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This review describes the difference between cow's milk allergy and lactose intolerance and offers insight into dealing with these dietary challenges.

A publication for health professionals

Dairy allergies and lactose intolerance

Animal milk has been part of the human diet for more than 9000 years. Although Hippocrates (460–370 BCE) already described adverse effects,[1,2] lactose intolerance has been formally recognised as such only in the last 50 years.[2] The terms 'cow's milk allergy' (CMA) and 'lactose intolerance' are often used interchangeably and are generally misunderstood by the general public. This creates many misconceptions and frequent self-misdiagnosis, which often lead to unnecessary exclusion of dairy products from the diet.[3,4]

Cow's milk allergy

A food allergy occurs when food protein or other large molecules elicit an immune response in the body. The immune system reacts by producing antibodies and histamine and activating various defence mechanisms, just as it would respond to any other antigen.[5] CMA is therefore an immune reaction to the proteins found in cow's milk.[6] Cow's milk contains about 30–35 g of protein per litre. Milk protein is made up of 25 different proteins, of which up to 20 have been identified as allergenic.[1,7,8]

The complexity of CMA is evident from the different proteins and epitopes that elicit a response, the different immunological mechanisms in place and the change in response over time, with IgE-mediated reactions more common in infancy and non-IgE-mediated reactions dominating in adults.[3]
IgE-mediated reactions are typically immediate (within a few minutes to two hours after consumption), whereas non-IgE-mediated reactions are delayed and typically occur between 48 hours and one week after ingestion.[6]

CMA is a common condition among children, with an estimated incidence of 2–7.5% in the first year of life. The prevalence of CMA decreases with age[8] and humans seem to be able to outgrow it.[7] Self-diagnosis of CMA is reported to be considerably higher than the actual clinically proven incidence,[3,8] and notably so among women.[8]

Risk factors for CMA include atopy, atopic dermatitis, atopic asthma, rhinitis, eczema, respiratory symptoms with skin or gastrointestinal symptoms.[6,8] Skin reactions are the most common clinical manifestations of CMA, followed by gastrointestinal and respiratory symptoms.[6,8]

Until suitable immunotherapies without side-effects become available, exclusion of milk protein from the diet will remain the preferred therapy for CMA.[1,6] Studies show that 30–50% of children have outgrown CMA by the age of 1 year and that 80–90% achieve tolerance within 5 years.[6,8] Although recovery does not always imply total tolerance, lifelong total exclusion of milk products might not be necessary.[6] Alternative approaches are continually being investigated, and Feehley et al.[9] suggest that interventions to modify intestinal bacterial communities may be a beneficial approach in preventing and treating food allergies.

Lactose intolerance

A food intolerance differs from a food allergy as it does not involve the immune system. A food intolerance occurs when a person does not have the necessary digestive enzyme(s) for a specific nutrient, such as lactose, or when there is a reaction to either natural or artificial substances in a food.[2,5] Lactose is present at a concentration of approximately 4.5 g per 100 mL in cow's milk.[10] This disaccharide is synthesised in mammary glands by the lactose synthetase system, which binds a D-galactose molecule to a D-glucose molecule with a β-1,4 glycosidic bond.[2,4]

A systematic review and meta-analysis by Storhaug et al.[11] shows that lactose malabsorption is common worldwide, although there is considerable variation both within and between countries. The prevalence of lactose intolerance differs widely across ethnic groups, likely owing to the influence of genetic differences.[5] The lowest prevalence is described among North-European populations and the highest prevalence in the Middle East, with an estimated global prevalence of 68%.[11]

Lactose intolerance is the result of low levels of intestinal lactase (also called lactase-phlorizin hydrolase), which is a β-D-galactosidase. The enzyme is exclusively expressed in mature enterocytes in the apical surface of the intestinal microvilli, with the highest expression in the mid-jejunum.[4,12] Lactase activity is typically highest just after birth, when the newborn relies on milk as exclusive form of nutrition.[5] Full-term infants have adequate lactase to digest about one litre of breastmilk (containing 60–70 g of lactose) per day.[4]

During childhood and adolescence, lactase activity declines to about 5–10% of that at birth in most populations.[4,5] The loss of lactase activity in adulthood results from an autosomal recessive gene, of which the occurrence is related to ethnicity.[12] It is described that lactase persistence beyond infancy is linked to several nucleotide poly-morphisms in the lactase gene promoter region of chromosome 2 (region 2q21).[4]

Primary lactase intolerance does not typically manifest clinically before the age of five years and early lactase deficiency may rather develop owing to the intestinal villi being damaged by disease (gastroenteritis, giardiasis, celiac disease, Crohn's disease), cow's milk enteropathy, medication, prolonged diarrhoea or malnutrition.[4,5] Depending on the cause, the type of damage and whether the cause is resolved, lactose malabsorption may be temporary or permanent.[4,5]

Reviews by Heine et al.[4] and Amiri et al.[13] distinguish between the following clinical categories of lactose intolerance:

