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Hypo-allergenic and therapeutic significance of goat milk

Y.W. Park

International Dairy Goat Research Center, Prairie View A&M University, The Texas A&M University System, P.O. Box 4079, Prairie View, TX 77446-4079, USA

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Abstract

Pathogenesis of cow milk allergy indicates that multiple immunological mechanisms exist. Two types of food allergy reactions occur in infants, children and adults. They are reaginic (IgE mediated) or nonreaginic. About 7% of children in the US have symptoms of cow milk allergy, even though almost all children under age 3 yr have circulating milk antibodies. β-Lactoglobulin (molecular weight 36 000) is the major whey protein of cow milk, not found in human breast milk and mostly responsible for cow milk allergy. Clinical symptomology for patients allergic to bovine milk proteins include: rhinitis, diarrhea, vomiting, asthma, anaphylaxis, urticaria, eczema, chronic catarrh, migraine, colitis and epigastric distress.

Goat milk has been recommended as a substitute for patients allergic to cow milk. Between 40 to 100% of patients allergic to cow milk proteins tolerate goat milk. Although some caprine milk proteins have immunological crossreactivity with cow milk proteins, infants suffering from gastrointestinal allergy and chronic enteropathy against cow milk were reportedly cured by goat milk therapy. The higher protein, nonprotein N and phosphate in caprine milk give it greater buffering capacity compared to cow milk. Some physico-chemical properties of caprine milk such as smaller fat globules, higher percent of short and medium chain fatty acids, and softer curd formation of its proteins are advantageous for higher digestibility and healthier lipid metabolism relative to cow milk. Goat milk also has a greater iron bioavailability in anemic rats than cow milk. Further studies of the hypo-allergenic and therapeutic significance of goat milk to humans are very much needed.

Keywords: Cow milk allergy; Clinical symptom; Goat milk; Hypo-allergenicity; Therapeutic value

1. Introduction

There is a shortage of scientific literature on the significance of goat milk in human nutrition, allergy, dietetics, pediatrics and medicine. On a world-wide basis, more people drink milk of goats than that of other species (Haenlein and Caccese, 1984; Park and Chukwu, 1989).

Goat milk differs from cow or human milk in higher digestibility, distinct alkalinity, higher buffering capacity, and certain therapeutic values in medicine and human nutrition (Gamble et al., 1939; Rosenblum and Rosenblum, 1952; Walker, 1965; Devendra and Burns, 1970; Haenlein and Caccese, 1984; Park and Chukwu, 1988; Park, 1991).

Goat milk has been recommended as a substitute for those who suffer from allergies to cow milk or other food sources (Rosenblum and Rosenblum, 1952; Walker, 1965; Van der Horst, 1976; Taitz and Armitage, 1984). Cow milk allergy (CMA) is a frequent disease in infants, but its etiologic mechanisms are not clear. Increased gastrointestinal absorption of antigens followed by adverse local immune reactions may constitute a major etiological factor in development of food allergies like CMA (Walker, 1987). Prolonged exposure of infants having CMA was associated with

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inflammatory response in the lamina propia and constant increase in macromolecular permeability and electrogenic activity of the epithelial layer, even in the absence of milk antigens (Robertson et al., 1982; Heyman et al., 1988). These clinical symptoms were transient, since all disease parameters returned to normal after several months on a cow milk-free diet (Heyman et al., 1990).

Potential of goat milk as the substitute for cow milk or the basis of cow milk-free diet is of importance to infants and other patients with CMA, goat milk consumers, producers and the goat milk industry. The purpose of this paper was to review research of hypoallergenicity and therapeutic values of caprine milk in humans.

2. Pathogenesis of food allergy

Food consumption presents the body with a myriad of antigens capable of causing an immunologic response. Food allergy is the clinical syndrome resulting from sensitization of an individual to dietary proteins or other food allergens present in the intestinal lumen (Firer et al., 1981; McClenathan and Walker, 1982; Heyman and Desjeux, 1992). Incidence of food allergy can increase with the introduction of cow milk early in infancy (Wood, 1986), which is probably due to the immaturity of the immune system of the intestine during the first month of life.

Milk allergy is not confined to infancy, but is also seen as persisting allergy in children and adults (Deamer et al., 1979; Heyman and Desjeux, 1992). Most children under 3 yr of age around the world have circulating milk antibodies (Eastam and Walker, 1977). However, approx. 7% of these children in the US (probably in all Western countries) have symptoms of milk protein allergy (Gerrad et al., 1973; Haenlein, 1992; Podleski, 1992). The type of immune response after intrusion of foreign proteins is very variable, depending on animal species, age of the host, quality and quantity of antigens absorbed, location of the absorption, pathophysiological state, and genetic background (Heyman and Desjeux, 1992).

Prolonged breast-feeding up to 6 months and delay in the introduction of cow milk and solid foods lessens the risk of the appearance of allergic manifestations in babies from atopic families (Saarinen et al., 1979; Foucard, 1985). Infants with minimal exposure to cow milk showed vastly increased total and milk specific IgE antibodies compared with milk-fed infants (Firer et al., 1981). Bovine milk allergy involves IgE responses, where β-lactoglobulin is a milk protein highly resistant to intestinal luminal hydrolysis and mostly responsible for cow milk allergy (Taylor, 1986; Robertson et al., 1982; Heyman and Desjeux, 1992).

