LACTOSE INTOLERANCE

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The phenomenon of lactose (milk sugar) intolerance, due to a deficiency of the intestinal enzyme lactase, which is essential for the absorption and utilisation of lactose, is drawing increasing attention as its implications in food aid programmes becomes clear. Lactose intolerance is manifest by abdominal pain, flatulence and diarrhoea following the ingestion of milk or milk products. (See Appendix A)

Lactose intolerance can be either primary or secondary. The secondary form occurs commonly in association with many gastro-intestinal diseases (Table I). The primary form occurs in otherwise healthy adults and has a varying incidence in different ethnic groups. There is a high incidence in American[1] and African Negroes[2], Australian Aborigines[3] and Greek Cypriots[4]. The initial report of Davis and Bolin[5] describing a high incidence of lactose intolerance in Asian students has been confirmed in other Asian populations[6-8].

This high incidence of lactose intolerance in Asian and other ethnic groups has important socio-economic implications when we realize the important role of milk and milk products in food aid programmes to underdeveloped countries. This aid may consist of the establishment of milk processing plants in Asian communities by government or international agencies, or the direct distribution of powdered milk. The United Nations Economic and Social Council[9] has reported the technical and economic difficulties of starting milk processing plants in under-

developed countries. The cost is high and establishment of a dairy industry is particularly difficult in a tropical climate[10]. One attempt to avoid these problems is to "tone" the extremely high fat content of the native buffalo milk with powdered milk from milk-producing countries such as Australia and New Zealand.

When there is co-existence of primary and secondary lactase deficiency, particularly in times of famine, milk in amounts contributing a significant part of the diet may induce or exacerbate diarrhoea. This could be particularly serious in malnourished and debilitated children. It is especially in this situation that aid in the form of milk tends to be given.

The pathogenesis of lactose intolerance is disputed. It could be an hereditary enzyme defect occurring with varying frequency in different ethnic groups. Another, and it would seem to us more likely possibility, is an acquired defect, due to lack of continued substrate challenge in the form of low level of milk consumption, resulting in a gradual adaptive decline in enzymatic activity. It is known that there is very little milk consumption after weaning in most of the population groups reported to have a high incidence of lactose intolerance.

Results of studies on both animals and humans are inconclusive, but seem to us to favour the theory that lactose intolerance is due to a gradual decline in enzyme activity because of the lack of a continued substrate challenge. This theory would account for the earlier appearance of the condition in some ethnic groups whose milk consumption after weaning is minimal, compared with Caucasians whose milk consumption declines at a later age. A direct relationship of age to an increased

incidence of lactose-induced cramps and diarrhoea in Negroes[7] supports this theory. However, a detailed survey of milk-drinking habits of the various ethnic groups is needed to verify it.

If lactose intolerance is a late manifestation of a genetically predetermined condition, it might be expected to occur in association with congenital lactose intolerance (a rare form of lactose intolerance occurring in infancy). There appears to be no such association, and there is also no convincing familial incidence of the condition, at least in Caucasians. A congenital defect should be manifest in childhood when the load is maximal. As compared to the congenital syndrome, these people tolerate milk well in infancy. Finally, the condition occurs too frequently to be explained as a genetic mutation.

There have been numerous attempts to induce intestinal lactase in the rat and other animals[11-21]. The results remain conflicting and controversial. Protagonists of the genetic aetiology of adult lactase deficiency rely largely on early reports of failure to adapt lactase in various animals to support this theory. However many of these experiments were performed before reliable methods of enzyme assay became available. Only Girardet et al (1964)[21] have shown adaptation to occur in the adult rat, but this paper is rarely quoted in the English literature. We thought it essential to confirm their work as the problem of adaptation assumes importance in relation to human lactase deficiency.

The detailed plan of this study can be found in Appendix B. In

summary it was found that after 5 to 8 weeks on a 30% lactose diet a significant increase in jejunal lactase activity was obtained. Intestinal lactase in the rat is therefore an adaptive enzyme and presumably adaptation can occur in the human.

Studies of human subjects again illustrate the controversy over the genetic or acquired nature of lactose intolerance. Cuatrecasas, Lockwood and Caldwell[22] showed in a group of 60 patients, 67 per cent Negro, a strong correlation between milk consumption and lactose absorption. Whereas 86% of "non-drinkers" of milk were lactose "non-absorbers", only 13% of milk "drinkers" were "non-absorbers". An attempt to increase lactose absorption in 7 "non-absorbers" was made by giving 150 g. lactose daily for up to 45 days. There was no increase in lactose absorption or jejunal lactase activity.

