

The practical significance of lactose maldigestion in institutionalised black children

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Summary

A study was carried out to determine the practical significance of a high prevalence of lactose maldigestion in institutionalised children whose diet included 500 ml milk daily.

Thirty of 34 children at a child welfare home were found to be lactose maldigesters as judged by a 2-hour rise in breath hydrogen of 20 parts per million or more after an oral load of lactose.

Breath hydrogen tests were also performed on the same group of children, before and up to 150 minutes after the routine mid-morning cup of milk. Sixteen of the 30 lactose-maldigesting children did not show increased breath hydrogen up to 2.5 hours after milk. No children were clinically intolerant of either the lactose or the milk.

In these children the degree of lactose digestion was much improved in the non-fasting state when measured by the breath hydrogen response to milk lactose. Lactose maldigestion *per se* is not a contraindication to institutional feeding routines, including regular moderate milk intake.

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Intestinal lactase deficiency develops progressively during childhood in genetically predisposed individuals. It is very common among black children, who can be shown to have lactose maldigestion from 3 years of age.¹

In the vast majority of cases, this is not considered to be of clinical significance. In North American studies, it was shown that the ingestion of 240 ml milk daily was tolerated well by lactose malabsorbers.² In addition, most children in communities with a high prevalence of lactose maldigestion do not drink significant quantities of non-human fresh milk. In Africa, fermented milk products with a low lactose content are usually preferred by the majority of the population.³ This may be due

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as much to difficulties in storage of fresh milk as to milk intolerance.

Institutional routines of child feeding in hospitals, school hostels and children's homes often include a fairly large intake of milk. In children with lactose maldigestion, this could potentially lead to bloating and jeopardise the nutritional state through its osmotic effect of increased intestinal motility and decreased carbohydrate absorption. On the other hand, practical experience indicates that milk can be used very successfully in most children.

A study was therefore performed on a group of institutionalised children with a previously documented high prevalence of lactose maldigestion,⁴ to determine any clinical consequences of a daily intake of approximately 500 ml milk and to compare the breath hydrogen response to a standard fasting lactose of 1 g/kg with that to a 200 ml cup of fresh dairy milk, given 2 hours after a normal breakfast.

Subjects and methods

The study was conducted at a children's home under the direction of the Durban African Child Welfare Society. For ethical reasons concerned with research on institutionalised children only totally non-invasive tests were considered, and appropriate ethics committee consent was obtained as well as permission from the agency controlling the institution.

The diet at this home is strictly controlled and includes approximately 500 ml of fresh milk daily, given with breakfast cereals and as a mid-morning and late afternoon drink of 200 ml. Children over the age of 2 years were studied when they were clinically well and had not suffered from diarrhoea or been on antibiotic treatment for the preceding 4 weeks. They were weighed barefoot in light indoor clothing on a superior-type bathroom scale. A portable measuring board was used to measure height to the nearest 0.5 cm. The values were compared with the National Center for Health Statistics (NCHS) tables⁵ and expressed as standard deviation scores (SDSs), calculated by the equation

$$\text{SDS} = (\text{actual measure} - \text{NCHS measure}) / \text{NCHS SD.}$$

In an attempt to obtain a measure of relative abdominal bloating, the ratio of chest circumference at the nipple line to abdominal circumference at the umbilicus was determined.

The occurrence of lactose maldigestion was studied by means of the breath hydrogen response to lactose 1 g/kg, given as a 10% solution in the fasting state. Mixed expiratory air was sampled by means of a face-mask sampling device and analysed immediately in a Hoekloos Hydrogen Breath Tester.⁶ A rise in breath hydrogen of 20 parts per million (ppm) or more within 2 hours was taken to indicate lactose maldigestion.⁷

Breath hydrogen tests were then performed on the same children when they had received their usual mid-morning cup of 200 ml fresh milk. This is given approximately 2 hours after breakfast, which also includes some milk, and 2 hours before lunch. The children were closely observed for the development of any symptoms and breath samples were analysed for hydrogen at 30-minute intervals for a period of 2,5 hours after the milk, the last sample being obtained after lunch.