Many foods are capable of causing allergic symptoms, as shown in Table 1 (Walker, 1965; Rapp, 1981). However, cow milk is the most frequent cause of food allergy, especially in children (Rosenblum and Rosenblum, 1952; Walker, 1965; Van der Horst, 1976; Firer et al., 1981; Robertson et al., 1982; Podleski, 1992; Heyman and Desjeux, 1992). Apparently, more than one mechanism exists for milk allergy, and more than one is involved in particular patients, even when there is a single clinical manifestation (Podleski, 1992), which has made it difficult to understand (Eastham and Walker, 1979; Deamer et al., 1979; Heyman and Desjeux, 1992; Podleski, 1992).

In understanding pathogenesis of food allergy, two integral aspects of disease mechanisms have been proposed: (a) antigen absorption by the gut, and (b) immune response by the host cell or animal.

Table 1
Major natural food causes of food allergy

Apples	Mustard
Beef	Nuts (oil and extract)
Berries	Onion
Buckwheat	Oranges and other citrus fruits
Cane sugar	Peanut butter
Chocolate (also cola)	Peas
Cinnamon	Pork
Coconut	Potatoes
Corn	Soy
Eggs	Potatoes
Fish (all types, including crab and shrimp)	Wheat
Food coloring	Yeast
Grapes (also raisins)	In adults only: Alcoholic beverages Coffee

Rapp (1981).

2.1. Mechanism of antigen absorption by the gut

The mechanism involved in intact protein absorption was first identified by cytochemistry, using macromolecular markers such as horseradish peroxidase (HRP) and recognized as an endocytotic–exocytotic process (Cornell et al., 1971; Heyman and Desjeux, 1992). Later studies showed that enterocytes were able to process these antigens inside their lysosomal system as non-specialized antigen-presenting cells due to their capacity to express class II histocompatibility antigens on their external membrane (Bland, 1987; Mayrhofer and Spargo, 1987).

Under normal physiological conditions, some amounts of macromolecules such as food antigens are constantly absorbed by the intestinal epithelium (Heyman and Desjeux, 1992). It is difficult to quantify the exact amount of protein that crosses the intestinal epithelium due to a number of interactions involved before and after epithelial transport.

Using in vitro methods in which intestinal fragments are tested in Ussing chambers, protein transport from the intestinal epithelium has been measured quantitatively (Marcon-Genty et al., 1989; Isolauri et al., 1990). Two functional pathways of protein antigen absorption by transcytosis have been proposed as shown in Fig. 1. (Heyman et al., 1982; Isolauri et al., 1990; Heyman and Desjeux, 1992). The main pathway is degradation in the lysosomal processing of protein. This does not imply total hydrolysis of the protein but generates new antigenic determinants with molecular weight of 2000–4000, which may still interact with the underlying immune cells (Heyman et al., 1982). More than 90% of the protein internalized passes in this way, and the magnitude of the absorption is about 2–4 $\mu g/h \cdot cm^2$ (Marcon-Genty et al., 1989). The second pathway of protein antigen absorption is direct transcytosis which is a minor one. This pathway involves the transport of intact protein which comprises $< 10\%$ of the total transport (Isolauri et al., 1990; Heyman and Desjeux, 1992).

2.2. Mechanism of immune response by host cell (animal)

Absorption of food antigens triggers the immune system of the host cell and releases various mediators, which are involved in the maintenance of epithelial permeability dysfunctions (Heyman and Desjeux, 1992). Reactions of food allergy can be classified according to immunological mechanisms as reaginic (IgE mediated) or nonreaginic (Deamer et al., 1979; McClenathan and Walker, 1982).

The first type reaction is immediate hypersensitivity. IgE-specific antibodies become bound to mast cells or basophils, which react on reexposure to the allergen, causing mediators such as histamine to be released (Worthington et al., 1974; McClenathan and Walker, 1982; Podleski, 1992). Mediators are stored in body cells and released when triggered by a local stimulus (Heyman and Desjeux, 1992; Podleski, 1992). The mediators act on local tissues, causing vasodilation, smooth muscle contraction, and secretion of mucus. Release of histamine also brings on a congestion of capillaries and flooding of intracellular spaces by lymphatic glands (McClenathan and Walker, 1982; Haenlein and Caccese, 1984). Stimulation of local nerve endings also occurs. On pathologic examination, affected areas show submucosal edema, dilated blood vessels, and eosinophilic infiltration. Mast cell degranulation and an increase in number of IgE-staining plasma cells may be seen in the intestinal interstitium (May and Bock, 1978; Firer et al., 1981). Persons with an allergic reaction are usually more sensitive to the release of histamine and tend to produce greater numbers of antibodies to certain proteins (Haenlein and Caccese (1984). Most milk allergy is not reagin (IgE) mediated (Deamer et al., 1979).

