies of camel milk in animals with artificially induced diabetes have shown a trend toward improved FBG and decreased insulin requirements, improvement in dyslipidemia, liver function, and diabetic nephropathy. These studies support the need for human trials of sound methodological quality in order to determine any potential clinical benefits of camel milk in diabetic patients.

Cancer. Two studies were identified that investigated the therapeutic effects of camel milk on angiogenesis and cell proliferation, as well as its potential antioxidant and antigenotoxic effects. Alhaider et al[15] used a randomized approach to determine the extent of vascularization of sponge implants between 3 groups of mice receiving increasing amounts of camel milk and controls (Table 1). Measured by the total amount of hemoglobin present in the tissue after 14 days, the study found higher amounts of camel milk administered (25 and 50 mg/kg/body weight daily) suppressed hemoglobin levels and therefore attenuated vascularization. Levels of vascular endothelial growth factor (VEGF) were also lower in the higher concentration camel milk groups.

The antigenotoxic and anticytotoxic effects of camel milk were investigated in a randomized study using seven groups of 10 mice administered different amounts and combinations of camel milk and cisplatin, as well as a control group[16] (Table 1). The objective of the study was to assess a potential protective role of camel milk against the genotoxic effects of cisplatin. Cisplatin was administered at subacute doses (0.5 mg/kg) in 3 groups and at acute doses (2.5 mg/kg) in 2 other groups. Camel milk was given orally at an amount of 33 mL/kg. Findings showed significant decreases in the frequency of micronucleated polychromatic erythrocytes and increases in the mitotic index in groups treated with camel milk, which are deemed to be markers of clastogenic and cytotoxic effects of cisplatin. However, the protective effect exhibited by camel milk did not maintain the same levels as control or camel milk alone groups.

Summary. The limited evidence from the animal studies identified suggests camel milk may have protective roles in both angiogenesis and reducing the cytotoxic and genotoxic effects of cisplatin therapy. While these findings are very preliminary, researchers should be encouraged to further explore potential oncology-related clinical applications of camel milk in human populations.

Colitis. It was anticipated that the antioxidant and anti-inflammatory properties of camel milk would provide benefit by decreasing the markers of colitis damage. Arab et al[17] randomized 32 adult rats with and without chemically induced colitis using 2,4,6-trinitrobenzene sulfonic acid (TNBS) to be treated with saline or camel milk (Table 1). The outcomes

included anthropomorphic, blinded histological examination, and biochemical markers of colitis. For all results, TNBS-induced rats showed statistically significantly worse results when compared with control groups. Whereas TNBS plus camel milk showed similar outcomes to control in body weight, TNF-α, capsase-3, reduced glutathione, and total antioxidant capacity (TAC) levels but showed statistically worse results in colon weight/length, colon macroscopic damage, area of colonic lesions, myeloperoxidase activity, IL-10, malondialdehyde, and nitric oxide. Despite these results, TNBS plus camel milk showed statistically significantly favorable results when compared with TNBS.

Summary. The findings of this animal study are intriguing and warrant further study of camel milk in colitis populations.

Infections. Two studies were identified relating to the treatment of infections[18,19] (Table 1). It is hypothesized that the high lactoferrin, an antioxidant and immunomodulator, and immunoglobulin G (IgG) content of camel's milk could protect the drinker from different infectious agents. In 2013, Cardoso et al[18] published the effects of camel milk saturated rat food compared with cow milk saturated rat food and controls on survival of mice infected with *Salmonella enterica* subspecies *enterica*. The authors found that all the controls groups worked appropriately. Two of the 10 mice treated with cow's milk and 6 of the 10 mice treated with camel milk survived to 31 days. Camel milk–treated mice showed statistically increased survival compared with the cow's milk group, but significantly decreased survival compared with mice not infected or those with heat killed Salmonella injections (100% survival). These results suggest improved survival outcome at 31 days in mice treated with camel milk.

A study by Maghraby et al[19] in 2005 looked at the effects of mature camel milk and camel colostrum on mice infected with *Schistosoma mansoni cercariae*. Mice were run in with treatment for 45 days prior to infection and followed up at 2, 4, and 6 weeks for the outcomes of total worm burden, IgG levels against soluble worm antigen, glutathione-*S*-transferase, aspartate transaminase, and alanine transaminase. Only numerical values were given in the results and no statistical tests reported. The authors state that there was no significant change in IgG levels at any time point between the groups without stating a *P* value.

Summary. Two animal model studies were identified that looked at clinically important outcomes of survival and surrogate outcomes. Limited data show that camel milk may have a protective role in Salmonella infections.

