Kefir improves lactose digestion and tolerance in adults with lactose maldigestion

STEVEN R. HERTZLER, PhD, RD; SHANNON M. CLANCY, MS, RD

ABSTRACT

Objective Kefir is a fermented milk beverage that contains different cultures than yogurt. The objective of this study was to determine whether kefir improves lactose digestion and tolerance in adults with lactose maldigestion.
Design Randomized block design.
Subjects Fifteen healthy, free-living adults with lactose maldigestion.
Main outcome measures Breath hydrogen excretion and lactose intolerance symptoms were monitored hourly for 8 hours after each test meal.
Intervention Subjects were fed test meals consisting of 20 g lactose portions of milk (2% reduced fat), plain and raspberry flavored kefir, and plain and raspberry flavored yogurt, each following an overnight (12 hour) fast.
Statistical analysis Mixed model ANOVA was performed on raw or transformed data, followed by Tukey HSD post hoc tests (when appropriate). Significance was defined as $P<.05$.
Results The breath hydrogen area under the curve (AUC) for milk (224±39 ppm · h) was significantly greater than for the plain yogurt (76±14 ppm · h, $P<.001$), the plain kefir (87±37 ppm · h, $P<.001$), and the flavored yogurt (76±14 ppm · h, $P=.005$). The flavored kefir had an intermediate response (156±26 ppm · h). The yogurts and kefirs all similarly reduced the perceived severity of flatulence by 54% to 71% relative to milk. Abdominal pain and diarrhea symptoms were negligible among the five treatments.
Applications/conclusions Because kefir improved lactose digestion and tolerance in this study, its use may be another potential strategy for overcoming lactose intolerance. Further studies of other types of kefir for improving lactose digestion are warranted. *J Am Diet Assoc. 2003;103:582-587.*

Lactose maldigestion is the inability to completely digest lactose, the major carbohydrate in virtually all mammalian milks. Lactose maldigestion affects approximately 75% of the world's adult population and occurs most often as the result of a genetically programmed decrease in intestinal lactose activity after the age of 3 to 5 years (1,2). Often the term "lactose intolerance" is used synonymously with lactose maldigestion, but this usage is not necessarily correct. Lactose maldigestion simply describes the incomplete digestion of lactose. Currently, the most common test for diagnosis of lactose maldigestion is the breath hydrogen test. This test operates on the principle that carbohydrate that escapes digestion in the small intestine is fermented by the colonic bacteria, resulting in the generation of hydrogen gas. A portion of this gas diffuses into the blood and is excreted via the pulmonary route.

Lactose intolerance, on the other hand, describes the presence of gastrointestinal symptoms such as abdominal pain, flatulence, bloating, nausea, or diarrhea resulting from lactose maldigestion. In recent years, it has been documented that a much smaller percentage of individuals who maldigest lactose actually develop clinical symptoms (3,4). Furthermore, there are many factors that influence the development of symptoms, including the following: (a) dose of lactose consumed (5,6); (b) consumption of milk with versus without meals (7); (c) use of fermented dairy foods, such as yogurt, that contain exogenous lactase, or β-galactosidase (8-10); (d) use of lactase enzyme supplements or lactase-treated milks (11); (e) colonic adaptation to lactose maldigestion (12-14); and (f) psychological factors (15) that are still unknown. Although it is true that lactose

S. R. Hertzler is an assistant professor in the Medical Dietetics Division, School of Allied Medical Professions, The Ohio State University, Columbus. S. M. Clancy is a clinical dietitian at the Toledo Hospital, Toledo, OH, and at time of research was a graduate student in the department of Medical Dietetics at The Ohio State University, Columbus.

Address correspondence to: Steven R. Hertzler, PhD, RD, Assistant Professor, 516 D Atwell Hall, 1583 Perry St, Columbus, OH 43210-1234. E-mail: Hertzler.4@osu.edu

0002-8223/03/10305-0004$35.00/0
doi: 10.1053/jada.2003.50111

intolerance remains much less common than lactose maldigestion, lactose intolerance is a significant problem worldwide.

The use of fermented dairy foods has long been employed as a strategy for overcoming lactose intolerance. For instance, yogurt has been demonstrated repeatedly to improve lactose digestion and tolerance in individuals with lactose maldigestion (8-10). This appears to be related to the presence of β-galactosidase in the yogurt starter culture bacteria (*Streptococcus salivarius* subsp. *thermophilus* and *Lactobacillus delbrueckii* subsp. *bulgaricus*).