However, animal experiments suggest that at least 8 weeks are required with lactose loading to result in enzyme induction. It is also possible that lactose alone is insufficient to provoke enzyme induction in humans. Lactose is a component of oligosaccharides in milk[23] and it may be that these or some other fractions of whole milk are essential for continued enzyme production.

A significant fall in "lactose absorption" was found in 2 patients deprived of milk for 5 months,[22] suggesting a decline in enzyme activity with the disappearance of continued substrate challenge.

McMichael, Webb and Dawson[4] concluded that there is an inherited basis for lactase deficiency on the grounds of finding lactase deficiency in 15 of 17 Greek Cypriots. Two of these patients had a family history of milk intolerance. No details of milk consumption were given.

Cook and Kajubi[2] found an "inherited congenital" difference in lactase levels between different African tribes. Lactase deficiency is very common in the Baganda, occurring in 89%. However, their diet is largely vegetable and mainly banana. Milk consumption was either nil or less than one-fourth pint daily. In contrast, the Bahima tribe "took between 2 and 7 pints of milk daily and little else". Only one of 11 Bahima patients had a flat lactose curve. Intestinal lactase levels were not done. Here, then, are two tribes, one of milk drinkers who are lactose tolerant and the other who do not drink milk and are nearly all lactose intolerant. We would interpret this as evidence more in favour of an acquired than of a congenital defect.

The differences between other tribes were not as marked but Cook and Kajubi[2] point out that "although every effort was made to obtain accurate histories of milk intake, limitations inherent in nutritional assessment in Africa must be appreciated". (This difficulty did not apply to the Baganda and Bahima tribes.)

Cook[24] found a gradual fall in the mean maximum rise of blood glucose after a lactose load in the first 4 years of age in an African population. These results were related to the previous paper and as "some infants with flat curves were breast fed and most of the infants had milk for many weeks before the test", an hereditary basis was again favoured. However, precise details of milk consumption were not obtained.

Bayless and Rosensweig[11,25] consider lactose intolerance to be

hereditary on the basis of the similar incidence in American and African Negroes. Shi-Shung Huang and Bayless[26] reach the same conclusions. Four of twenty Negro children and no white children had a maximum rise of blood sugar after lactose of less than 20 mg/100ml. The difference in incidence compared to older Negro groups is unexplained, but the majority of the children in this study consumed at least 1 glass of milk per day.

A detailed technical account of our own study will be found in Appendix C. Here we will give the general results.

Certain criteria should be met before a diagnosis of lactose intolerance is made[27]. (1) Diarrhoea upon ingestion of lactose (2) a flat lactose tolerance test response (3) normal tolerance curves for glucose and galactose (4) low levels of lactase in the intestinal mucosa.

The patients in this study complied with these criteria. Intestinal lactase activity was not estimated in all subjects but a "flat" lactose tolerance curve (maximum blood sugar rise over fasting of 0 - 20 mg/100ml) has been reported to be a reliable test for lactose intolerance and correlates well with intestinal lactase activity [28-30].

This study shows lactose intolerance to be common in Chinese students, New Guineans and a small group of Indians. A total of 29 out of 30 Chinese students were found to have lactose intolerance, an incidence of 97 per cent. The only Chinese subject without diarrhoea after lactose had a maximum rise in blood sugar of 25 mg/100 ml and her

jejunal lactase activity was 1 U./g. wet weight. All the New Guinean men were lactose intolerant as were four of the five Indians. One Caucasian had a maximum rise in blood sugar after lactose of 20 mg/ 100 ml, however he did not have diarrhoea, and had normal lactase activity. None of the well recognised causes of secondary lactase deficiency was evident in any of our subjects. Therefore in these Asians we are dealing with primary lactose intolerance.

As we have stated, we feel that the evidence favours the adaptive theory rather than the genetic. However there is as yet insufficient evidence. Regardless of which theory is correct, owing to the increasing importance of milk aid programmes to underdeveloped countries, particularly in times of famine, it is imperative to assess the overall incidence of lactose intolerance in recipient countries. What is required is a survey of lactose intolerance amongst Asian peoples in their own environment. This survey would need to ask certain questions. What is the overall incidence of lactose intolerance in the various communities? What is the incidence in relation to age and previous milk drinking habits? Is the defect congenital or acquired? The results of such a survey would show which communities (and which groups within an individual community) would benefit most from the introduction of milk products into the diet. If the defect could be shown to be an adaptive phenomenon, then milk could be introduced into the diet under a planned scheme which aimed at gradually increasing the lactose content of the various milk

products. If on the other hand the defect is genetically determined, then it would appear better to concentrate on other ways of improving the value of the basic diet.

