

Lactose Intolerance

Description

This article deals with lactose intolerance, ie enzyme deficiency, rather than lactose allergy, ie IgE-mediated reaction - which is covered in the separate [Food Intolerance and Food Allergy](#) article.

- Lactose is a disaccharide sugar found exclusively in milk. Absorption of lactose is dependent upon the enzyme lactase.
- Lactase is the enzyme that hydrolyses lactose to the monosaccharides, glucose and galactose, and is in the tips of the villi of the small intestine.
- This enzyme is essential in babies but tends to decrease in amount after the age of 2, although symptoms of lactose intolerance rarely occur before the age of 6.
- It is argued that it is only because we have the unusual habit of ingesting milk from other species, usually the cow, that the enzyme persists beyond the age of weaning.

Most milk intolerance in young children is due to allergy to cow's milk protein and not deficiency of lactase. The immature gut of the premature baby may be deficient in lactase but it soon increases as it is presented with milk.

Lactose enhances the absorption of calcium, magnesium, and zinc. It also promotes the growth of lactobacilli and provides galactose, which is essential for the formation of cerebral galactolipids and hence development of the brain.

Types of lactase deficiency

- **Primary lactase deficiency** - autosomal recessive and also known as: adult-type hypolactasia, lactase nonpersistence, or hereditary lactase deficiency. Deficiency of lactase develops at various ages.
- **Secondary lactase deficiency** - follows damage to the intestinal mucosa, eg acute viral or bacterial [gastroenteritis](#), and chemotherapy, and resolves when the disease process is over and the intestinal mucosa heals. It is more common in children and especially in the developing world.
- **Congenital lactase deficiency** - an extremely rare autosomal recessive disorder and associated with minimal, or complete absence of, lactase activity.^[1] It becomes apparent once milk is introduced, usually with intractable diarrhoea once milk or lactose formula has been introduced.^[2]
- **Developmental lactase deficiency** - occurs in premature babies (<34 weeks' gestation) and improves once the intestine matures.^[3]

Epidemiology

- Primary lactase deficiency - 70% of the world's population. Prevalence varies with ethnicity, eg 2% in Northern Europeans and up to 80% in Hispanic populations. This relates to the level of use of dairy products in the diet.^{[2] [4]}
- Age of presentation also varies, eg Hispanic, Asian and Afro-Caribbean children present before age of 5 years, whereas Northern Europeans usually present after the age of 5.^[2]
- Lactose intolerance in adults is very common and lactose may be found in many unexpected sources, including saccharine processed meats, bread, cake mixes, soft drinks, and lagers. This may account for unexplained symptoms, including irritable bowel disease.^[5]

Risk factors

Symptoms of intolerance depend upon how fast the small intestine is presented with the lactose load. They will be more marked if the lactose reaches the intestinal mucosa fast but less so if gastric emptying is delayed as by a fatty meal.

Presentation

History

Symptoms result from reduced absorption of lactose which is then broken down by intestinal bacteria leading to gas and short-chain fatty acids.^[3] Symptoms relate to the amount of lactose ingested.

Main symptoms

- Bloating.
- Abdominal pain.
- Flatulence.
- Loose watery stool - with a degree of urgency an hour or two after ingestion of milk.
- Perianal itching due to acidic stools.^[3]

Symptoms occur from one to several hours after ingestion of milk or dairy products. These symptoms are very nonspecific and occur with other disorders such as milk-protein sensitivity, allergic-type reactions to other substances in the meal, or intolerance of other saccharides.

Secondary lactase deficiency produces worse symptoms and dehydration may occur.

Examination

- In children there may be malnutrition and failure to thrive but this is uncommon.
- In adults there is usually nothing to find or perhaps a little bloating and discomfort during an attack.

Investigations

Diagnosis can be made on clinical features alone, eg reintroduction of lactose leads to symptoms. Specialised tests are rarely required.^[3]

- Reducing substances in the stool show that carbohydrates are not being absorbed.
- Another easy test is to measure stool pH which is below 5.5.

In both cases it is important to test the liquid portion of the stool and not the solid part.

