NutritionNews

February 1986
Vol. 49 No. 1
ISSN 0369-6464

Since 1937

An Update on Lactose Intolerance.

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Background

The following update deals with the complex and interesting issue of lactose intolerance, for which *scientific* knowledge spans barely two decades. The classic work of Cuatrecasas and his group in Baltimore in 1965 provided the first diagnostic test for the intestinal capacity to digest lactose to its component sugars: glucose and galactose. In 1971, Christopher and Bayless in Baltimore clarified the mechanism whereby undigested lactose could produce symptoms of gastrointestinal intolerance. Mapping of various ethnic groups for their genetic susceptibility to lactose-related problems began in 1970 at Stanford University. The mission continues into the present era with a flurry of recent activity from geneticists based in Hannover, Germany. Thus, despite its brief history as a subject of scientific scrutiny, important new insights into lactose intolerance, its occurrence, recognition, and control have been revealed in the past five years.

Definition of the Terms of Reference

The sugar of interest is, of course, lactose, the predominant carbohydrate in mammalian milk. The starting point of any contemporary discussion is a clarification of the terms most often used and abused. "Milk intolerance," "lactose intolerance," and "lactose maldigestion" are intimately related but have distinct connotations. The important point is that not all symptoms following milk ingestion are due to its lactose content.

Milk Intolerance is the experience of subjective symptoms, of a gastrointestinal nature or involving other systemic manifestations, after consuming milk. It can be due to the lactose content of the milk, but other chemical substances or contaminating toxins can also produce intolerance to milk in individuals capable of efficient digestion of lactose.

Lactose Intolerance is the experience of symptoms of gastrointestinal discomfort: nausea, gas in the abdomen and intestines, abdominal cramping and distention, belching or flatulence, and/or watery stools after ingestion of lactose (either in milk, or other dairy food, or the sugar itself).

Lactose Maldigestion is the incapacity to quantitatively digest an oral dose of lactose into its constituent simple sugars—glucose and galactose—during its passage through the small intestine. The diagnosis is made by objective measures of the failure to take up the carbohydrate from the gut.

In a study in Mexico, we found a small, but meaningful, portion of the subjects manifesting symptoms after ingesting lactose-free milk. Similarly, in the enrollment of volunteers for an experimental study at the University of Connecticut, Jorge Rosado, Lindsay Allen, and I identified a number of individuals who traditionally avoided milk and experienced symptoms of intolerance after drinking a challenge dose of milk. At the same time, there was incontestable evidence that they had completely digested and absorbed the lactose in the challenge dose.

Lactose intolerance is caused by the failure of an individual to digest completely the lactose ingested orally. Some of this carbohydrate then reaches the large intestine, is fermented, and provokes secretion of water and the release of gases. In those individuals susceptible to lactose intolerance, the phenomenon is dependent on the dose of lactose. Symptoms diminish in intensity with smaller amounts of lactose, and disappear altogether at a certain level of intake of the sugar.

Moreover, *not all subjects* who are classified as lactose-maldigesters experience discomfort when they consume lactose-containing foods or beverages. Dr. Albert Newcomer at the Mayo Clinic reviewed the literature for studies using challenges with eight ounces (one glass) of milk in individuals classifed as lactose-maldigesters. He found that only one in five subjects experienced intolerance at that level of consumption.

An important point arises in the clinical diagnosis of lactose intolerance, namely, the isolation of lactose as the agent responsible for the symptoms. A history of gastrointestinal discomfort after drinking milk is an insufficient criterion. True lactose intolerance can only be confirmed with certainty in double-blind challenges in which intact lactose is given on one occasion and a placebo (usually a glucose or galactose mixture) in another. Only a *selective* symptomatic response to the lactose—but not to the placebo—can be considered truly to be lactose-related in origin. Dosage is yet another consideration. Conventionally, since lactose *digestion* tests were performed with 50 grams of the sugar (the amount in a full quart of milk), intolerance was judged on the same basis. A more dietarily relevant challenge would be with the amount of lactose in an eight-ounce serving of milk.

The digestion of dietary lactose is carried out by the enzyme "lactase" in the intestinal wall. High levels of lactase activity assure the splitting of lactose to its absorbable constituent simple sugars. All infants are born with high intestinal lactase levels. But one's genetic make-up determines whether these levels will be maintained after early childhood or whether the lactase activity will decrease to levels below the critical quantity for efficient digestion of normal amounts of lactose.

Therapeutic Issues

Generally, most individuals with lactase deficiency, also known as lactase non-persistence, do not experience symptoms after consuming usual dietary amounts of lactose. Only about one-fifth will have symptoms. These are usually discomfort or irritation, and not of direct significance to health. However, in certain vulnerable groups, there is reason to believe that intolerance, if manifested by infectious diarrhea, would be a risk to health. This would include severely malnourished children, with acute gastroenteritis, and frail elderly persons with unstable circulatory status.

