Lactose digestion and maldigestion: implications for dietary habits in developing countries

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Abstract

Milk is an important source of high quality protein, energy, calcium, potassium, phosphorus and riboflavin. It also has good functional properties and a highly acceptable taste, making milk a good alternative for the nutrition of children and for food programmes in developing countries. However, in some instances it has been advocated that milk or milk based products might not be appropriate for nutritional support, given the high prevalence of lactose intolerance among populations in those countries. After reviewing studies in different regions of the world it is concluded that:

1. Prevalence of lactose maldigestion in children and adults is significantly reduced, to less than half in most studies, when assessed with a dose of milk similar to that normally taken in the diet, as distinct from a large dose of lactose used in the lactose tolerance test.
2. Lactose intolerance as a result of consuming a glass of milk occurs only in a small proportion (about 30% in most studies) of lactose maldigesters. Even this small group can ingest smaller amounts of milk without adverse symptoms and there is good evidence that colonic metabolism in these people adapts to the presence of lactose to reduce or eliminate adverse symptoms.
3. Lactose maldigestion and intolerance in children in developing countries is more likely to exist in association with gastrointestinal damage associated with diarrhoea, malnutrition and/or infection.
4. There is no evidence to support a reduction or elimination of milk intake for people in developing countries because of lactose maldigestion or intolerance.

Introduction

Milk is an important source of nutrients including high quality protein, calcium, potassium, phosphorus, and riboflavin. Milk also has good functional properties, it is heat stable and able to remain in solution in the presence of fats, minerals and water, and it has a highly acceptable taste. These characteristics make milk a good source of nutrition for children and in food programmes, particularly in developing countries. In some instances, however, government officials as well as certain research groups have advocated that milk or milk based products might not be a good alternative for nutritional support given the high prevalence of lactose maldigestion among children in those countries (Cifuentes *et al.* 1985). This concept has found

https://doi.org/10.1079/NRR19970008 Published online by Cambridge University Press

some support based on estimates that up to 70 % of the world's population have a genetically controlled limited ability to digest lactose, the carbohydrate in milk and other dairy products (Johnson, 1981). These figures greatly overestimate the incidence of intolerance to quantities of lactose usually consumed, since most studies on which these estimates are based have been carried out using a 50 g test dose of lactose, corresponding to the carbohydrate content of more than one l milk.

All young mammals and human infants are born with high levels of the enzyme lactase which enables them to digest lactose, except those rare conditions with a congenital defect. Lactase activity declines after weaning in most ethnic groups except for most white populations in Northern Europe and North America so that by approximately five years of age, when the child is consuming a variety of foods, lactase levels are lower than when it was born but not necessarily as low as not to tolerate habitual doses of milk. In fact many recent studies have failed to find an association between lactose maldigestion and the presence of symptoms of intolerance.

Lactose maldigestion can also occur owing to disease states that affect the integrity of the gastrointestinal mucosa. In many areas of developing countries this form of maldigestion may be prevalent, particularly associated with diarrhoea, malnutrition and some parasitic infections such as ascaris or giardia.

Studies of the prevalence of lactose maldigestion in developing countries as well as in developed countries have been extensively reviewed (Flatz, 1983). Scrimshaw & Murray (1988) made a detailed revision of most of the studies that associate the existence of lactose maldigestion with milk consumption. The present paper will concentrate on defining the actual role that lactose maldigestion and milk intolerance has in milk consumption of populations in developing countries and the potential for using milk or milk-based products for supplementary feeding.

