Eur J Nutr 38:218–226 (1999)

ORIGINAL CONTRIBUTION

S. Soontornchai
P. Sirichakwal
P. Puwastien
K. Tontisirin
D. Krüger
R. Grossklaus

Lactitol tolerance in healthy Thai adults

Summary The hydrogen breath analysis test was performed in healthy Thai adults to determine lactitol tolerance. The study was conducted in 39 individuals (11 males and 28 females) aged 18-41 years. All volunteers agreed to participate in this study after the risks and benefits had been fully explained. Subjects were requested not to consume milk, milk products, or high-vegetable diets for a day and to fast from 10 p.m. of the day preceding the test day. After consumption of the test diet (12 and 20 g of lactose or lactitol, respectively, in 250 mL water), the subjects recorded the severity of symptoms for 24 hours. Breath samples were collected after fasting and after consumption of the test diet at 30 min intervals over the 7-hour study period. Breath samples were analyzed for hydrogen using gas chromatography. After consumption of 12 g lactose, the prevalence of lactose malabsorbers was established. The increment of a peak breath hydrogen level of ≥ 20 ppm above the baseline level was used as an indicator of lactose malabsorption. The lactose malabsorbers were further classified as lactose tolerants or lactose intolerants according to the gastrointestinal symptoms observed. All 39 healthy Thai adults could be classified into 3 groups as follows: 9 (23%) lactose absorbers (LA), 15 (38.5%) lactose malabsorber/tolerants (LMT), and 15 (38.5%) lactose malabsorber/intolerants (LMI). Using the hydrogen breath test, 67% of the subjects were identified as lactitol intolerants after the consumption of 12 g lactitol. The lactitol intolerants comprised 53.8% of LMI, 34.6% of LMT, and 11.5% of LA. Among all subjects, one third of LA (33%), two thirds of LMT (60%), and 93% of LMI were lactitol intolerant. In addition, gastrointestinal symptoms such as flatulence and abdominal pain were most pronounced in LMI. Diarrhea was also a prominent manifestation after consumption of 12 g lactitol. Therefore, it was finally decided that 20 g lactose or lactitol were not given to LMI because of the risk of gastrointestinal symptoms. After high doses (20 g) of lactose and lactitol consumption, most LMT developed more symptoms than did LA and the main symptom was diarrhea. Consumption of 20 g lactose resulted in fewer symptoms than 20 g lactitol in both LA and LMT. On the basis of the hydrogen breath test, most LA tolerated 12 g lactitol without gastrointestinal symptoms except some flatulence whereas most LMT and LMI did not. Twenty g lactitol was not tolerated by both LA and LMT because there was diarrhea among the subjects, especially in LMT. Although the hydrogen breath analysis test is the best method for identification of lactose malabsorption, it is not the best method to identify lactitol intolerance. A hydrogen concentration of 15 ppm above the baseline level was found to be the best cut-off point to indicate lactitol intolerance although sensitivity was 85% and specificity only 38% in this study. It was further concluded that there is a greater susceptibility to lactitol in human lactose malabsorbers than in lactose absorbers. Our findings might be relevant for the limited use of lactitol in Thailand.

Key words Sugar alcohol – lactitol – breath hydrogen – lactose malabsorption – gastrointestinal symptoms – diarrhea

Received: 11 September 1998
Accepted: 8 September 1999

S. Soontornchai
School of Health Science
Sukhothai Thammathirat Open University
Pak Kret, Nonthaburi 11120, Thailand

P. Sirichakwal
P. Puwastien
K. Tontisirin
Institute of Nutrition
Mahidol University
Nakosuprathom, Thailand

D. Krüger
Robert Koch-Institut
Nordufer 20
D-13353 Berlin, Germany

Dir. u. Prof. Dr. med. R. Grossklaus (✉)
Federal Institute for Health Protection of Consumers
and Veterinary Medicine
Thielallee 88-92
D-14195 Berlin, Germany

Introduction

Lactitol is a synthetic disaccharide sugar alcohol produced by catalytic hydrogenation of the glucose moiety of lactose (1, 2). The relative sweetness of lactitol is about 35% as compared to sucrose and 110-140%, to lactose (1–3). Lactitol has been shown to be as effective as lactulose for the treatment of chronic stable hepatic encephalopathy but was more acceptable as a medication (4–6). Its laxative effect and other side effects (i.e., osmotic diarrhea, flatulence) diminish when administered regularly. The metabolism of lactitol is different from that of carbohydrates. In certain respects, lactitol is similar to dietary fiber. It is also largely metabolized by the bacterial gut flora (7).

Extensive studies of lactitol malabsorption have been conducted using the hydrogen breath test. However, most of the data were obtained from the studies in groups of subjects among whom lactose malabsorbers were not predominant (8–10). In Thailand, lactitol has not yet been used in food products or for medical treatment. The use of lactitol in some countries, especially in Thailand where more than 90% of the population are lactose intolerant, requires that lactitol absorption in such people be studied. This has been the objective of the present study.

Materials and methods

Test diet

Water (250 mL) was used as the test diet for the baseline study. In lactose and lactitol tolerance tests, two concentrations of each sugar, 12 and 20 g of lactose (BDH Limited, Poole, England) and 12 and 20 g of lactitol (Lacty[R]) (CCA, Biochem, Gorinchem, Netherlands) in 250 mL distilled water were prepared and used as test diets.