Another fermented dairy food for which claims of improved lactose digestion have been made is kefir. Kefir originated in the Caucasus Mountains of Russia centuries ago and has been credited with various health-promoting properties. Historically, kefir has been prepared using the milk of sheep, goats, and cows, although soy milk kefirs are now commercially available (16). The traditional method of making kefir consists of inoculating pasteurized milk, following the cooling of the milk to 20°C to 25°C, with kefir grains (2%-10% by weight), incubating the mixture for 18 to 24 hours, and then filtering the mixture to remove the grains (16). Recently, however, the manufacturing process has been modified to use bulk lyophilized cultures made from kefir grains to increase yield and decrease the potential for contamination. Kefir grains are small, irregularly shaped, yellow-white, hard granules that resemble miniature cauliflower blossoms. These grains are a mass of bacteria, yeasts, polysaccharides, and other products of bacterial metabolism, together with curds of milk protein. The species of bacteria and yeasts used to make kefir vary, depending on geographic location. Kefir typically has a larger and more diverse range of microorganisms in its starter culture than does yogurt. For example, the kefir used in this study (Lifeway Kefir, Lifeway Foods, Inc., Morton Grove, IL) contains the following cultures: *Streptococcus lactis*, *Lactobacillus plantarum*, *Streptococcus cremoris*, *Lactobacillus casei, Streptococcus diacetylactis, Saccharomyces florentinus*, and *Leuconostoc cremoris* (17). The dual fermentation by the lactic acid bacteria and yeasts in kefir results in the production of small amounts of carbon dioxide, alcohol (0.01 to 0.1 g/100 g using starter cultures), and aromatic molecules that give kefir distinctive organoleptic properties compared with yogurt (16). Kefir typically has a tart flavor, is slightly carbonated because of the naturally occurring carbon dioxide, and is somewhat thicker than milk.

Because several of the microorganisms in the kefir starter culture are not typically found in yogurt, it is unknown whether kefir can improve lactose digestion in a manner similar to yogurt. After conducting a search of the Medline database, we found only one study that directly evaluated the effect of kefir on lactose digestion. De Vrese and colleagues (18) studied 10 Göttingen minipigs fed 1.0 L kefir (containing 101.1 mmol lactose) that was inoculated with fresh versus heat-treated (control) grains. The effect of microbial β-galactosidase on intestinal lactose digestion was estimated postprandially by measuring venous plasma galactose concentrations periodically for 6 hours post-kefir ingestion. In the fresh kefir grain group, mean postprandial plasma galactose peak and AUC values were enhanced by 30% and 23%, respectively, indicating improved lactose digestion. This study also showed no induction of intestinal β-galactosidase activity or intestinal lactose-hydrolyzing bacteria by lactose feeding. These results, although interesting, have not yet been confirmed in a human study. The objectives of this study were to (a) determine whether plain and flavored kefir improve lactose digestion and tolerance in adults with lactose maldigestion and (b) compare lactose digestion from plain and flavored kefir with yogurt, a food which has a well-documented history of improving lactose digestion.

METHODS

Subjects

Subjects were recruited based on their responses to advertisements that were posted on the campus of The Ohio State University and the surrounding area. The subjects were 15 healthy adults (8 males and 7 females). Subject characteristics, displayed as the mean±SEM (range), were as follows: Age was 26±1 year (20-34 years); body weight was 61±3 kg (45-84 kg); and body mass index (BMI) was 22±1 kg/m^2 (18-27 kg/m^2). Of the 15 subjects, 10 were Asian, 2 were African American, and 3 were white. Subjects reported no history of gastrointestinal disease or diabetes and had not taken antibiotics within 3 weeks of the study. All subjects were confirmed as adults with lactose maldigestion prior to the study on the basis of an increase in breath hydrogen of at least 10 parts per million (ppm), or 0.45 μmol hydrogen per liter air, above the fasting level (19) following the ingestion of 400 mL of milk containing 20 g of lactose. Subjects gave informed consent, and the study was approved by the Institutional Review Board Human Subjects Committee at The Ohio State University.

Treatments

There were five treatments that were each administered on one occasion per subject in this randomized block protocol (each subject served as his/her own control). The treatments were each followed by an 8-hour breath hydrogen and symptom test. All treatments were administered following an overnight (12 hour) fast. The five treatments consisted of 2% reduced-fat cow's milk (Kroger Co., Cincinnati, OH), Lifeway plain kefir, Lifeway raspberry-flavored kefir (Lifeway Foods, Inc.), Dannon low-fat plain yogurt, and Dannon low-fat raspberry-flavored yogurt (The Dannon Company, Inc., Tarrytown, NY). The amount of lactose in each product was determined using a commercial lactose/D-galactose test kit (Roche Diagnostics, Indianapolis, IN), and the portions of each product were standardized to provide 20 g of lactose. The amounts of the treatments that were necessary to provide 20 g lactose were 407 g, 378 g, 508 g, 428 g, and 519 g for the milk, plain yogurt, plain kefir, flavored yogurt, and flavored kefir, respectively. The nutritional content of the five treatments is shown in Table 1. The test meals were administered to the subjects in random order. Because of obvious differences in the texture and flavor of the products, it was not possible to mask the subjects to the identity of the treatments. However, in an attempt to minimize the effect of product appearance on our results, the milk and kefir were served in opaque disposable cups fitted with lids and straws. Similarly, the yogurts were served in opaque paper dishes fitted with a lid that had an opening just large enough to insert and remove a spoon. The study personnel did not inform the subjects of the identity of the treatments, but no attempts were made to mask the flavor or the texture of the treatments. On the evening before the test day, subjects consumed an ad libitum meal of white rice and ground meat. It has been demonstrated in previous studies that a low-dietary-fiber meal such as this greatly reduces the amount of residual hydrogen that is detected in the baseline breath measurement at the beginning of the test (20).

