should have been picked up at the examination performed at 6 weeks of age. Interestingly one of these boys had no previous records of examination, the second was reported as having both testes palpable in the scrotum at 6 weeks, while the third had in fact had a left herniotomy at 5 months of age but required a left orchidopexy aged 5 years. In addition we know of at least one child in our sample who has had an orchidopexy who was recorded as having normally descended testes at the school medical.

One suggested advantage of the present unselective system is that of parental reassurance. Also the presence of a captive audience of parent and child in school has potential opportunities for health education. It is arguable, however, that little real value comes from a 10 minute physical examination, in addition to the questionnaire and the nurses' screening checks.

It has been suggested that this medical may be used for neurodevelopmental assessment.[4] However there are several difficulties with this. Firstly, children with appreciable problems will probably already have been identified by the age of 5. Secondly, because there is a wide variation of normal and many signs are 'soft' there is a danger of labelling a normal child as one with a problem. An additional factor to consider is one of time. Bax and Whitmore estimated that 15–20 minutes was required per medical.[4] This would increase by between 50–100% the estimated 500–600 sessions per year presently required in North Staffordshire and in our

opinion would require justification that real benefit would accrue.

The results of this study provide further evidence that a policy of selection of children for medicals at school entry is a reasonable one. This should be performed one term after school entry to allow time for teacher assessment. The selection process would be preceded by the screening of all children for hearing, vision, and growth problems by the school nurse. A class review would then be carried out by a named school doctor, school nurse, and the class teacher with the aid of a questionnaire completed by the parents and the preschool records. Criteria for selection for examination by the doctor should include: poor preschool records, previously identified problems requiring follow up, concern expressed by the nurse or teacher, and parental request. By adopting such a policy school doctors would be freed from performing unnecessary medicals on healthy children without fear of missing any important problems and this would allow more time to be concentrated on children in mainstream schools with special needs.

1 Kennedy FD. Have school entry medicals had their day? Arch Dis Child 1988;63:1261–3.
2 O'Callaghan EM, Colver AF. Selective medical examinations on starting school. Arch Dis Child 1987;62:1041–3.
3 Hadziselimovic F. Pathogenesis of cryptorchidism. In: Kogan SJ, Hafez ESE, eds. Pediatric andrology. Vol 7. The Hague: Martinus Nijhoff, 1981:147–62.
4 Whitmore K, Bax M. The school entry medical examination. Arch Dis Child 1986;61:807–17.

Isolated fructose malabsorption

J K H Wales, R A Primhak, J Rattenbury, C J Taylor

Sheffield Children's Hospital, Department of Paediatrics
J K H Wales
R A Primhak
C J Taylor

Department of Chemical Pathology
J Rattenbury

Correspondence to:
Dr C J Taylor,
Department of Paediatrics,
Sheffield Children's Hospital,
Sheffield S10 2TH.

Abstract
A patient with isolated fructose malabsorption presented with diarrhoea and colic during the first year of life and subsequently responded to a fructose free diet. Fructose malabsorption has been implicated in some cases of irritable bowel syndrome in adults and may also be an infrequently recognised cause of gastrointestinal symptoms in children.

Fructose is a monosaccharide and a component of the disaccharide sucrose that is increasingly being used as a 'natural' sweetener in processed foods. It was previously supposed that fructose was absorbed passively, and thus specific malabsorption states should not occur as they do for sugars that require brush border enzyme systems for their digestion and absorption. A low capacity, oxygen dependent transport system for fructose alone has been shown in the small bowel,[1] which is independent of the high capacity system used when fructose is absorbed together with glucose.[2]

Fructose malabsorption is a cause of recurrent abdominal pain in adults[3] but in children only a single case (diagnosed in a 12 year old) has been described.[4] In addition, asymptomatic adult volunteers have been shown to be intolerant of loading doses of fructose.[5] Fruit juices containing fructose have been implicated as a cause of 'toddler diarrhoea', but symptoms were thought to be more related to the sorbitol component of the drinks than the fructose they contained.[6]

We describe a case of isolated fructose malabsorption diagnosed in the first year of life.

Case report
A baby girl was the product of a healthy term pregnancy and the second child born to unrelated parents. Her birth weight was 4000 g and there were no serious neonatal problems. She fed poorly from the breast, so was given cows' milk formula complements from 2 days of age, and fennel or blackcurrant drinks from 1 week of age. She was changed entirely to formula feeds at 4 weeks and rice weaners were added at the age of 6 weeks.

Between 7 and 12 weeks of age she was admitted to hospital twice with a history of screaming after feeds, but was discharged after observation without a specific diagnosis having been made. A diet that contained no cows' milk, and various medications produced no relief of symptoms. The screaming bouts continued and she was reported to be 'windy' and have abdominal distension, the attacks being worse after she had had orange juice and bananas. At 7 months she was still thriving (weight >97th centile) with no abnormal findings on physical examination. A stool sample showed reducing substances with a pH of 5 on ward testing, yet chromatography failed to identify a specific sugar. In view of the association of symptoms with fruit and fruit juices the possibility of fructose malabsorption was raised and further investigations carried out.