If the theory of adaptation is correct then it should be possible to forecast which populations are likely to be lactose intolerant by reference to the known per capita milk consumption[31]. It can be seen from Table 2 which shows examples of per capita milk consumption in various countries that those populations who are lactose tolerant have a per capita milk consumption of the order of 600 g/day, while those that are lactose intolerant have a per capita milk consumption of approximately 100 g/day or less.

Therefore other countries with a similar per capita milk consumption of the order 100 g/day are likely to have a high incidence of lactose intolerance. The majority of these are in underdeveloped areas of the world and it seems essential that surveys such as that described above are carried out in these countries to assess the value of future milk aid programmes.

We are at present continuing our work on lactose intolerance by carrying out experiments in human volunteers and animals with the aim of proving whether the defect is acquired or genetic. In addition we plan to initiate a survey in an Asian community in the near future. (We are being supported in this work by the Australian Dairy Produce Board and the National Health and Medical Research Council of Australia.)

APPENDIX A

Lactose Absorption and Pathogenesis of Diarrhoea in Lactose Intolerance

Before being metabolised, lactose must undergo hydrolysis into its component monosaccharides, glucose and galactose. Formerly it was believed that lactase, the enzyme responsible for this hydrolysis, together with the other disaccharidases sucrase and maltase, was secreted by unspecified cells of the intestinal mucosa into the lumen of the gut where hydrolysis of the respective disaccharides occurred. However recent studies indicated that hydrolysis takes place in the intestinal wall[32-35]. It was also demonstrated by a variety of methods that the hydrolytic enzymes are localized in the brush border, or microvilli, of the epithelial cells facing the intestinal lumen. After hydrolysis the component monosaccharides are transported through the cell and into the blood stream (Figure I).

Two mechanisms produce the symptoms of lactose intolerance (Figure 2). When lactose remains unsplit in the small intestine there is a considerable flux of water into the lumen of the small bowel. In experiments where lactose has been perfused into the small bowel of lactase-deficient subjects, volumes of up to 2,200 ml have been recovered from the aspirate 100 cm. distally when only 780 ml of a 90% solution have been instilled[36]; a net flux of 1400 ml.

This aspirate remains isomolar due to diffusion of sodium, chloride and to a lesser extent potassium into the gut lumen. Symptoms of cramps and nausea began 5 minutes after the infusion commenced and may be due

to distention of the small intestine. This large volume of fluid containing unsplit lactose is rapidly transported to the colon, where the second mechanism comes into action when the lactose contacts the colonic bacterial flora. Some bacteria, especially E. coli, have marked lactase activity and split lactose into glucose and galactose. However, the greater part of the nonabsorbed lactose undergoes bacterial fermentation producing lower-molecular-weight organic acids and subsequently forming gas. These breakdown products exert a marked osmotic effect in the colon and again a massive inpouring of fluid occurs. The combination of fluid and gas causes bloating, abdominal distention, wind and diarrhoea.

APPENDIX B

Adaptation of Lactase in the Rat

This study was undertaken to determine whether intestinal lactase in the rat is an adaptive enzyme and, if so, how long adaptation takes to occur.

MATERIALS AND METHODS

Experiments Two experiments were undertaken.

1. Thirtyfour female Wistar rats aged approximately 12 weeks were used. Eighteen were started on an artificial diet containing 30% lactose[37]. Sixteen controls were fed commercial rat pellets (D.H.A.)*, these containing 3-4% lactose. The animals were killed after 95-218 days on the diet.
2. Thirtyeight female Wistar rats aged 4 to 5 months were divided into 2 groups. One group was given commercial rat pellets (D.H.A.) and libitum, and the other group was fed a diet containing 30% lactose.

Mucosal Homogenates The animals were killed after induction of ether anesthesia. 1 cm segments of intestine were excised at 25% and 90% of the total length of small intestine. They were cut open and the mucosa scraped off with a glass slide. This was immediately frozen on dry ice, weighed and homogenised in distilled water by hand, in a Bellco glass homogeniser, to produce a final concentration of 20 mg mucosa/ml.