Table 1
Nutritional composition and portion sizes of milk, plain yogurt, plain kefir, flavored yogurt, and flavored kefir (standardized to provide 20 g lactose) that were fed to 15 adults with lactose maldigestion

Treatment	Amount fed (g)	Amount of lactose (g)	Total carbohydrate (g)	Total protein (g)	Total fat (g)	Energy (kcal)
Milk	407	20.0	20.0	13.3	4.2	171
Plain yogurt	378	20.0	30.0	20.0	5.8	252
Plain kefir	508	20.0	20.8	29.1	5.2	246
Flavored yogurt	428	20.0	73.5	17.0	3.8	396
Flavored kefir	519	20.0	44.7	29.8	4.3	337

β-Galactosidase Activity

The β-galactosidase activity of each meal was determined on a single occasion using a method described previously (21). In brief, an aliquot (0.1 g) of sample was diluted in a standard β-galactosidase buffer (0.02 mol Na_2HPO_4/L, 0.01 mol $MgSO_4 \cdot 7\ H_2O$, 0.001 mol dithiothreitol/L) and sonicated for five separate 1-minute periods (placed in an ice water bath between sonication periods). A 1.0 mL portion of the sonicated sample was combined with 4.0 mL of 0.005 mol/L ortho-nitrophenol-β-D-galactopyranoside (ONPG; prepared in the buffer mentioned above), and β-galactosidase activity was determined spectrophotometrically. The substrate ONPG is cleaved by β-galactosidase, resulting in absorbance at 420 nm at 37°C. The absorbance was measured until the increase was linear for 10 minutes. A unit of activity was defined as 1 μmol ONPG cleaved · $minute^{-1} \cdot g^{-1}$.

Breath Hydrogen Analysis

The breath hydrogen response was monitored hourly for 8 hours following treatment administration. End-alveolar air samples were collected in 60-mL plastic syringes fitted with three-way stopcocks and were immediately frozen at −20°C. Samples were

