LACTOSE INTOLERANCE

by

JANICE O. HEATON

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Approved by:

Beth Fryer

Major Professor

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INTRODUCTION

Milk has always been the major food of the infant mammal. During the last 6000 years it has served as part of the diet beyond infancy and into adulthood. Humans first had the opportunity to obtain milk regularly as adults after herd animals were domesticated about 9000 B.C. However, dairying did not begin until about 4000 to 3000 B.C. Before this time, infants consumed the milk of their mothers from the time of their birth until weaning. After weaning, milk ceased to be included in the individual's diet. Today, milk is a major dietary constituent throughout life for individuals in the western world (1). Increased utilization of milk and milk products is recommended for all other parts of the world.

Lactose is the carbohydrate found in milk and must be broken down in the digestive tract into galactose and glucose before absorption can take place. Although there are several enzymes with lactase activity present in the human digestive tract, an enzyme referred to as neutral lactase is primarily responsible for the hydrolysis of lactose. Inadequate enzymatic digestion of lactose results in lactose malabsorption and intolerance. Lactose malabsorption usually refers to an abnormally small rise in blood glucose following an oral lactose load. When clinical symptoms, such as diarrhea or abdominal cramping occur in a person with lactose malabsorption, the condition is usually referred to as lactose intolerance (2). Intolerance may be influenced by the level of lactose ingested, gastrointestinal motility, intestinal length, and biochemical capacities of the intestinal flora, as well as the level of intestinal lactase (3). Therefore, not everyone with low intestinal lactase levels

has clinical symptoms after ingesting a moderate amount of lactose, such as that contained in one eight-ounce glass of milk (12 grams lactose) (4). Low lactase levels cause problems in all age groups, but clinical symptoms begin to appear in early childhood (age 3-5 years) and increase with increasing age.

Lactose intolerance may constitute a problem to a majority of the world's population. Prevalence of lactose intolerance is low among Caucasians of Scandinavian or West European extraction, with only 2-8% being intolerant. Non-Caucasians have a much higher incidence of intolerance, affecting from 60 to 90% of adults (5).

The purposes of this paper are a) to review the incidence of lactose intolerance, b) to discuss its causes, and c) to review methods of treatment.

NORMAL ABSORPTION AND DIGESTION OF LACTOSE

When lactose is ingested as a part of whole milk, milk fat is important in delaying gastric emptying. Therefore, under most dietary conditions, large amounts of lactose do not reach the small intestine at one time (6).

All of the dietary disaccharides, including lactose, arrive intact in the small intestine where they are hydrolyzed by disaccaridases. Dissaccharidase activity has been identified by electron microscopy and by chemical and immunofluorescent techniques to be localized in the brush border (microvilli) of the mucosal cells (7,8). Disaccharides are hydrolyzed within the brush border at a site from which diffusion and transport of the products into the cell is easier than diffusion back into the lumen (9).

After ingestion, lactose may be absorbed in small amounts without hydrolysis and then may appear in the urine. However, most lactose is

enzymatically hydrolyzed in the epithelial cells of the small intestine into its constituent monosaccharides, glucose and galactose (8).

Lactose is a β-galactoside and there are at least three enzymes with β-galactosidase activity in the human small intestine (10). The enzyme thought to be principally or totally responsible for the hydrolysis of lactose is referred to as neutral lactase. It is associated with the small intestinal epithelial cells and is most active in the 5.5-6.0 pH range. Although it will hydrolyze various synthetic β-galactosides and cellobiose, it most active toward lactose. Neutral lactase has its highest specific activity in the jejunum, but very low activity in the duodenum and ileum.

A second β-galactosidase with a pH optimum of 3.5-4.5 is concentrated in the lysosomes, or possibly in the cellular cytoplasm of the intestinal cells. It is most active toward synthetic β-galactosides. Its activity is highest in the ileum. This acid β-galactosidase does not participate in the ordinary digestion of dietary lactose (1,11).

A neutral pH is optimal for a third β-galactosidase. Also located in the small intestine, it is most active in the jejunum. It is active toward several synthetic β-galactosides and β-glucosides, but does not hydrolyze lactose. In fact, its enzymatic activity is partially inhibited by lactose (1, 10).

Concentration of disaccharidases in the intestinal mucosa is the rate-limiting step in disaccharide digestion (11). The level of lactase activity in the human is lower than that of any of the other disaccharidases (6).

All of the carbohydrates are absorbed in the form of monosaccharides. Monosaccharides are actively transported at a rate of about 1 gram

glucose per minute. The order of preference for the transport of the various monosaccharides and their rates of transport in relation to glucose is: galactose 1.1, glucose 1.0, fructose 0.4, mannose 0.2, xylose 0.15, and arabinose 0.1. Some coupled reaction between sodium transport and glucose transport provides the energy for moving glucose through the intestinal wall. The digested products are then immediately absorbed into the portal blood (12).

The lactase activity of the cells of the upper small intestine is usually adequate for the hydrolysis of all of the lactose in the average diet (9). An elevated concentration of lactose in the intestine will result in: 1) hydrolysis of a portion of the lactose 2) passive diffusion of a portion of the remaining lactose intact across the intestinal mucosa, finally appearing in the urine 3) passage of the remaining lactose into the large bowel 4) fermentation by bacteria of lactose in the large bowel and 5) diarrhea (13).

In the human, lactase activity is detected as early as the third month of gestation, but continues to increase throughout gestation, reaching a maximum only at the end of normal gestation. In a number of animals (such as the rat, rabbit, cat, and dog), the curve resulting from plotting age vs. lactase activity is similar. An example of a curve of this nature is seen in figure 1 (1,14,15). There are slight differences in the time at which intestinal lactase activity reaches a maximum, however the general shape of the curve for these different species is very similar (1). Maximal activity is observed in the perinatal period, followed by a decrease in activity, so that the lowest values are obtained at the time of weaning. The slight differences in

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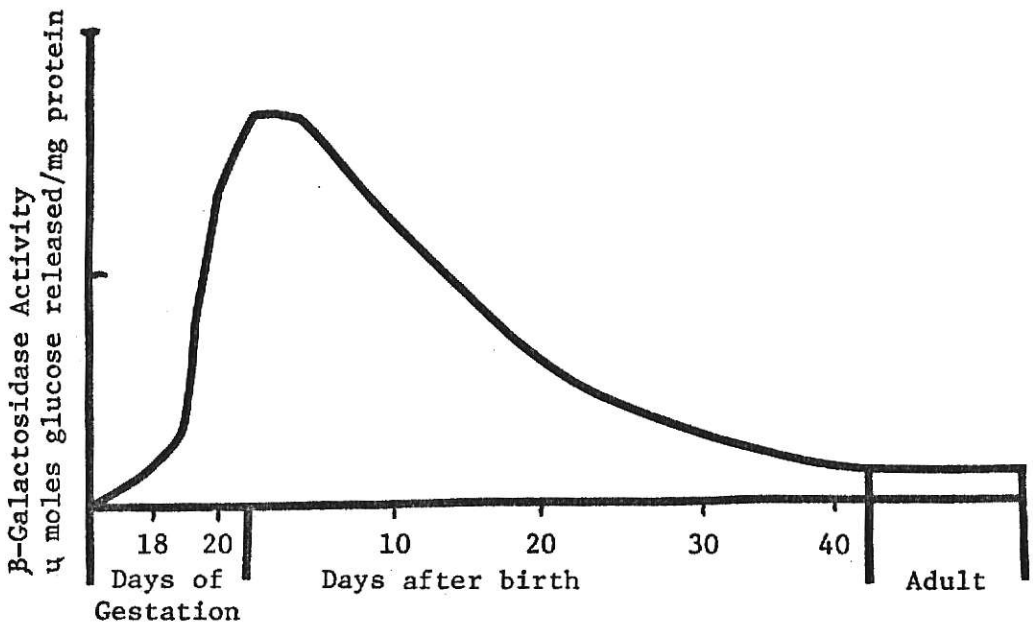

Fig. 1 β-Galactosidase activity in homogenates from the intestine of the developing rat (14).

the patterns from various mammals seems to depend on the state of the maturity of the species at the time of birth. For example, the guinea pig is born relatively mature and feeds on solid food from the time of birth. In this case, the activity of lactase declines only slightly following birth. In the rat, an animal born relatively immature, lactase activity appears late in gestation, reaches a peak at the age of 2-3 days, and remains high until weaning begins at about 14 days of age. By the 21st day, the time of complete weaning, the lactase activity has dropped to the low level found in the adult animal (1). It has been suggested that the same general pattern of lactase activity occurs in humans. The activity of lactase in the brush border of the mucosal cells of premature infants who have survived beyond twenty-four hours is greater than that in premature infants dying before twenty-four hours. This increased activity was not related to feedings (7).

