Adaptation to lactose in lactose malabsorbers – importance of the intestinal microflora

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ABSTRACT

At high intakes most lactose in lactose malabsorbers will be fermented by the intestinal microflora to hydrogen and other fermentable carbohydrates. By adaptation higher intakes of lactose could be tolerated partly due to a lower net formation of hydrogen. This shift in fermentation is at least partly caused by a change in the activities of the intestinal microflora.

Keywords: Adaptation, hydrogen production, lactose malabsorption, intestinal microflora

Lactose as a substrate to the intestinal microflora

Lactose malabsorbers will only digest minor amounts of lactose in the small intestine due to low lactase activity. At higher intakes most of the lactose will be fermented by the intestinal microflora. Lactic acid bacteria, and other bacteria able to ferment lactose, will perform the first step in the fermentation chain. They will form intermediate substances like galactose, lactic and acetic acid with only a minor amount of hydrogen produced. Some of the intermediate substances will be absorbed by the human intestine, some will be fermented to organic acids and other compounds such as hydrogen, methane and sulphite (1,2). When the formation of gaseous compounds is higher than the absorption through the intestine, the surplus could cause symptoms characteristic of lactose intolerance, such as flatulence, diarrhoea and abdominal pain. The symptoms are identical to those caused by other low-molecular, non-absorbable, fermentable carbohydrates as exemplified by lactulose, fructooligosaccharides and sorbitol partly used as sugar substitutes (3,4).

Adaptation to lactose

Adaptation to lactose has been investigated and demonstrated in at least four different studies (5-8). Healthy lactose-maldigesters of different ages have been given lactose for 10, 14, 21 and at most 88 days. Lactose intake in the different experiments was either constant at about 30 g daily or increased from 5–20 g at the beginning up to 8–65 g daily at the end of the studies. Hydrogen production decreased during the adaptation periods as measured both by breath test and by fewer symptoms of flatulence. Likewise the activity of faecal β-galactosidase increased during adaptation. Thereafter an improved capacity to ferment lactose without gastrointestinal symptoms was proven as the severity of the symptoms caused by lactose challenge was reduced in comparison to those reported before adaptation. In control groups given dextrose or sucrose as a placebo, no impact either on hydrogen production or on faecal β-galactosidase was documented after lactose provocation. However, results from one of the investigations indicated that factors other than adaptation of the microflora might be of importance. Symptoms after lactose intake in both the experimental and the placebo group were reduced at the end of the intervention period, indicating that the strength of the symptoms might be partly due to individual variations (8).

Evidence of adaptation of the intestinal microflora

The impact on the hydrogen excretion might be due either to a lowered production of hydrogen by the intestinal microflora. Hydrogen could be used to form methane or acetic acid or to reduce sulphate (2). To estimate the relative importance of hydrogen production and of consumption, faecal slurries were incubated with lactose (10). The faecal samples were taken after a 10-day period of lactose/dextrose adaptation. Hydrogen production was three times lower from slurries after lactose adaptation in comparison to dextrose. Hydrogen consumption was estimated by comparing data from slurries with or without inhibitors to known consumption reactions. Data indicated that consumption was of minor importance (10).

The net reduced hydrogen formation noticed after an adaptation to lactose could have several different explanations. A prolonged oroacetal transport time will result in an increased hydrolysis of lactose by remaining lactase and an increased absorption of galactose, lactic acid and acetic acid formed by homofermentative lactic acid bacteria and bifidobacteria. Although not proving by ex-vivo studies, hydrogen consumption might still be of importance. Studies indicate that acetogenesis, formation of acetic acid from hydrogen and carbon dioxide, might be stimulated by a low pH produced by fermentation of the lactose (2,12).

Conclusions

Adaptation to lactose allows lactose maldigesters to consume milk and other dairy products without symptoms caused by the formation of hydrogen. Further studies are needed to evaluate the different aspects of the adaptation process including the importance of the gastrointestinal microflora.

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