At the Institute of Nutrition of Central America and Panama (INCAP) in Guatemala, we conducted studies in which the formula fed to children recovering from severe protein-malnutrition (kwashiorkor) was either the standard formula based on intact milk, or the same formula incubated for 24 hours with an enzyme which split the lactose into its component sugars. Children were observed over 45 days of realimentation. There was more

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About the Author

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He received his medical degree at Harvard Medical School in 1970, having previously graduated from Harvard University, and has held teaching positions at the University of Chicago and the Department of Nutrition and Food Science at Massachusetts Institute of Technology.

His major areas of research interest are in trace mineral nutrition and absorption and protein-energy malnutrition.

lactose formula-fed group, but thereafter no differences between the two diets were observed. Recovery of proteins and body mass, weight gain, clinical recovery, and absorption of macronutrients and calcium were equivalent whether or not lactose was present in the diet.

However, the greater laxative effect of lactose in these children initially was of interest. It is known that one consequence of malnutrition is a reduction in lactase activity which recovers with nutritional rehabilitation. Early in the course, then, inefficient lactose digestion with some percentage of children showing additional loose stools due to the maldigested sugar would be expected in protein-energy malnutrition.

Another area of worldwide concern is the refeeding of children with acute gastroenteritis following intervention with intravenous or oral rehydration therapy. To prevent further deterioration of nutritional status, many have advocated the introduction of nutritive formulas as early as possible in the convalescent period. It is possible that the lactose in milk fed at a critical period of intestinal injury and acute infectious diarrhea would prolong the recovery or even cause a clinical relapse of dehydration. An alternative would be a lactose-reduced (prehydrolyzed-lactose) refeeding formula, one that could eventually be prepacked in sealed envelopes as in the present distribution systems for oral rehydration therapy itself. Studies on lactose intolerance in the post-diarrheal child are presently underway or planned in Peru, Brazil, and Guatemala.

The final question relates to the extent of the danger associated with lactose intolerance, manifest as diarrhea, in frail elderly. In these individuals, the optimal performance of vital organs such as the kidney, the brain, and the heart itself may be compromised by the sudden loss of blood pressure associated with dehydration through colonic fluid loss.

Conditions of Increased Susceptibility

In order to experience lactose intolerance, one must have a condition that leads to inefficient digestion. Low levels of lactase activity in the intestine are one cause. However, alterations in peristaltic regulation of the gut, propelling meals irregularly and rapidly along, could also result in a colonic bath of lactose. Thus, an intestinal condition predisposing to motility disorder would be a potential cause of incomplete lactose digestion. Moreover, the experience of lactose intolerance is an inherently subjective one. Just as individuals vary one from another in pain sensitivity, so too do they vary in sensitivity to intestinal discomfort. Certain intestinal disorders of a functional nature may in fact be in large measure due to a combination of motility derangement and enhanced sensitivity to discomfort.

Lactose and lactose intolerance play a role in two such disorders—recurrent abdominal pain syndrome in children and irritable (or spastic) colon in adults (also known as functional bowel disease). This has been the England, and France, the elimination of milk from the diet of children with recurrent pain and lactase deficiency has been tried. Only in Boston was the effect substantial. In early studies from Denmark, sensitivity to very small amounts of lactose (less than five grams) was reported in persons with functional bowel disease. This group also was reported to have more lactose intolerance, with up to 95 percent of patients affected.

Strategies for Relieving Lactose Intolerance

The simplest approach relies, in part, on the residual lactase capacity extant even in persons of the lactase non-persistent type. Breath-hydrogen studies in our laboratories at INCAP in Guatemala suggest that 13 grams of sugar were fermented after an oral lactose challenge with 18 grams of lactose in known maldigesters. That means that five grams (28 percent) of the dose was actually digested and absorbed during the passage through the intestine. If the lactose could be slowed down in its transit through the gut, providing a more prolonged contact with the residual lactase, then perhaps even a more efficient digestion would be produced. We have shown that a meal of solid foods—egg, banana and cereal—reduces the rate of fermentation of lactose from the same 12-ounce dose of milk. This slower and smaller deposition of undigested sugar in the colon could explain the intolerance experienced with a drink of milk alone, as opposed to the tolerance of milk taken as part of a mixed meal.

The enzymatic hydrolysis of lactose is a certain method for eliminating intolerance to the primary carbohydrate in milk. Several different fungi and yeasts can be used industrially to produce microbial 'lactases' capable of digesting lactose. One such enzyme works most efficiently in the conditions of native milk, and, if left in the refrigerator for 24 hours, it will work slowly but surely toward the elimination of 90 percent of the intact lactose. This, however, is inconvenient, requiring advance preparation. Also, it imparts the sweet, sugary taste of glucose and galactose, which may not be appreciated by the user. A less-sweet (70 percent-hydrolyzed lactose) commercial milk can be bought across the counter in some supermarkets in the U.S. and Canada; this resolves both of the disadvantageous features of the home preparation of lactose-hydrolyzed milk.

