Xylitol
Excerpted from “The Sugar Trap & How To Avoid It”
By Beatrice Trum Hunter

Xylitol has about the same sweetness as sucrose and leaves a pleasant cool taste in the mouth. While examining xylitol’s potential as a sucrose substitute, Finnish dental researchers made a startling discovery. In 1976, headlines concerning xylitol were ecstatic, from newspapers as widely divergent as *The Wall Street Journal*, *The National Enquirer*, and *Food Processing*. FINNISH RESEARCHERS HAIL SWEETENER XYLITOL AS “MIRACLE” THAT CAN HEAL TOOTH DECAY, MIRACLE SWEETENER ENDS TOOTH DECAY, and FIVE STICKS A DAY KEEPS THE DENTIST AWAY, were the respective headlines. This enthusiasm was generated by results of the Turku Sugar Studies of Dr. Kauko Makinen, a biochemist, and his colleague, Arje Sheinin, D.D.S., who had divided 125 dental students from the Finnish University of Turku into three feeding groups. The first consumed foods in which xylitol totally replaced sucrose; for the second, fructose totally replaced sucrose; and for the third, sucrose was eaten at an average level. After one year, the xylitol group had 90 percent fewer cavities than the sucrose group, and 30 to 40 percent fewer cavities than the fructose group.

In another Turku experiment, a hundred students were divided into two groups. One group chewed approximately five sticks of xylitol-sweetened gum daily while the other chewed sucrose-sweetened gum. Both groups were given free choice of food selections. After a year, the xylitol group showed an average decrease of one cavity per person; the sucrose group, an average increase of three cavities per person. In some students, the process of tooth decay appeared to be reversed, which suggested a possible therapeutic effect from xylitol.

The study demonstrated the role of fermentable carbohydrates in human caries formation and the fact that oral bacteria cannot ferment xylitol. Apparently, the breakdown of carbohydrates to acids in the mouth is arrested, partly because xylitol itself does not break down in the mouth but does so in the stomach. Also, xylitol raises the plaque pH above the acid range for prolonged periods, inhibits the action of *S. mutans*, and thus retards cavity formation.

In press meetings, Makinen’s statements were uncharacteristic of most scientific researchers. “It’s miraculous,” Makinen was quoted as saying. He claimed that xylitol “goes beyond” other non-cariogenic sweeteners that supposedly do not promote decay. Xylitol actually prevents it and will even heal incipient cavities.

The “miracle” sweetener generated enormous interest. The segment of American industry that would benefit most was an annual $3.5 billion market of chewing gums, candies, and chocolates.

The National Institute of Dental Health (NIDH) announced that it would launch a three-year study with about a thousand American schoolchildren to confirm the Turku findings. Both American Chicle and Life Savers decided cautiously to hold back and await the outcome of the study, while Wrigley decided to introduce a xylitol-sweetened gum. U.S. law did not allow advertisement of xylitol’s cavity-fighting ability until completion of the study. Wrigley made no dental claims, but merely described xylitol’s sweetening ability and its cooling effect in the mouth.

In November 1977, NIDH’s study was launched. Sticks of xylitol-sweetened chewing gum were
distributed to schoolchildren on Long Island. The gum was 50 percent xylitol, a level five times higher than in the Wrigley product. The children were instructed to chew three sticks daily, for a period of three years.

After day three, with only nine sticks chewed, parents were notified to return the remaining gum supplies. The experiment was halted unexpectedly and abruptly.

This action was triggered by news from the Huntingdon Research Center in Great Britain, where ongoing animal safety studies were conducted for xylitol, funded by Hoffmann-La Roche, the American distributor of imported xylitol. Preliminary results from a two-year mouse study showed that a substantial number of males fed high doses (10 to 20 percent levels) of xylitol developed urinary bladder stones late in life. Of these, some developed inflamed bladders and bladder tumors. In a two-year rat study, with animals fed at similar levels, no bladder stones or tumors developed. But some male rats, fed at the 20 percent level, developed growths on their adrenal glands; in some cases, malignant tumors developed in the adrenal glands.

FDA announced a review of the British findings but took no action to revoke xylitol’s food uses. Although some manufacturers, such as Wrigley and others, voluntarily withdrew xylitol from their American products, other companies continued to promote it.

The reality should have sobered health professionals, had they given any thought to the implications of the study. An anti-cavity substance would be carried mainly in a medium that should be used sparingly, if at all. Instead, with a new image of chewing gum as a prophylactic food, it would appear virtuous to chew gum frequently.

Xylitol’s use as a partial sucrose replacer may not reduce tooth decay. Yet xylitol as a total sucrose replacer in the typical American diet is neither practical nor desirable. Xylitol production is miniscule and costly compared to sucrose. Xylitol’s mouthfeel in processed foods and beverages is not identical to that of sucrose. As with sorbitol and mannitol, xylitol, too, if consumed at high levels, will induce osmotic diarrhea.

Scientists have reported that when more than 10 percent sucrose was replaced with xylitol in rat studies, liver-cell metabolism was damaged. Replacement of 20 percent sucrose with xylitol caused decreased liver glycogen and lipid levels, depleted liver RNA levels, and decreased liver cell growth.

In retrospect, it is shocking to realize that NIDH would launch long-range tests with young children before having carefully and systematically reviewed worldwide literature and research on xylitol. Obviously, if the Huntingdon Research Center had two-year studies in progress, adverse effects were being observed, noted, and discussed. Also, these long-range tests with children were launched with no regard to the diarrheal characteristic of xylitol. This feature of rare sugars was well recognized in medical literature, and it was established that children are especially at risk. In an eagerness to announce a “miracle” sweetener, were the warning signals ignored? To food and beverage processors, xylitol appeared to be one bright hope, being the sole alternative to saccharin, at a time when saccharin appeared doomed. What were the unheeded warning signals, even before the Huntingdon Research Center news?

