

Lactose digestion from yogurt: mechanism and relevance^{1–3}

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ABSTRACT

Yogurt is traditionally consumed throughout the world among populations who are seemingly unable to digest lactose. This review provides a historical overview of the studies that show lactose digestion and tolerance from yogurt by lactose-intolerant people. The lactose in yogurt is digested more efficiently than other dairy sources of lactose because the bacteria inherent in yogurt assist with its digestion. The bacterial lactase survives the acidic conditions of the stomach, apparently being physically protected within the bacterial cells and facilitated by the buffering capacity of yogurt. The increasing pH as the yogurt enters the small intestine and a slower gastrointestinal transit time allow the bacterial lactase to be active, digesting lactose from yogurt sufficiently to prevent symptoms in lactose-intolerant people. There is little difference in the lactase capability of different commercial yogurts, because they apparently contain *Lactobacillus bulgaricus* and *Streptococcus thermophilus* in sufficient quantities (10⁸ bacteria/mL). However, *Lactobacillus acidophilus* appears to require cell membrane disruption to physically release the lactase. Compared with unflavored yogurts, flavored yogurts appear to exhibit somewhat reduced lactase activity but are still well tolerated.

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INTRODUCTION

People with lactose intolerance experience gastrointestinal symptoms when consuming milk or milk products because they lack sufficient small intestinal lactase (β -galactosidase) activity to adequately digest the milk sugar lactose (which comprises galactose and glucose linked by a β -galactoside bond). Undigested lactose consequently enters the colon where it is fermented by the resident microflora, resulting in symptoms including abdominal pain, bloating, diarrhea, and flatulence. Lactase deficiency is common in nonwhite adults, with a prevalence of 50–70% or higher (1–3), because of a genetically programmed loss of lactase after weaning.

Yogurt is produced by incubating concentrated milk with *Lactobacillus bulgaricus* and *Streptococcus thermophilus* (4). The bacteria ferment the milk, reducing the pH and creating the tangy taste associated with yogurt. The lactose content of the finished product is approximately similar to that of unconcentrated milk (4), although there may be small differences (perhaps ~5%) between products and brands according to manufacturing processes. Traditionally, lactose-intolerant populations have consumed yogurt without experiencing symptoms; however, because yogurt contains lactose, this would appear to be counterintuitive. This review provides an overview of the studies that reported on how yogurt is well tolerated by people with lactose intolerance.

EARLY WORK

It was suggested as early as 1974 that fermented dairy foods would be beneficial for lactose intolerance, although, at the time, this was hypothesized to be attributable to a low lactose content (5). However, when natural (live culture) yogurt was fed to rats, they absorbed galactose more efficiently and had greater intestinal lactase activity than rats fed pasteurized yogurt or a simulated yogurt formulation (6). Furthermore, the yogurt bacteria survived for 3 h in the gastrointestinal tract of the rats, and the authors hypothesized that the bacteria contributed to the hydrolysis of lactose (6). These data from experimental animals suggested that there was something more going on than a simple lactose dose effect.

The first human study followed in 1982, although it was not designed to determine the mechanism. In contrast to low-fat milk, a test drink of yogurt or acidophilus milk resulted in no symptoms in lactose-intolerant individuals (1). The reduced lactose quantity in the yogurt/fermented milk was implicated, because the dose of lactose in the test drinks was greater in the low-fat milk (24.6 g) than in the acidophilus milk (18.1 g) or yogurt (11.4 g) (1). The author suggested that lactase-containing microorganisms within the yogurt and fermented milk could continue to be active in the intestinal tract, participating in the hydrolysis of lactose (1). However, with the confounding effect of dose it was not possible to establish the mechanism.