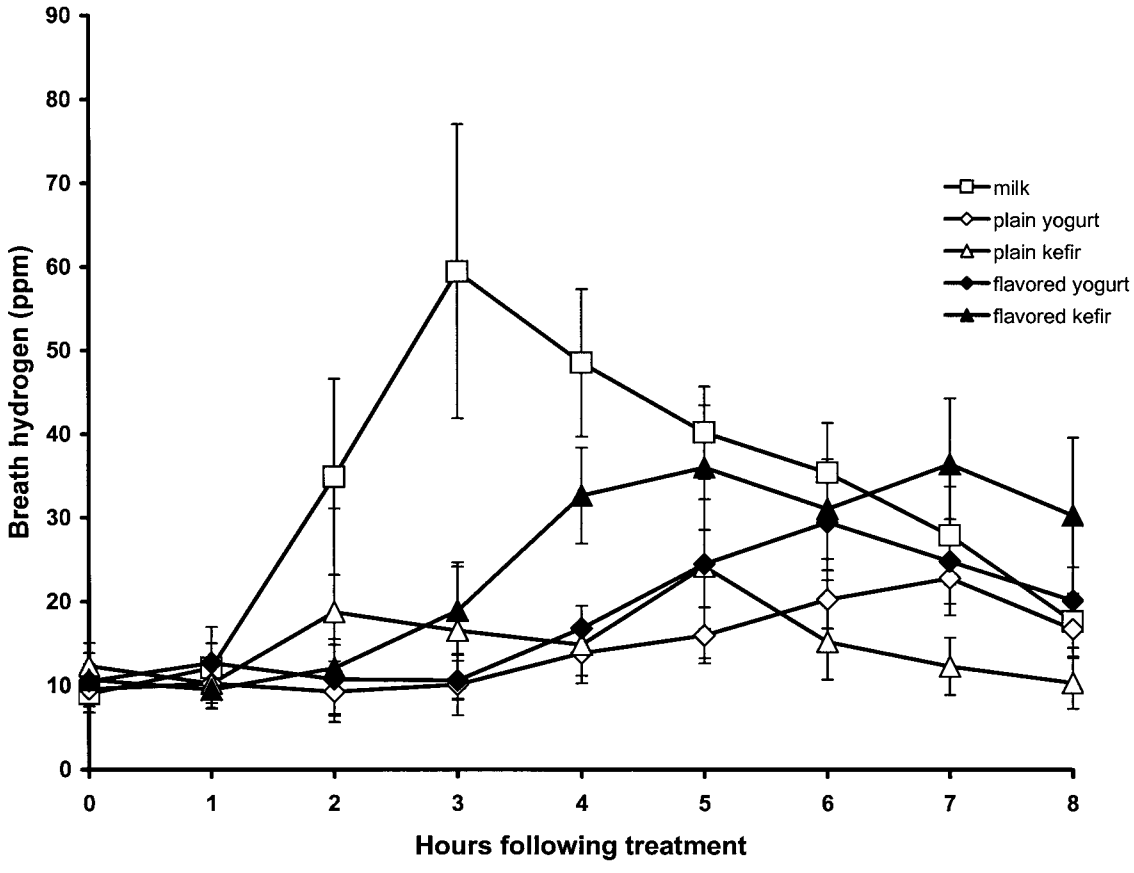

FIG 1. Hourly breath hydrogen production from 20 g lactose portions of milk, plain yogurt, plain kefir, flavored yogurt, and flavored kefir. Data are expressed as the mean±standard error. Number of subjects is 15.

