THE YALE JOURNAL OF BIOLOGY AND MEDICINE 65 (1992), 211–221

Politics and Pellagra: The Epidemic of Pellagra in the U.S. in the Early Twentieth Century

ALFRED JAY BOLLET, M.D.

Clinical Professor of Medicine, Yale University School of Medicine, New Haven, Connecticut; Vice-President for Academic Affairs, Danbury Hospital, Danbury, Connecticut

Received December 11, 1991

The epidemic of pellagra in the first half of this century at its peak produced at least 250,000 cases and caused 7,000 deaths a year for several decades in 15 southern states. It also filled hospital wards in other states, which had a similar incidence but refused to report their cases. Political influences interfered, not only with surveillance of the disease, but also in its study, recognition of its cause, and the institution of preventive measures when they became known. Politicians and the general public felt that it was more acceptable for pellagra to be infectious than for it to be a form of malnutrition, a result of poverty and thus an embarrassing social problem. Retrospectively, a change in the method of milling cornmeal, degermination, which began shortly after 1900, probably accounted for the appearance of the epidemic; such a process was suggested at the time, but the suggestion was ignored.

The story of the epidemic of pellagra in the United States, which occurred in the first half of the twentieth century, may remind people of contemporary events surrounding the AIDS epidemic, when social and political forces have affected medical research. To a very real extent, history has been repeating itself. The epidemic of pellagra, which caused over 3 million cases and 100,000 deaths in the U.S., has been largely forgotten, and the reason for the outbreak is not widely known, even though it is a phenomenon which caused a similar disease outbreak in the Far East (beriberi) beginning a few years earlier, and it could happen again.

THE HISTORY OF PELLAGRA

A brief biography of pellagra may help to put the events in the U.S. in the first half of this century into perspective. Pellagra was well known in Spain and northern Italy, beginning in 1735, with the first description of the disease by Don Pedro Casal, physician to King Philip V of Spain. Casal first observed the disease in the town of Oviedo, in the Asturias region, where it was known as "*mal de la rosa*" because of its characteristic red rash. Although Casal thought it was a peculiar form of leprosy, his descriptions were extremely accurate, and the rash in the exposed areas around the neck is still known as "Casal's necklace" [1].

Casal associated the disease with poverty and noted that the diet of pellagrins consisted largely of cornmeal and little meat. He suggested that the disease was due to spoilage of the corn, and the association of pellagra with corn remained the focus of etiologic speculation well into the twentieth century.

American Indian corn, or maize, when introduced into the Old World, increased

Abbreviation: PPF: pellagra preventive factor

Address reprint requests to: Alfred Jay Bollet, M.D., 22 Plum Beach Pt. Rd., Sands Point, NY 11050

the yield of food calories per acre well beyond that previously provided by rye and wheat. An increase in population per cultivated acre followed, beginning during the second half of the seventeenth century. The use of corn as a staple food crop spread from Spain to southern France, Italy, Romania, Russia, and Egypt; pellagra followed [2]. Cornmeal was the main source of calories for poor people, especially in the spring before new crops were ready; the resulting seasonal occurrence of pellagra led to the name "the springtime disease."

The association of pellagra with corn resulted in many theories to explain the origin of the new disease, often called "Zeism," based on the Latin name for maize, *Zea mays.* The "spoiled corn" theory was analogous to the cause of ergotism, which results from a toxin produced by a mold that contaminates rye. Repeated attempts were made to demonstrate an organism growing in moldy corn which could produce the symptoms of pellagra in men or animals. Despite lack of success, the theory remained popular, and laws were passed prohibiting the sale of spoiled or moldy corn. Thus the association of pellagra with poverty and corn was well established before the disease became common in the United States [2].

AN EPIDEMIC OF PELLAGRA IN THE UNITED STATES

Pellagra was a new disease to American physicians when the first cases began to be reported. A single case in a Georgia farmer in 1902 was reported to the state Medical Association [3] and attributed to spoiled corn, but it attracted little interest. In 1906, Dr. George H. Searcy recognized pellagra at the Mount Vernon annex to the Bryce Hospital for Alabama Insane in Tuscaloosa. He described 88 cases, with a mortality rate of 64 percent, in the Alabama state medical journal in 1907 [4], and in *JAMA* in 1907 [5]. Searcy reviewed hospital records and thought he had found a few cases each year since 1901.