Lactose and lactase

Lactose is a key nutrient in mammalian milk, comprising the major carbohydrate source during the neonatal period. From both evolutionary and biological viewpoints, lactose is a unique sugar as it exists as a free molecule only in milk. It is synthesized by lactose synthase (EC 2.4.1.22), exclusively in the mammary gland of virtually all placental mammals (except the sea lion; Kretchmer, 1981) during late pregnancy and lactation. Lactose concentration in milk is inversely related to the content of fat and protein (Newburg & Neubauer, 1995); human milk contains 7 % lactose, which is one of the highest concentrations compared with milk of other species; whole cows' milk contains 4·8 % lactose. Lactose is hydrolysed to glucose and galactose by lactase or, more precisely, by lactose phlorizin hydrolase, an intrinsic microvillus membrane glycoprotein with at least three characteristic enzyme activities: lactase (β-D-galactosidase, EC 3.2.1.23), glycosylceramidase (phlorizin hydrolase; glycosyl-*N*-acylsphingosine glycohydrolase, EC 3.2.1.62) and glucosylceramidase (D-glucosyl-*N*-acylsphingosine glucohydrolase; EC 3.2.1.45) (Jonas *et al.* 1985; Lau, 1987). In contrast to the other disaccharidases, sucrose-α-D-glucohydrolase (sucrase-isomaltase, EC 3.2.1.48) and α-D-glucosidase (maltase-glucoamylase, EC 3.2.1.20) lactase activity is rate limiting in the absorption of lactose (Saavedra & Perman, 1989). The location of the enzyme on the villus-crypt axis, with maximal expression at the upper villus, makes it particularly sensitive to villus injury (Boyle *et al.* 1982). Neither prolonged ingestion of lactose in humans nor exclusion of lactose from the diet influence the capacity of the small intestine to absorb lactose (Gilat *et al.*

1972; Kretchmer *et al.* 1971), strongly suggesting that lactase activity is not induced by its substrate lactose. Activity of the lactase enzyme is highest in the proximal ileum and very low in the first portion of the duodenum and in the terminal ileum (Grand *et al.* 1976). Of all dietary sugars, lactose is hydrolysed the most slowly, occurring at only half the rate of sucrose hydrolysis (Saavedra & Perman, 1989). The rate at which lactose is assimilated depends on the rate of hydrolysis. Lactose hydrolysis rather than glucose or galactose transport is the rate limiting step in its overall process of digestion and absorption (Gray & Santiago, 1969). The relatively slow rate of hydrolysis and the relative lack of reserve of lactase activity helps to explain why many people are vulnerable to lactose maldigestion.

Lactose maldigestion: definition and diagnosis

Much of the confusion in determining the real implications of lactose maldigestion arises because of an indiscriminate use of terms and definitions. A definition of terms is included in Table 1. Lactase deficiency has been used to refer to a reduction in lactase activity without any reference to the aetiology of the reduced activity. The decrease in the activity of the lactase enzyme could be attributed to a primary, genetically programmed, reduction in activity or it could be secondary, due to adverse factors in the gastrointestinal tract that affect the intestinal mucosa and consequently the activity of the enzyme. The logical consequence of a reduction in lactase activity is that a proportion of the lactose that is ingested will remain intact, without digestion in the intestinal lumen, a condition that is known as lactose maldigestion. This term should be used instead of lactase deficiency, because the method of measuring the amount of lactose not digested is less invasive and more commonly used than a direct measurement of enzyme activity which requires an intestinal biopsy.

Lactose maldigestion could be measured by determining the hydrogen concentration in expired air produced as a consequence of the colonic fermentation of the undigested lactose. The hydrogen breath test (HBT) has the advantage of being a non-invasive, highly sensitive tool for the determination of carbohydrate maldigestion. Its application, however, requires a series of considerations.

1. Given the presence of lactase, the capacity to digest lactose is associated directly with the amount of lactose that is ingested; this is a basic principle of enzyme kinetics. Many recent evaluations that utilize the HBT for diagnosing lactose maldigestion (Erinoso *et al.* 1992;

Table 1. Definition of terms

Primary lactase non-persistence. A normal, genetically controlled, age-related decrease in lactase activity
Secondary lactase deficiency. Acquired temporary reduction in lactase activity due to disease or medical conditions affecting the gastrointestinal tract
Congenital lactase deficiency. A rare genetic abnormality in which lactase is very low or absent at birth
Lactose maldigestion. Incomplete hydrolysis of lactose which then escapes into the large intestine
Lactose intolerance. The experience of adverse gastrointestinal symptoms, such as abdominal pain, bloating, flatulence, or diarrhoea following lactose maldigestion
Milk intolerance. The experience of adverse symptoms due to milk consumption

Medow *et al.* 1993; Montes *et al.* 1993) utilized 2 g lactose/kg body weight in small children and 50 g lactose in older children and adults. The application of such very high doses of the carbohydrate, in the pharmacological range, has its basis in the previous studies in which the lactose tolerance test was used to determine lactose maldigestion. The lactose tolerance test has poor sensitivity so that big doses of lactose are required, usually 2 g/kg body weight up to 50 g. Fifty grams of lactose is equivalent to the lactose content in 1·5 l of milk. The diagnosis of lactose maldigestion and/or intolerance with such very high doses does not necessarily reflect the response to amounts of lactose usually ingested. We strongly suggest that whenever there is an interest in knowing the implications of lactose maldigestion and intolerance in the ingestion of milk and dairy products, a maximum dose of lactose of 12 g in children and 18 g in adults should be used. This is the amount of lactose in 240 ml and 360 ml milk respectively.