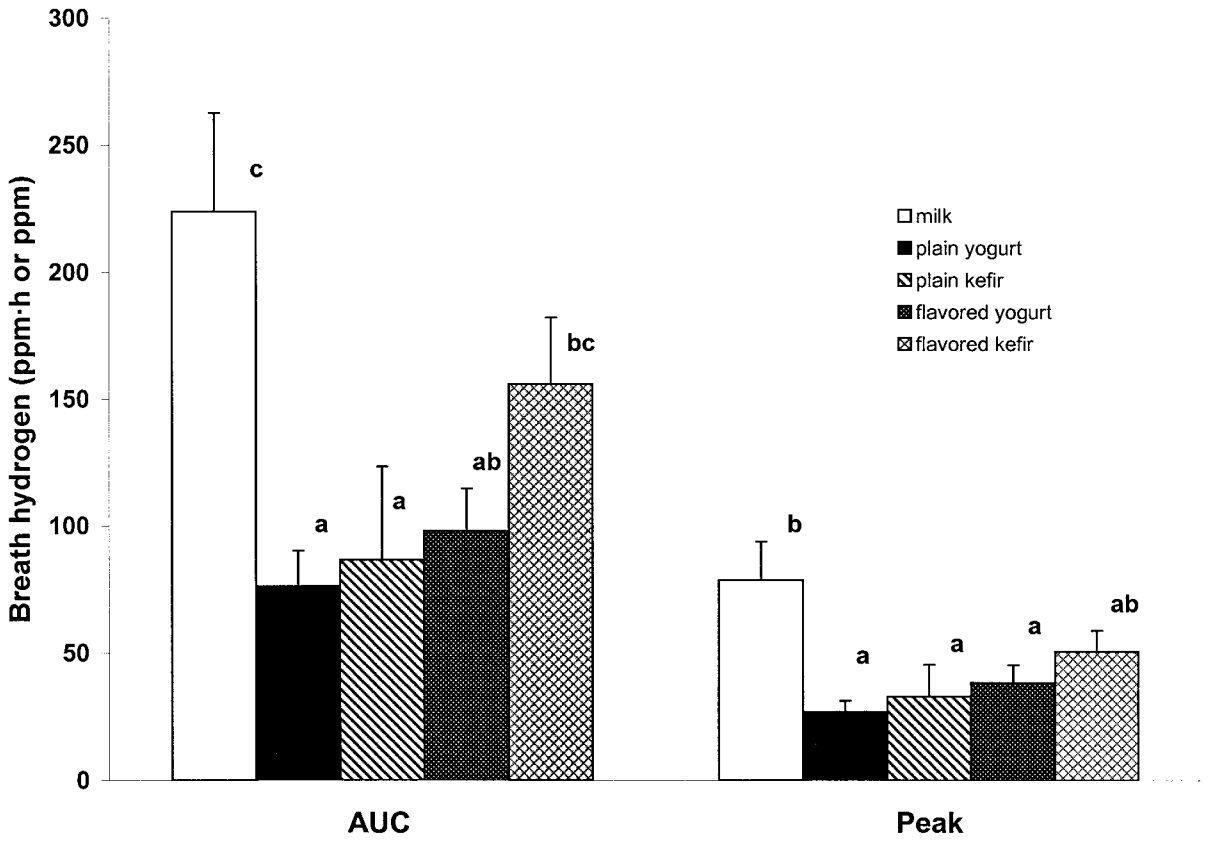

FIG 2. Breath hydrogen area under the curve (AUC) and peak responses to 20 g lactose portions of milk, plain yogurt, plain kefir, flavored yogurt, and flavored kefir. Data are expressed as the mean±standard error. Number of subjects is 15. Bars not sharing the same letter (within the AUC and Peak categories) are significantly different ($P<.05$) by the Tukey HSD paired comparisons method on square root transformed data.

analyzed within 48 hours. Freezing was shown to prevent leakage from syringes for at least 72 hours (unpublished data). Carbon dioxide and hydrogen concentrations were measured via gas chromatography using the Microlyzer SC instrument (Quintron Instruments, Inc., Milwaukee, WI). The observed hydrogen values were corrected for atmospheric contamination of alveolar air by normalization of the observed carbon dioxide concentrations to 40 mm Hg (5.3 kPa), the partial pressure of carbon dioxide in alveolar air (22). Positive, incremental breath hydrogen area under the curve (AUC) was calculated geometrically using the method of Wolever and colleagues (23).

Intolerance Symptoms

Subjects self-rated their perceived levels of severity for symptoms of flatulence, diarrhea, abdominal pain, and headache (a distracter symptom only) at each hourly breath sample during the breath hydrogen test. A ranked scale was used with the following definitions: 0 indicates no symptoms, 1 indicates slight symptoms, 2 indicates mild symptoms, 3 indicates moderate symptoms, 4 indicates moderately severe symptoms, and 5 indicates severe symptoms (5). Data are presented as the sum of the ratings for hours 1 to 8. Thus, the maximum possible symptom score was 40 for each symptom (a rating of 5 every hour for 8 hours). For determination of flatus frequency, subjects counted the number of rectal gas passages at hourly intervals over the 8-hour period. Again, data are presented as the sum of hours 1 to 8.

Statistical Analysis

Descriptive statistics and normality tests were performed for all variables using SPSS version 11.0, 2001 (SPSS, Inc., Chicago, IL). For those variables that were nonnormally distributed, data were transformed (ranks for symptom data and square root for breath H_2 data) prior to further analysis (24). When ranks were used, the sums of the symptom scores for the five treatments were ranked from 1 (least symptoms) to 5 (most symptoms). Mixed model ANOVA for a randomized block design (subject as random effect and treatment as fixed effect) was performed to detect global significant differences, followed by the Tukey HSD multiple comparisons procedure

Table 2
Lactose intolerance symptoms in 15 adults with lactose maldigestion to 20 g lactose from milk, plain yogurt, plain kefir, flavored yogurt, and flavored kefir[a]

	Flatus rating			Flatus frequency			Abdominal pain			Diarrhea		
	Mean rating[b]	Maximum	Mean rank[c]	Mean number[d]	Maximum	Mean rank[c]	Mean rating[b]	Maximum	Mean rank[c]	Mean rating[b]	Maximum	Mean rank[c]
Milk	7.8±2.0	20	4.2±0.3	12.9±3.7	46	3.9±0.4	4.7±1.6	22	3.6±0.3	0.8±0.6	8	3.1±0.2
Plain yogurt	2.5±1.0	13	2.8±0.2[g]	4.4±1.3[k]	19	2.7±0.3	1.3±0.7	11	2.6±0.3	0.5±0.5	8	2.7±0.1
Plain kefir	3.6±1.3	13	2.9±0.4[h]	6.5±2.4	32	2.7±0.4	2.7±1.5	20	3.0±0.3	1.5±1.0	15	3.5±0.3
Flavored yogurt	2.3±0.7	8	2.7±0.3[i]	5.3±1.3	16	3.0±0.3	1.8±0.7	9	3.0±0.1	0.3±0.2	3	2.8±0.2
Flavored kefir	3.1±1.3	14	2.5±0.3[j]	4.9±1.4	21	2.8±0.3	2.7±1.4	21	2.8±0.2	0.6±0.5	8	2.9±0.2
Global *P* value			.004[e]	.038[f]					.221[e]			.088[e]