YOGURT LACTASE ACTIVITY

The dose question was settled with a controlled study that showed that the lactose in yogurt is better digested than that in milk, apparently as a result of its lactase activity (4). In this study, the 10 participants were confirmed to be lactose-intolerant on the basis of elevated breath-hydrogen concentrations after a lactose challenge (4). This technique measures the hydrogen produced when undigested lactose is fermented by the colonic microflora from individuals who have low levels of gastrointestinal lactase

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activity. The subjects were given test drinks, each containing similar lactose loads, which comprised lactose in water (20 g lactose), milk (18 g lactose), commercial unflavored yogurt (18 g lactose), or lactulose (a nonabsorbable disaccharide, 10 g in water); and breath-hydrogen concentration was measured for 8 h afterward (4). The ingestion of 18 g of lactose in yogurt resulted in only approximately one-third as much hydrogen excretion as a similar load of lactose in milk or water, indicating a much better digestion of lactose from yogurt (4). The breath-hydrogen curves (**Figure 1**) showed a significantly smaller ($P < 0.01$) total AUC for yogurt (mean \pm SE: 108 ± 25 ppm/h) compared with milk (293 ± 33 ppm/h) or lactose solution (255 ± 33 ppm/h), with a smaller portion of yogurt (containing 11 g lactose) producing only 72 ± 22 ppm/h (4). The consumption of yogurt also resulted in fewer symptoms than did a similar quantity of lactose in milk or water, with diarrhea or flatulence reported by 20% of participants after yogurt and 80% of participants after milk (4). The lactase activity of duodenal contents was assessed indirectly in 3 individuals, by measuring lactose disappearance and galactose appearance. It was negligible before the yogurt test, but for at least 1 h afterward there was sufficient lactase activity to digest 50–100% of the lactose in 4 h (4), supporting the findings of the rat study (6). The measured lactase activity of yogurt decreased faster than would be expected from the measured rate of galactose appearance (4). This study suggested that the enhanced absorption of lactose in yogurt resulted from the intraintestinal digestion of lactose by yogurt-derived microbial lactase, with the survival of yogurt-derived lactase in the duodenum. The role of lactase-digesting bacteria in yogurt was further supported by findings that less breath hydrogen was produced by lactose-intolerant individuals after consuming unheated yogurt than when the product had been heated (7, 8).

YOGURT LACTASE ACTIVITY IN THE INTESTINE

The pH varies widely along the length of the gastrointestinal tract, being acidic (pH 1–2.5) in the stomach and increasing to 6.6 in the proximal small intestine and 7.5 in the terminal ileum (9). This pH variability affects the in vivo lactase activity from

yogurt. At 4°C and its final postfermented pH of 4, yogurt has minimal lactase activity (4). However, incubation at a pH of 7 and 37°C (and sonication) substantially increases its lactase activity, to 25 U/g, an amount sufficient to hydrolyze 95% of the lactose load in 4 h (4). Other studies have also documented pH effects (10, 11). The lactase activity of yogurt also increases in the presence of bile, as shown in vitro (7), perhaps by increasing the cellular permeability to allow more substrate to enter the bacterial cells (12). Thus, the activity of yogurt lactase is likely to vary at different gastrointestinal sites and should show maximal activity at an approximately neutral pH 7; it is no surprise then that lactase activity of duodenal contents was reported after ingestion of yogurt (4).

However, yogurt has a buffering capacity, requiring nearly 3 times as much acid to change its pH from 4.1 to 2.0 than is required to acidify milk (10). Gastrointestinal pH is influenced by this buffering capacity, as evidenced by the gastric pH remaining >2.7 for 3 h after ingestion of yogurt (10). This may also partly explain how lactase survives passage through the stomach; the integrity of the bacterial cell membrane may also play a role. By using a more direct approach than in previous studies (4), the lactase activity of duodenal contents was assessed after yogurt consumption (11). The fresh, unflavored yogurt contained ~ 10 g of lactose, and specific strains of *L. bulgaricus* and *S. thermophilus* (the “starter culture”), as well as being tagged with polyethylene glycol (a nonabsorbable internal standard for lactose) and spores of a marker bacterium, *Bacillus stearothermophilus* (an internal standard for bacteria, because it only germinates at 65°C). Lactose malabsorbers were given the yogurt either fresh ($n = 7$) or heated ($n = 3$). In duodenal samples taken after fresh yogurt ingestion, viable starter culture was detected for 60 min in 6 of 7 lactose malabsorbers, with large numbers of *L. bulgaricus* and *S. thermophilus* surviving passage through the stomach (11). The ratio of microbial lactase activity to the marker bacterium remained stable, showing that the enzyme is not degraded for at least 60 min after yogurt ingestion, despite some of the bacteria losing their viability (11). Yogurt ingestion affected duodenal pH, which decreased 15 min after ingestion and remained at <5.1 throughout