Experimental procedure

Thirty-nine healthy volunteers (11 males and 28 females) aged 18-41 years participated in the study. The volunteers agreed to participate after the risks and benefits had been fully explained. Ethical approval was provided by the Faculty of Medicine, Ramathibodi Hospital, Mahidol University. All subjects lacked history or symptoms of chronic gastrointestinal disease and had not received antibiotics for at least 4 weeks before the experiment. They were requested not to consume gas-forming foods such as milk and milk products, dried legume seeds, cabbage, onion, or high-fiber foods for one day, particularly at dinner on the day preceding the test. They were given a test diet after overnight fasting. 12 and 20 g of lactose and lactitol, respectively, were dissolved in 250 mL water and used as test diets. During the experimental period of 7 h, the subjects were allowed to have normal activities but smoking, sleeping, and excessive work or exercise were not allowed. No food was given to the subjects except water.

Breath samples were collected before and at 30 min intervals for 7 h after consumption of the test diet. A sample of expired air (30 mL) was collected at the end of the prolonged expiration period using a reusable air collection bag. Gastrointestinal symptoms were recorded after consumption of the test diets for 24 h. Four test diets were used and each diet required a period of 3-5 days for wash-out.

Measurement of hydrogen in breath

End expiratory breath hydrogen was measured directly using the Quintron Model 12i MicroLyzer[R], a special-purpose gas chromatograph equipped with an electrochemical cell. The monitor was calibrated to zero with atmospheric air and to 95 ppm of hydrogen with a standard gas mixture purchased from Quintron Company. The measurement of each sample was repeated three times. The stability of the calibration curve was checked every 2 h.

Calculation and statistics

The absolute magnitude of the peak H_2 concentration at 30 min intervals was measured and the peak H_2 concentration above the baseline was calculated. Cumulative H_2 excretion between 0 and 7 h was estimated by calculating the area under the curve of H_2 concentration plotted against time according to the following equation:

$$A = (\frac{1}{2} H_1 + H_2 + H_3 \ldots\ldots\ldots\ldots + H_{n+1} + \frac{1}{2} H_n) \quad t$$

where A is area under the H_2 peak, H is breath H_2 concentration (ppm), and t is 30 min.

Data derived after intake of the test diets were subjected to a Student’s t-test with a minimum significance level of 5% (11).

The relationships between hydrogen production and gastrointestinal symptoms observed after a dose of 12 g lactitol were tabulated in a 2 x 2 contingency table (Table 1) which represents the relationship of a particular disease (present or absent) to a particular predictor (positive or negative) (11).

The following terms can be defined on the basis of Table 1:

European Journal of Nutrition, Vol. 38, Number 5 (1999)

$$\text{Prior probability} = \frac{TP + FN}{TP + FN + FP + TN}$$

$$\text{Accuracy} = \frac{TP + TN}{TP + FN + FP + TN}$$

$$\text{Sensitivity (or true-positive rate)} = \frac{TP}{TP + FN}$$

$$\text{Specificity (or true-negative rate)} = \frac{TN}{TN + FP}$$

$$\text{False-negative rate (1-sensitivity)} = \frac{FN}{TP + FN}$$

$$\text{False-positive rate (1-specificity)} = \frac{FP}{TN + FP}$$

$$\text{Predictive value positive} = \frac{TP}{TP + FP}$$

$$\text{Predictive value negative} = \frac{TN}{TN + FN}$$

In this study the predictor is the breath hydrogen concentration. A 'case' is a subject classified as lactitol intolerant.

Results

Baseline breath hydrogen in healthy adults

The average baseline breath H_2 concentration is shown in Fig. 1. All subjects showed a low level of baseline peak H_2 concentrations ranging from 2.4 ± 0.1 to 5.9 ± 0.8 ppm (mean ± SEM). Frequently, breath hydrogen on waking up (15-30 min before the test started) was high but fell precipitously after 3-4 h. The hydrogen production during the first 2 h after drinking 250 mL water was significantly higher (P .05) than levels in the late morning over 4-6 h.

Lactose absorption in Thai adults

After intake of the aqueous solution of 12 g lactose which is equivalent to the amount of lactose in a glass of milk (250 mL), the hydrogen in expired air was measured. The peak hydrogen level above the baseline for each subject is shown in Fig. 2. The results show that 9 out of 39 subjects (23%) were lactose absorbers (LA) producing peak hydrogen concentration of less than 20 ppm above the baseline (13.9 ± 1.5 ppm). About 77% (30 out of 39 subjects) were lactose malabsorbers having a maximum increment in breath hydrogen concentration of more than 20 ppm (21-106 ppm) above the baseline. Those without gastrointestinal symptoms (50%) were lactose malabsorber/tolerants (LMT), and those with gastrointestinal symptoms (50%) were lactose malabsorber/intolerants (LMI). The mean ± SEM of peak hydrogen concentrations in lactose absorbers, malabsorber/tolerants, and malabsorber/intolerants were 13.9 ± 0.6, 43.6 ± 1.0, and 37.1 ± 1.0 ppm, respectively.