[a]Mean±standard error of the mean (SEM), number of subjects=15.
[b]The sum of the hourly ratings for hours 1-8 during the challenge test was used for perceived symptom severity ratings. The maximum possible score is 40 (a rating of 5 for each hour).
[c]The sums of the hourly values for hours 1-8 resulting from each treatment were ranked from 1 (least symptoms) to 5 (most symptoms). The mean ranks±SEM are displayed.
[d]Flatus frequency equals the number of rectal gas passages from hours 1-8 after the treatments.
[e]Mixed model analysis of variance (ANOVA) was performed on rank-transformed data, followed by the Tukey HSD post hoc test. A *P* value of <.05 was considered significant.
[f]Mixed model ANOVA was performed on nontransformed data, followed by the Tukey HSD post hoc test. A *P* value of <.05 was considered significant.
[g]Significantly different from milk (*P*=.024).
[h]Significantly different from milk (*P*=.043).
[i]Significantly different from milk (*P*=.020).
[j]Significantly different from milk (*P*=.004).
[k]Significantly different from milk (*P*=.047).

when significant differences were found. A *P* value of .05 was considered significant.

RESULTS

The hourly breath hydrogen results and the breath hydrogen AUC/peak breath hydrogen for the five treatments are shown in Figures 1 and 2, respectively. There were no significant differences in the fasting breath hydrogen levels among the five treatments (*P*=.338). The yogurts and the kefirs caused peaks in breath hydrogen that occurred at 5 to 7 hours postingestion, and, except for the flavored kefir, all were significantly lower than the peak observed for milk. The breath hydrogen AUC was also calculated because it represents a semiquantitative estimate of total the amount of intestinal hydrogen production over the 8-hour time period (25). Breath hydrogen AUC was low and not significantly different among the plain yogurt, plain kefir, and flavored yogurt treatments (76±14, 87±37, and 98±17 ppm · h, respectively). Milk significantly increased breath hydrogen AUC (224±39 ppm · h) compared with the plain yogurt (*P*<.001), the plain kefir (*P*<.001), and the flavored yogurt (*P*=.005). The breath hydrogen AUC for the flavored kefir (156±26 ppm · h) was not significantly different from milk (*P*=.425) or flavored yogurt (*P*=.331), but it was higher than for the plain yogurt (*P*=.043) or the plain kefir (*P*=.008).

Lactose intolerance symptom results are shown in Table 2. Flatulence is the most commonly reported symptom in most lactose intolerance studies (4,5), and milk significantly increased the perceived severity of flatus relative to all four treatments. There were no differences in flatus severity among the yogurts and the kefirs. Similar results were observed for flatus frequency. Symptoms of abdominal pain and diarrhea were quite low (a mean rating of <5 out of a potential maximum of 40) and were not significantly different among the five treatments. The β-galactosidase activities of the milk, plain yogurt, plain kefir, flavored yogurt, and flavored kefir were 0.0, 3.4, 5.4, 3.2, and 5.2 units, respectively.

DISCUSSION

It is well-documented that lactose digestion is improved from a fermented dairy product, such as yogurt, when compared with milk. Savaiano and colleagues (10) demonstrated that yogurt caused one-fourth as much breath hydrogen excretion as milk, and tolerance to the yogurt was greatly improved as well. The most likely explanation for this improved lactose digestion is that the starter culture bacteria for yogurt contain a β-galactosidase. Because yogurt has excellent buffering capacity (26), it allows some of the bacterial cells to survive the gastric acid and reach the duodenum intact. At that point, it appears that bile acids play a major role, either by causing the lysis of the bacterial cells, thereby releasing β-galactosidase into the lumen or by the altering of the permeability of the cell membrane so that lactose can easily enter into the cell (27). Whatever the mechanism, the β-galactosidase gains access to the lactose substrate, a process termed "autodigestion" (9). It is also known that the autodigestive capacity of yogurt is limited to the amount of lactose that is naturally present in yogurt (21). Finally, not all cultured dairy products may improve lactose digestion. For example, most studies report that commercially produced acidophilus milks do not improve lactose digestion (28-31). Possible explanations include that bacterial cell counts in these products are lower than in yogurt or that some commercial *Lactobacillus acidophilus* strains are insensitive to bile acids (27).

Numerous health claims exist for kefir, including the enhancement of the immune system and improved digestive health, particularly with regard to lactose digestion. Although it seems plausible that kefir might improve lactose digestion in a manner similar to yogurt, there is a lack of research to support such a claim. Kefir contains different starter culture microorganisms than does yogurt and the bile acid sensitivity, β-galactosidase activity, or lactose transport of these organisms may differ. To our knowledge, this is the first study to evaluate lactose digestion from kefir in adults with lactose maldigestion. This study demonstrated that plain kefir improved lactose digestion just as well as plain yogurt.

Part of the explanation for this could be the high level of β-galactosidase activity in the kefir, which was approximately 60% higher than the plain yogurt. Although not all of the microorganisms in kefir possess β-galactosidase activity (eg, a yeast such as *Saccharomyces florentinus*) (18), apparently the cell counts, β-galactosidase activity, and/or bile sensitivity of the other cultures remain high enough to permit significant lactose autodigestion. Further studies are required to provide more information regarding the cell counts and bile sensitivity of the kefir cultures. The improved lactose digestion from kefir was accompanied by a concomitant improvement in flatus symptoms that was comparable with the improvement observed from plain yogurt.