2. The HBT is based on evaluation of colonic fermentation of the carbohydrate; the capacity and rate of fermentation will depend on the amount and type of microflora in the colon. Thus any event that interferes with colonic fermentation of dietary carbohydrates will modify the result of an HBT. Antibiotics used will kill a proportion of the bacteria in the colon, may underestimate hydrogen production and may also lead to false negative results (Montes & Perman, 1991). When antibiotics have been consumed, at least three weeks may be necessary for recovery of the colonic flora. Acidification of contents by continued delivery of carbohydrates to the colon may depress hydrogen production (Vogelsang *et al.* 1988). Changes in intestinal transit time or in the rate of gastric emptying will modify the rate of hydrogen production, for example the ingestion of other nutrients or other foods with lactose, and modify intestinal transit time and the HBT results (Rosado *et al.* 1992).

3. The concentration of hydrogen in expired air is affected by the amount of air that is being expired; hyperventilation during exercise will reduce the concentration of hydrogen in expired air (Rumessen, 1992) and hypoventilation during sleeping (Perman *et al.* 1984) will increase it. Hypoventilation may also be responsible for high basal hydrogen values which may decrease the sensitivity of the test. All these factors should be considered when carrying on a BHT in children.

4. The rates of fermentation and hydrogen production are not the same for different types of carbohydrate (Levitt *et al.* 1987). This observation limits the quantitative applications of the HBT test, and it also complicates the interpretation of studies in which the HBT has been used to compare fermentation of different carbohydrates.

Lactose intolerance

Lactose intolerance consists of a series of subjective symptoms generally due to the incomplete digestion of lactose. The intact carbohydrate in the intestinal lumen produces an osmotic gradient attracting water, causing diarrhoea in some instances. Fermentation of the carbohydrate produces gas. Thus the undigested lactose may produce symptoms of carbohydrate intolerance such as flatulence, abdominal cramps and diarrhoea. Lactose maldigestion does not always produce lactose intolerance; there are individuals that could be diagnosed as maldigesters and do not have symptoms of intolerance. This lack of correlation between lactose maldigestion and lactose intolerance is specially true when pharmacological doses of lactose are used to determine maldigestion. Intolerance to lactose is dependent on the dose administered; most individuals if they are not sick can tolerate physiological doses of the sugar. This is specially true for children: lactose intolerance in children is mainly associated

with diseases that affect the gastrointestinal tract. The most common factors that produce lactose maldigestion and intolerance are shown in Table 2.

Primary lactase non-persistence

Primary lactase deficiency or lactase non-persistence, as it is more appropriately called, is inherited as a recessive trait, while genetically determined high lactase activity in some groups of the world is inherited as a single autosomal dominant gene (Montgomery *et al.* 1991). As indicated previously, lactase is not induced by the presence of the substrate: administration of lactose to a population with a reduced activity of the enzyme does not elevate lactase-specific activity. In humans, neither prolonged ingestion of lactose nor elimination of lactose from the diet alter reduced lactase activity.

The human lactase gene comprising 17 exons is located on chromosome 2 and covers approximately 55 kb, giving rise to a messenger RNA of slightly more than 6 kb (Boll *et al.* 1991). The translation product is composed, from initiation codon to stop codon, of 1927 amino acids. After glycosylation, this precursor protein of approximately 220 kDA is cleaved, either just before or upon insertion into the microvillus membrane of the enterocyte, yielding a mature protein of 160 kDA (Montgomery *et al.* 1991). No difference in structure of the chromosomal gene and complementary DNAs from humans with lactase persistence and non-persistence has been observed (Boll *et al.* 1991). Therefore the difference in lactase expression could not be attributed to a difference in the structure of the gene itself, supporting the hypothesis that differential gene regulation is responsible for the two phenotypes.