* Drug Houses of Australia

The homogenate was placed in polypropylene microcentrifuge tubes, frozen again on dry ice and stored at -4°C.

Enzyme Activity Assay Maltase, sucrase and lactase activities were assayed by the tris-glucose exidase method of Dahlqvist (1964)[38]. All enzyme activities are expressed as /u moles (units) substrate hydrolysed/ min. per g wet weight of mucosa.

Assay of Protein Concentration The protein content of the homogenates was assayed by the method of Lowry et al (1951)[39] with human serum albumin as a standard.

RESULTS

Experiment 1. An increase in jejunal lactase activity was found at all periods between 95-218 days in the group fed the 30% lactose diet when compared with the group fed rat pellets. On the 30% lactose diet, the mean activity was 2.3 units/g wet weight with a range of 0.3 to 4.3 units/g wet weight (Table I). In the group fed rat pellets, mean jejunal lactase activity was 1.1 u/g wet weight with a range of 0.6 to 2.1 u/g wet weight (Table 2). Lactase activity in the lower ileum was similar in both groups, the mean being 0.4 u/g wet weight with a range of 0.1 to 0.6 u/g wet weight in those rats on 30% lactose and in those fed rat pellets the mean was 0.3 with a range of 0.1 to 0.8 u/g wet weight. While the jejunal sucrase and maltase activities were the same in both groups these disaccharidases were much lower in the ileum in those rats fed rat pellets.

Experiment 2. Jejunal lactase activity was increased in those rats fed 30% lactose. This first became apparent between 5-8 weeks on the diet. The activity continued to rise, and at the end of 12 weeks the mean jejunal lactase activity was 3.4 units/g wet weight of mucosa (range 1.6 to 5.7 units) (Table 3). This was significantly higher than the rats fed 3% lactose ($P < 0.001$). In rats fed commercial rat pellets which contain 3-4% lactose, the mean jejunal lactase activity after 12 weeks was 1.1 units/g wet weight of mucosa (range 0.6 to 1.8 units). Protein estimations were performed on all homogenates at 12 weeks and no significant difference was found between the two groups (Table 4).

Discussion

These experiments show that adaptation of intestinal lactase occurs in the rat and this adaptation is related to diet. Rats on a lactose-rich diet had high jejunal lactase levels while those on a low lactose diet had significantly lower lactase activity. Adaptation occurred between the fifth and eighth week on the diet. Further work is in progress to define this time more accurately, but this agrees with the findings of Girardet et al (1964)[21] who found adaptation between 6-9 weeks on a lactose-rich diet.

APPENDIX C

Study of lactose intolerance in South East Asia

Subjects Studied

The control subjects were 23 asymptomatic Australian students of Caucasian background. Two admitted to vague abdominal symptoms, but these were not related to milk. The age range was 20 to 31, with a mean of 22 years. There were 3 females.

The test subjects included 30 Asian students, predominantly Chinese from Hong Kong, Malaysia or Singapore. Three were female. The age range was 22 to 26 years, with a mean of 23 years. Nine admitted to gastrointestinal symptoms on specific questioning; these were usually in the form of abdominal pain, bloating, and diarrhoea, and were frequently related to milk ingestion.

In addition there were 8 New Guinea men who were being investigated at Angau Memorial Hospital, Lae, for a variety of non-gastrointestinal complaints. Their ages ranged from 14 to 30 years, with a mean of 21 years.

Five male Indian students completed the group of test subjects. They were asymptomatic on specific questioning. The age range was 32 to 39 years with a mean of 35 years.

Methods

Lactose and glucose-galactose tolerance tests

A lactose tolerance test was performed using either 80 g. or 50 g. lactose dissolved in 400 ml water and given to the fasting subject. It was ingested within 5 minutes. The larger dose was given to 12

Caucasians, 15 Asians and all the Indians and New Guinea natives. In view of the severity of the diarrhoea, abdominal pain and bloating together with occasional vomiting, the smaller dose was given to the remaining 6 Asian students and 11 Caucasian students. Capillary blood samples were taken at 0, 30, 60, 90 and 120 min. Total blood sugar reducing substances were determined by a "Ferricyanide" method adapted to the autoanalyser. Any symptoms produced were noted. To test for glucose-galactose tolerance, the same procedure was repeated with all subjects after the oral administration of either 40 g. or 25 g. each of glucose and galactose. Stool pH was tested with Whatman-BDH indicator paper.