The second type of immunologic mechanism is considered to have several pathways: Nonreaginic antibodies react with antigen-forming complexes that in turn activate a complement system, causing inflammation and/or cytopathic effects (McClenathan and Walker, 1982). Another mechanism which is probably not immunologically mediated in food allergy may be direct intestinal mucosal toxicity for the protein or its breakdown fragments, as suggested in gluten enteropathy. Hydrolysis of the absorbed proteins allows the formation of peptides that might be implicated in lymphocyte activation (Bland, 1987; Mayrhofer and Spargo, 1987; Heyman and Desjeux, 1992). In a given patient, it is likely that several mechanisms operate simultaneously with one predominating and the others contributing to the reaction (Heyman and Desjeux, 1992; Podleski, 1992).

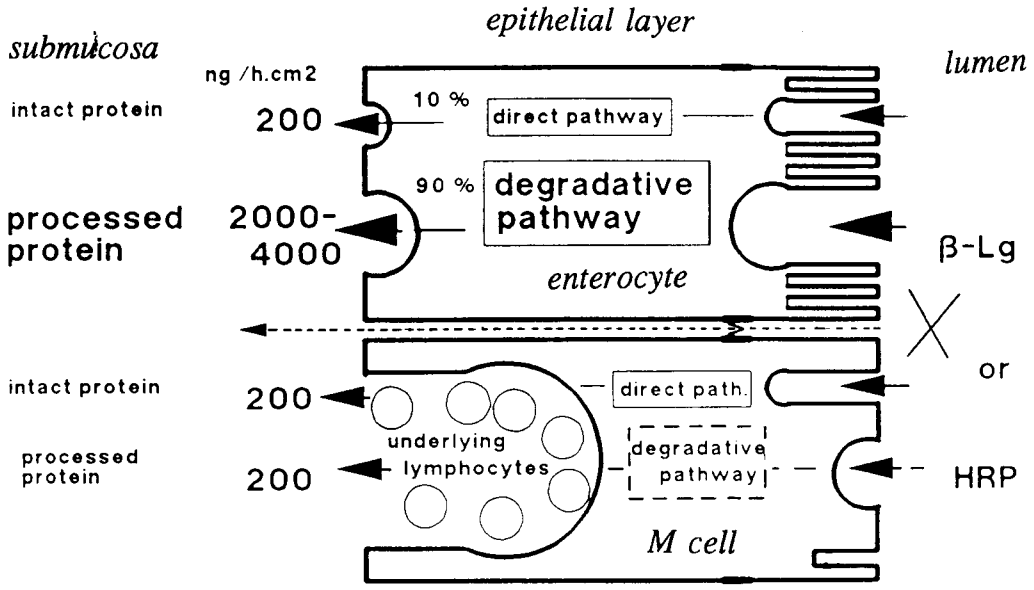

Fig. 1. β-Lactoglibulin (β-Lg) transcytosis across the intestinal epithelium: Total transport is measured by ^{14}C-β-Lg counting and antigenic β-Lg by enzyme-linked immunosorbent assay. Like most food-type proteins, β-Lg is absorbed along two functional pathways that comprise a main degradative pathway, implying the action of a lysosomal system, and a minor pathway that allows the transport of intact proteins. Paracellular leakage is very unlikely, except in certain pathological situations such as bacterial cytotoxin interactions or high evels of lymphokines such as interferon-γ and tumor necrosis factor α. Processing of absorbed proteins allows formation of peptides that might be implicated in lymphocyte activation. Protein absorption by Peyer's patches does not seem to increase more than absorption by the adjacent epithelium. However, the degradative pathway is greatly reduced, possibly due to the presence of M cells on the epithelium overlying the patch, because these cells have no lysosomal system. Another possibility is that degraded protein fragments are bound to the underlying lymphocytes and trapped inside the dome of the patch. HRP (horseradish peroxidase) (Heyman and Desjeux, 1992).

3. Clinical manifestations of cow milk allergy

Symptoms of milk protein allergy usually develop between 2 and 4 wk of age and almost always appear within the first 6 months of life (Deamer et al., 1979; Robertson et al., 1982). Sites of milk allergy, which are most often involved, are the gastrointestinal, respiratory, dermatologic and systemic local tissues. Symptoms of milk protein allergy are manifested as vomiting, diarrhea, colitis, epigastric distress, malabsorption, eczema, urticaria, rhinitis, asthma, bronchitis, anaphylaxis, hyperactivity, migraine (Walker, 1965; McClenathan and Walker, 1982; Husby et al., 1990).

Bovine milk eosinophilic induced colitis among children is well established (Wilson et al., 1990). Clinical symptomatology to bovine milk is related to bronchospasm, rhinitis, diarrhea, erythema, and eczema (Husby et al., 1990). Symptoms of documented bovine milk protein allergy have been found: rhinitis (43%), diarrhea (43%), abdominal pain (41%), anaphylaxis (10%), and urticaria (7%) (McClenathan and Walker, 1982).

In a study of 45 children having various gastrointestinal, dermatologic and respiratory symptoms suspected to be caused by cow milk allergy, Bahna (1991) gave oral challenge with whole bovine milk and skin testing supplemented with intradermal whole bovine milk, casein and α-lactalbumin. Tests were positive in 23 subjects: Concordance (both tests positive and negative) between the results of whole milk challenge and skin testing with bovine milk was 45%, with casein 51% and α-lactalbumin 31%. Pahud et al. (1985) observed that guinea pigs, which had been orally sensitized to demineralized whey were sensitized to several whey proteins (β-lactoglobulin, α-lactalbumin, and immunoglobulin). They reported highest titers in cutaneous anaphylaxis with β-lactoglobulin and lower titers with other whey proteins. Demineralized whey protein lost its sensitizing capacity when it was hydrolyzed with trypsin. Among children allergic to cow

milk, the group which was breast fed and had minimal exposure to cow milk showed lower titers of IgG, IgA, and IgM milk antibodies than the group fed substantial volumes of cow milk (Firer et al., 1981).