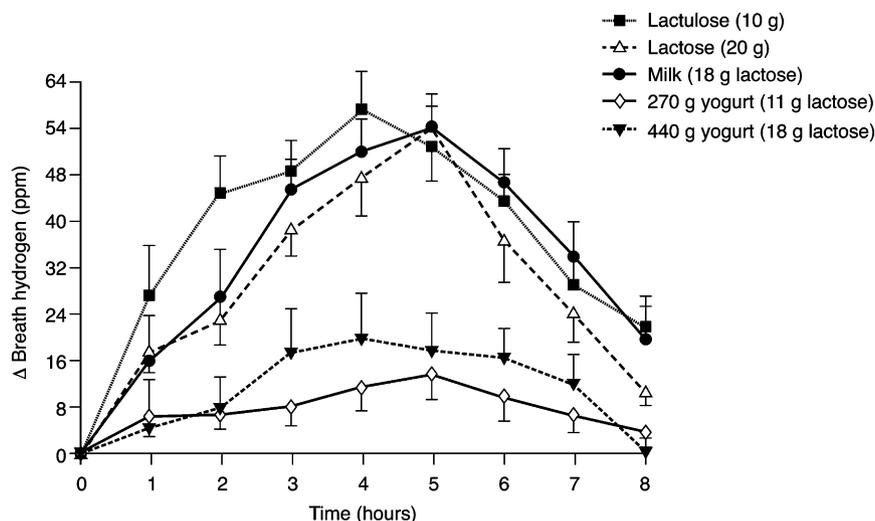


FIGURE 1. Mean (\pm SE) changes in breath-hydrogen concentrations after ingestion of lactose, milk, yogurt, or lactulose ($n = 10$). The amount of breath hydrogen expelled after ingestion of yogurt was one-third the amount expelled after ingestion of milk despite equivalent lactose loads. Reproduced with permission from reference 4.

(11). Whereas the *in vitro* lactase activity in yogurt was maximal at a pH of 7, it decreased by 80% when the pH was <5 (11). Hence, lactase activity in the duodenum increased after the ingestion of fresh yogurt, then decreased as duodenal pH lowered (Figure 2). Ratios of lactose to polyethylene glycol remained similar to preingested values for 90 min, suggesting that lactase could not hydrolyze the lactose (11). This study showed that after fresh yogurt ingestion, viable starter culture reaches the duodenum and contains lactase activity, confirming previous findings (4). However, it also suggests that the buffering capacity of the yogurt, which protects bacteria from the acidic gastric environment, may have an inhibitory effect on microbial lactase in the duodenum. The authors suggested that lactose digestion by microbial lactase might be occurring in the jejunum or ileum of the small intestine or (less likely) in the colon.

It was subsequently confirmed that >90% of the lactose in yogurt is digested in the small intestine, aided by a slow gastrointestinal transit time (13). This study, which collected ileal contents of lactase malabsorbers, found that the orocecal transit time (determined

from breath-hydrogen measurements) of fermentable components after the ingestion of yogurt (mean \pm SE: 165 \pm 17 min) and heated yogurt (206 \pm 19 min) was significantly longer than that with milk (103 \pm 19 min; $P < 0.01$ for comparisons of milk with yogurts, no significant difference between fresh and heated yogurt) (13). Significantly less lactose was recovered from the terminal ileum after yogurt (1740 \pm 260 mg) than after heated yogurt (2825 \pm 461 mg; $P < 0.05$) (Figure 3), with approximately one-fifth of yogurt lactase activity reaching the terminal ileum (13). This study showed that the small intestine is the site of most microbial lactase activity. The delay in transit time with yogurt compared with milk may be attributed to the difference in formulation—for example, increased osmolality or the physical thickening that occurs during fermentation (14). Together, these studies (11, 13) confirm that yogurt microbial lactase is detectable in the duodenum but is largely active in the distal small intestine, with only a small amount of lactose entering the colon.

