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Lactose intolerance

What is lactose intolerance?

This article deals with **lactose intolerance**, which is defined as symptoms that occur upon exposure to lactose in individuals with lactose malabsorption.^[1] It is the result of an enzyme deficiency, rather than lactose allergy, which is an IgE-mediated reaction. For information about allergy, see the separate Food Allergy and Food Intolerance 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 present in the tips of the villi of the small intestine.
- This enzyme is essential in babies but tends to decrease in amount after infancy, although symptoms of lactose intolerance rarely occur before the age of 6 years.

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Most milk intolerance in young children is due to allergy to cow's milk protein and not deficiency of lactase.

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**: also known as adult-type hypolactasia, lactase nonpersistence, or hereditary lactase deficiency. This is the commonest cause of lactose intolerance worldwide and is due to a decrease in lactase activity which usually becomes apparent between the ages of 5 and 20 years. Lactase activity rarely drops completely: around 10-30% of initial enzyme levels remain.^[2] Primary lactase deficiency is rarely a cause of symptoms in children under the age of 3 years.^[3] Lactase nonpersistence is the genetic wild-type (normal Mendelian inheritance). Lactase persistence is due to genetic mutation. Both are phenotypes that are seen in healthy humans.^[1]
- Secondary lactase deficiency: follows damage to the intestinal mucosa - eg, acute viral or bacterial gastroenteritis, parasitic infection, uncontrolled coeliac disease, inflammatory bowel disease, chemotherapy, long courses of antibiotics or severe malnutrition. This resolves when the disease process is over and the intestinal mucosa heals. It is more common in children and especially in lowerincome countries.
- **Congenital lactase deficiency**: an extremely rare autosomal recessive disorder associated with minimal, or complete absence of, lactase activity in an otherwise normal intestinal mucosa.^[4] It becomes apparent once milk or lactose formula is introduced, usually with severe intractable diarrhoea and faltering growth.
- **Developmental lactase deficiency**: occurs in premature babies (<34 weeks of gestation) and improves once the intestine matures.^[5]

How common is lactose intolerance? (Epidemiology)

- The global prevalence of lactose malabsorption is estimated to be 68%, ranging from 28% in Western, Southern, and Northern Europe, to 70% in the Middle East.^[6]
- There is wide geographical and ethnic variation in the age-related decline in lactase levels. This is related to the level of dairy consumption in the diet.

- The prevalence of high lactase levels in adulthood is highest in Northern European populations and lowest in black and East Asiatic communities where, traditionally, the adult diet does not include milk. People who lose lactase activity more quickly after weaning will present earlier with symptoms.^[2]
- Chinese and Japanese people lose 80% to 90% of lactase activity within 3 to 4 years after weaning; Jewish and Asian people lose 70% to 80% over several years after weaning; and in white Northern Europeans and North Americans it may take up to 18 to 20 years for lactase to reach its lowest levels.^[7]
- US data suggest that approximately 20% of Hispanic, Asian, and black children under the age of 5 years have evidence of lactase deficiency and lactose malabsorption, whereas white children typically do not develop symptoms of lactose intolerance until after the age of 4 or 5 years.^[8]
- Lactose intolerance in adults is very common and lactose may be found in many unexpected sources. These include saccharine, processed meats, bread, cake mixes, soft drinks and lagers. This may account for unexplained symptoms, including in some cases of irritable bowel syndrome.

Risk factors^[1] ^[2]

Symptoms are caused only by the ingestion of lactose. Whilst lactose malabsorption is necessary for lactose intolerance to occur, many individuals with lactose malabsorption will have no symptoms after exposure to lactose, meaning that other factors are at play. These factors include:

- The amount of lactose ingested.
- How fast the small intestine is presented with the lactose load.
 Symptoms will be more marked if the lactose reaches the intestinal mucosa fast (short gut transit time) and less so if gastric emptying is delayed (for example, by being ingested with a large meal).
- Gastrointestinal (GI) hypersensitivity (associated with psychiatric illness, stress, and functional GI disorders).
- History of previous GI disease or surgery.

• Colonic flora.

Individuals in countries where diet tends to include more dairy products (such as the UK) are more likely to inherit a retained ability to digest lactose and not experience symptoms.

Lactose intolerance symptoms

History

Symptoms result from reduced absorption of lactose which is then broken down by intestinal bacteria, forming gas and short-chain fatty acids.

Gas build up causes:

- Bloating.
- Flatulence.
- Abdominal discomfort.

The acidic and osmotic effects of undigested lactose may cause:

- Loose watery stool with a degree of urgency an hour or two after ingestion of milk.
- Perianal itching due to acidic stools.

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 may produce more severe symptoms and dehydration may occur.

Examination

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

Investigations

The diagnosis can be made on clinical features alone - eg, re-introduction of lactose leads to symptoms. Specialised tests are rarely required.

Start a trial of a two-week period of a strict lactose-free diet, with careful attention to food labels. If symptoms resolve but recur on re-introduction of lactose-containing foods, the diagnosis can be made.