LACTOSE INTOLERANCE

Physiology of lactose intolerance

When lactose is not hydrolyzed and absorbed properly, a small amount of the undigested lactose may diffuse across the intestinal mucosa and be excreted unmetabolized in the urine, causing lactosuria. The remainder of the unabsorbed sugar remains in the intestinal lumen, causing the movement of water and NaCl into the lumen until equilibrium is established between the intestinal contents and the extracellular fluid. The greater fluid load stimulates peristalsis and results in rapid transit time. As the undigested lactose passes into the terminal ileum and colon,

part of it is metabolized by bacteria to lactic and acetic acids and hydrogen gas. The organic acids, by exerting a further osmotic effect, may impair the reabsorption of water and electrolytes (1).

Clinical manifestations of lactose intolerance include watery, fermentative acid diarrhea, often accompanied by abdominal bloating and cramping pain. Symptoms usually occur within a few hours after the ingestion of milk and are related to the amount of milk ingested (6). Occasionally, nausea and vomiting will be manifested, but this occurs most often in infants. In infants with lactose malabsorption and intolerance, there is failure to thrive and even malnutrition if there is continued or prolonged ingestion of lactose (1).

Diarrhea is probably the most prevalent symptom of lactose intolerance. The association between the ingestion of lactose and the occurrence of diarrhea was recognized in the early 1900's by the pediatrician, Abraham Jocobi (1). The pathogenesis of diarrhea is summarized in figure 2. The speed of gastric emptying and individual motility may influence an individual response to lactose. It is possible that the other constituents in milk, such as protein, will affect gastric emptying and intestinal motility.

Symptoms occurring when a disaccharide is not digested completely (1,7) include:

1) fermentative diarrhea with pH of fresh stools less than 5.5. Acid stools result from the bacterial metabolism of lactose to organic acids.

2) watery, stringy stools with an acid odor. These stools contain an increased amount of lactic and other organic acids as well as the nonabsorbed sugar.

3) abdominal distention, irritability, and failure to thrive.

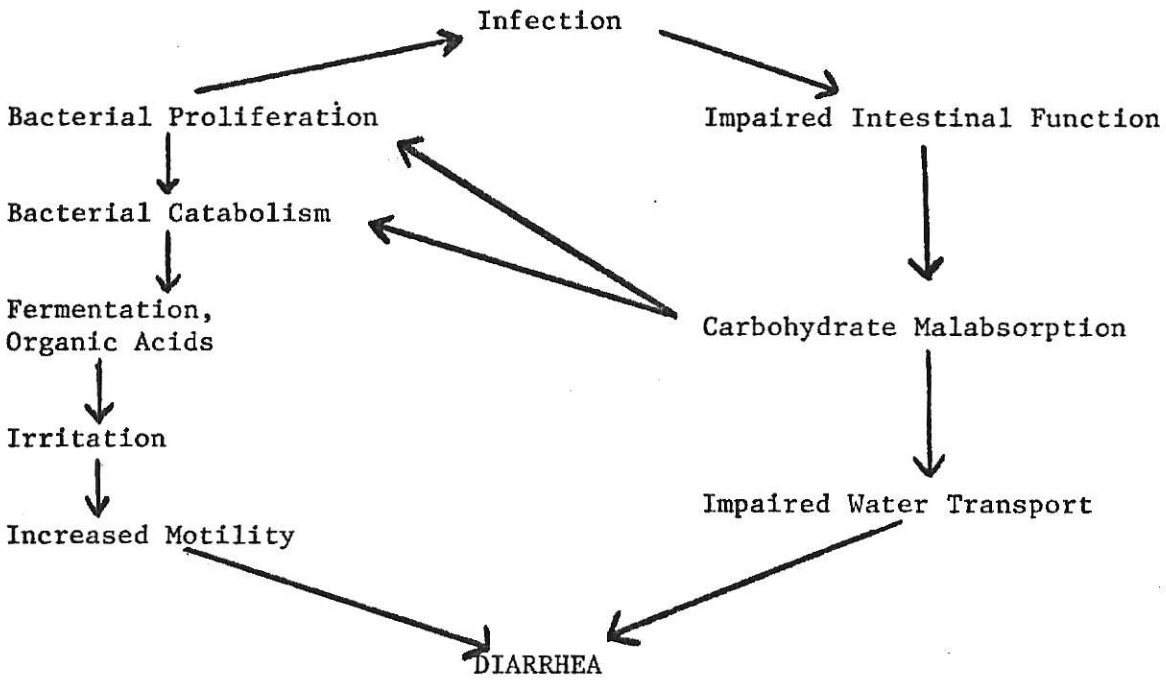

Fig. 2 The pathogenesis of diarrhea (16).

4) melliturıa may or may not be present

5) severe symptoms occurring after the ingestion of the specific carbohydrate which is not tolerated.

6) recovery following the removal of the offending sugar.

Diagnosis

The basis for diagnosis of lactose intolerance is the appearance of symptoms after lactose ingestion and their disappearance with the removal of lactose. Determination of this phenomenon is made by evaluation of a detailed history, or by results of a lactose tolerance test. Other methods have also been developed to aid in diagnosis.

History. Lactose malabsorption may be present when there is a history of diarrhea with onset in early infancy, especially when abdominal symptoms and diarrhea occur 30-60 minutes following the ingestion of milk or milk products (1, 17). Food allergy, hypermotility, and other apparent causes of diarrhea should be excluded before lactose malabsorption is considered (6).

Lactose Tolerance Tests. Lactose tolerance tests measure the rise in blood glucose after oral administration of lactose. A standard dose of lactose (2 mg/kg body weight for children, and 50-100 gm for adults) is given to fasting subjects who have been free from diarrhea for several days. Capillary blood glucose is determined at 1, 15, 30, 60, and 90 minutes after lactose ingestion. Blood glucose levels are frequently plotted against time, producing a "lactose tolerance curve." A rise in blood glucose over the fasting level of less than 20 mg/100 ml plus the occurrence of symptoms constitutes evidence of lactose malabsorption

and results in a flat lactose tolerance curve (1).

Gudmand-Hoyer and Jarnum (18) found that the relationship between jejunal lactase activity and the results of an oral lactose test was very good. They concluded that an oral lactose test is sufficient for the diagnosis of lactose malabsorption. Another investigator (19) stated that the lactose tolerance test is more valuable if compared to a similar test where an equivalent amount of glucose and galactose is administered in place of lactose.

Isokoski et al. (20) proposed a simpler screening method for lactose malabsorption, a one-point lactose tolerance test. Alcohol and lactose are given orally and blood galactose is determined forty minutes later. Sensitivity to this test was found to be approximately 96-100%. It gives results comparable to the four-point test and better than simply observing symptoms after an oral lactose load.