The breath hydrogen response to the flavored yogurt was somewhat higher than for plain yogurt, but an even greater difference was observed between the flavored and plain kefirs. It is not clear whether the increased breath hydrogen response to the flavored products was due to some impairment of lactose digestion in the flavored products or whether the presence of additional sweeteners and/or fruit contributed additional fermentable carbohydrate. To examine the latter possibility, we conducted breath hydrogen tests with flavored kefir in three adults who were confirmed lactose digesters. All three had positive breath hydrogen tests (rise in breath hydrogen of >10 ppm above the fasting baseline) with the flavored kefir, although the rise in breath hydrogen was relatively small (Clancy SM, unpublished data, November 2001). It is likely that the use of high-fructose corn syrup as the sweetener in the flavored kefir, coupled with any naturally occurring fructose or sugar alcohols in the fruit, was responsible for this increase in breath hydrogen (32-34). Regardless of what might have caused the increased breath hydrogen response to the flavored kefir relative to plain kefir, the flavored kefir was tolerated just as well as plain kefir or plain yogurt.

APPLICATIONS

■ The use of fermented dairy foods such as yogurt for the improvement of lactose digestion and tolerance has been recommended for many years. This study suggests that a brand of kefir that is commonly available in the United States can serve as an alternative to yogurt for improving lactose digestion. The greater breath hydrogen response to the flavored kefir was likely due to the additional sweeteners or fruit, rather than impaired lactose digestion, and did not affect the clinical tolerance of the product. Further studies are needed to elicit the mechanism of this improved lactose digestion and to determine whether brands of kefir containing different starter culture microorganisms have a similar impact on lactose digestion.