Urine was collected at the end of 2 hours and tested for reducing substances. If these were present urine chromatography was performed to elicit the nature of the sugar.

Disaccharidase Activity

Jejunal mucosal specimens were obtained by peroral biopsy using the Crosby capsule[40] from the upper jejunum at least 10 cm distal from the duodenal-jejunal junction. The specimens were divided into two parts. On one lactase activity was measured according to the method of Dahlqvist[39]. The other part of the biopsy specimen was examined under the light microscope for any histological changes.

Results

In the control group the mean maximum increase in blood sugar after 80 g. lactose was 34 mg/100 ml with a range of 20 - 50 mg/100 ml (Table 7). After 40 g. glucose and galactose the mean maximum rise was 43 mg/100 ml with a range of 20 - 105 mg/100 ml. Only 1 subject (no. 5) had diarrhoea after lactose.

The remaining control subjects were given 50 g. lactose and the mean maximum increase in blood sugar was 46 mg/100 ml with a range of 20 - 80 mg/100 ml (Rable 7). After 25 g. glucose and galactose the mean maximum rise was 40 mg/100 ml with a range of 10 - 70 mg/100 ml. No patient had diarrhoea after 50 g. lactose. The only patient with a rise of 20 mg/100 ml also had a rise of only 15 mg/100 ml after glucose and galactose. This indicates the importance of repeating the test with the monosaccharide mixture in those people who have a "flat" lactose curve.

In the Chinese students, the mean maximum increase in blood sugar after 80 g. lactose was 8 mg/100 ml with a range of 0 - 25 mg/100 ml (Table 8). The mean maximum rise in blood sugar after glucose and galactose was 56 mg/100 ml with a range of 30 - 105 mg/100 ml. All subjects had diarrhoea after lactose and the stool pH was below 6 in those in which it was tested (6). Whereas 13 of the 15 lactose curves were "flat" (rise of blood sugar 0 - 20 mg/100 ml), all subjects had a rise of blood sugar of more than 20 mg/100 ml when tested with glucose and galactose.

In the 15 Chinese students given 50 g. lactose the mean maximum rise in blood sugar was 8 mg/100 ml with a range of 0 - 25 mg/100 ml. The mean maximum rise after glucose and galactose was 44 mg/100ml with a range of 35 - 55 mg/100 ml (Table 2). All but one subjects had diarrhoea after lactose. Fourteen of the fifteen subjects had a "flat" lactose curve, and all had a normal curve after the monosaccharide mixture.

In the Indian group, the mean maximum rise in blood sugar was 8 mg/100 ml with a range of 0 - 30 mg/100 ml. After glucose and galactose, the rise was 32 mg/100 ml with a range of 20 - 45 mg/100 ml (Table 9). Four of the 5 subjects had a "flat" curve after lactose.

The New Guinea natives had a mean maximum rise in blood sugar after lactose of 3 mg/100 ml with a range of 0 - 15 mg/100 ml. The mean rise after glucose and galactose was 41 mg/100 ml with a range of 30 - 65 mg/100 ml (Table 10). Diarrhoea was not recorded in this group. All were regarded as lactose intolerant.

The mean lactase level in Caucasian students was 3.9 U/g. wet weight of mucosa with a range of 3.6 - 4.8 U/g. wet weight of mucosa. The mean lactase level in the Chinese students was 0.3 U/g. wet weight of mucosa with a range of 0.0 - 0.6 U/g. wet weight of mucosa. The results are shown in Table 11.

The histology of the biopsy specimens obtained from both the Caucasian and the Chinese groups was normal. There was no evidence of any abnormalities of the intestinal villi which are known to occur in a variety of intestinal diseases.

LEGENDS

TABLE I Conditions associated with Secondary Disaccharidase Deficiency.

TABLE 2 Examples of per Capita Milk Consumption in Different Countries (F.A.O. Yearbook 1967).

TABLE 3 Intestinal Disaccharidase Activity (Units/g wet weight of mucosa) in Rats fed a 30% Lactose Diet.

TABLE 4 Intestinal disaccharidase Activity (Units/g wet weight of mucosa) in Rats fed a 3% Lactose Diet (Rat Pellets).