- Developmental lactase deficiency occurs in preterm infants (<34 weeks of gestation). At 26–34 weeks of gestation, a human foetus has about 30% of the lactase activity compared with the peak lactase expression at birth. Lactase activity increases to 70% at 35–38 weeks and therefore premature babies often experience maldigestion of lactose. Developmental lactase deficiency is generally temporary and improves over time.
- Congenital lactase deficiency (alactasia) is a rare autosomal-recessive inherited disorder, presenting as a total absence of small-intestine lactase activity in newborns despite a normal small intestinal mucosa.
- Lactase non-persistence (hypolactasia) is the form of lactose intolerance that appears in adulthood and is caused by the absence of a lactase-persistent allele. A gradual decline of lactase activity after weaning typically occurs in approximately 70% of the global population. Symptoms usually do not before 5 years of age and the peak onset is during adolescence and early adulthood. Small mounts of lactose (about 24 g per day) are, however, tolerated if taken in divided amounts during the day. This form of lactose intolerance is regarded as the most common, affecting more than two-thirds of the world population.[13]
- Acquired or secondary lactose intolerance occurs because of small-intestine injury due to various conditions, e.g. acute gastroenteritis, chemotherapy or infections with intestinal microbes. Rare causes of secondary lactose intolerance include epithelial dysplasia syndromes. However, infants with glucose–galactose malabsorption have normal lactase activity but present with osmotic diarrhoea owing to the inability to absorb glucose and galactose (derived from lactose).

Both the amount of lactose consumed and the degree of lactase activity influence whether gastrointestinal symptoms such as diarrhoea, flatulence, nausea, gut distention and abdominal pain are experienced.[11] The intensity of symptoms generally correlates with the amount of lactose consumed: when more lactose is consumed than what the available lactase can digest, undigested lactose remains in the intestine and attracts water, causing bloating, abdominal discomfort and diarrhoea.[13] The undigested lactose also acts as a food source for intestinal bacteria and is fermented to produce short-chain fatty acids, carbon dioxide and methane, further worsening discomfort and diarrhoea.[2,4,5]

To effectively manage lactose intolerance, dietary adaptations might be necessary. Lactose-containing foods should be reduced, but total or lifelong exclusion of milk products might not be necessary. As lactose-intolerant individuals can absorb 42–77% of lactose in a 12.5 g dose, compared with 95% absorption in lactase-persistent individuals, many affected individuals can tolerate low levels of lactose in their daily diet. Lactose-intolerant adults and adolescents can tolerate up to 12–24 g of lactose daily if the amount is staggered over the course of the day and consumed as part of a meal to slow the release of lactose in the small intestine.[4]

As shown in Table 1, lactose-intolerant individuals are able to include dairy products in their diet without experiencing symptoms.

Table 1

Lactose content of various food items[14]

Food product	Serving size	Amount of lactose (g) per serving
Milk (3.5% fat)	150 mL	7.0
Skimmed milk powder	10 g	5.1
Whole-milk powder	10 g	3.5
Sour cream (10% fat)	25 g	0.9
Crème fraîche (30% fat)	25 g	0.6
Cream	15 g	0.5
Whey	150 mL	7.1
Butter	20 g	0.1
Mozzarella cheese (20% fat)	100 g	3.3
Mascarpone cheese	30 g	1
Cottage cheese	30 g	1
Cream cheese	30 g	0.9
Parmesan cheese	30 g	0
Most hard, sliced cheeses (e.g. Emmentaler, Gouda, Edam or Tilsit)	30 g	0
Latte macchiato	125 mL	5.4
Cappuccino	125 mL	2.9
Buttermilk	150 mL	6
Soured milk	150 g	6
Yoghurt	150 g	4.8
Kefir	150 g	5.4

The addition of probiotic bacteria has been shown to lessen symptoms of lactose intolerance;[12] formulations with β-galactosidase activity specifically have been shown to aid lactose digestion and so alleviate symptoms.[2] Fermented cow's milk products such as yoghurt and buttermilk have a lower lactose content than unprocessed milk products owing to the activity of lactose-fermenting organisms during the production process. Hard cheeses typically contain no lactose and the lactose content of butter is negligible.[4,15]

Avoidance or exclusion of dairy foods have notable nutritional consequences. The main concern associated with excluding milk and dairy foods from the diet is a reduced calcium intake, which may lead to rickets in children and low bone mineral density and increased risk of fractures later in life.[4] Milk is considered a complete food[2] and long-term exclusion of milk and milk products from the diet have been shown to result in individuals with CMA having a significantly lower intake of protein and several essential vitamins (e.g. vitamin A, vitamin B_{12} and riboflavin) and minerals (calcium, potassium, phosphorus, magnesium and zinc) compared with controls. Young adults who have had CMA from infancy have also been shown to be at risk of not reaching their full growth potential.[16] Reviews by Amiri et al.[13] and Szilagyi[17] also warn against total exclusion of milk and dairy products from the diet, given the possible role of biologically active compounds in milk and dairy products in cancer prevention.

Conclusion

A food allergy and an intolerance are not the same: the former involves an immune reaction to a food protein while the latter results from an enzyme deficiency that prevents proper digestion of a food substance. Although CMA is a common food allergy among children, most outgrow it within the first five years of life. CMA among adults is rare and often erroneously self-diagnosed. In contrast, lactose intolerance is fairly common in many populations across the world.

However, most people affected by lactose intolerance can include dairy in their diet by reducing the amount of lactose ingested at a time and gradually building up their tolerance. Milk and dairy products are nutrient dense and have an important role in health. Total avoidance of milk and dairy products is therefore rarely necessary and should be implemented only at the advice of a medical practitioner when true CMA has been diagnosed.

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