Other Indications. Two studies identified observed the effects of camel milk in rats with heavy metal induced anemia.[20,21] One study found that in cadmium-induced mice, camel milk treatment significantly improved anemia and markers of oxidative stress, except for no difference in glutathione levels.[20] The other study investigated aluminum chloride–induced kidney dysfunction, liver dysfunction, and markers of oxidative stress in each organ.[21] The camel milk–treated mice had significantly improved endpoints compared with the aluminum toxic group in all biochemical endpoints studied. No conclusions can be drawn between the camel milk–treated groups and controls in these studies as no statistical tests were reported.

It is anticipated that the antioxidant, anti-inflammatory, and antiapoptotic attributes of camel's milk may help ameliorate damage caused by alcohol induced liver injury. One study examined these results.[22] Rats treated prophylactically (for duration of alcohol ingestion) and therapeutically (half of alcohol ingestion duration) with camel milk showed improved surrogate markers of liver injury when compared with alcohol-treated rats. Therapeutically and prophylactically treating rats resulted in similar outcomes in alanine transaminase, aspartate transaminase, alkaline phosphatase, liver weight to body weight ratio, TNF-α, and caspase-3 serum levels when compared with controls. Camel milk ingestion resulted in higher serum triglycerides in both groups compared with controls. Therapeutically treated rats had no significant difference in TAC levels whereas prophylactically treated rats had statistically lower TAC levels when compared with controls.

Summary. Animal models that investigated the beneficial effects of heavy metal and alcohol-induced toxicity found improved outcomes in many of the surrogate markers of oxidative stress and anemias. Clinical implications for humans are currently unknown.

Human Trials

Diabetes. A total of 8 studies assessed the effects of camel milk on the prevention and treatment of type 1 and 2 diabetes[23-30] (Table 2). The effect of camel milk compared with cow milk in patients with type 2 diabetes was assessed in an randomized controlled trial conducted by Ejtahed et al.[23] After 2 months of therapy, they found a statistically significant increase in serum insulin concentration in the camel group and a nonsignificant trend toward increase in the cow milk group. There were no significant changes in any other parameters.

Mohamad et al[24] conducted another randomized controlled trial of 54 patients with type 1 diabetes randomized to receive usual care or usual care plus camel milk. The treatment groups were also compared to a group of 10 healthy patients.[24] After 16 weeks of therapy, they found that the group receiving camel milk had a significantly lower mean HbA1c, daily insulin dose, and FBG. However, this was at the expense of a significantly higher mean body mass index and c-peptide. There was no significant difference in lipid levels or renal function.

Three other studies were identified by Agrawal et al assessing various outcomes of camel milk supplementation in patients with type 1 diabetes through randomized controlled trial[25,26] and cohort designs.[27] They found a significant reduction in mean daily dose of insulin when camel milk was compared with usual care,[29] added to intensive insulin therapy for 12 months,[27] and for 24 months.[26] Two studies also found a significant decrease in mean blood glucose,[25,27] and 1 study

found a reduction in HbA1c.[25] Comparatively, only 1 study found a significant reduction in mean blood glucose in the usual care group, and none showed a reduction in HbA1c or daily insulin dose.[25] Although camel milk appears to have an effect on lowering blood glucose and decreasing the dose of insulin required, there were no statistical analyses done between the 2 groups (only before and after therapy in each group); therefore, it is difficult to assess the true benefit over usual care.

Agrawal et al[28] also conducted 1 crossover trial in type 2 diabetic patients in order to assess the effect of camel milk versus cow milk on general diabetes monitoring parameters. The outcomes were measured after a 1-month run-in period (education on diet, exercise, and insulin administration), 3 months of initial therapy (camel milk in diabetic patients and cow milk in nondiabetic patients), 1 month washout, and 3 months of the other milk product. They found, in the diabetic patients, that camel milk significantly reduced FBG, HbA1c, blood glucose 2 hours after an oral glucose tolerance test, area under the curve (AUC) glucose, and AUC insulin. All of these markers returned to baseline after the patients were crossed over to cow milk. In the nondiabetic group, there were no statistically significant differences in any parameters after 3 months of cow milk consumption, but after the crossover period, where they received camel milk, there was a statistically significant decrease in HbA1c and triglycerides.

One before-and-after study was conducted by Agrawal et al[29] to assess the outcome of microalbuminuria before and after camel milk supplementation in patients with type 1 diabetes. It was noted that there was a significant decrease in daily insulin dose and microalbuminuria after 6 months of camel milk therapy. However, there was no control group in this study and all the patients were initially entered into a 1-month run-in period in which they received teaching on a standardized diet, exercise regimen, and insulin administration. Therefore, it is unclear if the positive effect they saw on microalbuminuria and daily insulin requirement was a result of the camel milk or of the new lifestyle modifications.