Pathophysiological symptoms of milk allergy may be clinically manifested in two major sites: small intestine and colon. Typically patients having pathological reactions in the small intestine are irritable, fail to gain weight, and have bulky, foul-smelling diarrhea stools (Worthington et al., 1974; McClenathan and Walker, 1982). A 72-h fecal fat measurement for these patients often showed fat malabsorption and abnormal fat values in the stool. Observations on small-bowel biopsy may be indistinguishable from those in celiac disease. Histologic changes range from a moderate inflammatory cell infiltrate of the lamina propria to a totally flattened villous lesion with chronic inflammatory changes (Fontaine and Navarro, 1975). Bacterial infections, viral enteritis and malnutrition are often associated with histological intestinal lesions, which interact with pathological reactions of milk allergy in intestinal villi through increased permeability of antigen molecules (Isolauri et al., 1990; Heyman and Desjeux, 1992). Besides cow milk, foods that have been found to cause blunting of intestinal villi are soya, gluten and egg (Eastham et al., 1978).

The typical clinical symptom of pathological reaction in the colon is diarrhea with occult blood and mucus in the stool. Sigmoidoscopic findings showed erythema, edema, small ulcers, and spontaneous mucosal friability in the colon (McClenathan and Walker, 1982). Histologic characteristics on rectal biopsy revealed that there was infiltration of the lamina propria by lymphocytes, plasma cells, eosinophils and neutrophils, with destruction of the surface epithelium, crypt abscesses, and distortion of rectal glands (Gryboeld et al., 1966).

Food allergy can also be observed with delayed symptoms due to the activation of T lymphocytes. These activated lymphocytes release lymphokines, including various interleukins, tumor necrosis factor and γ-interferon that might affect intestinal epithelial permeability (Heyman et al., 1990).

In the manifestation of milk allergy, one should be cautious of the patients' symptomatology compared to that of lactose intolerance. Many humans in certain parts of the world gradually lose some or all of the intestinal enzyme lactase to digest lactose after infancy. Deficiency of lactase causes clinical symptoms, which can persist in some racial groups and are often confused with common symptoms of bovine milk allergy.

4. Hypo-allergenicity of goat milk

The use of goat milk as a hypo-allergenic infant food or cow milk substitute in human diets has been reported in much anecdotal literature about persons who suffer from eczema, asthma, chronic catarrh, migraine, colitis, hayfever, stomach ulcer, epigastric distress, and abdominal pain due to allergenicity of cow milk protein (Walker, 1965; Taitz and Armitage, 1984). Children who were reactive to bovine milk but not to goat milk, also reacted to bovine milk cheese but not to goat milk cheese (Soothill, 1987). Gastrointestinal allergy in infants with eosinophilia also improved after administration of goat milk (Rosenblum and Rosenblum, 1952). A case of chronic enteropathy in infants due to feeding cow milk formula was cured by shifting to goat milk (Maszewska-Kuzniarz and Sonta-Jakimczyk, 1973). Successful management of bovine milk allergy by substitution of goat milk formula has also been reported by Van der Horst (1976).

Brenneman (1978) reported that approx. 40% of allergic patients, sensitive to cow milk proteins, are able to tolerate goat milk proteins. These patients may have been sensitive to cow lactalbumin, which is species specific. Another milk protein, β-lactoglobulin, is mostly responsible for cow milk allergy (Zeman, 1982; Heyman and Desjeux, 1992). Walker (1965) reported that only one in 100 infants, who were allergic to cow milk, did not thrive well on goat milk. Of 1682 patients with allergic migraine, 1460 were due to food, 98 due to inhalants, 98 due to endogenous causes (bacterial), and 25 due to drugs (including tobacco). Among the 1460 patients with food allergy, 92% were due to cow milk or products, 35% to wheat, 25% to fish, 18% to egg, 10% to tomatoes, and 9% to chocolate. Some patients were allergic to more than one food.

Soy formula is the most frequent substitute for cow milk or cow milk formula for infants suspected of cow milk allergy, but approx. 20–50% of these infants will still have similar intolerance symptoms to soy formula (Halpla et al., 1977; Chandan et al., 1992). Evaporated goat milk or goat milk powder has been recommended for infant formula (McLaughlan et al., 1981; Juntunen

and Ali-Yrkko, 1983; Taitz and Armitage, 1984; Coveney and Darnton-Hill, 1985). Heat applied to manufacturing processes reduces allergic reactions (Perlman, 1977). Heat denaturation alters basic protein structures by decreasing its allergenicity (Macy et al., 1953), and high heat treatment removes the sensitizing capacity of milk (McLaughlan et al., 1981). Since α_{s1}-casein content of goat milk can be low, it is logical that children with sensitivity to α_{s1}-casein of cow milk may tolerate goat milk well (Juárez and Ramos, 1986; Chandan et al., 1992).