There are several pitfalls to the interpretation of lactose tolerance tests, however. A positive or abnormal test (blood sugar rise less than 20 mg/100 ml) may occur in subjects with normal lactase activity. A false positive test may be obtained when delayed gastric emptying is present or in the presence of monosaccharide malabsorption. The peak rise in glucose usually occurs between 30 and 90 minutes after oral lactose is ingested, but in a significant number of patients, the peak rise will occur at fifteen minutes. Omission of the fifteen minute time point can result in missing the peak blood sugar rise. A false negative or normal test result (blood glucose level rise greater than 20 mg/100 ml) can be obtained in a diabetic subject with true lactose malabsorption (1). Rossiter et al. (21) suggested that the absorption of an individual food

constituent and its uptake by the tissues is influenced by the presence of other food conditions. Therefore, the tolerance test of the individual nutrient, lactose, may not be a true test of the physiological state of the individual. If these limitations are kept in mind, there is generally a very good correlation between results of the lactose tolerance test and lactase activity as determined by examination of intestinal biopsy samples.

Biopsy. The intestinal biopsy also is frequently used to diagnose lactose malabsorption and is considered the most reliable method (1). Alpers and Isselbacher (6) suggest that a biopsy anywhere in the jejunum should be adequate to separate normal from lactase-deficient patients.

The lower limit for normal lactase activity ranges from 0.5 to 2.0 units lactase per gram of mucosa (1). A sucrase:lactase ratio can also be determined with a ratio of 4:1 or less present in normal individuals. An intestinal biopsy also enables one to determine whether the morphology of the intestine is normal or abnormal. The intestinal biopsy can, however, be hazardous in the case of small children and should usually be done only if absolutely necessary.

Fecal Examination. Fecal pH and lactic acid or sugar content of the feces following the ingestion of a lactose-containing diet may confirm the diagnosis of lactose malabsorption. If the concentration of reducing substances in the stool is greater than 0.25% and/or the pH is less than 6, the patient is considered to have lactose malabsorption (1,6,22). Fecal pH is more useful in children than in adults with lactose malabsorption. However, fecal pH also can be lowered by diarrhea unrelated to lactase deficiency.

Radiological Observation. Another helpful diagnostic tool is radiological observation, or a small bowel X-ray. Barium and lactose are ingested and the absorption pattern followed by the X-ray (6).

Lactose Loading. Alpers and Isselbacher (6) suggested that a "lactose loading test" could be utilized. Increasingly higher doses of lactose are administered orally to see which dose produces liquid stools. Normal subjects will tolerate forty grams of lactose well. This method takes many days to complete. Paige et al. (23) reported that in the lactose malabsorber, there is little difference in the ability to tolerate a lactose load when small or large doses are ingested. Symptoms of intolerance were present with 0.5 grams of lactose per kg body weight, but were more forceful and appeared earlier with 2.0 grams lactose per kg.

Expired Air. Examination of respired air after ingestion of a standard dose of lactose is still another method of diagnosis. Hansen (19) suggested that measuring the appearance of CO_2 derived from labeled lactose in the respired air could be helpful. Calloway et al. (24) utilized the concentration of hydrogen in the expired air to indicate lactose malabsorption. All hydrogen comes from fermentable substrates in the colon. Therefore, breath hydrogen accurately reflects lumen hydrogen (6). The peak of hydrogen expiration usually occurs about 5-6 hours after ingestion of lactose if the patient is lactose intolerant. A mean of the five highest hydrogen values recorded after a meal of ten grams milk is used to determine tolerance. An average of 30 ppm or above indicates intolerance to lactose, while an average of 20 ppm or less is normal. If the average hydrogen value falls between 20 and 30 ppm, the

test should be repeated, using a higher dose of lactose (24).

Etiology of Lactose Intolerance

There are four likely causes of decreased lactase activity and intolerance. Intolerance could be 1) related to disease, 2) part of normal development, 3) the result of hereditary factors, or 4) secondary to a lack of stimulation by the substrate, lactose (25).

Disease. Any disease affecting the small intestinal mucosa can lead to disaccharidase deficiency, especially of lactase (6). In any of a variety of situations, disaccharidases may be obliterated in cells as they migrate to the villi or may be lost as the cells containing them are rapidly lost into the intestinal lumen. Lactase seems to be the most sensitive of the disaccharidases to any stimulus that depresses disaccharidase activities (26).

Normal Development and the Aging Process. Often the cause of decreased lactase activity is attributed to normal development and the aging process. Infants usually have an adequate supply of lactase until the time of weaning. Lactase levels decrease soon after weaning.

Huang and Bayless (27) found that lactose-induced symptoms in Negro children and adults increased in frequency with increase in age. A gradual decrease in lactase activity began soon after weaning. This was more common in Negroes than in the whites.

In 98 Singapore subjects ranging in age from 1-42 years, Bolin et al. (28) found that after four years, there is increasing intolerance with increasing age (Table 1)

Hereditary Factors. After studying a group of Nigerian families,

TABLE 1

Relationship of age to lactose tolerance (28)

Age groups	Number of subjects	% of subjects lactose-tolerant
1-3	11	64
3-5	14	79
5-7	14	50
7-9	17	30
9-15	14	7
15-42	22	0

Ransome-Kuti et al. (29) found that if both parents were unable to digest lactose, all of their children were also unable to digest it. If one parent, usually of Northern European origin, could digest lactose, some or all of their children were able to digest the sugar. They concluded that the ability to digest lactose is transmitted as an autosomal dominant trait and represents a mutated gene or a polymorphism.

Desai et al. (30) suggested that hereditary factors were chiefly responsible for a high incidence of lactase deviciency in populations from India and Africa. Keusch et al. (31) studied 172 normal Thai infants and children. They suggested that one possible genetic factor could be that the enzyme, lactase, may be chemically or structurally different from that in Caucasians and that this somehow results in the early loss of lactase activity in the Thai. Many other studies contradict these results. As of yet, the genetic possibilities associated with lactose malabsorption have not been fully explored.

Lack of Stimulation by Substrate. It has been hypothesized that

the continued presence of lactase activity in humans after weaning may be the result of adequate utilization of the milk supply. This is one explanation for the life-long presence of lactase in most Western European adults and in those ethnically derived from Europe (32).

Cain et al. (33) found that lactase and cellobiase activity were stimulated significantly when rats were given increasing doses of lactose in the diet. Bolin et al. (34) found that a decrease in lactase activity occurred when lactose was withdrawn from the diet of rats. When lactose was reintroduced, lactase activity increased. The time taken for these adaptive responses varied between five and ten weeks. The changes in jejunal lactase activity were accompanied by similar alterations in both sucrase and maltase activities.

Bolin et al. (28) hypothesized that if intolerance were due to decreased lactose intake, intolerance should appear at an earlier age in non-milk drinkers than in milk drinkers. This was found to be true in their Singapore subjects (Table 2). Thirteen percent of milk-drinkers were intolerant of milk at five years of age, while sixty-four percent of non-milk drinkers were intolerant at that age.

TABLE 2

Classification of subjects as lactose-tolerant or intolerant and the relation to milk-drinking habits (28)

Subjects	Lactose tolerant		Lactose intolerant	
	Number	%	Number	%
Milk-drinkers	21	75	11	39
Non-milk drinkers	7	25	17	61
Total	28	100	28	100

Individuals with lactose malabsorption may develop the ability to "tolerate" lactose after prolonged ingestion without developing diarrhea or symptoms. This is due, perhaps, to a change in intestinal flora. Gilat et al. (35) studied ten lactase deficient volunteers who drank gradually increasing amounts of milk several times a day, up to at least one liter per day. This amount was consumed for 6-14 months. After an initial adjustment period, the patients tolerated the increasing amounts of milk well. Individuals who develop this ability, however, continue to have low levels of lactase and there is no increase in blood glucose following lactose administration. The tolerance of lactose in those individuals with low levels of intestinal lactase also depends on the amount of lactose ingested (1).

Other researchers have found that lactase activity in no way depends upon the amount of lactose ingested. Sriratanaban et al. (36) used litters of rats maintained on control (8% dextrose) or lactose enriched (8% lactose) diets. The 8% carbohydrate level was used because it supplies the same number of calories as the lactose in the maternal milk of rats. A decrease in intestinal lactase in rats on both diets made it appear that, in the rat, post-weaning decline of intestinal lactase activity was <u>not</u> a result of a decrease in dietary substrate.