TABLE 5 Jejunal Disaccharidase levels (Units/g wet weight of mucosa) in Rats on 30% or 3% Lactose Diets.

TABLE 6 Jejunal mucosal Protein Concentration (mg/g wet weight of mucosa) after 12 Weeks in Rats on either 30% or 3% Lactose Diets.

TABLE 7 Maximum Rise in Blood Sugar in Australian Students after the Test Dose of Lactose and Glucose-Galactose.

TABLE 8 Maximum Rise in Blood Sugar in Chinese Students after the Test Dose of Lactose and Glucose-Galactose.

TABLE 9 Maximum Rise in Blood Sugar in Indian Students after the Test Dose of Lactose and Glucose-Galactose.

TABLE 10 Maximum Rise in Blood Sugar in New Guinea Natives after the Test Dose of Lactose and Glucose-Galactose.

TABLE 11 Lactase Levels in Jejunal Biopsy Specimens in Caucasian and Chinese Students.

FIGURE I Mechanism of Lactose Hydrolysis and Absorption.

FIGURE 2 Pathogenesis of Diarrhoea in Lactose Intolerance.

TABLE I

SECONDARY DISACCHARIDASE DEFICIENCY

1. Gastro-enteritis
2. Protein-calorie Malnutrition (Kwashiorkor)
3. Coeliac Disease
4. Tropical Sprue
5. Idiopathic Steatorrhoea
6. Ulcerative Colitis
7. Whipples Disease
8. Regional Enteritis
9. Cystic Fibrosis
10. Intestinal Lymphangiectasia
11. Beta-lipoprotein Deficiency
12. Drugs - Neomycin
    Conovid
13. Giardia Lamblia Infestation
14. Infectious Hepatitis
15. Post-surgical : Post-gastrectomy
    Gastroenterostomy
    Blind Loop
    Shortened Small Intestine

TABLE 2

A. Lactose Tolerant

Country	Per Capita milk consumption (g/day)
U.S.A.	673
Canada	646
U.K.	593
Australia	614
New Zealand	771
Denmark	728

B. Lactose Intolerant

Uganda	63
Nigeria	18
Ghana	8
China (Taiwan)	11
India	110
Japan	100
Malaysia	112
Korea	5

TABLE 3

DISACCHARIDASE ACTIVITY
(units/g wet weight of mucosa)
IN RATS FED A 30% LACTOSE DIET

LACTASE		SUCRASE		MALTASE	
Jejunum	Ileum	Jejunum	Ileum	Jejunum	Ileum
2.6	0.4	9.4	3.4	29.8	24.1
0.4	0.4	4.3	3.1	17.4	28.4
2.9	0.4	8.4	2.3	35.0	22.6
0.3	0.3	7.4	2.0	33.3	25.0
3.0	0.6	10.0	4.0	41.9	32.6
2.3	0.3	9.3	2.3	40.4	25.6
3.4	0.4	10.1	2.7	39.4	28.1
2.1	0.2	10.1	4.1	42.6	48.1
1.2	0.1	8.6	4.9	27.3	29.4
1.2	0	6.4	2.2	22.1	17.4
2.4	0.1	8.3	3.8	34.4	28.3
2.7	0.4	8.4	5.1	32.8	37.0
2.4	0.6	8.7	5.1	33.3	36.3
4.3	0.3	9.7	3.9	30.7	35.7
1.8	0.3	9.2	3.0	40.7	26.1
3.7	0.3	9.8	2.6	47.4	25.0
2.6	0.1	2.1	1.1	36.8	13.7
2.8	0.4	8.9	1.1	36.7	11.8
Mean 2.3	0.4	8.3	3.2	34.9	27.8

TABLE 4

DISACCHARIDASE ACTIVITY
(units/g wet weight of mucosa)
IN RATS FED A 3% LACTOSE DIET (RAT PELLETS)

LACTASE		SUCRASE		MALTASE	
Jejunum	Ileum	Jejunum	Ileum	Jejunum	Ileum
0.6	0.4	6.8	0.3	30.9	8.0
0.8	0.8	6.9	2.4	30.0	11.3
0.9	0.4	6.1	1.0	31.1	13.0
0.7	0.3	6.2	0.2	31.1	7.0
0.8	0.3	7.9	0.2	36.3	5.9
1.5	0.1	11.1	0.3	45.9	7.8
1.1	0.4	10.2	1.1	44.1	12.4
0.9	0.3	6.0	0.9	30.7	10.3
1.2	0.2	12.0	0.7	51.9	13.8
0.6	0.2	9.9	0.5	41.1	8.4
1.1	0.2	10.7	1.0	44.1	13.5
2.1	0.1	11.1	0.4	44.4	8.7
0.9	0.2	7.8	0.8	37.4	13.3
1.8	0.2	11.4	0.5	50.0	7.3
1.1	0.1	6.7	1.1	31.3	9.3
0.8	0.1	4.4	0.5	25.9	9.0
Mean 1.1	0.3	8.5	0.7	37.9	10.0