There is no single agreed diagnostic test but the following can be used where appropriate: $\begin{bmatrix} 9 \end{bmatrix} \begin{bmatrix} 10 \end{bmatrix}$

- **Hydrogen breath test** if carbohydrate is unabsorbed in the gut, it is fermented by bacteria in the large intestine. This leads to the production of hydrogen gas, which is 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. Antibiotic use may produce false negative results. To diagnose lactose intolerance, oral lactose is given after overnight fasting and an increase greater than 20 parts per million (ppm) of hydrogen is diagnostic.
- **Stool tests** rarely, faecal pH and reducing substances might be tested in infants with diarrhoea in whom lactose or other carbohydrate intolerance is suspected. Stool may also be analysed for infection, if this is suspected as a differential diagnosis.^[8]
- Lactose tolerance test (LTT) this involves giving a lactose load after fasting, and noting the rise in blood glucose at subsequent intervals

 rather like a glucose tolerance test. A positive test is reproduction of symptoms and rise in serum glucose. For many reasons, including the high rate of false negative and positive results, and the need for multiple blood tests, LTTs have now been superseded by breath hydrogen tests.
- **Genetic tests** are available but so far cannot cover all the genetic mutations that exist within different populations.

• 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. The gold standard test is analysis of enzyme and carbohydrate levels and ratios from biopsy; however, it is usually too invasive a test for a mild condition.

Differential diagnosis

- Recurrent abdominal pain of childhood.
- Irritable bowel syndrome.
- Allergy to milk proteins or other constituents of milk.
- Deficiency of other disaccharidases.
- Infantile colic.
- Diverticular disease.
- Ulcerative colitis.
- Coeliac disease.
- Cystic fibrosis.

Lactose intolerance treatment and management

A low lactose diet will relieve symptoms in most cases. A strict lactose-free diet is not required, as most people will tolerate up to 12 grams of lactose in a single dose (this equates to a 250 mL glass of milk).^[11] However, long-term lack of milk and dairy products can result in the loss of a vital source of calcium, especially in people who are also vegetarian, and you should consider referring the patient to a dietician if you are concerned about nutritional deficiency.^[12]

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 and with meals.

- Yoghurt and curds may be tolerated due to their thicker consistency leading to slower gastric emptying. Live culture or fermented products may be better tolerated, as they contain bacteria which partly hydrolyse their own lactose.
- Dairy products with a higher fat content, such as ice cream, chocolate milk, cheese, and full-fat rather than skimmed milk, are better tolerated. The fat content slows gastric emptying.
- Hard cheeses, such as Cheddar, Edam, Parmesan and Emmental, contain very little lactose and may be well tolerated.
- Milk substitutes (eg soya, oat, nut milk) can also be used but they contain fewer nutrients compared with cow's milk.
- Lactose-free dairy products in which lactase enzyme is added to the product are widely available and considered to be safe.
- Oral lactase enzyme supplements available commercially can be combined with lactose-containing foods. Alternatively, probiotics that produce lactase in the gut can be taken. The evidence for the effectiveness of these is variable.^[1]
- Consider the need for calcium or vitamin D supplementation.^[13]

Secondary lactase deficiency^[3] ^[12] ^[14]

- Treat the underlying cause.
- Resuscitation with intravenous rehydration may occasionally be required .
- Antibiotics should be avoided unless there is strong evidence for a bacterial cause.
- Mothers who breastfeed should be advised to continue.
- Parents of formula-fed infants with likely post-gastroenteritis lactase deficiency (diarrhoea lasting beyond two weeks) can make a temporary switch to lactose-free formula, with review after two weeks and an aim to re-introduce lactose at eight weeks.
 Improvement in symptoms should be seen after 2-3 days. Soya based formula is not suitable for those under the age of 6 months.

• Infants on solid foods should temporarily avoid and then gradually re-introduce lactose. This may need to be done under the supervision of a dietician.

Developmental lactase deficiency

All premature infants should be breastfed where possible. Tube feedings with milk containing lactose in premature infants usually contain breast milk, or if not possible, reduced-lactose milk. Full-strength lactose formula is more likely to induce intolerance. Evidence for adding lactase to feeds in this time period remains weak. ^[15] Breast milk contains components which aid lactose absorption.

Congenital lactase deficiency

Babies with severe lactase deficiency require a diet full of essential nutrients but excluding lactose. They cannot be breastfed; they need lactose-free formula milk and must be weaned on to lactose-free foods.

Complications

Most people with lactase deficiency suffer very little. Transient lactase deficiency affects a significant number of infants following severe gastroenteritis. 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, which is extremely important for bone growth and subsequent maintenance of bone mineral density.^[16]

Lactose intolerance prevention

Patients and carers need to be advised to monitor food labels. The following may contain unexpected lactose:^[12]

- Bread.
- Cakes and biscuits.
- Cereals.
- Margarine.

- Dressings.
- Sweets.
- Snacks.
- Ready meals.
- Quorn-based products.
- Baked beans.
- Various drugs, whether prescribed or over-the-counter.

Further reading

 Deng Y, Misselwitz B, Dai N, et al; Lactose Intolerance in Adults: Biological Mechanism and Dietary Management. Nutrients. 2015 Sep 18;7(9):8020-35. doi: 10.3390/nu7095380.

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