Gilat et al. (37) divided a group of 76 children aged 2 1/2 to 16 years into groups according to daily intake of milk and other lactose-containing foods. The groups were: low consumption (less than 200 ml), regular consumption (200-400 ml), and high consumption (more than 400 ml). Lactose tolerance tests were performed on all children. Lactose intolerance appeared after age 6 1/2 in all groups, with the proportion

of lactose-intolerant children not differing significantly among the various groups. The researchers concluded that lactose tolerance is not affected by the amount of lactose consumed during childhood. Lactase was therefore thought to be a non-adaptable enzyme (unlike sucrase and maltase) even in childhood (25,37).

Kogut et al. (38) examined ten patients with galactosemia who had excluded lactose from their diet since early infancy. These ten patients and six control patients were given oral lactose loads. Nine of the ten patients with galactosemia were able to hydrolyze the lactose. The results suggest that intestinal lactase activity does not appear to be dependent upon lactose intake.

Total lactose abstinence for forty-two days by seven subjects did not induce any deficiencies of intestinal lactase or clinical lactose intolerance during a study conducted by Knudsen et al. (39). They concluded that dietary lactose does not appear to be an important factor in determining the level of intestinal lactase in humans for this period of time (42 days). Paige et al. (40) studied Peruvian children and found that the youngster's early nutritional experience (such as nutritional reinforcement with milk, or deficiencies) had no effect on the lactose tolerance of the child. Rosensweig and Herman (41) also found that lactose feeding does not specifically alter lactase activity. They did find, however, that fasting will lower all disaccharidase activity. Carbohydrates specifically, not just calories, are needed to return this activity to normal.

Delay of the symptoms of lactose intolerance has been found to be furthered by lactose feeding. Jones & Latham (42) performed lactose

tolerance tests on 34 children of African, Asian, or Latin American origin who normally consumed at least 1.5 cups of milk per day. They speculated that the expected fall in intestinal lactase activity after weaning may be delayed in children who continue to have high levels of milk intake. Thus, maintenance of adequate nutritional status by continued milk ingestion and avoidance of intestinal damage during early childhood is thought by some to delay the onset of inadequate lactose digestion (43).

Types of lactose intolerance

The two basic types of lactose intolerance are congenital and acquired. The congenital form is usually specific, affecting one enzyme or function. The acquired forms tend to show a more unspecific involvement of several processes (22,44).

Congenital. The only positively identified congenital lactose intolerance is a congenital lactase deficiency. Intestinal lactase activity is totally absent and remains so throughout life. Onset is with milk feeding during the first week. Severe acid fermentative diarrhea and mild-to-moderate vomiting is present. There is an overall failure of the infant to thrive. When the infant is placed on a lactose-free diet, symptoms rapidly disappear. Symptoms tend to grow milder with time (6,22,26). This rare condition was originally described by Holzel et al. in 1959 (45). By 1967, Holzel was able to tabulate only eighteen additional cases since his original description (1). Lifshitz (46) described two patients with a congenital lactase deficiency. Other associated disaccharidase deficiencies also occurred, but were thought

to be caused by lactose-induced diarrhea.

Acquired. Acquired forms of lactose intolerance are far more prevalent than the congenital form. One acquired form is characterized by lactosuria and is often referred to as severe infantile lactose intolerance (22). Onset is within the first month of life, generally as soon as milk feeding is well established. Intolerance is more severe in the breast-fed baby and in the artificially fed baby whose formula is augmented with lactose (17). Vomiting is a more prominent symptom than diarrhea and dehydration develops quickly. There are frequently signs of renal involvement including renal acidosis and aminoaciduria. Lactosuria may be severe and is occasionally accompanied by sucrosuria and glucosuria. Haemorrhagic manifestations have been observed (6). Infants suffering from this condition are restless and irritable and cry a lot because of hunger and intestinal colic. Commonly used formulas do not satisfy them as they lose approximately 40% of the calories from vomiting and malabsorption (17). Prompt recovery follows a removal of lactose from the diet. A return to less than 1% of lactose in the food can lead to death in severe cases. Nearly half of the severe cases reported in the literature have died. In survivors, spontaneous recovery occurs after 7-18 months, after which lactose can be tolerated without any discomfort or ill health. The cause of this unusual disorder may be a generalized membrane defect (6,17,22).

A second type of acquired lactose intolerance is referred to as primary lactase deficiency. Lactase levels are only about 10% of normal. Milk usually is tolerated in early childhood but symptoms seem to begin around the age of three years (6). This is the period of life when

ability to hydrolyze lactose varies from one individual to another (1). There is a complete, or nearly complete, lack of small-intestinal lactase activity (22). Sunshine & Kretchmer (47) examined six intolerant infants and found that clinical symptoms (diarrhea, vomiting, failure to gain weight) of the patients with primary lactose intolerance were identical to those of patients with congenital absence of intestinal lactase.

Secondary lactose intolerance is a third type of acquired intolerance and is presumed to be caused by intestinal mucosal damage induced by some primary cause, with secondary loss of the enzyme activity. This condition is usually associated with 1) infectious or non-specific diarrhea in infancy, 2) malnutrition in infancy, 3) gluten-induced enteropathy, and 4) a wide variety of conditions that cause abnormal intestinal morphology (1). In milk-drinking young children, gastroenteritis may produce secondary complications, one of which is faulty disaccharide absorption and a subsequent unavailability of sugar (19). The clinical features of children with lactose intolerance due to secondary lactase deficiency are those of the underlying disorder plus manifestations similar to those of a primary lactase deficiency (3).

INCIDENCE

Colostrum, the earliest secretion of the mammary glands during lactation, is the first food naturally ingested by the newborn infant who is breast fed. Colostrum is progressively diluted with newly secreted milk for five to fourteen days until mature milk is produced. The change from colostrum to mature milk results in increasingly higher intakes of lactose. The rise in lactose content continues for several weeks following the secretion of mature milk (48).

Breast milk has a high lactose content (7% or 70 g/1), which accounts for approximately 40-45% of the total caloric intake of the infant. Therefore, during the first weeks of life, an infant may digest as much as 8-9 grams of lactose per feeding or 50-60 grams per day.

Cow's milk contains less lactose than human milk (4-5% or 40-50 g/1). It usually is diluted, and is supplemented with dextrins, maltose, or sucrose when used in infant formulas (6,7,11,15).

Low levels of lactase seem to be the norm in most population groups (49). Cuatrecasas et al. (50) examined sixty hospitalized Caucasian adult subjects and found 55% were intolerant to lactose due to a deficiency of jejunal B-galactosidase. Bayless and Huang (51) studied five otherwise healthy school-aged children (6-13 years old) with episodes of abdominal pain. They experienced symptoms after a lactose tolerance test, but symptoms ceased after they were placed on a diet low in milk products. None had complained of diarrhea and all had been able to drink milk as infants. Bayless and Huang also found that many individuals with lactose intolerance will have symptoms if they consume one or two glasses of milk at one time. This is equivalent to twelve to twenty-five grams of lactose.

In 1975, Lebenthal et al. (52) studied 172 individuals ranging in age from six weeks to fifty years. They found that after age five, the groups could be divided into two groups. One group, comprising approximately 24.6% of the population had low lactase activity and exhibited clinical intolerance after consuming small amounts of milk. The second group retained the same mean value for lactase activity as they had in their first three years of life. Individuals in this group

consumed as much as one quart of milk per day with no discomfort. It was concluded that low lactase activity in the Caucasian population may make its appearance around five years of age.

Healthy individuals of many ethnic groups, but particularly non-Caucasians frequently experience lactose malabsorption and intolerance (2). Generally it is agreed that there is a higher percentage of individuals with lactase deficiency and lactose intolerance in the Negroid than in the Caucasian population (53).

