



City Research Online

City St George's, University of London

Citation: Coutts, A. M. (2013). Lactose intolerance: Causes, effects, diagnosis and symptom control. *Gastrointestinal Nursing*, 11(2), pp. 18-21. doi: 10.12968/gasn.2013.11.2.18

This is the unspecified version of the paper.

This version of the publication may differ from the final published version. To cite this item please consult the publisher's version.

Permanent repository link: <https://city-ref-test.eprints-hosting.org/id/eprint/2377/>

Link to published version: <https://doi.org/10.12968/gasn.2013.11.2.18>

Copyright and Reuse: Copyright and Moral Rights remain with the author(s) and/or copyright holders. Copies of full items can be used for personal research or study, educational, or not-for-profit purposes without prior permission or charge, unless otherwise indicated, provided that the authors, title and full bibliographic details are credited, a hyperlink and/or URL is given for the original metadata page and the content is not changed in any way. For full details of reuse please refer to [City Research Online policy](#).

Lactose intolerance

Introduction

Lactose is a carbohydrate found mostly in dairy products: it is a disaccharide, and is composed of galactose and glucose. For a simple sugar, lactose is rather unusual, as it is less sweet to taste and less water soluble than would normally be predicated. Lactose is digested to its component monosaccharides by the enzyme lactase. Lactase is a brush-border enzyme, which is synthesised by the cells lining the small bowel, and released onto the outside of the plasma membrane; the enzyme works at the cell surface, immediately prior to the absorption of the monosaccharides. If this enzyme is deficient or absent altogether, then lactose intolerance occurs. This is quite different from an allergy to lactose (or milk). Such an allergy would be a disorder of the immune response, and represent a significant, systemic disease. Furthermore it is possible that many people wrongly diagnose themselves as allergic or intolerant to milk, and try to control this by denying themselves milk and other dairy products (NHS Choices 2011). Such people are unnecessarily missing out on an important and nourishing food. For this reason if lactose intolerance is suspected, it should be carefully investigated and diagnosed, prior to the introduction of dietary management.

Types of lactase deficiency

Primary lactase deficiency is the most common type of lactose intolerance (NHS Choices 2011), accounting for 70% of all sufferers (BMJ Best practice). Lactase production decreases in the young child as they become less dependent upon milk in their diet, at around the age of two or three years, although problems may not become apparent until adulthood. It is largely genetically controlled. An individual's response to lactose can thus be classified as lactase persistent (LP), who are able to digest lactose; and lactase non-persistent (LNP) who are not able to digest lactase, and thus experience lactose intolerance (Shrier et al 2008).

As a general rule, populations who trace their origins to equatorial regions and in the East tend to be more prone to lactose intolerance than those from the Polar Regions (Shrier et al 2008). The traditional explanation for this was that individuals living in those regions where the population was dependent upon milk in the diet would experience a severe disadvantage in being lactose intolerant, so that the gene was discouraged. However, populations from polar regions were at risk of vitamin D deficiency, and being able to consume and digest milk may have made calcium available, thus offsetting a deficiency in vitamin D. It has been suggested more recently that the gene for a lack of lactase could also have protected populations from malaria (Shrier et al 2008).

Secondary lactase deficiency is when another condition causes a reduction in the availability of lactase. Such conditions include Crohns disease, gastroenteritis and surgery, and it can also occur as a complication of anti cancer drugs. In these cases the lactase deficiency may be temporary, but it can also be permanent.

Congenital lactase deficiency is present from birth. It is extremely rare; only 40 cases have ever been reported world-wide. Familial lactase deficiency is similar but in this case the lactase is released but does not have the expected effect. Both are inherited autosomal recessive conditions – that is to say that both genes need to be affected for the individual to suffer from the condition.

Developmental hypolactasia sometimes occurs in the premature infant (less than 34 weeks) and improves rapidly as the infant matures. It has further been suggested (Kanabar et al 2001) that transient lactose intolerance is the cause of infant colic in some cases.

Genetics of lactase deficiency

The gene for lactase production is located on the long arm of chromosome 2. Alterations in the promoter region of this gene can lead to LNP. The promoter region is that part of the chromosome that enables the gene itself to function. In LNP people alterations appear that reduce or eliminate the gene's ability to synthesise lactase. These alterations are recessive, which means that the person has to be homozygous for LNP in order to develop lactose intolerance. Furthermore, the alteration can be complete, so that the person does not produce any lactase (anlactasia), or incomplete (hypolactasia), which means that they do not synthesise lactase in the usual quantities. The amount of lactase present will directly influence how intolerant they are to lactose.

The numbers of people affected varies greatly around the world: about 5-15% of people of northern European origin are affected, and in these people LNP tends to occur later, whilst in African American and Asians LNP is almost universal and occurs from a few months old (BMJ Best practice). Overall 70% of the world's population are LNP, and this may be the "natural state" (Roxas 2008), with LP developing later, as populations migrated.