TABLE 5

Relationship between Jejunal Disaccharidase Levels
(units/g wet weight of mucosa)

Diet and Time

WEEK	DIET					
	30% Lactose			Rat Pellets (3% Lactose)		
	Lactase	Sucrase	Maltase	Lactase	Sucrase	Maltase
0	0.9	7.8	35.4	0.8	7.2	32.9
	1.1	9.2	37.2	0.9	8.0	34.0
2	1.0	7.1	34.3	0.6	6.8	30.9
	2.3	8.8	37.4	0.8	6.9	30.0
5	0.6	9.7	37.8	0.9	6.1	31.1
	1.2	9.2	37.0	0.7	6.2	31.1
8	1.8	9.7	35.2	1.1	6.7	31.3
	2.0	9.8	37.4	0.8	4.4	25.9
10	4.0	10.4	40.7	0.8	7.9	36.3
	2.0	11.4	46.7	1.5	11.1	45.9
12	3.4	10.7	45.2	1.1	10.2	44.1
	1.6	10.7	43.3	0.9	6.0	30.7
	3.8	11.3	45.2	1.2	12.0	51.9
	3.7	11.1	45.2	0.6	9.9	41.1
	2.9	10.0	43.9	1.1	10.7	44.1
	3.1	10.8	46.7	1.1	11.1	44.4
	2.6	9.6	42.2	0.9	7.8	37.4
	3.8	10.5	43.0	1.8	11.4	50.0
	3.6	10.2	43.7			
	5.7	10.5	41.5			
MEAN	3.4	10.5	44.0	1.1	9.9	43.0

TABLE 6

Jejunal Mucosal Protein Concentration
(mg/g wet weight of mucosa) after 12 weeks

DIET	
30% Lactose	Rat Pellets (3% Lactose)
102	115
151	124
156	197
106	138
123	113
119	162
128	110
152	130
124	
151	
Mean 131	136

TABLE 7

Maximum Rise in Blood Sugar
(mg/100 ml. blood)

Subjects	After 80 g. of Lactose	After 40 g. Glucose 40 g. Galactose
CONTROLS Caucasian		
1	30	20
2	50	45
3	40	35
4	35	30
5	30	105
6	20	30
7	30	45
8	25	30
9	35	50
10	40	40
11	35	35
12	35	50
	Mean 34	Mean 43
	After 50 g. of Lactose	After 25 g. Glucose 25 g. Galactose
13	80	15
14	75	45
15	60	50
16	20	15
17	55	70
18	25	10
19	60	-
20	30	50
21	45	40
22	30	30
23	25	70
	Mean 46	Mean 40

TABLE 8

Maximum Rise in Blood Sugar
(mg/100ml. blood)

Subjects	After 80 g. of Lactose	After 40 g. Glucose 40 g. Galactose
Chinese		
1	10	55
2	0	50
3	0	40
4	25	55
5	5	30
6	15	55
7	10	65
8	25	85
9	15	50
10	5	45
11	10	60
12	5	35
13	5	105
14	0	35
15	0	65
	Mean 8	Mean 56
	After 50 g. of Lactose	After 25 g. Glucose 25 g. Galactose
16	10	55
17	5	45
18	25	35
19	0	50
20	10	40
21	0	40
22	5	-
23	10	-
24	0	-
25	5	-
26	15	-
27	5	-
28	20	-
29	0	-
30	10	-
	Mean 8	Mean 44

TABLE 9

Maximum Rise in Blood Sugar
(mg/100 ml. blood)

Subjects	After 80 g. of Lactose	After 40 g. Glucose 40 g. Galactose
Indian		
1	0	20
2	0	45
3	0	35
4	10	30
5	30	30
	Mean 8	Mean 32

TABLE 10

Maximum Rise in Blood Sugar
(mg/100 ml. blood)