BACTERIAL LACTASE ACTIVITIES

Having identified that yogurt microbial lactases digest lactose in the small intestine, the logical next question was “Are all bacteria equal?” Most commercial yogurts contain $\sim 10^8$ bacteria/mL, and strains may vary by product. Several bacterial strains and doses were compared in lactose-intolerant individuals (15). Yogurt (containing *S. thermophilus* and *L. bulgaricus*) and acidophilus milk (containing *Lactobacillus acidophilus*) were prepared by using commercially processed 2% low-fat milk, with 10^7 or 10^8 bacteria/mL. Lactose maldigestion was monitored by measuring breath-hydrogen excretion at hourly intervals for 8 h after consumption of each test drink containing ~ 20 g of lactose. The study found that, compared with the milk control (30.78 ppm breath hydrogen), there was little difference between *L. acidophilus* (either of the doses) or yogurt bacteria (10^7 /mL), whereas the standard dose of yogurt bacteria (10^8 /mL) resulted in significantly less hydrogen (9.81 ppm; $P < 0.05$) (15). This study showed that a 10-fold reduction in the dose of yogurt bacteria rendered their lactase activity ineffective and that the acidophilus milk had no lactase activity. However, sonicating acidophilus milk restores the lactase activity, presumably by releasing the enzyme from the cells (16). In contrast, sonication of yogurt bacteria appears to render them susceptible to gastric acid, reducing their lactase activity (10). It is possible that these differences may be attributable to species- or strain-specific characteristics of the bacteria/enzyme (eg, location of enzyme, cell structure).

Another study evaluated the ability of different strains and species of bacteria to digest lactose *in vivo*, comparing yogurts (containing mixtures of strains of *Streptococcus salivarius* subsp. *thermophilus* and *Lactobacillus delbrueckii* subsp. *bulgaricus*) and fermented milks (containing individual species of *S. thermophilus*, *L. bulgaricus*, *L. acidophilus*, or *Bifidobacterium bifidus*) that varied in lactase activity (14). All of the yogurts had similar lactose content, and all of the yogurts performed similarly in lactase-deficient individuals (Figure 4), regardless of their total or specific lactase activity and any variation in cell counts ($2.7\text{--}15 \times 10^8$ /g product) (14). The response to fermented milks was more varied (14). The results suggested that, rather than total lactase activity or microbial cell count, another factor (perhaps intracellular substrate transport) was the rate-limiting factor in determining lactose hydrolysis from yogurt.