In our laboratories, there has been interest in the development of microbial lactases as a potential remedy for lactose intolerance in people who would prefer to take their treatment at mealtime directly. Vega-Franco in Mexico and Mizote in Japan led teams of investigators who first demonstrated that lactose digestion could be enhanced, and symptoms of intolerance diminished, by the use of enzyme-modified milk. Also, we recently found that an enzyme in tablet form was highly efficient.

Thus, "enzyme replacement therapy" for lactase non-persistent individuals, using dairy products.

Digestion of lactose assisted by microbial lactases has been documented by workers at the University of Minnesota led by Dr. Dennis Savaiano. His discovery relates to yogurt. It has long been observed that yogurt does not produce distress when consumed by lactose-intolerant individuals. It was thought that this was related to a destruction of the lactose during the fermentation process. Analyses of yogurt revealed, surprisingly, that yogurt contains as much lactose per gram as whole milk. Then why the better tolerance of this product by lactase non-persistent individuals than of milk?

The answer to this question came from the Minnesotans, who confirmed the release of free 'lactases' from the culture bacteria of yogurt during its passage through the human stomach. Gastric acid and gastric enzymes open up the bacteria and release lactases that are effective against the lactose in the same yogurt: this is called "auto-digestion." Only *live* bacteria will provide the effect, as prepasteurized yogurt produces as much maldigestion and discomfort in lactase non-persistent subjects as does milk itself.

Some *Acidophilus* milk, if specially treated to disrupt the bacterial walls of the culture organisms, will also release enzymes capable of acting against the milk's lactose within the intestine, as shown by a research team at the USDA laboratory at Beltsville, Maryland.

Diagnostic Approaches in the Clinical Management of Lactose Intolerance

We have come a long way from the time, only 20 years ago, when the glucose rise after the intake of 50 grams of oral carbohydrate was the only mode of investigating in-vivo digestion and absorption. At least seven additional methods have been developed to assess the function of lactase. Of course, the "gold standard" for identification of lactase status is intestinal biopsy and enzyme assay of intestinal tissue. But this has obvious disadvantages as a routine procedure in a problem for which simple avoidance of milk and dairy products is a sure, if troubling, solution. Some of the other procedures have elaborate aspects or radiation hazards which also detract from their routine application.

The hydrogen breath-analysis test has a high degree of accuracy in establishing states of both complete and incomplete lactose digestion. The mode of sample collection—taking exhaled air into bags or syringes—is totally innocuous and noninvasive, making it ideally suited for children. It can detect the incomplete absorption of as little as 2 to 4 grams of lactose, so that the ordinary dietary amounts of lactose in the form of real foods can be used as challenge doses with the breath-hydrogen technique. It is based on the principle that when a fermentable carbohydrate reaches the bacteria of the human large intestine, the ensuing fermentation releases gaseous hydrogen (H_2). Notwithstanding some pitfalls, the H_2-breath-analysis test has emerged as the procedure of choice for the quantitative determination of the com-

pleteness of lactose digestion in clinical settings, as well as in survey work.

Research Needs

Lactose intolerance after drinking one glass of milk is not experienced by 80 percent of lactase non-persistent or lactase-deficient individuals. For them, lactose intolerance is a non-issue. However, the lactose intolerance experienced among the non-white** races of the world affects an extraordinarily large number of people. They have the option not to drink milk (and thus lose its nutritional benefits), or to choose from the strategies outlined above that permit them to enjoy milk without fear of discomfort.

The theory that the gastrointestinal symptoms that accompany milk-drinking in some persons is based on the digestibility of the lactose has a tenure of only 20 years. Thus, it is not surprising that all of the answers to either the biological or the practical aspects of lactose intolerance are not at hand. The most urgent area for research would be in situations where milk might be undesirable, as in the early period of convalescence from acute gastroenteritis. The limitations imposed by lactose intolerance in the feeding of intact milk to the post-diarrheal infant are worthy of investigation. It was cited with a high priority by the WHO Working Group. The convenience and practicality of the alternative, lactose-hydrolyzed milk formula, need further study.

Another area that has been addressed as a research need is the degree to which individuals who are both lactose maldigesters and lactose-intolerant, when fed milk sporadically, "adapt" to a regular, daily intake of milk with the disappearance of symptoms. Clearly established is the fact that human lactase cannot be *induced* by regular feeding.