Lactalbumin from goat milk shows a different skin reaction in comparison to bovine milk. Perlman (1977) reported the variation of skin test reactions to allergenic fractions of bovine milk and goat milk (Table 2). The data indicate that some proteins of bovine milk gave higher incidence of positive skin test reactions than goat milk. Inconsistency in cross-allergenicity among milks of different species may be qualitative and quantitative (Podleski, 1992). A few reports using gel electrophoretic precipitation analysis also suggested that there was a certain immunological crossreactivity between cow and goat milk proteins (Saperstein, 1960; Parkash and Jenness, 1968; Saperstein, 1974; McClenathan and Walker, 1982). However, little clinical research has demonstrated that goat milk is not suitable for patients allergic to cow milk due to immunological crossreactivity between the two milks and their protein.

Much anecdotal evidence of goat milk value as a hypo-allergenic substitute for children allergic to bovine milk has been and is reported (Podleski, 1992), but there are limited data on the basic immunology and biological mechanisms to support the clinical observations, why goat milk can substitute for cow milk in allergic patients.

5. Therapeutic and special nutritional merits of goat milk

Goat milk fat contains significantly greater contents of short and medium chain length fatty acids (C4:0–C12:0) than cow milk fat (Babayan, 1981; Juarez and Ramos, 1986; Chandan et al., 1992; Haenlein, 1992). This difference may contribute to more rapid digestion of goat milk fat, since lipase attacks ester linkages of such fatty acids more readily than those of longer chains (Jenness, 1980; Chandan et al., 1992). Caproic (C6:0), caprylic (C8:0), capric (C10:0) and medium chain length fatty acids (MCT) have been utilized for treatment in a variety of malabsorption patients suffering from chyluria, steatorrhea, hyperlipoproteinemia, and in cases of intestinal reaction, coronary bypass, premature infant feeding, childhood epilepsy, cystic fibrosis and gallstones. These fatty acids are metabolically unique in providing energy in growing children as well as hypocholesterolemic effects on tissues through inhibition of cholesterol deposition and dissolution of cholesterol in gallstones (Greenberger and Skillman, 1969; Kalser, 1971; Tantibhedhyanangkul and Hashim, 1975; Haenlein, 1992). Goat butter, ghee and related products with higher concentration of MCT than even goat milk have not been studied in relation to the physiological well-being of human subjects.

Average size of goat milk fat globules is smaller than that of cow and other species milks. Comparative average diameters of fat globules for goat, cow, buffalo and sheep milk were reported as 3.49, 4.55, 5.92, and 3.30 μm, respectively (Fahmi et al., 1956; Juarez and Ramos, 1986). The smaller fat globule size of goat milk would have better digestibility compared to cow milk counterparts (Haenlein and Caccese, 1984; Stark,

Table 2
Variations in skin test reactions to fractions of cow milk and goat milk

Patients	Fractions			Bovine plasma albumin	Goat milk albumin
	α-lactalbumin	β-lactoglobulin	Casein		
GF	+ + + +	+	–	–	–
VWW	+ +	+	–	–	–
DK[a]	+	+ + + +[a]	+	–	–
VDB	+ + + +	+ + +	+ + +	not done	+ + +

[a] β-Lactalbumin heated to 100°C still gave a + + reaction, but after heating to 120°C for 20 min all skin test reactions disappeared.
Perlman (1977).

Table 3
Concentration of total N, NPN, and phosphate in goat, cow milk and soy-based infant formulas[1]

	n[2]	Total N		NPN		P_2O_5	
		$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
Goat milk							
Alpine	25	0.390[c]	0.032	0.048[b]	0.008	0.166[a]	0.020
Nubian	25	0.556[a]	0.013	0.061[a]	0.013	0.212[a]	0.015
Cow milk							
Holstein	25	0.392[c]	0.058	0.033[c]	0.002	0.173[a]	0.022
Jersey	25	0.505[b]	0.043	0.038[c]	0.004	0.211[a]	0.118
Formula milk							
Brand A	5	0.227[d]	0.026	0.020[d]	0.003	0.211[a]	0.008
Brand B	5	0.259[d]	0.016	0.019[d]	0.003	0.192[a]	0.053

[a,b,c,d]Means with different superscripts within same column are significantly different ($P<0.01$).
[1]Expressed in g/100 ml.
[2]Number of determinations per mean value.
Park (1991).

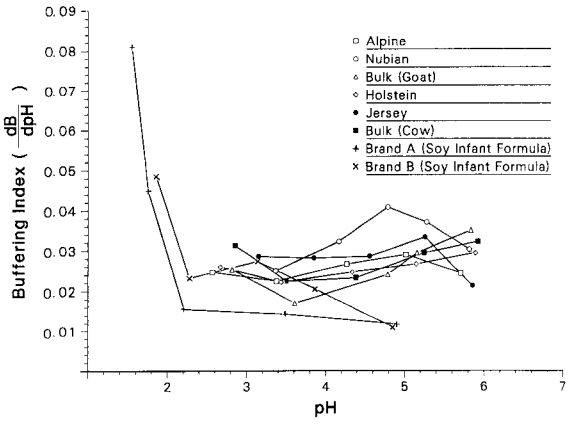

Fig. 2. Buffering capacities of goat and cow milk compared with soy-based infant formula. Number of observations for Alpine, Nubian, Holstein, Jersey, brand A, B, C formula milks were 25, 25, 25, 5, and 5. (Park, 1991).