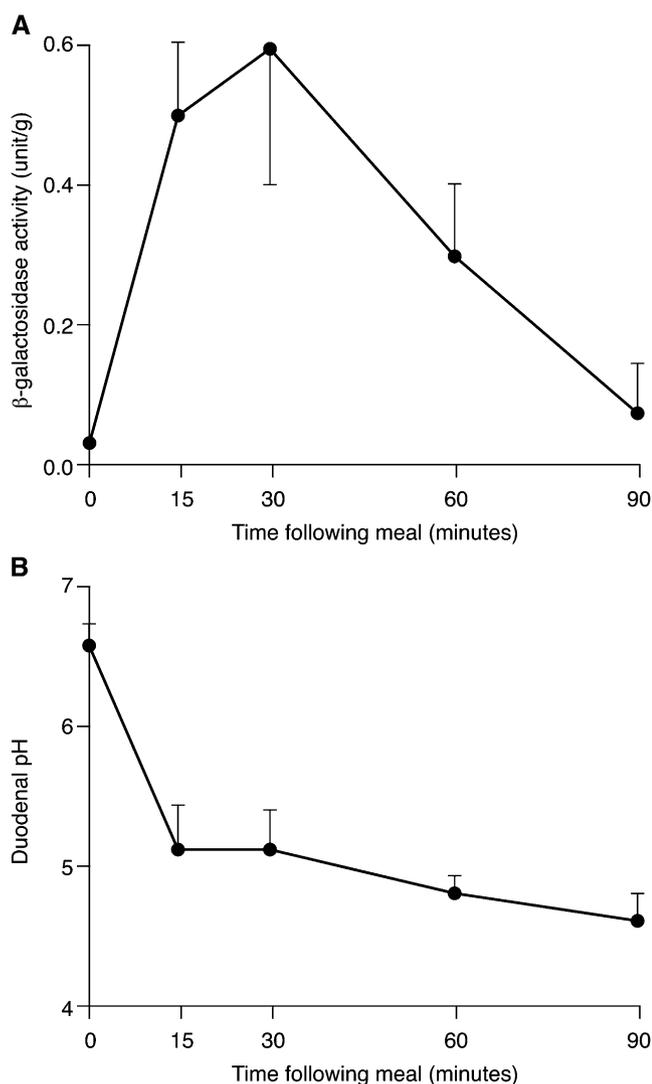


FIGURE 2. After fresh yogurt ingestion, duodenal lactase (β -galactosidase) activity increases (A) before falling again with decreasing duodenal pH (B). Values are means \pm SEs; $n = 7$. Reproduced with permission from reference 11.

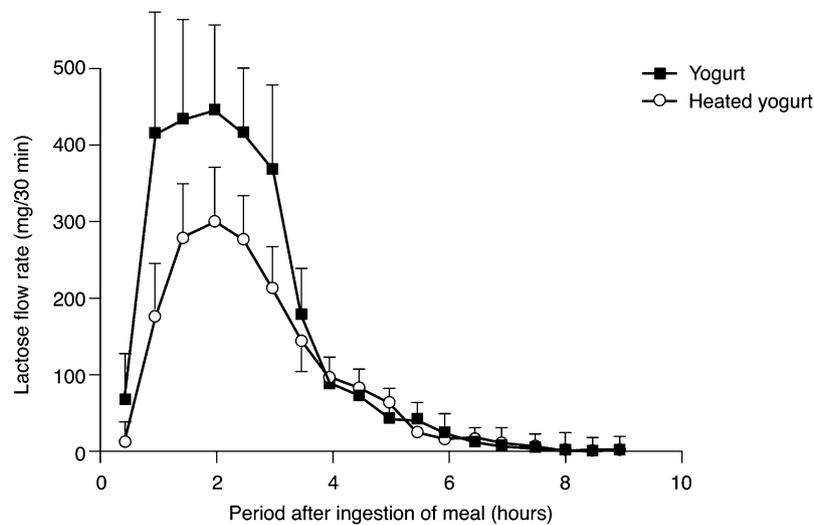


FIGURE 3. Mean (\pm SE) lactose flow rate through the ileum after ingestion of fresh yogurt or heated yogurt ($n = 8$ lactose-deficient subjects). Reproduced with permission from reference 13.

EFFECT OF YOGURT CHARACTERISTICS ON LACTOSE HYDROLYSIS

One factor that could influence the lactase activity of yogurt is concurrent food intake. This was investigated by examining the effect of consuming a meal with yogurt (17). Breath-hydrogen expiration, incidence of symptoms, and enzyme and lactose content of gastric aspirates indicated that concurrent food intake does not inhibit, and may slightly improve, lactose digestion from yogurt (17).