The milk-lactose intake was related to the children's body weight and to the breath hydrogen response.

Statistical evaluations were performed at the Institute of Biostatistics of the South African Medical Research Council.

Results

Thirty-four children were included in the study, of whom 26 were boys and 8 girls. Their mean age was 53 ± 15 months (range 36 - 106 months). They had been living at the home for a mean period of 31 ± 17 months (range 8 - 83 months) before the study. Fifteen of the children had been admitted to the home after hospital recovery from kwashiorkor; 6 were known to have suffered from recurrent diarrhoea; and the other 13 had been committed because of a variety of social reasons including abandonment, abuse or neglect.

On comparison with the NCHS tables, the children were stunted (height SDS ± SD -1,36 ± 1,03) and underweight (weight SDS -0,69 ± 0,96), but the weight-for-stature SDS of + 0,26 ± 0,87 suggested a normal recent nutritional state.

TABLE I. BREATH HYDROGEN TESTS AFTER LACTOSE AND AFTER MILK

	Lactose digesters	Lactose maldigesters	Total
Lactose (1 g/kg)	4	30	34
Milk (200 ml)	16	18	34

P < 0,001; McNemar test of symmetry.

The fasting lactose breath hydrogen tests showed 30 of the 34 children to be lactose maldigesters. In these 30 children, mean breath hydrogen rose from 2 ± 4 ppm to 73 ± 43 ppm at 90 - 120 minutes.

The non-fasting milk-lactose breath hydrogen tests showed a significantly lower prevalence of elevated breath hydrogen levels (*P* < 0,001; McNemar test of symmetry) (Table I). Sixteen of the 34 children had breath hydrogen responses less than 20 ppm at 150 minutes, but no lactose-digesting child was unable to digest milk-lactose. The positive breath hydrogen responses occurred later after milk-lactose than after lactose (Table II). The maximal breath hydrogen response was expectedly lower after milk-lactose than after lactose (42 ± 19 v. 93 ± 46 ppm; *P* < 0,001; Student's *t*-test), but 200 ml milk provided only 0,64 g/kg milk-lactose (range 0,45 - 0,83 g/kg), less than the 1 g/kg used in the lactose breath test.

TABLE II. BREATH HYDROGEN RESPONSES AFTER LACTOSE AND AFTER MILK

	No. of subjects and time of hydrogen response			Total
	< 20 ppm	≥ 20 ppm at 120 min	≥ 20 ppm at 150 min	
Lactose (1 g/kg)	4	30		34
Milk (200 ml)	16	8	10	34

Both lactose 1 g/kg and fresh milk 200 ml were very well accepted and tolerated by all children, and no abnormal symptoms were recorded.

When the children were divided into milk-lactose-digesting and maldigesting groups on the basis of the breath hydrogen response at 150 minutes (Table III), the comparison showed a trend towards a poorer nutritional state among the milk-lactose-maldigesting children, as indicated by lower weight and weight-for-height SDSs. There was no difference in the ratio of chest to abdominal circumference. There was no difference in the amount of milk-lactose ingested relative to body weight in the two groups, but the comparison showed a higher maximal breath hydrogen level after lactose in those who were milk-lactose maldigesters than in the milk-lactose-digesting children (93 ± 46 v. 62 ± 39 ppm; *P* = 0,058; Student's *t*-test).

