Lactose maldigestion — age-specific prevalence in black and Indian children

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Summary

A study was performed to determine the prevalence and age of onset of primary lactose maldigestion in healthy black and Indian children, and to determine whether this was of clinical significance.

More black (22 of 44 — 50%) than Indian children (10 of 45 — 22,2%) had lactose maldigestion ($P < 0,02$), the development of which was age-related and occurred earlier in blacks than in Indians; 6 of 19 black children less than 5 years old (31,6%) were lactose maldigesters, compared with 8 of 10 (80%) over 10 years old, while only 1 of 16 Indian children aged under 8 years (6,3%) were maldigesters, compared with 5 of 13 (38,5%) aged over 10 years. Most children had a very low intake of milk and lactose maldigestion was of no clinical significance to them.

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Adult lactase deficiency is considered to be under genetic control and not influenced by the amount of dietary lactose.[1,2] It has been shown to be very common in South African black adults,[3,4] and has also been documented in children.[5,6] Most communities world-wide show a very high prevalence of lactose maldigestion in adults. Some populations, including most Caucasians, have maintained a predominant ability to digest lactose. This has been considered to have evolved as a beneficial mutation in these traditionally milk-drinking communities.[7] In India, a difference was found between a high prevalence of lactose maldigestion of 66,6% observed in the south and a low prevalence of 27,4% in the north.[8]

A declining level of lactase activity during childhood results in a progressive increase in the prevalence of lactose maldigestion in most communities. In North American black children it rose from 11% at 4 - 5 years of age to 72% at 8 - 9 years.[9] In Japan and Israel lactose maldigestion was found to develop from 3 years onwards,[10,11] whereas white children in the study by Welsh *et al.*[12] manifested it only after 5 years of age. The age at which primary lactase deficiency develops therefore has a genetic basis, but this may in addition be influenced by other factors such as previous malnutrition or mucosal damage. These may hasten the permanent decline of lactase activity, which would normally be expected later in life.[13]

As the prevalence and age of onset of lactose maldigestion has not been documented in South African children, a study was performed to compare these in healthy black and Indian children in Natal and to determine their clinical significance.

Subjects and methods

One hundred and seventy-five black children at a primary school in Umlazi and 96 children attending a day care centre in Lamontville were measured and weighed. The homes of randomly selected, clinically well-nourished children from these groups were then visited and a detailed questionnaire was administered regarding early life experiences including hospitalisation and nutrition, milk intake and related symptoms, previous gut parasitism and any symptoms of milk intolerance in the parents. Subjects with a history of hospitalisation for malnutrition or for diarrhoeal episodes lasting longer than 2 weeks, or of diarrhoea or antibiotic therapy in the preceding 4 weeks, were excluded.

Forty-four black children were studied by means of a lactose breath hydrogen test. Their median age was 76 months (mean (± SD) 75,9 ± 43,3 months, range 29 - 199 months).

The Indian group comprised 45 children, randomly selected from pupils attending a primary school in Chatsworth, Durban, with a median age of 100 months (mean 99,4 ± 25,4 months, range 67 - 133 months).

A Hoekloos Lactoscreen hydrogen breath tester was used for the breath hydrogen tests.[14] Mixed expiratory breath was collected with a face-mask sampling device at baseline and at 30-minute intervals up to 120 minutes after an oral load of lactose (1 g/kg body weight), given as a 10% solution. Care was taken to ensure an adequate breath sample, which was analysed immediately. Paired readings were taken at random intervals to confirm reproducibility of results. A rise in breath hydrogen of 20 parts per million or more was taken as evidence of lactose maldigestion.[15]

The anthropometric data for the black and Indian children were compared with the National Center for Health Statistics (NCHS) tables[16] and expressed as standard deviation scores (SDS), as well as with control values established in local populations.[17]

The statistical evaluation was performed at the Institute of Biostatistics of the South African Medical Research Council.

Results

History, clinical assessment and anthropometric measurements indicated that both groups of children were adequately nourished. Compared with the NCHS tables, the black children had mean height and weight SDS (± SD) of −0,55 ± 0,93 and −0,37 ± 0,98 respectively. They conformed to or exceeded local standards of normal growth,[17] and their mean weight for height was normal with a SDS of 0,03 ± 1,12.