Secondary lactase deficiency

Since lactase is a brush border enzyme, any condition that injures the intestinal mucosa is likely to induce a decrease in lactase activity (Nichols *et al.* 1992; Jiang & Rui-Yun, 1991). Thus, secondary lactase deficiency is the result of mucosal injury. Of the causes of secondary lactose maldigestion listed in Table 2, acute diarrhoea is important, particularly in children in developing countries. Acute diarrhoeal disease is commonly due to infectious organisms which may disrupt the mucosa and produce an inflammatory response. The activities of lactase and

Table 2. Most common aetiology of lactose maldigestion and lactose intolerance in children

Primary lactose maldigestion
Congenital
Primary lactase non-persistence
Secondary lactose maldigestion
Malnutrition
Diarrhoeal disease
Infectious diseases
Parasitosis (giardiasis and ascariasis)
Coeliac sprue
Inflammatory bowel disease
Cows' milk protein intolerance
Recurrent abdominal pain of childhood
Premature infants

other disaccharidases will be decreased in consequence. Lactase activity is more affected than the other brush border enzymes, and several studies have demonstrated increased rates of lactose maldigestion during and for variable periods after acute enteric infections (Villiers, 1995).

Secondary maldigestion of lactose and in some instances of other carbohydrates during gastroenteritis poses a potential problem for the management of diarrhoeal disease of infancy, particularly when diarrhoea is also accompanied by malnutrition. This is the case in regions of the world where the incidence of malnutrition is high.

Previous studies on the effectiveness of including or eliminating lactose from the diet therapy of children with diarrhoea were reviewed by Brown & Lake (1991). Of 20 studies reviewed, 12 studies detected increased severity with the ingestion of milk or lactose and 10 studies detected no difference in severity of the disease. The same group (Brown *et al.* 1994) carried out a meta-analysis of 29 clinical studies and found that the inclusion of lactose in the diet of children with diarrhoea is detrimental only when children were also dehydrated. Other recent studies (Erinoso *et al.* 1992; Gregorio *et al.* 1992; Penny & Brown, 1992) suggest that the need to eliminate lactose from the diet in children with diarrhoeal disease occurs in only a few cases; most children can safely continue receiving milk during acute diarrhoea. In the newborn baby continued breast feeding during diarrhoea results in decreased stool output and is highly recommended even though the mother's milk contains more lactose than cows' milk. It is important to avoid fasting during diarrhoea; the inclusion of other foods such as cereals with milk or fermented foods is highly recommended. Feeding of solutions with a low osmotic gradient, e.g. those prepared with more complex carbohydrates such as rice (Martínez-Salgado *et al.* 1991) or maltodextrin (Santos-Ocampo *et al.* 1993), instead of sucrose or glucose have also been shown to be beneficial during rehydration in acute diarrhoea.

Aside from diarrhoeal disease, there are other factors that affect the intestinal mucosa and have been demonstrated to decrease lactase activity (Table 2). Parasitosis of children with ascaris (Taren *et al.* 1987) and giardia (Tolboom *et al.* 1987) was shown to increase lactose maldigestion; a more recent study in well nourished African children (Gendrel *et al.* 1992) showed a marginal effect of ascaris and no effect of giardia on lactose digestion. Perhaps the overall nutritional status of children is a more important determinant of the capacity to digest lactose. Malnutrition affects the integrity of the intestinal mucosa and reduces lactose digestion capacity. Other clinical conditions that affect lactose digestion include coeliac disease, inflammatory bowel disease, allergy to milk protein, recurrent abdominal pain of childhood and chemotherapy.

Effect of lactose maldigestion and milk intolerance on milk consumption

It is clear from many studies that primary lactose maldigestion has very little effect on lactose intolerance when the dose of the carbohydrate is reduced to amounts commonly found in habitual diets (Scrimshaw & Murray, 1988; Rosado *et al.* 1994). Even so, some subjects who are diagnosed as lactose intolerant are not lactose maldigesters (Rosado *et al.* 1987; Johnson *et al.* 1993; Suarez *et al.* 1995). In a recent study in Finland there was no difference in the mean severity of symptoms between lactose-free milk and lactose-free milk with up to 7 g lactose added in a group of lactose maldigesters (Vesa *et al.* 1996). However, the important question of how lactose maldigestion and lactose intolerance affect dietary habits related to milk consumption has been addressed in only a few studies. This question is specially relevant in many developing countries where, in general, primary lactase deficiency is more prevalent and