Subjects	After 80 g. of Lactose	After 40 g. Glucose 40 g. Galactose
New Guinea Natives		
1	0	35
2	0	40
3	15	30
4	0	45
5	5	30
6	5	65
7	0	40
8	0	40
	Mean 3	Mean 41

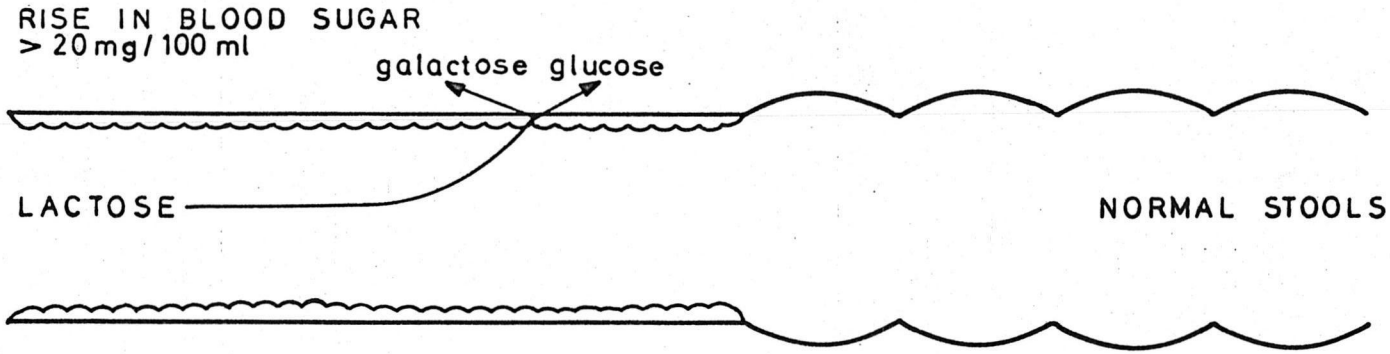
TABLE 11

Lactase Levels in Jejunal Biopsy Specimens
(U/g. wet wt. of mucosa)

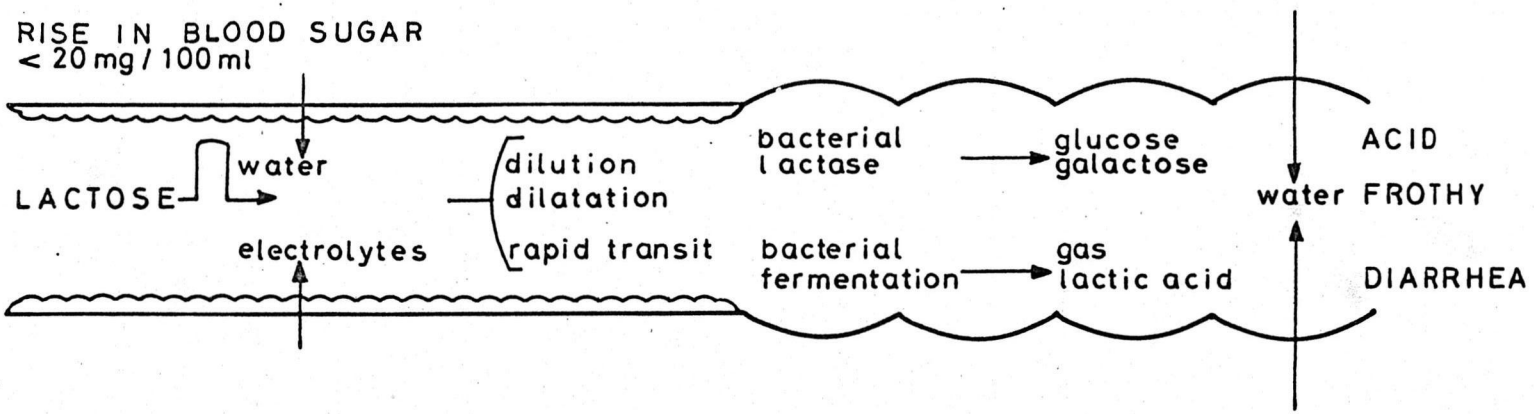
Caucasian Subjects	Chinese Subjects
3.8	0.3
3.6	0.3
4.3	0.1
3.0	0.3
4.8	0.6
3.8	0.0
Mean 3.9	Mean 0.3

FIGURE 2

NORMAL LACTASE ACTIVITY



LACTASE DEFICIENCY



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