Bayless and Rosensweig (54,55) studied twenty Negro and twenty Caucasian subjects. A deficient level of lactase activity was present in 70% of the Negroes and in only 5% of Caucasians (Table 3). Females were affected as well as males. Surprisingly enough, the lactase deficiency did not result in any clinical symptoms in the subjects, as they had all learned to limit their intake of milk to amounts which were tolerated - usually one glass or less with a meal. Several did use milk as a laxative.

Huang and Bayless (27) performed lactose tolerance tests in twenty Negroes and twenty Caucasians aged eleven months to eleven years. Symptoms were evident in seven of the Negroes and two Caucasians. Fourteen of the twenty Negroes and two of the twenty Caucasians had a family history of milk intolerance.

Although symptoms in both Negro and Caucasian children increased in frequency with advancing age, they were not as common in the Caucasian subjects. Paige et al. (56) also studied Negro children in the United States aged six to eleven years and found that 45% were intolerant to a lactose load equivalent to three or four glasses of milk.

TABLE 3

Lactose intolerance and malabsorption in Negro and Caucasian subjects (55)

	Negro subjects	Caucasian subjects
Total in experiment	20	20
Milk intolerant	19	2
Symptoms with lactose tolerance test	18	2
Lactose tolerance test rise less than 25 mg%	15	2
Lactase activity less than 2 units	14	1

In 1973, Paige et al. (57) studied thirteen healthy Negro and Caucasian women and their children aged 18-24 months. Thirty-eight percent of the Negro toddlers were lactose intolerant. All of the lactose intolerant Negro children were born to lactose intolerant women. Only one Caucasian child was intolerant and all of the Caucasian mothers were tolerant. Paige et al. (58) also studied thirty-two Negro and thirty-two Caucasian women. Twenty-one, or sixty percent of the Negro women were malabsorbers and seventeen had symptoms and were considered intolerant. Three, or only nine percent, of the Caucasian women were malabsorbers with two developing symptoms after the lactose tolerance tests.

In 1975, Paige et al. (4) studied 89 black elementary school children, 6.3-13.5 years of age. Forty-eight (54%) had a flat lactose tolerance curve. Twenty-eight (58%) of these forty-eight were non-milk drinkers. They concluded that some lactose malabsorbing children may have sufficient levels of lactase to hydrolyze moderate amounts of milk.

Paige et al. (59) studied 58 black and white children and found that more blacks had a problem with lactose absorption than whites (Table 4). It was also noted that more non-milk drinkers were intolerant than milk drinkers.

TABLE 4

Lactose malabsorption and intolerance in black and white children (59)

	Number		
	Total	Abnormal blood sugar rise	Symptoms
White milk drinkers	15	3 (20%)	0
White non-milk drinkers	12	2 (17%0	3
Black milk-drinkers	14	5 (35.7%)	7
Black non-milk drinkers	17	13 (76.5%)	12
Total	58		

Other non-Caucasians also have a higher incidence of lactose malabsorption and intolerance. Keusch et al. (31) studied 172 normal Thai infants and children. They found that abnormalities in lactose absorption became manifest after infancy, and by two years of age, virtually all children studied were abnormal. Varavithya et al. (60) also studied Thai infants. Lactose tolerance tests were done on malnourished Thai infants less than two years old. Abnormal tests were found in sixty percent of the infants suffering from malnutrition alone, and in sixty-nine percent of those who had malnutrition and diarrhea. They also found that lactose tolerance decreased rapidly with age in Thai infants until almost all children over two years of age were lactose intolerant.

A comparison of the ability to hydrolyse lactose in Caucasians, Negroes, and Thais is given in figure 3.

Huang and Bayless (61) examined twenty Orientals, aged 23 to 38 years, living in America, and twenty Caucasians 18 to 54 years old. Two of the twenty Caucasians were intolerant to milk and lactose. Nineteen of the twenty Orientals developed abdominal cramps and diarrhea after ingesting an amount of lactose equivalent to one quart of milk. Fourteen reported similar symptoms after ingesting one or two glasses of milk. All had consumed milk as infants without symptoms.

Jones and Latham (42) performed lactose tolerance tests on 34 subjects of African, Asian, and Latin American origin. Eleven (72%) of the adults and four (25%) of the children were found to be lactose intolerant or lactose malabsorbers as determined by a less than 25 mg/100 ml blood glucose rise after lactose administration. All the children under age five, however, were lactose tolerant.

Stoopler et al. (2) found that lactose malabsorption occurred in 56% of a random sample of 94 rural Jamaican children under four years of age. There was a significant decrease in the percentage of children able to absorb lactose after the first year of life. When the original malabsorbers were retested seven to eight months later, 21% had normal lactose tolerance curves. They concluded that a high percentage of rural Jamaican children under four years of age cannot absorb lactose and that this percentage increased significantly after the first year of life. A similar lactose tolerance test run on 20 urban Jamaican children revealed that 14, or 70%, were lactose malabsorbers (2). Neither sex, anthropometric status, milk consumption, symptoms of lactose

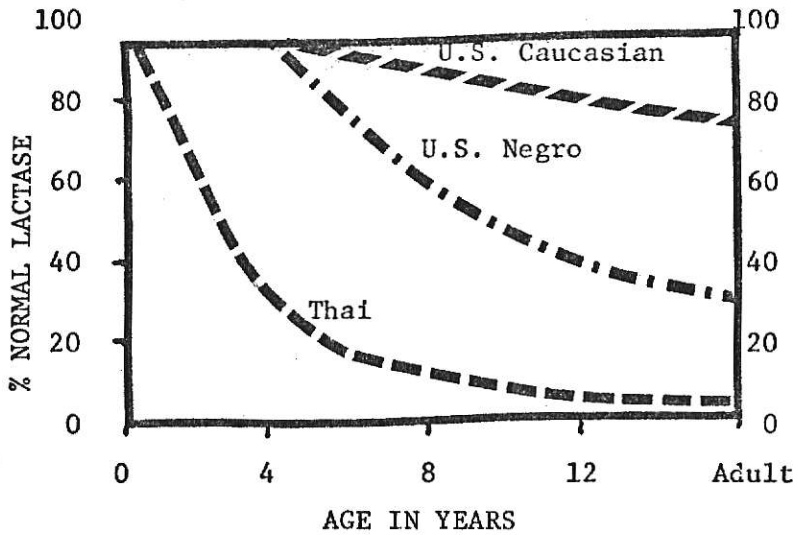

Fig. 3 Decrease in capacity to hydrolyze lactose relative to age in three populations (59).

intolerance, nor duration of breast feeding correlated with the occurrence of lactose malabsorption or its persistence. From these two studies, Stoopler et al. proposed that an abrupt transformation in absorptive status occurs after age one and that some children at every age could regain a previously lost capacity to absorb lactose.

Reddy and Pershad (62) performed intestinal biopsies, disaccharidase assays, and lactose tolerance tests on Indian adults and children. A lactase deficiency was found in all adults, with lactose intolerance occurring in 50% of them. A skim milk supplement for four weeks had no effect on lactase activity. Twenty out of 54 children were intolerant to a lactose load of 2 g/kg, but when 8 of the 20 were retested with 1 g/kg, none had symptoms. Only 4 of the 20 intolerant children had symptoms when milk containing an equivalent amount of lactose was given. Even in those children who developed symptoms, the symptoms disappeared when the milk was given in divided doses. Leichter and Lee (63) also found a high incidence of lactose intolerance among Indians during adolescence. David and Bolin (64) studied asymptomatic volunteer Chinese and Indian students and found that intolerance to lactose was very common in this group.

Paige et al. (65) studied ninety Mesitizo (Peruvian) children. Seventy-three percent of children below three years had normal tolerance curves. All children over age twelve had abnormal tolerance curves. Seventy-five percent of those with abnormal curves had symptoms. Ransome-Kuti et al (29) studied ten Nigerian families of the Yoruba tribe. One

parent was a lactose digestor and the other a lactose nondigestor. Of the 29 children, 18 were lactose digestors and 11 were nondigestors.

A summary of lactose intolerance in different populations is given in figure 4.