where secondary lactose maldigestion due to malnutrition and infections would be expected to be higher. In order to answer this question we studied populations known to differ in their habitual consumption of milk; this is the case for the rural and urban populations in Mexico. The urban population consume 1·5–2·5 times more milk than those in the rural areas, depending on the region (Rosado *et al.* 1994). Table 3 shows the prevalence of lactose maldigestion at different ages from newborn to the elderly. Lactose maldigestion in these studies was defined by breath hydrogen excretion after the ingestion of 240 or 360 ml milk in children less than 8 years old or in older children and adults respectively. The information in the Table shows several important conclusions that could be extrapolated to other developing countries. (1) The prevalence of lactose maldigestion is much lower when it is evaluated with doses of lactose habitually consumed: previous studies in this population have reported that lactose maldigestion was present in 36 % of those 4–5 years old, 58 % of those 6–7 years old, 57 % of those 8–9 years old, 77 % of those 10–11 years old and 74 % of subjects 13–72 years old (Lisker *et al.* 1974; Lisker & Meza-Calix 1976). These figures were obtained after administration of higher doses of lactose. (2) Prevalence of lactose maldigestion is very low (about 5 %) in children less than 4 years old, increasing with age and reaching a plateau after 13 years at values around a 33 % incidence of lactose maldigestion characteristic of adulthood. (3) The existence of lactose maldigestion is not significantly different between rural and urban populations in any of the age groups.

Table 4 shows milk consumption scores of rural and urban populations in the different age groups. Milk consumption was significantly higher in the urban population in all age groups. In fact this was expected from previous information. The score was from 0 when practically no milk was included in the diet to 5 when more than two servings of milk were included daily. From the information in both Tables it can be concluded that lactose maldigestion is not an important factor for milk consumption. Other factors such as cultural belief, buying power, availability or taste may be more important determinants of milk consumption. Few subjects, apparently healthy, perceive symptoms with a glass of milk. These symptoms could have a psychological origin (Rosado *et al.* 1987; Suarez & Levitt, 1996); they could also represent lower adaptation of the colon to metabolize undigested lactose, making these subjects more sensitive to smaller amounts of lactose (Hertzler & Savaiano, 1996), or they could be due to primary or secondary lactose maldigestion. Most investigations in developing countries

Table 3. Prevalence of lactose maldigestion in rural and urban populations. The study included subjects of all age groups

	Rural population			Urban population			Total population		
		Lactose maldigesters			Lactose maldigesters			Lactose maldigesters	
Age Group	*n*	*n*	(%)	*n*	*n*	(%)	*n*	*n*	(%)
0– < 4	42	1	(2·4)	65	5	(7·7)	107	6	(5·6)
4– < 8	122	30	(24·6)	110	13	(11·8)	232	43	(18·5)
8– < 13	118	26	(22·0)	102	24	(23·5)	220	50	(22·7)
13– < 25	94	28	(29·8)	91	33	(36·3)	185	61	(33·0)
25– < 60	49	14	(28·6)	91	29	(31·9)	140	43	(30·7)
60	3	–	–	41	12	(29·3)	44	12	(27·3)
Total	428	99	(23·13)	500	116	(23·2)	928	215	(23·2)

Adapted from Rosado *et al.* (1994), Lopez *et al.* (1996) and Palma *et al.* (1996)

https://doi.org/10.1079/NRR19970008 Published online by Cambridge University Press