In 1963, xylitol was given a limited clearance as a sweetener by the FDA. Its use was restricted to special dietary purposes as a sugar substitute for diabetics, in gums, jams, jellies, and chewable vitamin coatings. Unlike sorbitol and mannitol, xylitol was not granted GRAS status. Hence, it is legally classified as a food additive and regulated as such.

The first warning signal concerning xylitol came in the early 1970s. In Great Britain, a water solution
Of 20 percent xylitol became available for patients being fed intravenously who required additional calories. *The Lancet* carried a report of adverse effects suffered by patients infused with xylitol. Of 23, eight developed metabolic acidosis, severe enough so that measures needed to be taken to restore normal blood balance. Seven patients suffered osmotic diuresis, and later four of them developed serious kidney problems that included swellings, diminished urine secretion relative to fluid intake, and calcium oxalate crystal deposits in the organ. Effects ranged from mild nausea, to mental confusion, stupor and complete loss of consciousness. Subsequently, six patients died. At autopsy, unidentified crystal deposits were found in the brains. In Great Britain, medical use of intravenous xylitol infusions was halted. Patients suffered similar adverse effects in New Zealand and Australia, where both organ and intravenous xylitol uses were banned.

After these findings were reported, FDA favored a total ban on xylitol. However, since there had been no indication of harm from low levels of xylitol consumption, FDA favored limiting xylitol’s use to chewing gum. The agency was just about to make this proposal when the Huntingdon results were reported. But Hoffman-La Roche requested that xylitol’s uses not be further restricted, since the company was interested in having xylitol available for use in jams, jellies, candies, and other products. FDA acquiesced and never imposed further limitations.

Although intravenous xylitol infusions had been prohibited elsewhere, the practice continued in the United States. In 1976, an American medical report noted similar adverse effects already reported in the literature, and a few additional ones encountered after xylitol infusions were given to surgery patients. New findings included liver injury and calcium oxalate crystal deposits in the artery walls of the midbrain.

Animal studies provided further confirmation. In one study, xylose was found lethal to rats, even within a few days, when fed at normal dietary levels.

In another study, xylitol was examined for its ability to form oxalate crystals in normal rats and in rats deficient in some B vitamin fractions. Oxalate formation increased significantly in B6-deficient rats infused with xylitol. The study suggested that B6 deficiency may be a factor contributing to oxalate crystal deposits observed in some patients infused with xylitol.

During the Turku studies, some Finnish students who chewed xylitol gum and ate large amounts of xylitol in prepared food suffered from diarrhea. This side effect was noted also in 1977 by a researcher at National Institute for Dental Research (a division of the National Institutes of Health), who reported that, in some persons, even moderate xylitol doses have a cathartic effect. A single diarrheagenic dose is usually from 30 to 40 grams. However, large individual differences exist in xylitol tolerance, and its sustained use may lead to adaptation so that higher doses may be tolerated.

Subsequent to that fiasco, additional adverse findings were reported. In 1979, rats fed xylitol at high levels developed severe diarrhea and gas. This experiment, conducted by Dr. Marleen Wekell, a nutritional science professor at the University of Washington, demonstrated how a substance such as xylitol can adversely alter the bacterial composition of an organism’s intestinal tract. Symptoms of diarrhea and gas were attributed to the prolific growth of *Clostridium perfringens*, a disease-causing bacterium normally found only at very low levels in healthy rats. *C. perfringens* causes similar intestinal distress in humans and is one of the bacterial species implicated in metabolizing harmless substances into carcinogenic ones. Since the formation of some carcinogens may depend on the gut’s bacterial environment, this finding underscored the critical role of diet in health.

At present, xylitol chewing gum is marketed in the United States. In addition, xylitol is being marketed as a crystalline sugar. In one advertisement it was suggested that …
“SUGAR IS GOOD FOR YOU, IF YOU USE THE RIGHT KIND … not all sugars are alike. The sugar that you were warned against is over-refined and adulterated cane sugar. Fortunately, there is an alternative, a natural sugar that provides the delightfully sweet taste of cane sugar without the side-effects. This sugar is xylitol – the delicious fruit sugar that is everything sugar should be – nutritious, energizing and healthful … [brand name] is made from 100 percent pure crystalline xylitol … good for dieters. The reduced insulin secretion works to reduce the appetite. And tests indicate this sweetener may decrease fat accumulation in tissues thereby adding weight loss … It’s the completely natural sugar that’s actually good for you.”

Despite the hype, xylitol is as refined as cane sugar, and has approximately the same number of calories and sweetening power. Although the advertisement mentions “fruit sugar”, most likely the product has been derived from wood sugar. The purported “tests” were neither cited, nor supplied upon request. And the price? $8.50 for a half-pound bottle, which totals $17.00 per pound. The saving grace is that at $17.00 a pound, the buyer may use this sweetener parsimoniously.

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The use of these rare sugars may be safe when they are consumed infrequently, or at low levels, for dietetic use. However, this former limited application has changed radically. Food and beverage processors, constantly searching for sugar alternatives, now seek new applications for these rare sugars, which are being used increasingly in numerous products intended for general consumption. FASEB noted that, by 1970, the total amount of sorbitol used in foods was approximately seven times greater than the amount used in 1960; and the total amount of mannitol, about ninety times greater than in 1960. This escalation continued throughout the 1970s. The trend is undesirable. Consumption of rare sugars needs to be limited and carefully monitored.

[Editor Note: Excerpted from pages 152-159 with book study cites and references 29-40 deleted.]