Effects of lactase deficiency

When an LNP individual takes in significant quantities of milk, they are unable to digest the lactose. This disaccharide then passes, unaltered, into the large bowel, where there are two possible outcomes.

1. This disaccharide in the gut is osmotically active, and attracts water into the large bowel, causing the LNP person to experience abdominal cramps and diarrhoea.
2. The lactose is fermented by the micro-organisms that normally inhabit the gut. The result of this fermentation is short chain fatty acids, carbon dioxide, hydrogen and methane. This is often perceived as mitigating the effects of LND (Shrier et al 2008), but it is also possible that these gasses that cause people uncomfortable feelings of being "bloated" and passing unacceptable quantities of flatus.

Individuals who are LNP can, in fact, tolerate some milk; the amount they can manage is very individual, and is best worked out on a case-by-case basis. It is important to stress that a person who is LNP taking milk may experience unpleasant and unacceptable symptoms, but they are not in danger: the symptoms will wear off as the lactose passes through or is fermented. Furthermore, milk is a useful food, and it is worth discovering how much an individual can tolerate, rather than just excluding it from the diet "just to be sure". It is also important to note that many dairy products, for example cheese, butter and yoghurt have minimal quantities of lactose remaining in them, and can be consumed without risk of any discomfort.

Diagnosing lactose intolerance

The simplest diagnostic test is to avoid lactose in the diet, which should lead to an improvement in their condition, which deteriorates again when the lactose is re-introduced. However, this is quite a subjective test, and the diagnostic test of choice is the lactose hydrogen breath test. The patient is fasted then given an oral load of lactose and then a breath test for hydrogen is conducted at regular

intervals over the next two or three hours. Hydrogen is a by-product of colonic anaerobic bacteria acting on un-digested dietary carbohydrate. This hydrogen is released into the lumen of the bowel and is absorbed into the bloodstream to be excreted in the lungs: a value of over 20 parts per million is suggestive of LNP, and a value of 10-20ppm is non-determinate, and needs to be interpreted in view of the presence of symptoms (Roxas 2008). A false negative result can be obtained if the person has recently had oral antibiotics, which will have killed the relevant organisms, and in some people who are resistant to producing hydrogen (BMJ Best practice). False positives can be obtained when bacterial overgrowth has occurred into the small bowel, and indeed the hydrogen breath test is sometimes used to test for this. Raised hydrogen has been found in some infants severely affected by colic (Kanabar et al 2001).

Control of symptoms

Treatment is only required in the presence of symptoms – asymptomatic people should not receive treatment. The mainstay of treatment is to exclude lactose from the diet; this mainly means milk and cream. Cheese and yoghurts can cause symptoms but often do not, as they have less lactose present. Lactose is usually only found in milk and milk products, but it can be incorporated into some prepared meals, particularly foods sold to dieters. People who find they need to exclude all dairy products may need nutritional advice to ensure they obtain all the nutrients they need, particularly Calcium and vitamin D. People who continue to experience symptoms despite apparently eliminating lactose from their diet may also need nutritional advice to help locate any “hidden lactose” (BMJ Best practice). Lactose-free milks, such as soya milk are also commercially available.

Some foods have been pre-treated with lactase, but the evidence of the effectiveness of these in controlling symptoms is unclear (BMJ best practice). However, oral lactase supplementation has been shown to allow even people who are severely LNP to take milk and dairy products without discomfort. A double-blind, placebo controlled trial involved giving babies who were badly affected by colic some lactase, either mixed with the formula milk or after a breast feed. The results of this research was adversely affected by poor compliance to the lactase regime, but some of those babies who experienced severe colic and received lactase cried less and had less hydrogen on their breath than those who received a placebo, but some did not respond. The authors conclude that lactose intolerance is the cause of severe colic in some babies, but others experience colic for other reasons (Kanabar et al 2001).

Conclusion

Lactose intolerance is a very common complaint; it leads to a number of distressing symptoms in those affected. It can be quite easy to control by reducing milk in the diet, and possibly by adding lactase to the diet. However, particularly in the indigenous white population we should be wary of over-diagnosing this condition, and thereby depriving the individual of a useful, cheap and nutritious food.

References

BMJ best practice lactose intolerance <http://0-bestpractice.bmj.com.wam.city.ac.uk/best-practice/monograph/798> accessed February 2013

Kanabar,D.; Randhawa,M.; Clayton,P. (2001) improvement of symptoms in infant colic following reduction of lactose load with lactase *Human nutrition and dietetics* 14 359-363

NHS Choices (2011) Lactose intolerance <http://www.nhs.uk/conditions/Lactose-intolerance/Pages/Introduction.aspx> accessed January 2013

Roxas M (2008) the role of enzyme supplementation in digestive disorders *Alternative medicine review* 13 4 307-14

Shrier, I; Szilagyi, A; Correa, J (2008) Impact of Lactose Containing Foods and the Genetics of Lactase on Diseases: An Analytical Review of Population Data *Nutrition and Cancer*, 60(3), 292–300