Finally, one study was identified that assessed the benefits of camel milk in preventing diabetes.[30] Agrawal and colleagues had previously identified that the Raica community of northern India, who consume camel milk, had low prevalence of diabetes. Subsequently, they conducted a cross-sectional study to compare the prevalence of impaired fasting glucose (IFG), impaired glucose tolerance (IGT), and overt diabetes mellitus (DM) in 1055 Raica and 1044 non-Raica Indian community consuming and not consuming camel milk. The results show a trend to lower rates of IFG, IGT, and DM independently in the Raica community and in communities consuming camel milk.

Summary. There are limited studies, with few patients, and of poor methodological quality looking at the effects of camel milk in humans with type 1 or type 2 diabetes. They consistently show a potential benefit of camel milk in reducing FPG, HbA1c, and daily insulin dose required.[23-25,27-30] However, this appears to come at the expense of an increase in body mass index.[27,30] One study also demonstrated a potential decrease in microalbuminuria, but this study had no control group and so conclusions are not firm.[25] Unlike the animal studies, human trials have not shown a benefit in improved lipid levels, and liver function was not assessed. All these observations are limited by a lack of statistical comparison between the camel milk group and control groups. Also, for purported randomized controlled trials, the randomization process, allocation concealment, baseline characteristics, and blinding were either not documented[27,28] or not adequately reported.[29,30] Despite the potential benefits of increasing insulin levels and decreasing serum glucose, camel milk should not be recommended as a therapeutic agent for diabetic patients. However, there does not appear to be major safety risks associated with normal dietary use based on these studies.[23-30]

Autism. Because of its unique composition, camel milk is being investigated for potential effects of oxidative stress along with therapeutic effects in patients with autism spectrum disorder (ASD). Two studies identified in the search sought to evaluate the effect of camel milk consumption on the clinical outcomes of autism and on oxidative stress markers. Al-Ayadhi et al[31] used a randomized, double blind, placebo-controlled approach to determine the effects of raw and boiled camel milk on the Childhood Autism Rating Scale (CARS) and oxidative stress biomarkers including glutathione, superoxide dismutase, and myeloperoxidase (Table 2). Study participants aged 2 to 12 years were randomized into 3 different groups (boiled camel milk, raw camel milk, cow milk [placebo]) and received 500 mL of their respective milk product daily for 14 days. Participants were assessed prior to the introduction of milk into their diet and after receiving the milk product for 14 days. Both raw and boiled camel milk groups had statistically significant reductions in the CARS and oxidative stress markers. In the placebo group, the cow milk intervention did not significantly reduce any measured outcomes. The authors stated that participants were not allowed to add or remove any interventions such as diet plans, supplements, or pharmacotherapies throughout the study period.

In a second study, Bashir et al[32] used a randomized, double blind, placebo-controlled design to assess the effects of camel milk on the CARS assessment and thymus and activation-regulated chemokine (TARC) serum levels (Table 2). Study participants aged 2 to 12 years were randomized to receive boiled or raw camel milk or cow milk 500 mL for 2 weeks. Prior to randomization a baseline CARS evaluation was performed by a child psychiatrist and blood samples were collected at an autism research and treatment center. Findings from this study showed that raw camel milk was associated with significant improvements in the CARS score compared with baseline, while both raw and boiled camel milk were associated with significant decreases in serum levels of TARC. Cow milk was not associated with any significant changes in either measurement. Camel milk in both preparations was generally well tolerated with only irritability and stomach

discomfort noted, although the true numbers of children experiencing these adverse effects were not described.

Two recent reports have also been published highlighting the anecdotal benefits of camel milk in children and youth with ASD. Shabo and Yagil[33] reported that in a population of 4- to 21-year-olds with ASD, the introduction of camel milk into the regular diet was associated with a subjective improvement in behavior and functioning ability. A case report by Adams[34] in 2013 presented a mother's experience with using camel milk in her autistic son stating improvement after a single ingestion of camel milk through behavioral changes. These beneficial effects were sustained throughout the period of camel milk use but dissipated on cessation of camel milk use.

Summary. Available studies have demonstrated significant improvements in the clinical measurement of ASD through the CARS assessment.[31,32] However, as this measurement was only completed once after the 2-week period, it is unclear whether the beneficial effects were maintained. Furthermore, it remains unclear from the current studies as to whether or not the numerical improvements identified in the CARS assessment with the use of camel milk correlated with clinically relevant improvements in patients with autism. Future studies should attempt to identify the potential long-term benefits of camel milk in ASD. Based on current evidence, camel milk should not replace current available treatment modalities.

Limitations

The current evidence is limited by the lack of well-designed trials to decrease the influence of biases. Of the animal models studied, most only reported on surrogate outcomes and many used inappropriate statistical tests increasing the risk of multiplicity errors. Of the human studies, there were incomplete efficacy and safety outcomes reported.

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All authors contributed to the design, execution, analysis, and write-up of this article.

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Ethical approval was not required for this review.

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