When these subjects, except for lactose malabsorber/intolerants, were given 20 g lactose, 5 out of 24 subjects produced peak hydrogen concentrations of less than 20 ppm above the baseline (data not shown). Of these, 3 subjects suffered from diarrhea. Therefore, 22 out of 24 subjects (92%) could not absorb 20 g lactose. The average hydrogen peak level (Table 1) after intake of 20 g

Fig. 1 Composite fasting breath hydrogen concentration in 39 healthy adult subjects measured at 30 min intervals for 7 hours after getting up and intake of 250 mL water

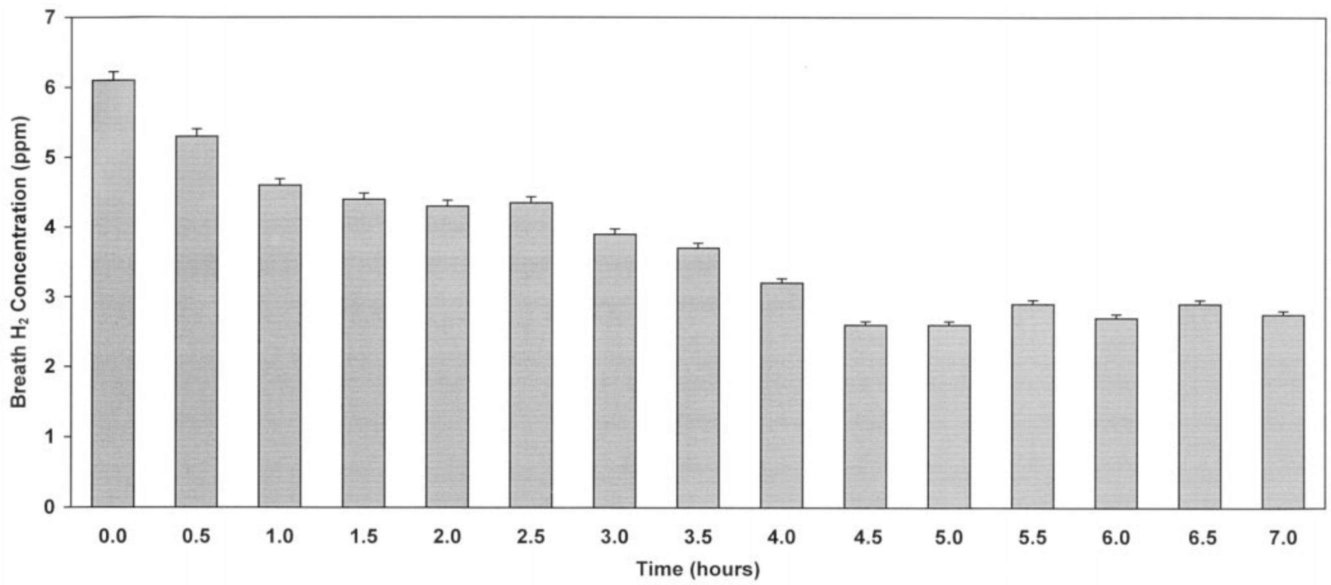
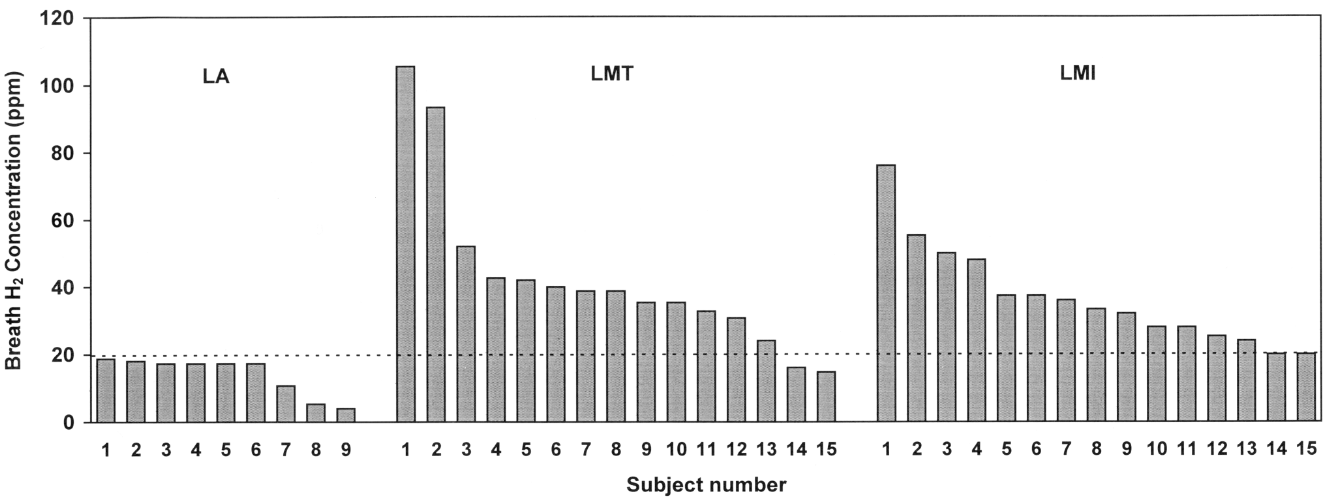


Fig. 2 Peak hydrogen concentration (ppm) in expired air above fasting level after intake of 12 g lactose in 39 healthy adult subjects



The dotted line represents a rise in breath hydrogen concentration of 20 ppm, taken as the cut-off criterion for identification of lactose malabsorbers. LA = Lactose absorbers; LMT = Lactose malabsorber/tolerants; LMI = Lactose malabsorber/intolerants.