Whereas most studies have been conducted using plain (unflavored) yogurt, measuring breath hydrogen after ingestion of flavored yogurt shows that this may be associated with less lactase activity (more malabsorption). In a study measuring breath-hydrogen production in lactase-deficient individuals, unflavored yogurt caused significantly less (37 ppm/h; $P < 0.005$) hydrogen production than milk (185 ppm/h), whereas hydrogen production with flavored yogurt was intermediate (77 ppm/h) (18). The plain and flavored yogurts both contained significant lactase activities (18), so the increased breath hydrogen may be a result of dilution of the yogurt with the flavoring or sugar, an osmotic effect of the sugar in the stomach, or possible end-product inhibition by glucose. However, the subjects had no symptoms after consuming the flavored yogurt (18).

It is possible that there is an influence of shelf life on the lactase activity of yogurt. Whereas most research suggests that all yogurts are effective, there are exceptions. For example, a study that used commercial products off the shelf found considerable differences between them (19). Eight lactose-malabsorbing individuals were challenged with 3 different brands of yogurt (Borden, Dannon, and Royal Maid), each of which contained 20 g lactose (19). Breath-hydrogen measurements were significantly higher for Borden, both in terms of total ppm and peak ppm, although there was no relation with symptoms (19). The authors implicated their small sample size for the observed mild symptoms but suggested that other factors may be involved, including temperature changes during transportation of the products from manufacturer to retailer (19). It is not currently known if yogurts that sit on the shelf for a longer time have diminished lactase activity.

LONG-TERM BENEFITS

Evidence suggests that colonic adaptation to lactose consumption may occur over days to weeks in lactose maldigesters (20, 21), although there may also be a placebo effect (22).

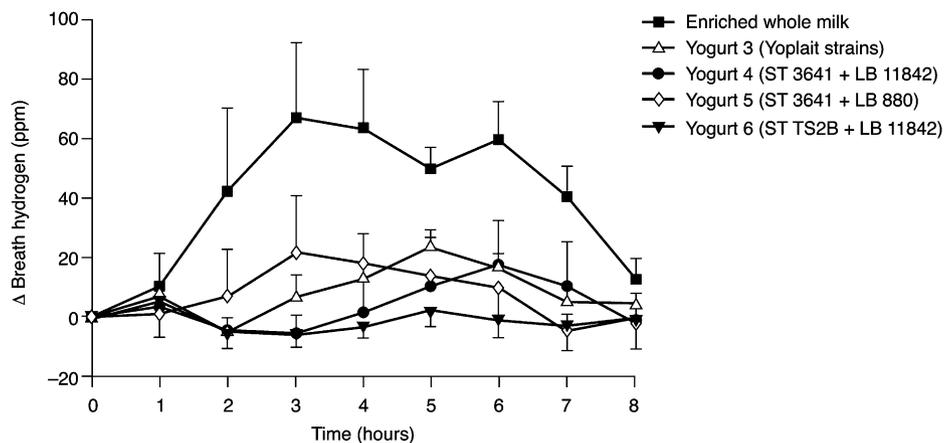


FIGURE 4. Mean (\pm SE) changes in concentration of breath hydrogen after ingestion of enriched whole milk or yogurts containing various strains of ST and LB or Yoplait strains ($n = 7$ lactase-deficient subjects). Reproduced with permission from reference 14. LB, *Lactobacillus delbrueckii* subsp. *bulgaricus*; ST, *Streptococcus salivarius* subsp. *thermophilus*.

Adaptation was apparent in a double-blind study that repeatedly provided yogurt (either fresh or heat-treated) to lactose malabsorbers for 15 d and measured breath hydrogen on days 1 and 15 (23). Whereas breath-hydrogen production was minimal and similar on days 1 and 15 for fresh yogurt, the response was improved for heated yogurt after 15 d of consumption (23). This suggests that regular consumption of small doses of lactose might be part of the management strategy for people with lactose intolerance, and that the benefit of yogurt consumption is maintained with regular consumption.

CONCLUSIONS

Autodigestion of lactose by yogurt bacteria improves its absorption, compared with other dairy products, in lactase-deficient people. Yogurt with sufficient numbers of *S. thermophilus* and *L. bulgaricus* (as is the case in most commercial yogurts) is very well tolerated by lactose maldigesters, because it is effectively analogous to taking an enzyme supplement with a dairy food.

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