References

1. Swaggerty DL, Walling AD, Klein RM. Lactose intolerance. *Am Fam Physician*. 2002;65:1845-1850, 1855-1856.
2. Sahi T. Genetics and epidemiology of adult-type hypolactasia. *Scan J Gastroenterol*. 1994;29(suppl)202:7-20.
3. Carroccio A, Montalto MG, Notarbatolo A, Lactase Deficiency Study Group. Lactose intolerance and self-reported milk intolerance: Relationship with lactose maldigestion and nutrient intake. *J Am Coll Nutr*. 1998;17:631-636.
4. Suarez FL, Savaiano DA, Levitt MD. A comparison of symptoms after the consumption of milk or lactose-hydrolyzed milk by people with self-reported severe lactose intolerance. *N Engl J Med*. 1995;333:1-4.
5. Hertzler SR, Huynh B-C L, Savaiano DA. How much lactose is low lactose? *J Am Diet Assoc*. 1996;96:243-246.
6. Vesa TH, Korpela RA, Sahi T. Tolerance to small amounts of lactose in lactose maldigesters. *Am J Clin Nutr*. 1996;64:197-201.
7. Martini MC, Savaiano DA. Reduced intolerance symptoms from lactose consumed during a meal. *Am J Clin Nutr*. 1988:47:57-60.
8. Pelletier X, Laure-Boussuge S, Donazzolo Y. Hydrogen excretion upon ingestion of dairy products in lactose-intolerant male subjects: Importance of the live flora. *Eur J Clin Nutr*. 2001;55:509-512.
9. Kolars JC, Levitt MD, Aouji M, Savaiano DA. Yogurt: An autodigesting source of lactose. *N Engl J Med*. 1984:310:1-3.
10. Savaiano DA, AbouElAnouar A, Smith DE, Levitt MD. Lactose malabsorption from yogurt, pasteurized yogurt, sweet acidophilus milk, and cultured milk in lactase-deficient individuals. *Am J Clin Nutr*. 1984;40:1219-1223.
11. Lin M-Y, DiPalma JA, Martini MC, Gross CJ, Harlander SK, Savaiano DA. Comparative effects of exogenous lactase (β-galactosidase) preparations on in vivo lactose digestion. *Dig Dis Sci*. 1993;38:2022-2027.
12. Hertzler SR, Savaiano DA. Colonic adaptation to daily lactose feeding in lactose maldigesters reduces lactose intolerance. *Am J Clin Nutr*. 1996;64:232-236.
13. Briet F, Pochart P, Marteau P, Flourie B, Arrigoni E, Rambaud JC. Improved clinical tolerance to chronic lactose ingestion in subjects with lactose intolerance: a placebo effect? *Gut*. 1997:41:632-635.
14. Pribila BA, Hertzler SR, Martin BR, Weaver CM, Savaiano DA. Improved lactose digestion and intolerance among African-American adolescent girls fed a dairy-rich diet. *J Am Diet Assoc*. 2000;100:524-528.
15. Suarez FL, Savaiano D, Arbisi P, Levitt MD. Tolerance to the daily ingestion of two cups of milk by individuals claiming lactose intolerance. *Am J Clin Nutr*. 1997;65:1502-1506.
16. Saloff-Coste CJ. Kefir. Danone World Newsletter 1996;11:1-19. Available at: www.danonevitapole.com/extranet/vitapole/portail.nsf/ACCUEIL?OpenForm&Seq=1. Accessed March 6, 2003.
17. Lifeway Foods, Inc. Lifeway kefir research reports. Available at: http://www.kefir.com/research/research_reports.html. Accessed March 6, 2003.
18. De Vrese M, Keller B, Barth CA. Enhancement of intestinal hydrolysis of lactose by microbial β-galactosidase (EC 3.2.1.23) of kefir. *Br J Nutr*. 1992;67:67-75.
19. Strocchi A, Corazza G, Ellis CJ, Gasbarrini G, Levitt MD. Detection of malabsorption of low doses of carbohydrate: accuracy of various breath H_2 criteria. *Gastroenterology*. 1993;105:1404-1410.
20. Anderson IH, Levine AS, Levitt MD. Incomplete absorption of the carbohydrate in all-purpose flour. *N Engl J Med*. 1981;304:891-892.
21. Martini MC, Kukielka D, Savaiano DA. Lactose digestion from yogurt: influence of a meal and additional lactose. *Am J Clin Nutr*. 1991;53:1253-1258.
22. Holum JR. *Fundamentals of General, Organic, and Biological Chemistry*. 2nd ed. New York, NY: John Wiley and Sons; 1982:111.
23. Wolever TMS, Jenkins DJA, Jenkins AL, Josse RG. The glycemic index: Methodology and clinical implications. *Am J Clin Nutr*. 1991;54:846-854.
24. Dawson-Sanders B, Trapp RG. *Basic and Clinical Biostatistics*. 2nd ed. Norwalk, CT: Appleton and Lange; 1994:105-106.
25. Levitt MD, Gibson GR, Christl SU. Gas metabolism in the large intestine. In: Gibson GR, Macfarlane GT, eds. *Human Colonic Bacteria: Role in Nutrition, Physiology, and Disease*. Boca Raton, FL: CRC Press, Inc.; 1995:135-154.
26. Martini MC, Bollweg GL, Levitt MD, Savaiano DA. Lactose digestion by yogurt β-galactosidase: Influence of pH and microbial cell integrity. *Am J Clin Nutr*. 1987;45:432-436.
27. Mustapha A, Jiang T, Savaiano DA. Improvement of lactose digestion by humans following ingestion of unfermented acidophilus milk: Influence of bile sensitivity, lactose transport, and acid tolerance of *Lactobacillus acidophilus*. *J Dairy Sci*. 1997;80:1537-1545.
28. Dehkordi N, Rao DR, Warren AP, Chawan CB. Lactose malabsorption as influenced by chocolate milk, skim milk, sucrose, whole milk, and lactic cultures. *J Am Diet Assoc*. 1995;95:484-486.
29. Newcomer AD, Park HS, O'Brien PC, McGill DB. Response of patients with irritable bowel syndrome and lactase deficiency using unfermented acidophilus milk. *Am J Clin Nutr.* 1983;38:257-263.
30. Onwulata CI, Rao DR, Vankineni P. Relative efficiency of yogurt, sweet acidophilus milk, hydrolyzed-lactose milk, and a commercial lactase tablet in alleviating lactose maldigestion. *Am J Clin Nutr*. 1989;49:1233-1237.
31. Saltzman JR, Russell RM, Golner B, Barakat, Dallal GE, Goldin BR. A randomized trial of *Lactobacillus acidophilus* BG2FO4 to treat lactose intolerance. *Am J Clin Nutr*. 1999;69:140-146.
32. Riby JE, Fujisawa T, Kretchmer N. Fructose absorption. *Am J Clin Nutr*. 1993;58(suppl):S748-S753.
33. Rumessen JJ, Gudmand-Hoyer E. Absorption capacity of fructose in healthy adults. Comparison with sucrose and its constituent monosaccharides. *Gut*. 1986;27:1161-1168.
34. Perman JA. Digestion and absorption of fruit juice carbohydrates. *J Am Coll Nutr*. 1996:15:S12-S17.

This study was funded by a grant (solicited by S. Hertzler) from Lifeway Foods, Inc., 6431 West Oakton Avenue, Morton Grove, IL 60053. Lifeway Foods also supplied the kefir used in the study.