TREATMENT

In the treatment of an infant or person with lactose intolerance, the diarrhea, if present, must be controlled first before any tolerance tests are done to define the enzyme defect. This is accomplished by removing all the disaccharides from the diet and replacing them with simple sugars (monosaccharides). Cornblath and Schwartz (7) have suggested a careful and detailed history and a well-planned elimination dietary trial as one means of completing knowledge before treatment is instigated.

Dahlquist et al. (22) suggested that the daily intake of milk be limited and divided into small portions. It is possible to add lactase to the milk some time before ingestion so that the lactose is completely hydrolysed to glucose and galactose before the milk is fed. Milk consumed without a meal or very cold seems to cause more symptoms than milk eaten with food or slightly warm. Bayless (5) has suggested that milk might be diluted by a meal, or a meal may delay gastric emptying and release only a small amount of lactose to the small intestinal enzyme system at any one time.

For infants, a lactose-free soybean formula or a diluted cow's milk formula whose caloric strength has been reconstituted with sucrose or glucose instead of lactose may be satisfactory (3,22).

Fig. 5

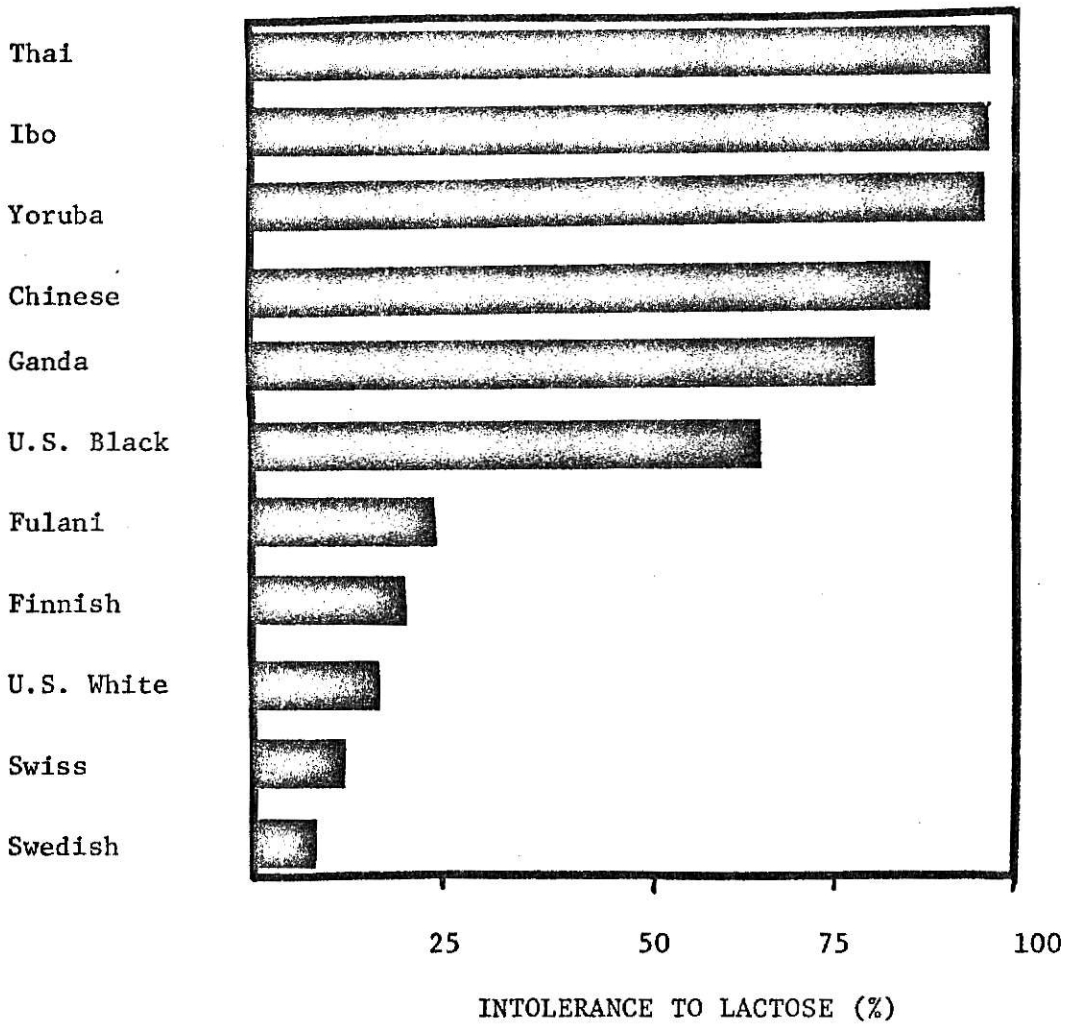

Fig. 4 Intolerance to lactose of selected populations (15)

Low lactose or lactose-free diets also have been suggested for the routine treatment of malnutrition and diarrhea. Varavithya et al. (60) proposed that since acquired lactase deficiency could be caused by chronic diarrhea and malnutrition of Thai infants, nonlactose milk formulas and/or other lactose-free foods may be best for infants suffering from these conditions.

Bartrop and Hull (66) found lactose intolerance difficult to diagnose in 100 infants with bowel disorders. They proposed that all malnourished infants under the age of six months, who suffer from persistent diarrhea, be treated with a lactose-free diet. Three out of four infants in their study tolerated lactose when it was reintroduced two months after the illness. Only if diarrhea returns with the reintroduction of lactose should a lactose-free diet be maintained for a three month period or longer.

Bowie et al. (67) studied 27 children with protein-calorie malnutrition. Diarrhea was reduced when their diet was changed from milk to a carbohydrate-free one. Lactic acid drops to normal levels and sugar disappears from the stool also with the carbohydrate free diet. They concluded that because milk provides the protein essential for the recovery of these children, a reduction in lactose can be achieved by diluting the milk and adding supplementary protein as casein.

Lactose Free Diets

If the severity of the lactose intolerance necessitates, a lactose-free diet may be prescribed. It should include the following foods daily (68):

7 oz. meat, fish or poultry
1 egg
3 or more fruits including:
    1-2 servings citrus fruit
    1-2 other fruits
3-4 servings vegetables including:
    1 dark green or deep yellow
    1 potato
    1-2 other vegetables, raw or cooked, as tolerated.
6 servings enriched bread or cereals
6 tsps. fortified milk-free margarine
Other foods are needed to provide calories.

The lactose-free diet is designed to eliminate all sources of lactose (68). Common foods containing lactose are given in table 5. All milk and milk products must be eliminated. Lactose is used in the manufacture of many foods and medicines, making it essential to read the labels of all commercial products before being used. The diet is inadequate in calcium and riboflavin, therefore supplements of these nutrients should be prescribed. The protein intake may be increased by adding meat, fish, poultry, or eggs, lactose-free milk substitutes, or breads and cereals from those allowed. Adding carbohydrate foods such as fruits, sugar, jelly, and desserts free of lactose will help to increase the caloric level of the diet. A meal pattern is given below:

Meal Pattern

Breakfast
    Fruit
    Cereal with milk substitute and sugar
    Egg
    Bread or roll made without milk - 2 slices
    Margarine, milk free - 2 teaspoons
    Beverage with cream substitute and sugar

TABLE 5

Foods containing lactose (6)

Source of lactose	Lactose content gm/100 gm
Large amounts	
Milk, cow	5.0
dried, skim	52.0
dried, whole	38.0
skim	5.0
sweetened condensed	14.0
Milk chocolate	8.0
Ice cream	3.5
Whey	5.0
Small amounts	
Cakes and sweet rolls	
Caramels and fudge	
Cheese foods and spreads	
Cookie, sandwich fillings	
Liqueurs	
Cottage cheese and creamed cheese	
Instant coffee	
Potatoes	
Sherbets and ices	
Sour cream	
Custards	
Powdered soft drinks	

Luncheon or Supper
- Lean meat, fish, or poultry - 3 ounces
- Potato or substitute
- Cooked vegetable
- Salad
- Bread made without milk - 2 slices
- Margarine, milk free - 2 teaspoons
- Jelly
- Fruit
- Beverage

Dinner
- Lean meat, fish, or poultry - 4 ounces
- Potato
- Vegetable
- Bread or roll made without milk - 2 slices
- Margarine, milk free - 2 teaspoons
- Jelly
- Fruit or dessert
- Beverage

Fruit juices or water can be substituted for milk in many recipes. Meals eaten away from home should include foods prepared without breading, cream sauces, gravies, etc. Broiled or roasted meats, baked potato, vegetables without added fat, salads, and desserts such as plain angel cake, fresh fruit, and gelatin are good choices (68).

