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When Does Lactose Malabsorption Matter in Adults?

Generations of medical students have been taught that dietary carbohydrates are completely broken down in the intestinal lumen and the resultant monosaccharides (mainly glucose) then absorbed. This is now known to be wrong; in fact hydrolysis in the bowel lumen is incomplete, so that a mixture of small sugar molecules, especially disaccharides, is produced, which are finally split by their own specific enzymes located on the microvilli (brush border) of the intestinal epithelial cells—so-called membrane digestion. Milk sugar (lactose) accounts for 10% of the daily carbohydrate intake in Western countries,[1] and it is digested by the enzyme lactase into its two component monosaccharides, glucose and galactose. Absence of this enzyme has aroused much interest and controversy in recent years.

In 1959 Holzel described[2] congenital absence of lactase in two siblings whose intestines were otherwise normal. In this very rare condition, which is probably inherited as an autosomal recessive trait,[3] chronic watery diarrhoea followed by vomiting develops in infants during the first few days of life, with subsequent failure to thrive. Severe malnutrition may be caused, but it will respond dramatically to the elimination of lactose from the diet. This congenital lactose malabsorption must be distinguished from another variety of lactose malabsorption, found in older children and adults, described in 1963 by both Dahlqvist[4] and Auricchio.[5] The condition has a confusing array of names emphasizing either lactose malabsorption, lactase deficiency, or alactasia—primary lactose malabsorption, selective lactose malabsorption, constitutional hypolactasia, and acquired lactase deficiency in the adult. Intestinal lactase levels are high at birth but decrease in childhood or adolescence; the other disaccharidases are not affected and the small intestine is otherwise structurally and functionally normal. Lactose malabsorption may appearas early as six months of age or as late as 20 years.[6] Lactase disappears in a similar way in all mammals in association with weaning.

Yet another type of lactase deficiency—secondary lactose malabsorption—reflects damage to the small intestinal mucosa by gastroenteritis, coeliac disease, or protein malnutrition. It is part of a general malabsorption syndrome affecting other disaccharides and monosaccharides, though lactose tends to be the first enzyme to disappear and is the last to recover with treatment. The diarrhoea of lactose malabsorption is superimposed upon that of the primary disease. Treatment is of the underlying bowel disorder, but elimination of milk from the diet may be necessary to reinforce conventional treatment, especially in children.[7]

When dietary lactose is not absorbed it remains in the intestinal lumen, where it acts as an osmotic load, attracts fluid into the bowel,[8] and increases motility. When lactose reaches the colon it is degraded by bacteria into short-chain organic acids, which further increase the osmotic pressure and produce watery acid stools with a characteristic odour. The clinical symptoms are abdominal colic, bloating, borborygmi, flatulence, and frothy diarrhoea. This fermentative diarrhoea may be suspected if the stool pH is found to be less than 6. Unfortunately this is a useful screening test only in children; adult stools usually[9] have a normal pH. Lactose malabsorption should, therefore, be diagnosed by the lactose tolerance test, in which 50 g of the sugar is given by mouth and a blood glucose curve plotted; a peak rise of less than 20 mg/100 ml is diagnostic. The accuracy of the test may be improved by measuring blood galactose levels after blocking its metabolism with ethanol.[10] However, measurement of blood sugar levels is really a refinement, because patients with lactose malabsorption will usually develop symptoms after ingesting such a large load of lactose, and a simple screening test may be performed by giving the patient 50 g lactose on one day and 50 g glucose on the next and asking him to report any symptoms induced. Secondary lactose malabsorption is identified by repeating the tolerance test with a mixture of 25 g glucose and 25 g galactose. Final proof of the diagnosis has to be obtained by measuring the lactase activity of a jejunal biopsy specimen. Other screening tests include the lactose-barium meal[11] and a carbon-14 breath test,[12] but these are usually unnecessary. Treatment is by a milk-free diet; a low lactose diet may be prescribed, but it is much more difficult to adhere to and needs to be supplemented with extra calcium.

Soon after the discovery of lactose malabsorption in adults it was noted that it was much more common in Cypriots living in Camden Town than in native Londoners[9]; negroes in the U.S.A. and Africa also showed a high incidence.[13 14] In the years that followed a flood of publications from all over the world indicated a definite racial variation. In most ethnic groups the prevalence of lactose malabsorption is over 70%, whereas people of North European stock tend to have a low prevalence, always less than 20% and often about 6%. Is this due to genetic differences or does it reflect an adaptation to the lack of milk in the diet of most races? Though lactase

may be an adaptive enzyme in rats, there is no evidence that lactose feeding prevents lactase deficiency in man or that lactose exclusion will produce it.[15] Moreover, several small family studies have suggested a genetic aetiology,[16 17] and a large family study with detailed analysis performed in Finland has recently indicated that inheritance is by a single autosomal gene.[6 18] The differences in frequency of the gene around the world are probably due to evolutionary selection associated with milk consumption. North Europeans may have become lactose tolerant owing to the advantage of lactose-induced enhancement of calcium absorption in an environment with little ultraviolet light and insufficient vitamin D in the diet.[19]