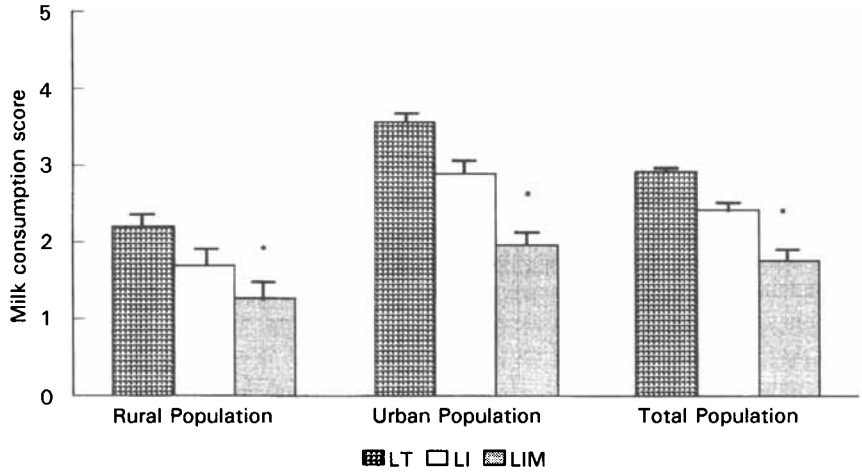

Fig. 1. Effect of the capacity to tolerate lactose on milk consumption by rural and urban individuals. Values are means ± SEM of milk consumption scores in lactose tolerant (LT) ($n = 803$), lactose intolerant with mild symptoms (LI) ($n = 91$) and lactose intolerant with major symptoms (LIM) ($n = 66$). * Significantly different from LT ($P < 0.05$). From Rosado *et al.* (1994) and Palma *et al.* (1996).

studying the prevalence of lactose maldigestion and/or lactose intolerance do not also assess any diseases affecting the gastrointestinal tract, so that cases of lactose maldigestion attributed to a genetic decrease in activity may in fact be due to an affected intestinal mucosa.

Fig. 1 shows how the presence of symptoms after ingestion of 240 or 360 ml milk (milk intolerance) affect milk consumption. The subjects in these studies were divided into those that presented symptoms (LI) and those that presented major symptoms (LIM) (Palma *et al.* 1996). Milk consumption in both rural and urban populations was affected only when major symptoms of intolerance were present; the experience of mild symptoms did not significantly affect milk consumption. The proportion of the population that presented major symptoms of intolerance with one glass of milk (240 ml in individuals of ≤8 years and 360 ml in those > 8 years) and that significantly reduced milk consumption is shown in Table 5. It should be mentioned that even these subjects could tolerate lower amounts of lactose in milk (Tuula *et al.* 1996).

Table 4. Average (± SEM) of milk consumption scores in individuals of different age groups from rural and urban populations

	Rural population		Urban population		Total population	
Age group	*n*	Milk consumption (score)	*n*	Milk consumption (score)	*n*	Milk consumption (score)
0– < 4	42	3·4 ± 0·2	70	4·2 ± 0·1	112	3·9 ± 0·1
4– < 8	123	2·2 ± 0·1	116	3·6 ± 0·1	239	2·9 ± 0·1
8– < 13	121	2·0 ± 0·1	106	3·5 ± 0·1	227	2·7 ± 0·1
13– < 25	95	1·8 ± 0·1	100	2·7 ± 0·1	195	2·3 ± 0·1
25– < 60	49	1·5 ± 0·1	93	2·6 ± 0·2	142	2·2 ± 0·1
60	3	2·3 ± 0·3	42	3·9 ± 0·1	45	3·7 ± 0·1
Total	433	2·1 ± 0·1	527	3·4 ± 0·1	960	2·8 ± 0·1

Adapted from Rosado *et al.* (1994), Lopez *et al.* (1996) and Palma *et al.* (1996)

https://doi.org/10.1079/NRR19970008 Published online by Cambridge University Press

Other aspects of lactose metabolism relevant to populations in developing countries

Effect of nutritional status and diet on lactose in human milk

Lactose is considered to be the most consistent in level of the macronutrients in human milk (Butte *et al.* 1988); as mentioned, human milk contains ~7 % lactose, while cows' milk contains 4·8 %. Lactose in milk is not affected by nutritional status. Milk composition of poorly nourished women has been reviewed (Jelliffe & Jelliffe, 1978; WHO, 1985). One study found that lactose levels of a 65-year-old malnourished woman were within the normal range (Gindler *et al.* 1985). Some investigations found significantly elevated lactose levels in poorly nourished women (Ojofeitimi *et al.* 1983; Van Steenbergen *et al.* 1983; Gindler *et al.* 1987) while others (Khin-Maung-Naing *et al.* 1980) found no such difference. Lactose content of milk from undernourished women studied in a metabolic ward was not correlated with maternal weight, arm circumference, or triceps skinfold measurements (Brown *et al.* 1986). Race did not affect lactose concentration in milk (Prinsloo *et al.* 1970).