Lactose-Free Milk and Milk Products

Although lactase deficient individuals are most likely unable to tolerate non-fermented dairy products such as milk and ice cream, many are able to tolerate fermented dairy products in which some or all of the lactose has been fermented to lactic acid and other short-chain acids, such as yogurt, cottage cheese, and buttermilk (5). Gallagher et al (69) found that three lactase deficient patients tolerated fermented dairy products without symptoms, but consumption of non-fermented milk products caused moderate to severe symptoms. The bacteria added in the process of culturing may continue to exert lactase activity in the

intestinal tract after ingestion. Patients should be encouraged to test their tolerance of fermented dairy products. Such products as yogurt, cottage cheese, buttermilk, sour cream, and a wide variety of cheeses will add variety and calcium to the diet.

Lactose-free formulas have been used successfully in the treatment of lactose intolerant infants. The formulas have also been used for the treatment of children with kwashiorkor, where the absence of lactose is of definite benefit and lactose intolerance is suspected (70). Lida-Lac, a lactose free milk, is tolerated satisfactorily by the lactose intolerant. It has been used successfully as a milk substitute in the preparation of foods (71). Lida-Lac is basically the powdered product of pure milk containing 85% protein, 1% milk fat, 1% calcium, and practically no carbohydrate. Skala et al. (71) compared Lida-Lac with a dried powdered lactose-containing milk with 33% protein, 0.5% fat and 51.5% lactose. Lactose intolerant individuals consuming the lactose-free milk had much less frequent signs of intolerance than when consuming the lactose-containing milk. Complaints, it any, were very mild. Skala found, however, that the taste of Lida-Lac is not acceptable due to its unpleasant casein flavor. It is acceptable after flavoring with such things as coffee extract, cocoa, and banana. Ten grams of Lida-Lac in 200 ml of water was found to produce the most acceptable milk product. No scum was formed when the milk was boiled. A buttery tasting whipped cream was prepared from 10 grams Lida-Lac in 12 grams water and 80 grams butter. Small amounts could be added to gravy, but did not work well in mashed potatoes. Lida-Lac could be one solution for the lactose-intolerant, as it adds variety to the diet and the protein and calcium

content of the lactose-free diet is improved. Another recent development which may help the lactose-intolerant is the use of the synthetic disaccharide, lactulose (galactose + fructose). When consumed, many enzyme-deficient patients can tolerate over 50 grams (equivalent to two pints of milk) daily with very little change in stool weight (19).

At the ARS Eastern Regional Research Laboratory in Philadelphia, dairy products are being created with lactases derived from molds and bacteria. There is a disadvantage, however, in that these enzymes hydrolyse lactose only under acid conditions, which cause milk to coagulate. Cottage cheese whey can be treated successfully with an enzyme from Aspergillus niger. To reduce the cost of the enzyme, the lactase is bound to porous glass beads and then the liquid whey is pumped through a column, using the same lactase over and over. They have produced low-lactose fluid, concentrated and powdered milks, and whey-containing ice cream. With these enzymes, 90% or more of the lactose is hydrolyzed. Quantities of the enzyme-treated products have been supplied to industrial firms which have expressed interest in them. At the ARS Dairy Products Laboratory in Washington, D.C., yeast lactases, which operate near the neutral pH range are being used to split the lactose of whole and skim milk into glucose and galactose. The only change in flavor is an increase in sweetness (72).

Paige et al. (73) compared carbohydrate absorption in lactose tolerant and intolerant adolescents consuming untreated whole and lactose hydrolyzed milk. Thirty-two black Baltimore City volunteers 13-19 years of age participated in the study. Twenty-two of the 32 participants (69%) were lactose malabsorbers as confirmed by a lactose tolerance test.

They were given 8 oz.untreated whole milk (12 grams lactose), 8 oz. 50% hydrolyzed whole milk (6 grams lactose), and 8 oz. 90% hydrolyzed whole milk (1.2 grams lactose). The test milk was fresh mixed raw whole milk treated with a fixed enzyme, lactase, isolated from the yeast saccharomyces lactis. The lactase hydrolyzed up to 90% of the lactose to glucose and galactose. Although the lactose-intolerant subjects had a lower peak blood sugar rise than the tolerant subjects after ingesting the untreated milk, the difference was eliminated when the malabsorbers were given milk in which 90% of the lactose had been converted by hydrolysis to glucose and galactose (table 6).

TABLE 6

Mean maximum blood sugar rise (73)

Subjects	Number of subjects	Blood sugar rise		
		Untreated	50%	90%
Intolerant	22	4.4	8.8	14.5
Tolerant	10	12.2	13.3	13.7

The same study indicated a slight alteration in taste when the lactose was hydrolyzed (table 7). The reported increase in sweetness of the 90% hydrolyzed milk did not seem to interfere with the acceptance of the products in the study, however.

TABLE 7

Alteration in taste of hydrolyzed milk (73)

Type of milk	Taste response	
	Sweeter than milk (number responding)	Just like milk (number responding)
Untreated	12	88
50% hydrolyzed	19	81
90% hydrolyzed	56	44

LACTOSE INTOLERANCE AND CALCIUM ABSORPTION

A lactose intolerant individual who consumes little or no milk products may not meet his daily requirements for calcium (69). The danger may be reduced by consumption of fermented, lactose-free, and synthetic dairy products.

The presence or absence of lactose in the diet may affect calcium absorption. Lengemann et al. (74) studied radionuclides in the femur after the oral administration of labeled calcium to rats. Lactose enhanced calcium absorption when given as the chloride, gluconate, lactate, or acetate salt. Lactose increased the absorption of calcium in the absence or presence of vitamin D and did so as effectively as vitamin D. Finlayson (75) found that in rats fed a constant lactose diet, the binding of calcium to the intestinal wall was enhanced.

Fifteen healthy infants aged 2-8 months were examined by Kobayashi et al. (76). They were divided into three groups and given 1) formula milk, 2) lactose free milk, and 3) a formula to which a lactase preparation

was added. The intestinal absorption of calcium and magnesium was reduced in infants on the lactose-free diet and enhanced in infants fed the formula with added lactase.

Condon et al. (77) performed calcium and phosphorus balance tests on four volunteers with normal lactose tolerance and one patient with lactose intolerance. When lactose was ingested by the lactose tolerant individuals, calcium and phosphorus balance improved. However, when ingested by the lactose-intolerant patient, fecal calcium rose and calcium balance became more negative. Their results suggested that the beneficial effect of lactose on intestinal calcium absorption is absent in patients with lactase deficiency. They cautioned that the administration of lactose may induce a negative calcium balance large enough to be a factor in the eventual production of osteoporosis.

Kocian et al. (78) found that the heights of calcium absorption curves after lactose-free milk was given to healthy lactose tolerant subjects were lower than after the ingestion of milk with a normal lactose content. If lactose-free milk was given to lactose intolerant subjects, however, the calcium absorption curves were higher than after milk with a normal lactose content was ingested. Gallagher et al. (69) found that in three lactase deficient subjects, an increase in fecal calcium paralleled an increase in the symptoms of lactose intolerance. When fermented dairy products were ingested, calcium excretion was decreased. These results suggested that calcium absorption was increased when the calcium was consumed in fermented dairy products.