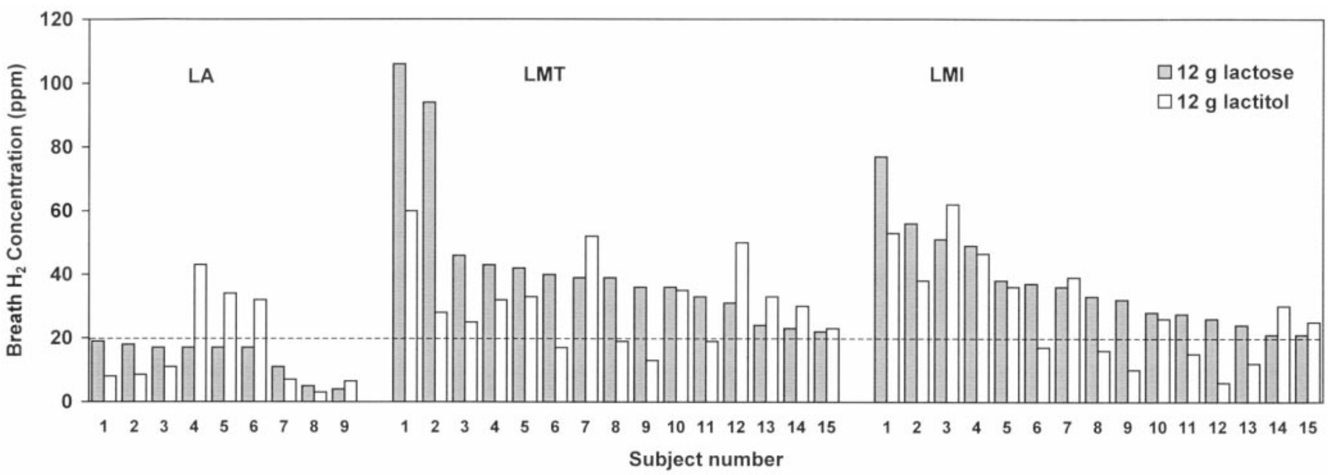
lactose was 40.9 ± 1.1 ppm which was significantly higher ($P < 0.05$) than that produced after intake of 12 g lactose (34.2 ± 0.5 ppm).

Lactitol absorption in Thai adults

After intake of the aqueous solution of 12 g lactitol, 16 out of 39 subjects produced peak H_2 concentrations of less than 20 ppm above the baseline (Fig. 3). However, 5 out of the 16 subjects experienced diarrhea. Therefore, only 11 in 39 or 28% of the subjects can be counted as lactitol absorbers and 72% as lactitol malabsorbers. Of lactitol malabsorbers, 11, 46, and 43% had formerly been identified as LA, LMT, and LMI, respectively, with 12 g lactose. The average levels of peak H_2 concentrations produced after ingestion of 12 g lactitol (27.1 ± 0.4 ppm) were significantly lower than those produced after ingestion of 12 g lactose (Table 2).

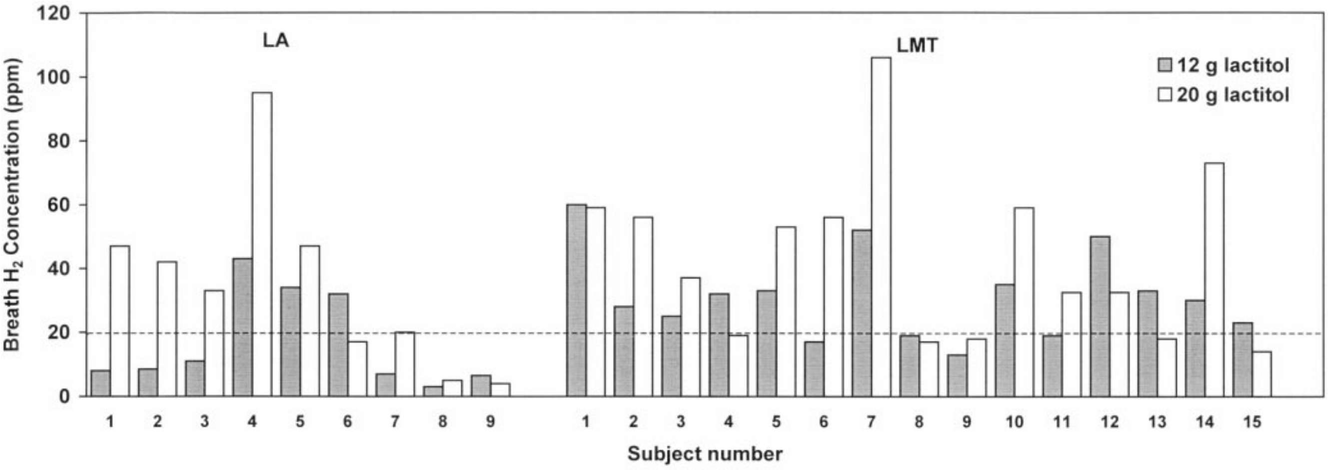
Fig. 4 shows the peak H_2 concentrations above the baseline produced after intake of 20 g lactitol. They were compared with those produced after 12 g lactitol intake. A total of 8 out of 24 subjects produced peak H_2 concentrations of less than 20 ppm above the baseline but 7 of these suffered from diarrhea. Therefore, 96% of the sub-

Fig. 3 Peak hydrogen concentration (ppm) in expired air above fasting level of individuals after intake of 12 g lactose and 12 g lactitol



The dotted line represents a rise in breath hydrogen concentration of 20 ppm, taken as the cut-off criterion for identification of lactose malabsorbers. LA = Lactose absorbers; LMT = Lactose malabsorber/tolerants; LMI = Lactose malabsorber/intolerants.