Conclusion

The most fruitful discussion of lactose intolerance develops when the terms of reference are considered and the distinctions are made between milk intolerance, on the one hand, and lactose maldigestion on the other. At best, lactose intolerance is an annoyance that will make an individual avoid drinking milk; at worst it could be a reaction that would threaten the health of an at-risk individual, such as a dehydrated child or a frail elder who is given a substantial oral meal of lactose-containing food. The latter situations are to be understood so they can be avoided.

Dietary strategies to reduce lactose-induced discomfort, by slowing transit of a milk meal, or by using a 'lactase' enzyme either before or with the meal, are available for the lactose-malabsorbing, lactose-intolerant person who still wishes to drink milk or have dairy items from time to time. Paradoxically, yet another approach for the lactase non-persistent individual with lactose-related symptoms is not to avoid the sugar, but to indulge in it so often that the colon no longer reacts to it.

** Lactase non-persistence is also prevalent among certain Caucasian ethnic groups including Mediterranean peoples, hemetic groups, and semetic groups.

Reviews

Weight Watchers Quick Start Program Cookbook. Jean Nidetch. New American Library Books, New York, N.Y., 1984, $17.95, 455 pages, hardcover.

The Weight Watchers Quick Start Program Cookbook presents three progressively liberal Quick Start programs and the Weight Watchers Full Exchange program, plus over 500 recipes in an easy-to-read, matter-of-fact manner. The book features: a recipe-keyed menu planner; recipes designated as "fast" or "budget stretchers;" exchange information on each recipe; and carbohydrate, protein, fat, sodium, and cholesterol content per serving for each recipe.

The Quick Start program, based on the original Weight Watchers diet, was developed for three reasons—it offers a plan that is simple to follow, is flexible, and gives a fast start to losing weight.

The information presented is accurate. The calorie levels may seem quite restrictive, ranging from 940 calories (Quick Start 1) to 1115 calories (Quick Start 3) for women. However, Quick Start 1 is to be followed for only one week. Unfortunately, the time periods for progression from Quick Start 2 to the Full Exchange Plan are not given.

The book can be recommended for persons wanting to lose weight and to get off to a "quick start." However, the dieter should probably progress to the Full Exchange Plan within a month's time. It should be recognized that the program of Weight Watchers International includes a plan for moderate exercise, although this book does not mention its role in weight control.

The above review was provided by the Chicago Nutrition Association.

Tools and Techniques

The Fitcomp Nutrition, Exercise, and Weight Control System

Victor Katch, Ed.D., University of Michigan, and Frank Katch, Ed.D., University of Massachusetts at Amherst.

Background

FITCOMP is the acronym for "Fitness by Computer," a system we developed in the early '70s to provide a rapid method of anthropornetric data analysis for various professional sports teams.

An interactive computer system for input, analysis, and output to provide timely reports on individuals, FITCOMP was designed to answer the need to integrate information about body compostion (chiefly recommendations on weight loss and an optimal playing-weight range) with nutritional recommendations and such parameters of physical fitness as muscular strength and endurance, cardiovascular capacity, and flexibility.

Early on, a decision was made against assessment of nutrient intake from daily recall of foods consumed, or 3- to 7-day food diaries. Instead, the position was taken that a computer could be programmed to plan daily menus based on an individual's food preferences. It is our firm belief that individuals respond more favorably to weight-loss programs when they are allowed to make choices about their food consumption and exercise. Instead of selecting specific calorie menus from cookbooks, we devised a computer program that constructs nutritionally balanced breakfasts, lunches, and dinners from a basic list of foods selected by the individual.

Design of the Program

Designed to be used by the clinician, as well as educators and consumers in general, the FITCOMP program includes an easy-to-read and easy-to-fill-out questionnaire that can be completed in private or with the help of a health professional.

The questionnaire includes a section on exercise habits and capabilities, current and desirable body weight (included is an option for input and output for body composition analysis from girth, skinfolds, hydrostatic weighing, and bioelectric impedance), and food preferences. A list of over 200 foods is presented and the clients choose those foods they would eat. Also included are "menu" foods such as lasagna, different casseroles, and desserts. As an option, we permit the client to choose three different levels of fat as a percent of the total caloric input. The normal dietary prescription includes 30 percent or less of fat, the low-calorie option includes 20 percent or less, while a very-low-fat option (12 percent fat) is also available.

The answers to the questionnaire can be read into the computer via different methods —keyboard terminal cards ("batch"), code-bar reader, or digitizer. Recently, we installed the program on a Hewlett Packard (HP-150) touch-screen computer, where it is possible for clients to sit at the computer screen and actually touch the images of foods they would want to eat. With the computer connected to the new HP Laser-Printer, printouts are obtained within minutes (a typical 20-page letter-quality printout is produced in less than four minutes).

Individualized Printouts

Each printout contains the following: Analysis and explanation of the individual's body composition (fat weight, lean body weight, percent body fat, body surface area), a

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