In some circumstances, lactose concentration has been altered by dietary manipulation. Reduction in calorie intake of well nourished breast feeders did not influence milk lactose concentration (Strode *et al.* 1986) nor did protein supplementation of poorly nourished women (Deb & Cama, 1962). However, lactose decreased significantly in fasting Gambian women, perhaps as a result of formation of a paracellular pathway (Prentice *et al.* 1984). Also energy supplementation of Gambian nursing women whose dietary intakes were below recommended levels resulted in a significant decrease in lactose concentration (Prentice *et al.* 1983). Increasing carbohydrate intake from 35 to 65 % of the diet, with consequent lowering of fat intake from 50 to 15 %, resulted in significantly less lactose in milk (Harzer *et al.* 1984). A vegetarian diet did not affect lactose concentration in milk (Finley *et al.* 1985; Dagnelie *et al.* 1992).

It is expected that lactose in milk of mothers in developing countries will be constant and independent of nutritional status and diet. The relatively high content of lactose in human milk and its incomplete digestion in the newborn could be a protective factor against gastrointestinal infections. *Lactobacillus*, the predominant intestinal flora in the newborn, competes for colonization against *Escherichia coli* of which some types are pathogenic (Solomons, 1996). Undigested lactose could reach the colon and stimulate *Lactobacillus* proliferation (MacLean & Fink, 1980).

Lactase appears very late in fetal development. It is estimated that at 35–38 weeks of gestation lactase concentrations are ~70 % of full-term level, and full-term lactase levels are 2–4 times those found in infants 2–11 months of age (Antonowicz & Lebenthal, 1977). Despite the higher lactase levels in the newborn, the fact that some lactose from human milk is undigested in the infant is indicated by the rise in breath hydrogen excretion after breast feeding found in some studies (MacLean & Fink, 1980; Lifshitz *et al.* 1983).

Nutrient supplementation in vulnerable populations

Milk can provide an inexpensive source of several nutrients such as carbohydrate, protein, calcium and riboflavin to populations where there is a need for supplementary feeding. Many developing countries with a high prevalence of malnutrition also have a high incidence of lactose maldigestion. The evidence however, suggests that individuals in poor regions of the world can tolerate amounts of milk within the physiological range (i.e. habitual amounts such

Table 5. Population with major symptoms of intolerance that significantly reduce milk consumption

Age group	*n*	Population with major symptoms of intolerance	
		n	(%)
0– < 4	112	0	0
4– < 8	239	12	(5·0)
8– < 13	227	13	(5·7)
13– < 25	195	18	(9·2)
25– < 60	142	17	(12·0)
60≤	45	6	(13·3)

Adapted from Palma *et al.* (1996)

as one glass) without any adverse effects. In a study involving Gambian children, lactose maldigestion was not associated with growth failure, milk consumption was common among the children and was rarely associated with adverse effects (Erinoso *et al.* 1992). The authors recommended that cows' milk be given to Gambian children as a means of supplementing their diet. Even for nutritional rehabilitation the elimination of milk or the use of lactose-free milk has not shown any additional advantage. In a study involving Guatemalan preschool children with protein-energy malnutrition, recovery after a 45 d period of treatment was similar with lactose-hydrolysed milk than with lactose-containing milk. There was no difference between the two dietary treatments in rates of growth, body protein repletion, restoration of energy reserves, or intestinal function (Solomons *et al.* 1984). Similar conclusions can be drawn from studies in other countries with children recovering from malnutrition and diarrhoea (Lozano & Céspedes, 1994; Beau *et al.* 1990; Villiers, 1995) and in malnourished adults (O'Keefe *et al.* 1990).

Given the nutritional quality, price and functional properties of milk, we have developed a good-tasting milk-based formula with some micronutrients added as a means of nutrient supplementation in children 5–24 months of age and pregnant and lactating women (unpublished information). The product is being distributed as an important part of a National programme in Mexico in which about 2·5 million people will receive the supplement every day.

Conclusions

1. Prevalence of lactose maldigestion in children and adults is significantly reduced, to less than half in most studies, when assessed with an amount of milk which is habitually included in the diet.

2. Lactose intolerance when a glass of milk is consumed occurs in only a limited proportion of lactose maldigesters (around 30 % of maldigesters in most studies); but even this group can ingest smaller amounts of milk lactose. In addition there is the important possibility that this group of people could adapt colonic metabolism to cope with increased doses of lactose.

3. Lactose maldigestion and intolerance in children in developing countries is more likely to exist associated with a deterioration of the gastrointestinal tract due to diarrhoea, malnutrition and/or infection.

4. There is no scientific evidence to support the reduction or elimination of milk intake in populations in developing countries owing to lactose maldigestion or lactose intolerance.

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