- A lactose tolerance test involves a test dose of 2 g of lactose per kg body weight up to a maximum of 50 g, given after a fast, and noting the rise in blood glucose, rather like a glucose tolerance test. A positive test is reproduction of symptoms and rise in serum glucose by <1.11 mmol/L, 60-120 minutes after ingestion.^[3] However, this rather higher dose than is presented in a normal meal has been criticised as representing an atypical situation. This has now been superseded by breath hydrogen tests.
- Breath hydrogen test - if carbohydrate is unabsorbed in the gut, it is fermented by bacteria in the large intestine and hydrogen gas is produced, absorbed into the blood and excreted by the lungs. Thus, carbohydrate malabsorption can be determined by measuring the exhaled hydrogen concentration after a carbohydrate load. Normally, the fermenting bacteria are confined to the large intestine but, when bacterial overgrowth in the small intestine occurs, upper small bowel fermentation of ingested lactose occurs and causes an early rise in the exhaled hydrogen concentration. There will still be a later rise in exhaled hydrogen during large bowel fermentation. Antibiotics may produce false negative results. For diagnosis of lactose intolerance, 0.5 to 1.0 g/kg to a maximum of 12 to 25 g of lactose is given and an increase greater than 20 ppm of hydrogen is diagnostic.
- If difficulty remains, a small intestinal mucosal biopsy can be obtained by endoscopy for direct assay of lactase activity as well as that of other brush border disaccharidases.

Differential diagnosis

Recurrent abdominal pain of childhood and **irritable bowel syndrome** can mimic the symptoms of lactose intolerance and vice versa. Other differentials include:

- Allergy to milk proteins or other constituents of milk - see article dealing with **intolerance and allergy**.
- Deficiency of other disaccharidases.
- **Infantile colic**.
- Irritable bowel syndrome.

Management

Avoiding milk and dairy products will relieve symptoms in most. However, lack of milk and dairy products will result in the loss of a vital source of calcium, especially if the patient is on a vegetarian diet.^{[3] [4]}

Furthermore a recent systematic review concluded that most patients could tolerate a glass of milk per day.^[6]

Primary lactase deficiency

- Varying amounts of lactose can be tolerated - this needs to be determined. Things can be further improved by taking the lactose in divided portions throughout the day.
- Lactase from yeast can be added to milk or live yoghurt and tends to be well tolerated. Furthermore, some foods, eg yogurt and curds, may be better tolerated. This is due to the fact that gastric emptying is slower, resulting in these foods having a thicker consistency, and their preparation means that the lactose is partially hydrolysed.^[3]
- Milk substitutes can also be used but they contain fewer nutrients compared with cow's milk.
- Lactase enzyme preparations can be combined with lactose foods but these are expensive.

Secondary lactase deficiency

- Resuscitation with intravenous rehydration may be required in secondary lactase deficiency.
- Antibiotics should be avoided unless strong evidence for a bacterial cause.
- Parents/carers should be advised to continue giving formula or **breast milk** or regular milk during an acute diarrhoeal illnesses. However, some authorities recommend withdrawing lactose for 3 weeks in prolonged diarrhoea after gastroenteritis.

Developmental lactase deficiency

- Lactase production can be induced by lactose during the newborn period but not later in life.^[7] Tube feedings with milk containing lactose in premature infants usually contain half-strength lactose formula or breast milk. Full lactose strength formula is more likely to induce intolerance. Infant feeding, when breast-feeding, starts with colostrum and it is 2 or 3 days before milk comes in.^[8]

Novel therapies in the research phase

- Peroral beta-galactosidase preparations can be used in the management of lactose intolerance. They are given as large doses just before lactose ingestion is to occur. There is hope of developing a new long-acting formulation.^[8]

Complications

Most people with lactase deficiency suffer very little but babies with severe deficiency require a diet full of essential nutrients but excluding lactose. Transient lactase deficiency affects a significant number of infants with severe gastroenteritis and diarrhoea. Improper early feeding with lactose-based products without the recognition of lactose malabsorption can lead to **chronic diarrhoea** and malnutrition.

Lactose enhances the absorption of several minerals, including calcium, magnesium, and zinc. In addition, milk products are high in calcium that is extremely important in bone growth. Children can quickly become deficient and so calcium supplements are required if there is restriction of eating dairy products.^[4]

Prevention

The following may contain unexpected lactose and patients and carers need to be advised to monitor food labels:

- Bread
- Cakes
- Cereals
- Margarine
- Dressings
- Sweets
- Snacks
- Various drugs, whether prescribed or over-the-counter

Further reading & references

- Foodreactions.org; List of products that may contain lactose
1. [Congenital Lactase Deficiency](#), Online Mendelian Inheritance in Man (OMIM)
 2. [Heyman MB](#); Lactose intolerance in infants, children, and adolescents. *Pediatrics*. 2006 Sep;118(3):1279-86. [abstract]
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 5. [Matthews SB, Waud JP, Roberts AG, et al](#); Systemic lactose intolerance: a new perspective on an old problem. *Postgrad Med J*. 2005 Mar;81(953):167-73. [abstract]
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