The results of breath hydrogen tests carried out when she was 8 months old using loading doses of 2 g/kg sugar orally are shown in fig 1. A loading dose of sucrose gave an increase in hydrogen production (>20 ppm) at 45 minutes but no symptoms. A higher peak was seen at 75 minutes after a loading dose of fructose; this was associated with symptoms of bloating and screaming. Lactose produced a small peak at 15 minutes suggestive of bacterial overgrowth, but this was not confirmed on culture of jejunal juice.

A jejunal biopsy specimen showed normal villous architecture and normal assays of disaccharidases (all values expressed as U/g protein): lactase 59 (normal 6–55), trehalase 27 (normal 10–80), sucrase 120 (normal 40–152), and maltase 403 (normal 130–456). The tissue activities of fructose-1-phosphate aldolase and

fructose 1, 6–diphosphate aldolase were 122 (normal 107–517) and 226 (normal 218–1330) μmol/min/mg protein, respectively, and the ratio between these enzymes was 1·85 (normal 1·22–4·26). An intravenous loading dose of 2·5 g fructose failed to cause a fall in plasma glucose or phosphate concentrations, excluding the diagnosis of hereditary fructose intolerance.

After the introduction of a diet containing no fructose or sucrose there was a remarkable improvement, but her symptoms recurred* immediately when she was challenged with fructose. The child remained free of symptoms when sucrose alone was subsequently added to her diet. Further bouts of bloating and screaming occurred after accidental ingestion of fructose. When she was challenged with 20 g fructose at 2 years of age, she developed colic and loose stools, but a satisfactory breath sample was not obtained.

She presented to the clinic again at 4 years and 9 months of age with a six month history of episodic abdominal pain and distension that was followed by bouts of offensive stools that scalded the perineum. She had continued to avoid fruit, but had generally relaxed her other dietary restrictions. Her height and weight remained above the 90th centile.

A challenge with 15 g fructose orally caused a bout of abdominal pain but no alteration in the appearance or acidity of the stool. Measurement of her blood fructose concentration showed a minimal rise compared with a normal response of 0·7–0·9 mmol/l at 30 minutes to one hour, confirming malabsorption of the sugar (fig 2). A dose of 15 g glucose produced a normal rise in blood sugar of 4·0 mmol/l at 30 minutes. A jejunal biopsy specimen again showed normal villous architecture and levels of brush border disaccharidases. The patient was again placed on a strict diet containing no fructose with good effect.

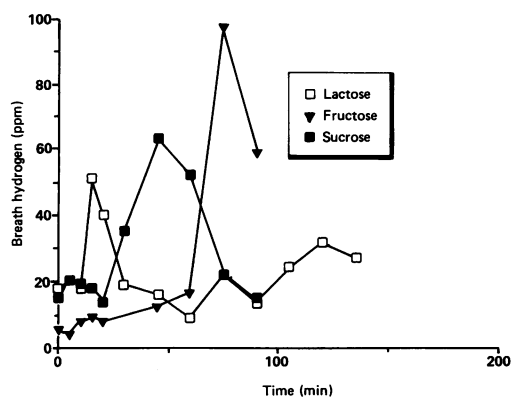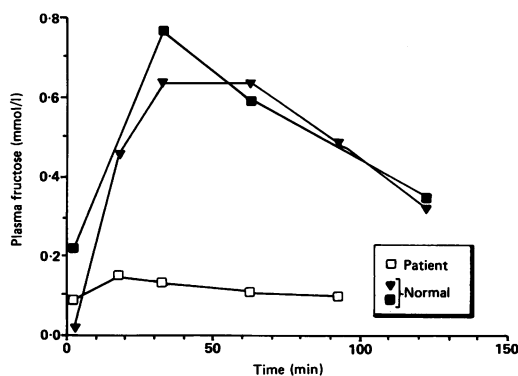*Figure 1 Patient's breath hydrogen response to 2 g/kg oral sugar loads at 8 months of age.*

Figure 2 Response of patient at 4·8 years old compared with normal response to 2 g/kg load of oral fructose.

Discussion

Specific malabsorption of a monosaccharide can only occur if there is active transport of the sugar by the enterocyte. Although it was thought that fructose absorption was passive, Gracey et al showed an oxygen dependent transport system for fructose that was able to maintain a concentration gradient across perfused rat intestine.[1] Recently Rumessen and Gudmand-Hoyer, using absorption studies in human adults, have shown both a high capacity system that allows fructose absorption in the presence of free glucose, and a low capacity transport mechanism for the free monosaccharide that is selectively inhibited by sorbitol (a naturally occurring alcohol present in many fruit preparations).[2 3 5] The high capacity system may simply represent solvent drag resulting from active glucose transport, and would explain the initially puzzling observation that adults who have fructose malabsorption can tolerate both sucrose, and fructose combined with glucose. The inhibition by sorbitol may, in part, arise from a negative osmotic effect on passive fructose absorption. This could account for the symptoms in a proportion of children with