TABLE III. COMPARISON OF MILK-LACTOSE DIGESTERS AND MILK-LACTOSE MALDIGESTERS

	Milk-lactose digesters (<i>N</i> = 16)	Milk-lactose maldigesters (<i>N</i> = 18)
Dose of milk-lactose (g/kg) (mean ± SD)	0,61 ± 0,11	0,64 ± 0,11
Height SDS (± SD)	-1,12 ± 1,04	-1,51 ± 1,03
Weight SDS (± SD)	-0,36 ± 0,94	-0,99 ± 0,93
Weight by stature SDS (± SD)	+0,57 ± 0,97	-0,02 ± 0,68
Maximum breath hydrogen after lactose (ppm) (mean ± SD)	62 ± 39	93 ± 46
	(<i>N</i> = 12)	
Maximum breath hydrogen after milk (ppm) (mean ± SD)	7 ± 6	42 ± 19
Chest/abdomen circumference ratio (mean ± SD)	1,05 ± 0,07	1,05 ± 0,06

Discussion

Many studies have confirmed that lactose maldigestion is not a contraindication to the use of milk in the refeeding of malnourished patients.⁸ Bowie⁹ noted that nitrogen retention and fat absorption is normal in kwashiorkor patients with lactose-induced diarrhoea. In a study of lactose-maldigesting school-children on marginal diets, Brown *et al.*¹⁰ found that low-dose milk supplements were well utilised, although studies in malnourished children suggested a tendency to decreased weight gain with lactose-containing milk, compared with lactose-hydrolysed milk feeds.¹¹

In children who are institutionalised because of poor social circumstances, the prevalence of lactose maldigestion is increased by an additional influence of malnutrition-induced or mucosal injury-induced lactase deficiency. We found the usual age-related progressive rise to be replaced by a uniformly high prevalence of lactose maldigestion in a population of institutionalised children above 2 years of age, most of whom had a history of previous malnutrition or recurrent diarrhoea.⁴

The children studied here were on a documented adequate diet including milk 25 - 40 ml/kg/d, and the question posed was whether lactose maldigestion in such children was of any practical significance, and whether the breath hydrogen response to milk given as part of the routine feed, rather than to a fasting lactose challenge, could explain why milk is used so successfully in the feeding of lactose-maldigesting children.

None of the lactose-maldigesting children developed symptoms of intolerance on a lactose dose of 1 g/kg, equivalent to 20 ml/kg of milk. This appears to be a tolerable level of a single dose of lactose in the fasting state, and has been confirmed by others.¹² When the children were fed milk in the non-fasting state this was also well tolerated, and no child experienced symptoms of discomfort. The chest/abdomen circumference ratio in the maldigesting children was also not different from that of healthy, lactose-digesting children of similar age, suggesting that there was no excess abdominal bloating in the former.

The breath hydrogen responses to fasting lactose and to non-fasting milk-lactose confirm the much slower and lesser rate of hydrogen production in the latter circumstance. Twelve of the lactose-maldigesting children did not produce increased breath hydrogen in 2,5 hours after drinking milk. Even though a test period of 5 hours is required to identify delayed breath hydrogen increases, this finding does suggest significantly improved digestion of milk-lactose.

The presence of protein and fat in milk delays gastric emptying and results in a more gradual exposure of milk-lactose to mucosal lactase, thus allowing more complete hydrolysis. The lowered peak hydrogen levels in our milk tests compared with the lactose tests are compatible with a lowered amount of undigested lactose in the milk tests.

Those children with breath hydrogen evidence of milk-lactose maldigestion within 150 minutes were more stunted and lighter than the children without such evidence. However, even though they had a lower weight-by-stature SDS than the milk-lactose digesters, this measure was nevertheless normal, indicating a normal recent nutritional state. They also had a higher mean rise of breath hydrogen after a standard dose of lactose. This may suggest a more complete loss of intestinal mucosal lactase in these children. However, other possible causes of a difference in the nutritional state are not completely excluded in these children, many of whom had suffered from a variety of nutritional conditions.

In conclusion, institutionalised lactose-maldigesting children with a milk intake of 25 - 40 ml/kg/d were found to suffer no symptomatic effects. The degree of lactose digestion in these children is much improved in the non-fasting state when measured by the milk-lactose breath hydrogen response. Milk can safely and effectively be included in feeding schedules for institutionalised black children, provided it is given in reasonable quantities not exceeding 20 ml/kg at any one time.

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