Fig. 4 Peak hydrogen concentration (ppm) in expired air above fasting level after intake of 12 g and 20 g lactitol in 9 LA and 15 LMT



The dotted line represents a rise in breath hydrogen concentration of 20 ppm, taken as the cut-off criterion for identification of lactose malabsorbers. LA = Lactose absorbers; LMT = Lactose malabsorber/tolerants; LMI = Lactose malabsorber/intolerants.

jects (23 out of 24) did not absorb 20 g lactitol which included those who produced peak H_2 concentration of more than 20 ppm above the baseline and those who had diarrhea associated with low hydrogen production. The average levels of peak H_2 concentrations produced after intake of 20 g lactitol (40.0 ± 1.10 ppm) were quite similar to those produced after intake of 20 g lactose (40.9 ± 1.10 ppm) (Table 2).

Average cumulative H_2 concentrations exhaled by each study group in different periods are shown in Fig. 5. In the 9 LA, the average cumulative H_2 excretion was found to differ significantly after consumption of 20 g lactose from that obtained after fasting. Average cumulative H_2 excretion after consumption of 12 g lactitol was slightly higher than that generated after consumption of 12 g lactose. Average cumulative H_2 excretion after consumption of 20 g lactitol was slightly higher than that produced after consumption of 20 g lactose but clearly higher than that produced by 12 g lactitol.

In the 15 LMT (Fig. 5), cumulative H_2 excretion after ingestion of 20 g lactitol, 20 g lactose, 12 g lactitol and 12 g lactose was significantly higher than the corresponding fasting level. Cumulative H_2 excretion after consumption of 12 g lactitol was slightly lower than that produced by 12 g lactose and that produced by 20 g lactitol was also slightly lower than that produced by 20 g lactose but higher than that produced after consumption of 12 g lactitol.

Table 1 A 2 x 2 contingency table

Predictor	Present ('Case')	Absent ('Non-case')
Positive	True-positives (TP)	False-positives (FP)
Negative	False-negatives (FN)	True-negatives (TN)

Table 2 Average peak hydrogen concentration (ppm) in expired air above baseline after intake of 12 and 20 g lactose and 12 and 20 g lactitol by healthy subjects

Test diet	Total subjects	Peak H_2 (ppm) (Mean ± SEM)
Lactose 12 g	39	34.2 ± 0.54
Lactose 20 g	24	40.9 ± 1.10
Lactitol 12 g	39	27.1 ± 0.41
Lactitol 20 g	24	40.0 ± 1.10

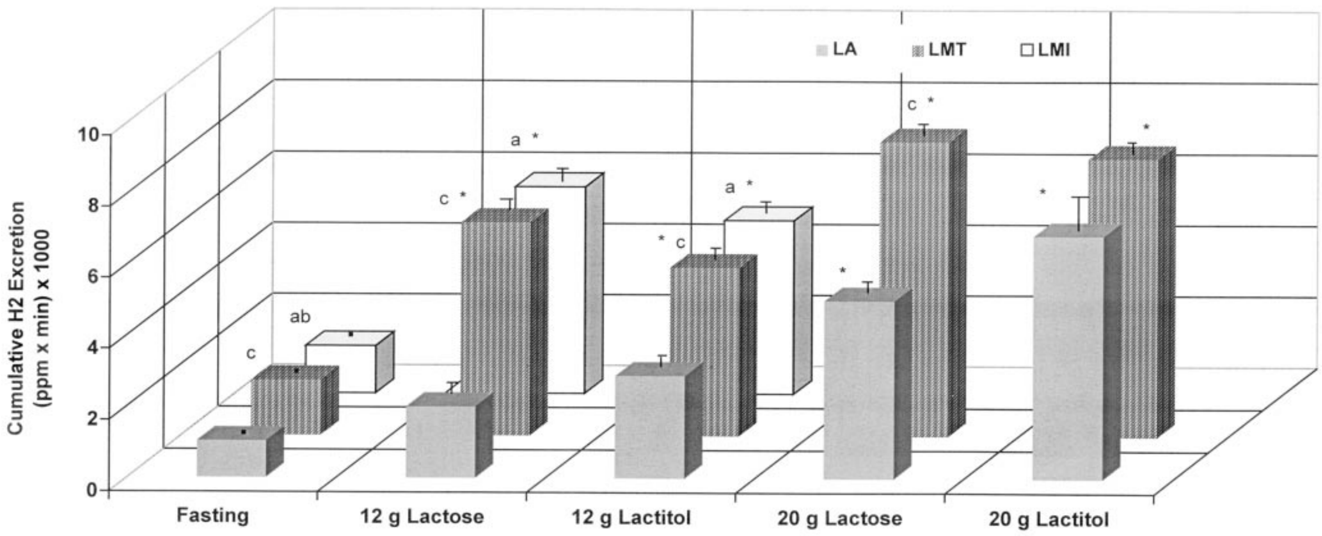
In the 15 LMI (Fig. 5), average cumulative H_2 excretion after consumption of 12 g lactitol and 12 g lactose was significantly higher than that produced during fasting. Average cumulative H_2 excretion produced by 12 g lactitol was slightly lower than that produced by 12 g lactose.

