Lactose intolerance and lactase activity

INTRODUCTION

Lactose intolerance is a disorder in which the patient experiences unpleasant gastrointestinal symptoms as a consequence of the ingestion of milk or dairy products. It is estimated that up to 15% of the UK population is lactose intolerant. This increases to as many as 95% of Asian populations.

Symptoms of lactose intolerance are usually abdominal pain, nausea, bloating and diarrhoea. These symptoms are believed to be a direct effect of undigested lactose arriving in the distal gastrointestinal tract. Lactose is the main sugar in milk and is broken down in the small intestine by the disaccharidase lactase to glucose and galactose. The failure to breakdown lactose results in the lactose being delivered directly to the more distal gut. To understand the situation further it is necessary to discuss the recognised differences in lactase activity; we can then attempt to explain the variability of symptomatology of at-risk individuals.

Lactase and lactose

Lactose is the main sugar in milk and is vital for the nutrition of all young mammals. In 1901, Reid pointed out that the mucosal lining of the small intestine was able to break down disaccharides. In 1907, Plimmer noted that the ability of the mucosa to break down lactose decreased as infant mammals aged. Following this early work on intestinal lactase activity there was a hiatus for almost half a century. Interest in the subject was revived following the description in the late 1950s by Holzel and Durand of hereditary lactase deficiency. This is a rare condition in which neonates lack small intestinal lactase and develop diarrhoea and malnutrition when fed milk. This condition is rapidly fatal unless lactose is excluded from the diet.

These clinical studies promoted a period of active research into the subject of human intestinal lactase activity and it became clear that, in the human, lactase deficiency occurs in three forms:

i) Congenital lactase deficiency;
ii) Acquired (secondary) lactase deficiency as a result of small intestinal damage, e.g. infective diarrhoea, protein malnutrition, coeliac disease and tropical sprue;
iii) Isolated adult-type primary lactase deficiency.

The description of primary lactase deficiency is technically incorrect, as it has become clear in the last 30 years that this state is the norm for the majority of adults. In spite of this the use of the term primary lactase deficiency has remained.

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Racial differences in intestinal lactase activity

In 1966, a study on prisoners in the US found that 95% of black prisoners were lactase deficient compared to 10% of white prisoners. In a subsequent study on children it was found that intestinal lactase activity was similar in black and white children, indicating a decline in lactase activity in blacks with age. In a subsequent study on white, black and American Indian people it was shown that all children had "normal" lactase levels but that in the non-white groups lactase activity declined at 3-4 years. This decline was not secondary to mucosal damage.

Data from Africa have provided some interesting insight into the variability of lactase activity. In a study of tribespeople in Uganda and neighbouring Rwanda a striking difference in the prevalence of lactase deficiency in neighbouring tribes was identified. They found that the agricultural Ganda people of Uganda had a 90% incidence of lactase deficiency, whereas the nearby Hima of Uganda and the Tussi of Rwanda had incidences of 9% and 16% respectively. These latter two tribes have a tradition of milking cows whereas the Ganda do not keep dairy herds. In a subsequent study of Ganda children it was shown that newborn infants had normal lactase levels but this gradually declined so that most developed lactase deficiency between 3 and 4 years of age, as had been found elsewhere.

The correlation between milk-drinking traditions and lactase persistence in populations led to the geographic evolutionary hypothesis of Simoons, who hypothesised that as lactase "deficiency" after infancy represents the usual mammalian condition, mutant lactase persistence in adulthood would represent a survival advantage to milk drinkers and these would be naturally selected in such societies. Others, however, believe that lactase persistence is the wild type and that lactase deficiency allowed survival advantage by protecting from malaria. They point out that on a global basis, areas of adult lactase deficiency coincide geographically with areas where falciparum malaria is endemic. However, this hypothesis assumes that lactase deficiency discourages milk drinking and there is evidence that this may not be the case.

It has been found that lactase deficiency does not directly correlate with the amount of milk ingested. It was originally believed that all individuals with lactase deficiency would limit their milk ingestion because they would develop unpleasant symptoms as a consequence. How, therefore, do we explain the fact that some individuals with lactase deficiency experience significant symptoms and others do not? Without doubt those with lactase deficiency will deliver a greater amount of undigested lactose to the distal intestine for a given ingested amount. It would therefore appear that each individual has a threshold for tolerating undigested lactose in the distal gut, and only when this is exceeded do they get symptoms. These two factors would account the variable correlation of symptoms after milk and lactase deficiency.

The clinical setting

It can be difficult to identify patients with lactose intolerance. The symptoms are often identical to irritable bowel syndrome, and failure to enquire about milk and dairy product ingestion will result in a missed opportunity. If a patient ingests considerable amounts of dairy product and has troublesome symptoms a trial excluding all dairy is worthwhile. If it is successful, a diagnosis is possible although often it is not so clear cut. Removing dairy products is not ideal as milk offers significant nutritional value. So tests to exclude causes of secondary lactase deficiency are indicated before undertaking a lactose tolerance test or a hydrogen breath test to confirm the diagnosis of lactase deficiency.

Once a diagnosis has been confirmed, the management of lactase deficiency is not straightforward. Not all individuals will have lactose intolerance, but a supervised period of lactose free is a logical approach to assess response. The period of lactose-free is difficult to maintain. The provision of lactose-free products is not patient friendly, and limits the patient's calcium and riboflavin intake. A new product has been launched which improves the situation for individuals with lactose intolerance. Lactofree is a product in which lactose has been actively removed from pasteurised semi-skimmed milk. It results in a 99.95% lactose-free drink that can be used as an alternative to milk without the loss of other useful nutrients. It is anticipated that this product will improve the quality of life of patients with lactose intolerance significantly.

lactofree