The Indian children were well grown but thin, with a height SDS of −0,13 ± 1,01 and a weight SDS of −0,62 ± 1,32. This is in agreement with previous findings for South African Indian children (A. Moosa and H. Coovadia — unpublished data). Overall, 50% of the black children (22 of 44) and 22,2% of the Indian children (10 of 45) were found to be maldigesting lactose on the basis of an abnormal rise in breath hydrogen at 60 - 120 minutes ($P < 0,02$; Yates' corrected χ^2 test) (Table I). The prevalence varied with age. Six of 19 black children under 5 years of age were maldigesters (31,6%), compared with 8 of 10 children over 10 years of age. Whereas lactose maldigestion appeared between the ages of 2 and 3 years in blacks, only 1 of 16 Indian children younger than 8 years manifested the condition. In the age group 5 - 10 years, 8 of 15 blacks and 5 of 32 Indians were affected ($P < 0,02$, Fisher's exact test). Five of

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TABLE II. ANTHROPOMETRIC DATA FOR HEALTHY BLACK AND INDIAN CHILDREN

	Blacks		Indians	
	Lactose digesters ($N = 22$)	Lactose maldigesters ($N = 22$)	Lactose digesters ($N = 35$)	Lactose maldigesters ($N = 10$)
Height SDS (± SD)	−0,67 ± 0,88	−0,52 ± 0,96	−0,23 ± 1,06	+0,19 ± 0,73
Weight SDS (± SD)	−0,56 ± 0,86	−0,17 ± 1,06	−0,88 ± 1,41	−0,23 ± 1,48
Weight for height SDS (± SD)	−0,23 ± 0,9	+ 0,27 ± 1,24	−0,68 ± 1,48	−0,29 ± 0,53

13 Indian children older than 10 years were lactose maldigesters.

The lactose breath hydrogen tests were well tolerated, with only a few complaints of abdominal discomfort; no diarrhoea was reported in the 2 hours following the test.

Analysis of the health questionnaires revealed no differences between lactose digesters and maldigesters with respect to the frequency of previous episodes of diarrhoea. Similar numbers of black children in both groups had a history of infestation with intestinal parasites, but stools were not examined.

Most black and Indian children had a very low intake of milk — the majority drank less than a cup a day. This was independent of their lactose digesting status, since only a few children were considered to be milk-intolerant by their parents. Even so, 4 of 21 black lactose maldigesters claimed daily intake of more than 2 cups of milk. There was no significant difference in nutritional status or measurements of growth between lactose digesters and maldigesters (Table II).

TABLE I. PREVALENCE OF LACTOSE MALDIGESTION IN HEALTHY BLACK AND INDIAN CHILDREN

Age (mo.)	Blacks No.	Blacks %	Indians No.	Indians %	*P* value
< 60	**6/19**	**31,6**	—	—	
60 - 120	**8/15**	**53,3**	**5/32**	**15,6**	**< 0,02***
> 120	**8/10**	**80**	**5/13**	**38,5**	
Total	**22/44**	**50**	**10/45**	**22,2**	**< 0,02†**

* Fisher's exact test.
† Yates' corrected χ^2 test.

Discussion

The black children in this study had a high prevalence of lactose maldigestion by the age of 10 years, similar to published figures for adults in South Africa and elsewhere.[18] Care had been taken to ensure that these children were well nourished and had not suffered from conditions likely to result in acquired lactose maldigestion. A history of intestinal parasitic infestation did not exclude subjects from the study, even though *Ascaris* infestation has been shown to result in decreased absorption of lactose,[19] as there were equal numbers of children with a history of worm infestation in the digesting and the maldigesting groups. An acquired effect on lactase activity can therefore not entirely be excluded.

The Indian children in this study showed a prevalence of lactose maldigestion intermediate to the rates described for north and south Indians.[8] This is fully compatible with the mixed geographical origins of the local Indian population.

The ability to digest lactose was clearly age-related in both the black and the Indian children. Lactose maldigestion presented earlier in the blacks, with an onset by 3 years of age. In contrast, the Indian children had a very low level of lactose maldigestion under 8 years of age. This difference in the age of development of the condition between populations with high and low prevalence rates has been described previously.[10–12] Lactose maldigestion is of no significance in the daily life of these children, most of whom have a very low intake of milk, and a number of children with maldigestion appeared to suffer no untoward symptoms from consumption of milk in excess of two cups per day. This is well recognised.[20] Few children drink enough milk to achieve a lactose intake of 2 g/kg; this is equivalent to an intake of milk of 40 ml/kg. A lactose load of 0,5 - 1 g/kg, even in one dose, is not usually associated with symptoms of lactose intolerance. Also, milk lactose enters the small gut at a slower rate than lactose in aqueous solution,[21] especially if taken with other food; this allows time for hydrolysis by intraluminal bacteria or by residual brush border lactase. Furthermore, nitrogen retention and fat absorption have been shown to be unaffected by lactose malabsorption.[22]

In conclusion, the high prevalence of lactose maldigestion in black children has been confirmed and contrasted with a much lower prevalence in South African Indian children. This has no effect on the nutritional state or daily life of these children.