These studies indicate that lactose does indeed enhance the absorption of calcium <u>except</u> in the lactose-intolerant person. Lactose-free products

enhance calcium absorption to a greater degree than lactose-containing products in lactose intolerant individuals.

INFLUENCE ON MILK PROGRAMS

Developing Countries

Milk is a nearly complete human food, and in powdered form can be stored conveniently and shipped long distances. For these reasons, it has long been a popular source of protein and other nutrients in many aid programs for nutritionally impoverished children (15). Worldwide interest in kwashiorkor, and its prevention and cure through the use of dried skim milk in child feeding, has served to confirm and publicize the unique place of milk in child feeding schemes (79). Milk consumption, however, is less than 100 grams per day in many Asian and African countries where lactase levels are generally low. In Northern European, Australian, and North American countries where lactase levels are usually higher, milk consumption is around 600 grams per day. Low consumption is also a function of the difficult production, distribution, and storage of milk (43).

Many developing countries receive dry skim milk to be used as a protein supplement. The amount consumed usually does not exceed 200 ml at one time. Reddy and Pershad (62) believe that even a child with lactose intolerance is unlikely to develop symptoms on this supplementation. They believe that the high incidence of lactase deficiency in many developing countries should not be used as an argument against the distribution of skim milk to undernourished populations. Others are more concerned about lactose intolerance and its effect upon milk programs.

It is important for those individuals who are participating in milk programs to consume milk without symptoms and to utilize its nutrients without interference and loss.

Paige et al. (59) found that fifty percent of the population in developing countries is intolerant by the age of three years. Bayless et al. (43) found that milk and lactose intolerance is not a problem in most parts of the world if individuals are allowed to follow their natural dietary habits which include small quantities of milk and more fermented milk forms. Supplying milk as a nutritional aid might, they warn, change this situation.

United States

Lactose intolerance also may threaten milk programs in the United States. In the U.S., race and nationality certainly do influence milk consumption. Native white American and Northern Europeans consume much more milk than people of Southern European extraction, Negroes, and Orientals (43). It is becoming evident that lactose intolerance exists in a significant number of Negro children. Public health nutrition programs have traditionally stressed milk as a staple in the diet of target populations. School-age children are the target of some nutritional programs utilizing milk as a source of protein and calories. Therefore, it is important to know the acceptance or rejection of milk and the physiological basis for the rejection (if any) in populations of children destined to be lactose intolerant. There does not appear to be any long-term biological effects on the growth and development of healthy children who are lactose intolerant and/or lactase deficient.

If, however, lactose is present to a large extent in the diets fed to Negroes and there is rejection because of an underlying enzymatic deficiency, then a problem would exist. It is possible that we are attempting to upgrade the nutritional status of disadvantaged Negroes with food products that a large number of the individuals cannot physiologically tolerate.

Paige et al. (65) conducted two independent observations of milk consumption in two Baltimore elementary schools. Both schools were located in lower socioeconomic areas, one being predominantly Negro and one predominantly white. A type A lunch, including 1/2 pint of unflavored whole cow's milk was fed. The children were categorized as either milk drinkers or non-milk drinkers, milk drinkers being those who consumed fifty percent or more of their 1/2 pint container. Twenty percent of the black children studied were classified as non-milk drinkers, while only ten percent of white were classified as such. Eight-five percent of the Negro non-milk drinkers had an abnormal rise in blood sugar after the ingestion of a lactose load. Fifty-three percent of the Negro and 42% of the white non-milk drinkers were also non-drinkers at home. Ninety-three percent of the Negro and 100% of the white children who drank milk at school also drank milk at home. All children categorized as non-milk drinkers had been able to drink milk as infants. There were no past histories of milk allergy or milk feeding problems in any of the children.

The results of this study (65) indicate that milk rejection by Negro elementary school children in feeding programs is significantly

higher than in similarly matched white children. Overall, the frequency of non-consumption of moderate amounts of milk in Negroes is approximately twice that of whites. A definite association was demonstrated between poor consumption of milk in the lunchroom and at home, lactose induced symptoms, and lactose malabsorption in the Negro children. Fifty-eight percent of all the Negro children studied were shown to be inadequate digestors of lactose as evidenced by flat lactose tolerance curves. Even though many of these children may continue to consume milk, they are probably not realizing its full nutritive value. It appears that race does influence milk consumption.

Aid in the form of milk and milk products with subsequent diarrhea probably is not the most efficient way to help a malnourished community (64). The nutritional contribution of the school lunch, as presently planned and served, depends in a large part on milk, particularly with regard to calcium, magnesium, phosphorus, riboflavin, and protein (80). It is generally assumed that a glass of milk, once given to a child, will be consumed. It is often thought that low levels of milk consumption are usually the result of financial or educational inadequacies. Paige et al. (56) suggested that is milk is not being accepted by a significant segment of a child population, other options include: 1) hydrolysis of lactose into monosaccharides before milk is distributed for general consumption, 2) increased availability of milk substitutes in the form of soy or 3) increased use of combinations of milk and other juices and liquids to lower lactose content.

The Food and Nutrition Board of the National Research Council issued a statement concerning these problems. They emphasized that

reactions to test doses of lactose, which are usually equivalent to the lactose present in one quart of milk, do not necessarily mean intolerance if milk is consumed in a normal manner. Persons with low intestinal lactase activity will spontaneously limit their milk consumption to amounts they can tolerate without difficulty. They concluded that, provided milk is introduced into the diet gradually, there are no significant difficulties associated with the consumption of supplementary amounts of milk. Authorities and supervisors working with the milk supplementation programs should, however, be aware of the current knowledge concerning lactose intolerance, as there may be circumstances in which the gradual introduction of milk is advisable (80, 81).

SUMMARY

Lactose, the carbohydrate in milk, must be hydrolyzed by lactase in the digestive tract to galactose and glucose before absorption can take place. Inadequate enzymatic digestion of lactose results in lactose malabsorption and intolerance. Lactose intolerance affects all age groups and races, but specifically 60-90% of non-Caucasian adults.

Intolerance is characterized by diarrhea and cramping pain. Diagnosis may be made by use of dietary history, lactose tolerance tests, intestinal biopsy, fecal examination, radiological observation, lactose loading, or expired air. Lactose tolerance tests are the most commonly used method.

The cause of lactose intolerance is not certain, but is probably related to disease, normal development, hereditary factors or lack of lactose in the diet. Intolerance can be either congenital or acquired. The congenital form is usually specific, while acquired forms involve several

After control of the diarrhea has been established, treatment for lactose intolerance may include a lactose free diet, low lactose diet, or use of lactose free milks and milk products. Care should be exercised to include adequate sources of calcium in the diet.

Lactose intolerance greatly affects milk programs abroad and in the United States. Supervisors and authorities working with any kind of milk supplementation should be aware of the problems lactose intolerance may present.

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LACTOSE INTOLERANCE

by

JANICE O. HEATON

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Lactose, the carbohydrate in milk, must be hydrolyzed by lactase in the digestive tract to galactose and glucose before absorption can take place. Inadequate enzymatic digestion of lactose results in lactose malabsorption and intolerance. Lactose intolerance affects all age groups and races, but specifically 60-90% of non-Caucasian adults.

Intolerance is characterized by diarrhea and cramping pain. Diagnosis may be made by use of dietary history, lactose tolerance tests, intestinal biopsy, fecal examination, radiological observation, lactose loading, or expired air. Lactose tolerance tests are the most commonly used method.

The cause of lactose intolerance is not certain, but is probably related to disease, normal development, hereditary factors or lack of lactose in the diet. Intolerance can be either congenital or acquired. The congenital form is usually specific, while acquired forms involve several processes.

After control of the diarrhea has been established, treatment for lactose intolerance may include a lactose free diet, low lactose diet, or use of lactose free milks and milk products. Care should be exercised to include adequate sources of calcium in the diet.

Lactose intolerance greatly affects milk programs abroad and in the United States. Supervisors and authorities working with any kind of milk supplementation should be aware of the problems lactose intolerance may present.