'toddler diarrhoea' when given apple juice or other fruit drinks containing sorbitol.[6]

The incidence of clinical fructose malabsorption is uncertain. Rumessen and Gudmand-Hoyer showed that eight of 10 healthy adult volunteers failed to absorb 50 g of fructose, but only one of 10 failed to tolerate 15 g. They all absorbed 100 g of sucrose or a mixture of 50 g fructose and glucose. None had symptoms related to their malabsorption.[2] In a further study they showed malabsorption of fructose in seven of 10 adult volunteers who were given 5 g of fructose and sorbitol concurrently.[5] A connection between 'irritable bowel syndrome' and fructose malabsorption has also been proposed. The same authors studied the absorptive capacity of 25 patients with functional bowel symptoms aged between 31 and 77 years, and found that 13 of the 25 showed abnormal breath hydrogen peaks and symptoms after taking 25 g fructose.[3] The symptoms were made worse by the addition of sorbitol, but there was no reaction to sucrose or sorbitol alone.

Fructose intolerance—reproducible unpleasant symptoms in response to an oral challenge—does not therefore always follow fructose malabsorption. Our patient had both malabsorption and intolerance of fructose but tolerated glucose. The child clinically tolerated sucrose, but showed an intermediate degree of sucrose absorption on breath hydrogen testing. Presumably the normal sucrase activity hydrolysed the disaccharide and liberated free glucose, which then facilitated fructose transport (as was shown in studies in adults).[2] This suggests that the low capacity oxygen dependent transport system was defective in our patient.

Abdominal colic and chronic non-specific diarrhoea are common complaints in infancy and childhood. One similar case of diarrhoea secondary to fructose malabsorption has previously been described, but the diagnosis was not recognised until the age of 12 years, although the symptoms began in early childhood as did those of our patient.[4] The apparently high incidence of this condition in adults, and its possible relevance to 'functional' bowel symptoms suggest that it should be more actively considered in children with these symptoms, especially if they follow the ingestion of fruits or fruit juices.

1 Gracey M, Burke V, Oshin A. Intestinal transport of fructose. *Lancet* 1970;ii:827–8.
2 Rumessen JJ, Gudmand-Hoyer E. Absorption capacity of fructose in healthy adults. Comparison with sucrose and its constituent monosaccharides. *Gut* 1986;27:1161–8.
3 Rumessen JJ, Gudmand-Hoyer E. Functional bowel disease: malabsorption and abdominal distress after ingestion of fructose, sorbitol, and fructose-sorbitol mixtures. *Gastroenterology* 1988;95:694–700.
4 Barnes G, Mc Kellar W, Lawrance S. Detection of fructose malabsorption by breath hydrogen test in a child with diarrhea. *J Pediatr* 1983;103:575–7.
5 Rumessen JJ, Gudmand-Hoyer E. Malabsorption of fructose-sorbitol mixtures. Interactions causing abdominal distress. *Scand J Gastroenterol* 1987;22:431–6.
6 Hyams JS, Etienne NL, Leichtner AM, Theuer RC. Carbohydrate malabsorption following fruit juice ingestion in young children. *Pediatrics* 1988;82:64–8.

Dietary management of D-lactic acidosis in short bowel syndrome

A J Mayne, D J Handy, M A Preece, R H George, I W Booth

Institute of
Child Health,
University of
Birmingham and
The Children's
Hospital, Birmingham
A J Mayne
D J Handy
M A Preece
R H George
I W Booth

Correspondence to:
Dr I W Booth,
Institute of Child Health,
Francis Road,
Birmingham B16 8ET.

Accepted 26 October 1989

Abstract
Manipulation of carbohydrate intake was used to treat severe, recurrent D-lactic acidosis in a patient with short bowel syndrome. Dietary carbohydrate composition was determined after assessment of D-lactic acid production from various carbohydrate substrates by faecal flora in vitro. This approach may be preferable to repeated courses of antibiotics.

D-lactic acidosis is a well recognised, but rare, complication of short bowel syndrome and intestinal bypass surgery.[1 2] To date, seven children have been reported in whom this disorder occurred as a complication of short bowel syndrome. Non-absorbed carbohydrate is fermented by colonic organisms, but lactobacilli when present, unlike most other bacteria, produce D-lactic acid, which cannot be metabolised by D-lactate dehydrogenase. Absorption leads to a severe metabolic acidosis. Treatment has been directed at altering the colonic flora with antibiotics,[2 3] or by administering a standard bacterial flora orally.[3]

Recently we have successfully treated a girl with this disorder by dietary carbohydrate manipulation alone.

Case report
After massive small bowel resection for a spontaneous small intestinal volvulus, which followed a vigorous session of disco dancing, a 9 year old girl was left with 14 cm of jejunum beyond the duodenojejunal flexure; the ileocaecal valve was preserved. After prolonged parenteral nutrition she was discharged nine months later on a normal diet together with energy supplements given as sip feeds.