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REFERENCES

1. Rosenzweig N. Diet and intestinal enzyme adaptation: implication for gastrointestinal disorders. *Am J Clin Nutr* 1975; **28:** 648-649.
2. Newcomer A, McGill D. Clinical importance of lactase deficiency. *N Engl J Med* 1984; **310:** 42.
3. Segal I, Gagjee PP, Essop PP, Noormohamed AM. Lactase deficiency in the South African black. *Am J Clin Nutr* 1983; **38:** 901-905.
4. O'Keefe S, Adam J. Primary lactose intolerance in Zulu adults. *S Afr Med J* 1983; **63:** 778-780.
5. De Villiers FPR. Relationship between milk lactose tolerance and body mass in teenage Tswana schoolchildren. *S Afr Med J* 1988; **74:** 499-501.
6. Tolboom JJ, Kabir H, Molatseli P *et al.* Lactose malabsorption and giardiasis in Basotho school children. *Acta Paediatr Scand* 1987; **76:** 60-75.

7. Simoons FJ. Primary adult lactose intolerance and the milking habit: a problem in biologic and cultural interrelations: 2. A culture historical hypothesis. *Am J Dig Dis* 1970; **15:** 695-710.
8. Tandon RK, Joshi YK, Singh DS *et al.* Lactose intolerance in North and South Indians. *Am J Clin Nutr* 1981; **34:** 943-946.
9. Garza C, Scrimshaw N. Relationship of lactose intolerance to milk consumption in young children. *Am J Clin Nutr* 1976; **29:** 192-196.
10. Nose O, Iida Y, Kai H *et al.* Breath hydrogen test for detecting lactose malabsorption in infants and children: prevalence of lactose malabsorption in Japanese children and adults. *Arch Dis Child* 1979; **54:** 436-440.
11. Bujanover Y, Katz A, Peled Y, Gilat T. Lactose malabsorption in Israeli children. *Isr J Med Sci* 1985; **21:** 32-35.
12. Welsh JD, Poley JR, Bhatia M, Stevenson DE. Intestinal disaccharidase activities in relation to age, race and mucosal damage. *Gastroenterology* 1978; **75:** 847-855.
13. Leslie J, MacLean WC, Graham GG. Effect of an episode of severe malnutrition and age on lactose absorption by recovered infants and children. *Am J Clin Nutr* 1979; **32:** 971-974.
14. Douwes AC, Fernandes J, Rietveld W. Hydrogen breath test in infants and children: sampling and storing expired air. *Clin Chim Acta* 1978; **82:** 293-296.
15. Solomons NW, Barillas C. The cut-off criterion for a positive hydrogen breath test in children: a reappraisal. *J Pediatr Gastroenterol Nutr* 1986; **5:** 920-925.
16. NCHS Standards. In: *Measuring Changes in Nutritional Status.* Geneva: World Health Organisation, 1983.
17. Coovadia HM, Adhikari M, Mthethwa D. Physical growth of negro children in the Durban area. *Trop Geogr Med* 1978; **30:** 373-381.
18. Scrimshaw NS, Murray EB. The acceptability of milk and milk products in populations with a high prevalence of lactose intolerance. *Am J Clin Nutr* 1988; **48:** suppl, 1083-1159.
19. Carrera E, Nesheim MC, Compton DWT. Lactose maldigestion in preschool children. *Am J Clin Nutr* 1984; **39:** 255-264.
20. Newcomer AD, McGill DB, Thomas PJ, Hofmann AF. Tolerance to lactose among lactase deficient American Indians. *Gastroenterology* 1978; **74:** 44-46.
21. Bayless TM, Rothfeld B, Massa C *et al.* Lactose and milk intolerance: clinical implications. *N Engl J Med* 1975; **292:** 1156-1159.
22. Bowie MD. Effect of lactose-induced diarrhoea on absorption of nitrogen and fat. *Arch Dis Child* 1975; **50:** 363-366.