1988; Chandan et al., 1992). Goat milk proteins may be digested more readily and their amino acids absorbed more efficiently than those of cow milk. Goat milk is considered to form a softer, more friable curd when acidified, which may be related to lower contents of α_{s1}-casein in the milk (Jenness, 1980; Haenlein and Caccese, 1984; Chandan et al., 1992). Smaller, more friable curds would be attacked more rapidly by stomach proteases (Jenness, 1980).

Goat milk also had a greater iron bioavailability in anemic rats than cow milk (Park et al., 1986). Anemic rats fed on goat milk grew significantly better, had higher liver weights and hemoglobin regeneration efficiency than those on cow milk. Mack (1953) also observed that children on goat milk surpassed those on cow milk in weight gain, stature, skeletal mineralization, bone density, blood plasma vitamin A, calcium, thiamine, riboflavin, niacin, and hemoglobin concentrations. However, goat milk has been blamed for development of goat milk anemia due to deficiencies of folic acid and vitamin B_{12} in the milk (György, 1934; Collins, 1962; Nicol and Davis, 1967; Davidson and Townley, 1977; Park et al., 1986).

Goat milk has better buffering capacity, which is good for the treatment of ulcers (Devendra and Burns, 1970; Haenlein and Caccese, 1984; Park, 1991; 1992). Proteins, primarily casein and phosphate systems in milk, influence its buffering capacity (BC) (Watson, 1931). Nubian goat milk showed a higher BC compared with Alpine, Holstein and Jersey cow milks (Park, 1991). Major buffering entities of milks were influenced by species and breeds within species (Table 3). Nubian goat milk had highest levels of total N, protein, non-protein N (NPN) and phosphate (P_2O_5) among four breeds of goats and cow milks. Regardless

of breed, goat milk contained significantly higher non-protein N than cow milk. The higher levels of nitrogen moieties and phosphate in goat milk were positively correlated with higher BC (Park, 1991). Soy-based infant formulae contained less total N and NPN compared with natural goat and cow milks, and BC of the formulae were also lower than those of natural milks (Fig. 2). This suggests that higher BC in Nubian goat milk compared to cow milk can be of importance in human nutrition.

References

Babayan, V.K., 1981. Medium chain length fatty acid esters and their medical and nutritional applications. J. Am. Oil Chem. Soc., 59: 49A–51A.

Bahna, S.L., 1991. New aspects of diagnosis of milk allergy in children. Allergy Proc., 12: 217–220.

Bland, P.W., 1987. Antigen presentation by gut epithelial cells: secretion by rat enterocytes of a factor with IL-like activity. Adv. Exp. Med. Biol., 216: 129–135.

Brenneman, J.C., 1978. Basics of Food Allergy. Charles C. Thomas Publ., Springfield, IL, USA, pp. 170–174.

Chandan, R.C., Attaie, R. and Shahani, K.M., 1992. Nutritional aspects of goat milk and its products. Proc. V. Intl. Conf. on Goats. New Delhi, India. Vol. II. Part I. pp. 399–420.

Collins, R.A., 1962. Goat's milk anemia in retrospect. Am. J. Clin. Nutr., 11: 169–170.

Cornell, R., Walker, W.A. and Isselbacher, K.J., 1971. Small intestinal absorption of horseradish peroxidase: a cytochemical study. Lab. Invest., 25: 42–48.

Coveney, J. and Darnton-Hill, I., 1985. Goat's milk and infant feeding. Med. J. Aust., 143: 508–511.

Davidson, G.P. and Townley, R.R.W., 1977. Structural and functional abnormalities of the small intestine due to nutritional folic acid deficiency in infancy. J. Pediatr., 90: 590–594.

Deamer, W.C., Gerrard, J.W. and Speer, F., 1979. Cow's milk allergy: A critical review. J. Fam. Pract., 9: 223–232.

Devendra, C. and Burns, M., 1970. Goat Production in the Tropics. Commonwealth Bur. Anim. Breeding and Genetics, Tech. Commun. No. 19.

Eastham, E.J. and Walker, W.A., 1977. Effect of cow's milk on the gastrointestinal tract: A persistent dilemma for the pediatrician. Pediatrics, 60: 477–481.

Eastham, E.J. and Walker, W.A., 1979. Adverse effects of milk formula ingestion on the gastrointestinal tract: An update. Gastroenterology, 76: 365–374.

Eastham, E.J., Lichauco, T., Grady, M.I. and Walker, W.A., 1978. Antigenicity of infant formulas: Role of immature intestine on protein permeability. J. Pediatr., 93: 561–564.

Fahmi, A.H., Sirry, I. and Safwat, A., 1956. The size of fat globules and the creaming power of cow, buffalo, sheep and goat milk. Indian J. Dairy Sci., 9: 80–86.

Firer, M.A., Hosking, C.S. and Hill, D.J., 1981. Effect of antigen load on development of milk antibodies in infants allergic to milk. Br. Med. J., 283: 693–696.