Gastrointestinal symptoms

After consumption of the test diets, the subjects reported various gastrointestinal symptoms which have been summarized in Table 3. Types and severity of the symptoms depended mainly on the amount and types of sugars consumed.

After intake of 12 g lactose, about 38% of the subjects (15 out of 39) experienced gastrointestinal symptoms, mainly abdominal pain and flatulence. Diarrhea was reported by 6 out of 15 subjects (40%) and nausea was found in a few.

Fig. 5 Cumulative H_2 excretion after intake of different amounts of lactose and lactitol in 9 LA, 15 LMT, 15 LMI.



Significant differences are indicated: [a,c] versus LA, [b] versus LMT, * versus fasting

About 54% of the subjects (13 out of 24) reported gastrointestinal symptoms after intake of 20 g lactose. Their symptoms, mainly diarrhea (in 6 out of 13 subjects, i.e., 46.2%), abdominal pain, and flatulence, were more severe than those produced after consumption of 12 g lactose.

Diarrhea was the main symptom reported by the subjects when lactitol had been given. After ingestion of 12 g lactitol, about 67% of the subjects (26 out of 39) reported gastrointestinal symptoms, and 42.3% (11 out of 26) reported diarrhea.

After 20 g lactitol, about 79% (19 out of 24 subjects) experienced gastrointestinal symptoms and 63.2% of the subjects (12 out of 19) suffered from diarrhea. Other minor uncomfortable gastrointestinal symptoms reported were flatulence, nausea, and abdominal pain.

As shown in Table 4, any change in the cut-off point that makes the test more sensitive (increase of the true-positive rate) will make it less specific (increase of the false-negative results) and vice versa. Thus, the best cut-off point should be high enough to take account of both sensitivity and specificity.

The receiver operating characteristic (ROC) curve reproduced in Fig. 6 shows that the hydrogen concentration of 15 ppm above the baseline level has been the best cut-off point for diagnosing lactitol intolerance. This cut-off point permitted a high sensitivity, a high predictive value positive, a high predictive value negative and a high accuracy even though specificity was not quite high. Sensitivity and specificity of the hydrogen breath test at this cut-off point were 85 and 38%, respectively. At this level of prior probability (67%), sensitivity (85%), and specificity (38%), the predictive value positive and accuracy were 73 and 69%, respectively.

Discussion

Lactitol has successfully been used in various countries in Europe and in the USA. In general, more than 20 g of the sugar substitute is administered for medical treatment and variable amounts are used in food industries. A problem limiting the role of this and other alternative carbohydrate sweeteners is their laxative action which requires that la-

Table 3 Number of subjects showing gastrointestinal and other symptoms after intake of 12 and 20 g lactose and 12 and 20 g lactitol

Test diet	Total subjects	Subjects with gastro-intestinal symptoms	Flatulence	Flatulence	Abdominal pain	Diarrhea	Cramps	Other symptoms Headache	Dizziness
Lactose 12 g	39	15	10	3	13	6	0	4	3
Lactose 20 g	24	13	5	2	4	6	2	0	2
Lactitol l12 g	39	26	28	8	9	11	0	4	4
Lactitol 20 g	24	19	4	3	4	12	4	0	0

Table 4 Effect of various increments in breath hydrogen concentration on sensitivity and specificity of the test for detecting lactitol intolerance (calculated from Table 1 – 2 x 2 contingency table)

Increase in breath H_2	Sensitivity (true-positive rate)	Specificity (true-negative rate)	Predictive value positive	Predictive value negative	Prior probability	Accuracy
(ppm)	(%)	(%)	(%)	(%)	(%)	(%)
≥ 60	8	100	100	35	67	38
≥ 55	12	100	100	36	67	41
≥ 50	19	92	83	36	67	44
≥ 45	19	85	71	34	67	41
≥ 40	31	85	80	38	67	59
≥ 35	35	62	64	32	67	44
≥ 30	54	54	70	37	67	54
≥ 25	58	46	68	35	67	54
≥ 20	65	38	68	36	67	56
≥ 15	85	38	73	56	67	69
≥ 10	88	15	68	40	67	64
≥ 5	100	0	67	0	67	67

beling of products containing such sweeteners provides cautionary information including limitations of the amounts consumed (12). In order to explore the use of lactitol in Thailand, a tolerance test for the sugar alcohol should be developed. Since lactitol is a lactose analogue and no recent data on lactose intolerance among Thai people are available, a tolerance test for lactose was therefore performed in this study prior to lactitol testing. A low dose of lactose, equivalent to a glass of milk, was used as the test diet in the lactose tolerance test and the effect of the same dose of lactitol was studied. Since a very high percentage of Thai people was reported to be lactose malabsorbers (13, 14), 20 g of lactitol was chosen as the high dose for lactitol tolerance testing which was performed in parallel to testing with an identical dose of lactose.