British patients with lactose malabsorption rarely present with chronic diarrhoea unless they suffer from a second gastrointestinal disorder such as Crohn's disease. If a patient with long-standing abdominal symptoms is found to have lactase deficiency it is likely to be a coincidental finding from thorough investigation; such patients often have symptoms due to psychological disturbance. The clinical significance of lactose malabsorption depends on how much milk the patient drinks and how quickly he consumes it. A 50 g test dose of lactose almost invariably produces symptoms. Nevertheless, a litre of milk contains only 40 g lactose, and if milk is taken in smaller amounts throughout the day symptoms are unlikely to occur. A study[20] in the U.S.A. not surprisingly showed that the smallest amount of lactose which would cause symptoms varied: 240 ml milk produced symptoms in 75% of subjects but 60 ml only occasionally caused diarrhoea and abdominal pain. Rarely individuals may be found who are sensitive to minute quantities such as those found in the lactose coating of drugs.[21]

In view of the rather vague symptoms and their uncertain relation to milk, it has been postulated that the irritable bowel syndrome might be due to occult lactose malabsorption. One study from Oxford suggested that patients with an irritable colon did not have a higher incidence of lactose malabsorption than the normal population[22]; but lactose malabsorption was found more often in Danish patients with an irritable colon, though a history of milk intolerance had no diagnostic value.[23] Nevertheless, elimination of milk often improved their symptoms. A controlled study from Finland has also shown that when lactose malabsorption was diagnosed in patients with nonspecific abdominal symptoms there was an excellent chance of improvement on a lactose-free diet.[24] A lactose tolerance test is, therefore, worthwhile in patients with irritable bowels who do not respond to the usual measures; the effect of milk withdrawal should be tried if the curve is flat. Conversely, chronic abdominal symptoms should not be attributed to lactose even in the presence of lactase deficiency if they do not respond to milk withdrawal; another possible cause should always be sought.

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A Criminal Approach to Abortion

Discussion and debate about abortion might have been expected[1] to fade away after the publication a year ago of the report[2] of the Lane inquiry into the working of the 1967 Abortion Act. The committee of doctors, lawyers, nurses, and laymen had made a detailed, exhaustive examination of practices in the N.H.S. and private hospitals and clinics and had concluded that the positive gains resulting from the Act had much outweighed any disadvantages for which it had been criticized. In particular, the report stated that most N.H.S. abortions and many in the private sector had been on grounds within the terms of the Act; and its main criticism was of the inequalities in the Service offered to individuals throughout the country: "too many women," it said, "have been forced to pay for abortions when they had legitimate medical grounds for termination of pregnancy."

Yet a year later an Abortion (Amendment) Bill has received a second reading, and a select committee of the House of Commons is holding what is in effect another inquiry into the working of the Act.[3] If made law, the amendments would restrict the legal indications for abortion (other than in cases of the likelihood of fetal abnormality) to grave risks to the life of the woman and risks of serious injury to the physical and mental health of the woman or any existing children of her family. It would also, among other changes, make it an offence for a doctor to give advice on obtaining an abortion to a girl under the age of 16 unless her parent or guardian (if ascertainable and willing to be present) was included in the consultation; or for a doctor to discuss abortion without giving advice as to available alternatives. Furthermore, if a doctor who had terminated a pregnancy was charged with contravention of the (amended) Act, "the onus of proof that the regulations had been complied with . . . would rest with the accused person." Finally the Bill would guarantee anonymity for anyone giving evidence in legal proceedings under the Act.

These provisions represent a serious threat to the professional freedom of doctors. In assessing an individual case the question would no longer be what was best for the patient: the legal test would be whether or not a court would regard the risks to life or health as "grave" or "serious." And what is the general practitioner to do when faced with a pregnant 15-year-old who refuses to let him tell her parents of her condition? The Lane Committee had suggested that every effort should be made to involve parents in the discussion; the amendment Bill requires a doctor to tell such a girl that he could not discuss even the possibility of termination of her pregnancy unless her parents were present; and he might later be called on for proof of his version of the consultation in court. The extraordinary provision on onus of proof is, indeed, a denial of the fundamental legal presumption of innocence until guilt has been proved.

Clearly the intention of the sponsors of the Bill is to reduce the number of terminations of pregnancy by making the