Fontaine, J.L. and Navarro, J., 1975. Small intestine biopsy in cow's milk protein allergy. Arch. Dis. Child., 50: 357–360.

Foucard, T., 1985. Development of food allergies with special reference to cow's milk allergy. Pediatrics, 75: 177–181.

Gamble, J.A., Ellis, N.R. and Besley, A.K., 1939. Composition and properties of goat's milk as compared with cow's milk. US Dept. Agric. Tech. Bull. 671, pp. 1–72.

Gerrard, J.W., Machenzie, J.W.A. and Goluboff, N., 1973. Cow's milk allergy: Prevalence and manifestations in an unselected series of newborns. Acta Paediatr. Scand. (Suppl.), 243: 3.

Greenberger, N.J. and Skillman, T.G., 1969. Medium chain triglycerides. Physiologic considerations and clinical implications. N. Engl. J. Med., 280: 1045–1058.

Gryboeld, J.D., Burkle, F. and Hillman, R., 1966. Milk induced colitis in an infant. Pediatrics, 38: 299–302.

György, P., 1934. Beitrag zur Pathogenese der Ziegenmilchanämie. (Contribution to the pathogenesis of goat milk anemia.) Z. Kinderheikld., 56: 1–12.

Haenlein, G.F.W. and Caccese, R., 1984. Goat milk versus cow milk. In: G.F.W. Haenlein and D.L. Ace (Eds.) Extension Goat Handbook. USDA Publ., Washington, DC, E-1, pp. 1–4.

Haenlein, G.F.W., 1992. Role of goat meat and milk in human nutrition. Proc. V. Intl. Conf. Goat. New Delhi, India. Vol. II: Part I, pp. 575–580.

Halpla, T.C., Byrne, W.J. and Ameat, M.E., 1977. Colitis persistent diarrhea and soy protein intolerance. J. Pediatr., 97: 404–407.

Heyman, M., Ducroc, R., Desjeux, J.F. and Morgat, J.L., 1982. Horseradish peroxidase transport across adult rabbit jejunum in vitro. Am. J. Physiol., 242: 558–564.

Heyman, M., Grasset, E., Duroc, R. and Desjeux, J.F., 1988. Antigen absorption by the jejunal epithelium of children with cow's milk allergy. Pediatr. Res., 24: 197–202.

Heyman, M., Andriantsoa, M., Crain-Denoyelle, A.M. and Desjeux, J.F., 1990. Effect of oral and parental sensitization to cow's milk on mucosal permeability in guinea-pigs. Int. Arch. Allergy Appl. Immunol., 92: 242–246.

Heyman, M. and Desjeux, J.F., 1992. Significance of intestinal food protein transport. J. Pediatr. Gastroenterol. Nutr., 15: 48–57.

Husby, S., Hist, A., Teisner, B. and Suehag, S.E., 1990. Infants and children with cow milk allergy/intolerance. Investigation of the uptake of cow milk protein and activation of the complement system. Allergy, 45: 547–551.

Isolauri, E., Gotteland, M., Heyman, M., Pochart, P. and Desjeux, J.F., 1990. Antigen absorption in rabbit bacterial diarrhea (RDEC-1): in vitro modifications. Dig. Dis. Sci., 35: 360–366.

Jenness, R., 1980. Composition and characteristics of goat milk: Review 1968–1979. J. Dairy Sci., 63: 1605–1630.

Juàrez, M. and Ramos, M., 1986. Physico-chemical characteristics of goat milk as distinct from those of cow milk. Intl. Dairy Fed. Bull. No. 202, pp. 54–67.

Juntunen, K. and Ali-Yrkko, S., 1983. Goat's milk for children allergic to cow's milk. Kiel. Milchwirt. Forschungsber., 35: 439–40.

Kalser, M.H., 1971. Medium chain triglycerides. Adv. Intern. Med., 17: 301–22.

Mack, P.B., 1953. A preliminary nutrition study of the value of goat's milk in the diet of children. Yearbook Am. Goat Soc., 1952–1953.

Macy, I.G., Kelly, H.J. and Sloan, R.E., 1953. The composition of milks. National Academy of Sciences, Washington, DC, Publication No. 254, pp. 50.

Marcon-Genty, D., Tomé, D., Kheroua, O., Dumontier, A.M., Heyman, M. and Desjeux, J.F., 1989. Transport of β-lactoglobulin across rabbit ileum in vitro. Am. J. Physiol., 256: G943–8.

Maszewska-Kuzniarz, K. and Sonta-Jakimczyk, D., 1973. Chronic enteropathy in infants due to feeding with cow's milk formulae. Dairy Sci. Abstr., 35: 2567.

May, C.D. and Bock, S.A., 1978. A modern clinical approach to food hypersensitivity: Allergy, 33: 166–188.

Mayrhofer, G. and Spargo, D.J., 1987. Subcellular distribution of class II major histocompatibility antigens in enterocytes of the human and rat small intestine. Immunol. Cell Biol., 67: 251–260.

McClenathan, D.T., and Walker, W.A., 1982. Food allergy. Cow milk and other common culprits. Postgrad. Med., 72: 233–239.

McLaughlan, P., Widdowson, K.J. and Coombs, R.R.A., 1981. Effect of heat on the anaphylactic sensitizing capacity of cow's milk, goat's milk, and various infant formulae fed to guinea pigs. Arch. Dis. Child., 56: 165–171.