The hydrogen breath test has become the gold standard for the diagnosis of carbohydrate malabsorption. It is sensitive, non-invasive, and can be performed in subjects of all ages. It was chosen for lactose and lactitol tolerance testing in this study.

All subjects followed the request not to consume gas-forming foods during the day preceding the test day. This resulted in a low variation of breath hydrogen during the baseline study. A high baseline of peak H_2 concentration was observed among the subjects after waking up. This is a normal condition which reflects a general hypoventilation and decrease of colonic motility during sleeping (15, 16).

As in previous studies, the level of peak hydrogen production of ± 20 ppm above the baseline is considered as carbohydrate malabsorption in this study. The high peak H_2 production by the malabsorbers could indicate that a normal microflora was present in the intestine to digest unabsorbed carbohydrates which generated high concentrations of hydrogen in the air expired by the subjects (14, 17).

Various symptoms of different degrees of intensity found in the malabsorber/intolerants were probably due to the variation in the normal flora, lactase level, the net secretory response to an osmotic load, the intestinal motor response to an increase of the fluid load, and the irritability response of the small intestine and colon among individuals (18). The severity of the symptoms may affect the level of hydrogen in breath. As can be seen in this study, about 50% of the lactose absorbers showed peak hydrogen levels after consumption of 12 g lactose which were lower than those produced after consumption of 12 g lactitol (see Fig. 3, 5 and Table 2). However, the mean peak values and time courses of hydrogen production after consumption of these sugars were significantly different. The results implied that the lactose absorbers had the same level of enzyme activity to digest lactose and lactitol at 12 g doses.

It was found that about 33% of LMT and about 40% of LMI developed acute diarrhea after consumption of 12 g lactitol. Consumption of 12 g lactitol resulted in a significantly lower peak hydrogen production in both groups (see Fig. 3). The gastrointestinal symptoms of the LMT were less severe than those of the LMI. It can therefore be concluded that LA were able to tolerate 12 g lactitol and that LMT were able to tolerate it slightly better than LMI.

In the diagnosis of lactitol intolerance, the evaluation of specificity and sensitivity of the hydrogen breath test showed that a rise in the hydrogen concentration of 15 ppm above the baseline was the best cut-off point because it produced the lowest false-negative results. However, at

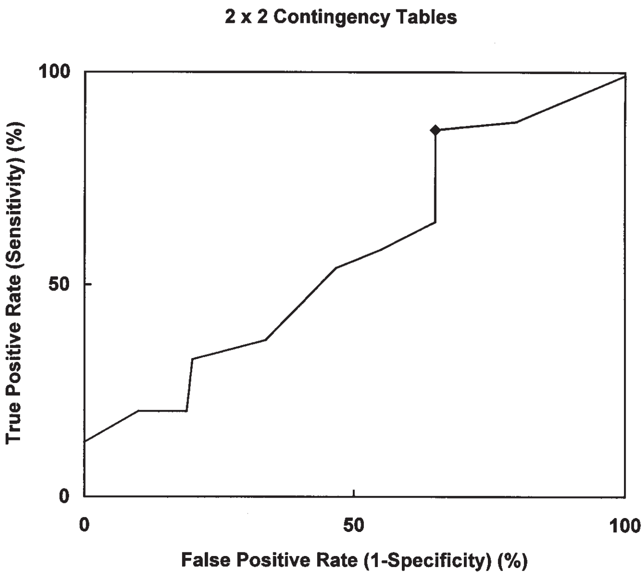


Fig. 6 Receiver operating characteristic (ROC) curve

this point, the optimum indicator of lactitol intolerance has a specificity of only 38% and a sensitivity of 85% (see Table 4 and Fig. 6).

According to the criterion that lactitol intolerants are subjects who develop the five specific gastrointestinal symptoms of flatulence, diarrhea, abdominal pain, nausea, and cramps after consumption of 12 g lactitol, about 67% of the subjects were identified as lactitol intolerants. The percentage of total symptoms were calculated from these five specific gastrointestinal symptoms which could be found in one subject. The number of subjects with lactitol intolerance included 11.5% of LA, 34.6% of LMT and 53.8% of LMI. It could be concluded that LA are less susceptible to lactitol than LMT and LMI. Lactose and lactitol have structural similarities. Therefore, lactase can also digest lactitol, but to a lesser degree. Lactitol causes less cumulative H_2 excretion than the expected amount since most of LMT and LMI had various gastrointestinal symptoms such as flatulence and diarrhea, which cause loss of H_2.

In 1969, Keusch et al. (13) reported on low levels of lactase in mucosal biopsy specimens found in 97% of Thai adults. Benjapong (14) reported in 1989 that, after consumption of a glass of milk (250 mL), 88% of the subjects were lactose malabsorbers on the basis of the hydrogen breath test. Based on the lactose tolerance test using 12 g lactose in this study, a lower rate of lactose malabsorbers (77%) among adult subjects was found. It remains unclear whether this small difference (88% versus 77%) is or is not due to the design of these experiments. It could also be a consequence of increased milk drinking among Thai people due to government programes since 1977.

When higher amounts of lactose were given, a very high percentage of lactose malabsorbers (more than 90%) and a considerably higher percentage of intolerants (more than 90%) were obtained as expected. Similar figures of malabsorbers were found when low and high doses of lactitol were given. However, numbers of intolerants were higher after consumption of low and high doses of lactitol as compared to lactose (67 and 79% vs. 38 and 54%, respectively). In addition, the severity in terms of the percentage of intolerants suffering from diarrhea was higher when a high dose of lactitol (20 g) was given.

Therefore, intolerance is not only a question of whether a person is MA or not, since cleavage rate and absorption rate of both sugar/sugar alcohol will eventually reach the maximal capacity. Although the hydrogen breath analysis test is the best method for identification of lactose malabsorption, it is not the best method to identify lactitol intolerance. A hydrogen concentration of 15 ppm above the baseline level was found to be the best cut-off point to indicate lactitol intolerance although sensitivity was 85% and specificity only 38% in this study.

Based on the results obtained in this study, it is suggested that 12 g of lactitol or less be used for Thai people which would generate about the same degree of severity of gastrointestinal symptoms as after consumption of one glass of milk. In comparison with Thailand, the prevalence of lactose intolerance in Scandinavia and northwest Europe is much lower (3-8%). Therefore, a higher dose of lactitol (40 g/day) was well tolerated by normal adult subjects (19, 20).

In conclusion, there is a greater susceptibility to lactitol in lactose malabsorbers than in lactose absorbers. These findings might be relevant for the limited use of lactitol in Thailand.

▶

References

1. den Uyl CH (1987) Technical and commercial aspects of the use of lactitol in foods as a reduced-calorie bulk sweetener. In: Developments in Sweetener. 3.; Grenby TH (ed) Elsevier Applied Science, London, New York, pp. 65–81
2. van Velthuijsen JA (1979) Food additives derived from lactose: lactitol and lactitol palmitate. J Agric Food Chem 27:680–686
3. Siijonmaa T, Heilonen M, Kreula M, Linko P (1978) Preparation and characterization of milk sugar alcohol, lactitol. Milchwissenschaft 33:733–736
4. Patil DH, Westaby D, Mahida YR, Palmer KR, Rees R, Clark ML, Dawson AM, Silk DBA (1987) Comparative modes of action of lactitol and lactulose in the treatment of hepatic encephalopathy. Gut 28:255–259
5. Scevola D, Zambelli A, Concia E, Perversi L, Candiani C (1989) Lactitiol and neomycin; monotherapy or combined therapy in the prevention and treatment of hepatic encephalopathy? Clin Ther 129:105–111
6. Lanthier P, Mosgan MY (1985) Lactitol in the treatment of chronic hepatic encephalopathy: an open comparison with lactulose. Gut 26:415–420
7. Dills WL (1989) Sugar alcohols as bulk sweeteners. AnnRevNutr. 9:161–186
8. Morgan MY, Hawley KE (1987) Lactitol vs. lactulose in the treatment of acute hepatic encephalopathy in cirrhotic patients: a double-blind, randomized trial. Hepatology 7:1278–1284
9. Heredia D, Caballeria J, Arroyo V, Ravelli O, Rodes J (1987) Lactitol versus lactulose in the treatment of acute portal systemic encephalopathy. J Hepatol 4:293–298
10. Wrong OM, Vince AJ (1987) Lactitol, lactulose, and blood ammonia. Lancet ii:1280–1281
11. Sachs L (1972) Statistische Auswertungsmethoden. 3rd ed; Springer Verlag, New York, Heidelberg, Berlin
12. Levin GV, Zehner LR, Saunders JP, Beade JR (1995) Sugar substitutes: their energy values, bulk characteristics, and potential health benefits. Am J Clin Nutr 62 (suppl):1161S–1168S
13. Keusch GT, Troncale FJ, Miller LH, Promadhat V, Anderson PR (1969) Acquired lactose malabsorption in Thai children. Pediatrics 43:540–545
14. Benjapong W (1987) Effect of solid foods and reduction of milk quantity on postprandial colonic lactose fermentation. M.Sc. Thesis in Nutrition. Faculty of Graduate Studies, Mahidol University
15. Tadesse K, Eastwood MA (1978) Metabolism of dietary fiber components in man assessed by breath hydrogen and methane. Br J Nutr 40:393–395
16. Permann JA, Molder S, Barr RG (1984) Fasting breath hydrogen concentration: normal values and clinical application. Gastroenterology 87:1358–1363
17. Griessen M, Bergoz R, Balant L, Loizeau E (1986) Effet du lactitol sur la production d'hydrogène expiré chez l'homme normal. Schweiz med Wschr 116:469–472
18. Nicholas L, Christopher MD, Theodore M, Bayless MD (1971) Role of the small bowel and colon in lactose-induced diarrhea. Gastroenterology 60:845–852
19. Gudmand-Høyer E (1994) The clinical significance of disaccharide maldigestion. Am J Clin Nutr 59 (suppl):735S–741S
20. Patil DH, Grimble GK, Silk DBA (1987) Lactitol, a new hydrogenated lactose derivative: intestinal absorption and laxative threshold in normal human subjects. Br J Nutr 57:195–199