Nicol, D.J. and Davis, R.E., 1967. Folate and vitamin B_{12} content of infant milk goods with particular reference to goat's milk. Med. J. Aust., II: 212.

Pahud, J.J., Monti, J.C. and Jost, R., 1985. Allergenicity of whey proteins: Its modification by tryptic in-vitro hydrolysis of the protein. J. Prediat. Gastroenterol. Nutr., 4: 408–413.

Park, Y.W., 1991. Relative buffering capacity of goat milk, cow milk, soy-based infant formulas, and commercial non-prescription antacid drugs. J. Dairy Sci., 74: 3326–3333.

Park, Y.W., 1992. Comparison of buffering components in goat and cow milk. Small Rumin. Res., 8: 75–81.

Park, Y.W. and Chukwu, H.I., 1988. Macro-mineral concentrations in milk of two goat breeds at different stages of lactation. Small Rumin. Res., 1: 157–166.

Park, Y.W. and Chukwu, H.I., 1989. Trace mineral concentrations in goat milk from French-Alpine and Anglo-Nubian breeds during the first five months of lactation. J. Food Composit. Anal., 2: 161–169.

Park, Y.W., Mahoney, A.W. and Hendricks, D.G., 1986. Bioavailability of iron in goat milk compared with cow milk fed to anemic rats. J. Dairy Sci., 69: 2608–15.

Parkash, S. and Jenness, R., 1968. The composition and characteristics of goat's milk: A review. Dairy Sci. Abstr., 30: 67.

Perlman, F., 1977. Food Allergens. Immunological aspects of foods. N. Catsimpoolas (Ed.). AVI Publ. Co., Inc. Westport, CT. pp. 279–316.

Podleski, W.K., 1992. Milk protein sensitivity and lactose intolerance with special reference to goat milk. Proc. V Intl. Conf. Goats. New Delhi, India. Vol. II, Part I. pp. 610–613.

Rapp, D.J., 1981. Practical approach to diagnosis and management of food allergy. In: B.A. Berman and K.F. MacDonnell (Eds.). Differential diagnosis and treatment of Pediatric Allergy. Little, Brown & Co., Boston, MA. pp. 467.

Roberton, D.M., Paganelli, R., Dinwiddie, R. and Levinsky, R.J., 1982. Milk antigen absorption in the preterm and term neonate. Arch. Dis. Child., 57: 369–72.

Rosenblum, A.H. and Rosenblum, P., 1952. Gastrointestinal allergy in infancy. Significance of eosinophiles in the stools. Pediatrics, 9: 311–319.

Saarinen, U.M., Backman, A., Kajosaari, M. and Siimes, M., 1979. Prolonged breast-feeding as prophylaxis for atopic disease. Lancet, ii: 163–166.

Saperstein, S., 1960. Antigenicity of the whey proteins in evaporated cow's milk and whole goat's milk. Ann. Allergy, 18: 765–773.

Saperstein, S., 1974. Immunological problems in milk feeding. In: B.L. Larson and V.R. Smith (Eds.) Lactation: A comprehensive treatise. Vol. III. Academic Press, New York, pp. 257–280.

Scothill, J.F., 1987. Slow food allergic disease. Food Allergy In: Chandra, R.K. (Ed.) Nutrition Research Education Found. St. John's, Newfoundland. pp. 305–310.

Stark, B.A., 1988. Improving the quality of goat milk. Dairy Ind. Intl., 53: 23–25.

Taitz, L.S. and Armitage, B.L., 1984. Goat's milk for infants and children. Br. Med. J., 288: 428–429.

Tantibhedhyangkul, P. and Hashim, S.A., 1975. Medium-chain triglyceride feeding in premature infants: Effect on fat and nitrogen absorption. Pediatrics, 55: 359–370.

Taylor, S.L., 1986. Immunologic and allergic properties of cow's milk proteins in human. J. Food Prot., 49: 239–250.

Van der Horst, R.L., 1976. Foods of infants allergic to cow's milk. S. Afr. Med. J., 5: 927–28.

Walker, V.B., 1965. Therapeutic uses of goat's milk in modern medicine. Br. Goat Society's Yearbook 24–26. pp. 23–26.

Walker, W.A., 1987. Pathology of intestinal uptake and absorption of antigens in food allergy. Ann. Allergy, 59: 7–16.

Watson, P.D., 1931. Variation in the buffer value of herd milk. J. Dairy Sci., 14: 50–58.

Wilson, N.W., Self, T.W. and Hamburger, R.N., 1990. Severe cow's milk induced colitis in an exclusively breast fed neonate. Case report and clinical review of cow milk allergy. Clin. Pediatr. (Phil.), 29: 77–80.

Wood, C.B.S., 1986. How common is food allergy? Acta. Paediatr. Scand., Suppl., 323: 76–83.

Worthington, B.S., Boatman, E.S. and Kenny, G.E., 1974. Intestinal absorption of intact proteins in normal and protein-deficient rats. Am. J. Clin. Nutr., 27: 276–286.

Zeman, F.J., 1982. Clinical Nutrition and Dietetics. Callamore Press, D.C. Health and Co., Lexington, MA, USA. pp. 75.