The disease then began to be diagnosed in other mental hospitals. In 1908, Dr. James Babcock, the superintendent of the State Hospital for the Insane in Columbia, South Carolina, realized that patients in that hospital were dying of a new disease, which he had not seen before. Because of the earlier reports of the outbreak of pellagra in mental hospitals, he diagnosed the new disease as pellagra and reported it in the *American Journal of Insanity* [6]. The new disease rapidly increased in frequency, and it began to be recognized in inmates of prisons, orphanages, and in the general population. Records for 1907–11 for eight southern states reveal 15,870 cases, with a fatality rate of 39.1 percent [7]. Physicians aware of the mounting epidemic became concerned.

During the summer of 1908, Babcock took a vacation tour of Europe with the U.S. Senator from South Carolina, Benjamin R. Tillman. Troubled by the cases of pellagra in Columbia, they visited several hospitals for pellagrins in Italy. Tillman was horrified by what he saw and asked the U.S. Vice-Consul in Milan to prepare a report about the disease and government attempts to control it [7].

Back in South Carolina, the State Board of Health became concerned about the spread of the epidemic of pellagra. Influenced by Babcock and Senator Tillman, Governor Martin Ansell called interested people together for a conference on pellagra in Columbia, on December 9, 1908. Seventy-two physicians attended, mostly from within the state.

Both the Governor and Senator Tillman spoke at the meetings, lavishly praising Babcock for identifying the disease and calling attention to the developing epidemic.

That meeting, later known as the first National Conference on Pellagra, led to press reports of the spread of the disease, which aroused great alarm, the beginning of the "pellagra scare."

When a second National Conference on Pellagra was held in Columbia on November 3, 1909, 394 physicians attended, reporting outbreaks of the disease in many parts of the country, especially in orphanages, mental hospitals, and prisons. The National Association for the Study of Pellagra was formed; Babcock was elected first president. The conference recognized Babcock as "the father of the movement for the study and control of pellagra in America" [8]. The Association met again in 1912 and 1915.

WAS PELLAGRA A NEW DISEASE IN THE UNITED STATES?

Pellagra was relatively easy to recognize, since it produced characteristic skin and mucous membrane lesions, plus diarrhea and mental changes. The syndrome was referred to as the "three D's": dermatitis, diarrhea, and dementia; the fourth "D" was death. Nineteenth-century American physicians who had trained or traveled abroad knew the disease, but only a few isolated cases had been diagnosed in this country, and those primarily in mental hospitals. In Osler's first seven editions of his classic text, published between 1892 and 1909, he gave an excellent description of the clinical manifestations of pellagra and described its association with corn, but stated: "It has not been observed in this country."

New disease or not, the incidence of pellagra markedly increased during the first few years after it was recognized and brought to medical and public attention. And the epidemic continued to spread and increase in virulence for several decades. During the first four years after recognizing the disease, Babcock found 435 cases in his hospital. The Georgia State Asylum for the Insane in Milledgeville recorded 40 deaths from pellagra in 1908; by 1909, there were 73, and pellagra moved ahead of tuberculosis as the most frequent cause of death in that and other mental hospitals. In 1915, Milledgeville had 1,433 cases with 220 deaths—35 percent of all deaths in the institution [7]. The epidemic was not limited to the South, however; Peoria State Hospital in Illinois reported 135 cases in 1909, with 45 deaths [7]. Retrospectively, it was estimated that there were 1,000 cases of pellagra in the entire country in 1909, and, between 1907 and 1911, a total of 16,000 cases were reported in 18 states. Reporting was never complete, however, each state deciding whether it would recognize and publicly admit the existence of this embarrassing plague. The reported cases never included pellagrins in mental hospitals and thus were always underestimates of the incidence of the disease.

During 1915, estimates of the total number of cases reached 75,000, and, by the 1920s, there were about 100,000 cases per year. Thus, a disease affecting patients in a few mental hospitals developed into a huge epidemic. In 1924, reports of pellagra came from a total of 36 states and the District of Columbia; the nine southern states included in the reporting area—Florida, Georgia, Kentucky, Louisiana, Mississippi, North and South Carolina, Tennessee, and Virginia—accounting for 90 percent of the cases. Populous southern states known to have a high incidence of pellagra, including Alabama, Arkansas, Texas, and West Virginia, were not included in the reporting area, and the true extent of the epidemic always was greatly underestimated.

During the period from 1915 to 1925, the first decade of reporting, 27,648 deaths

from pellagra were recorded. House-to-house surveys during that period, seeking unhospitalized cases, suggested that there were at least 35 non-fatal cases for each death. On the basis of that estimate, a total of 967,000 cases of pellagra occurred during that decade alone. Another million cases are thought to have occurred between 1925 and 1930, and, in the decade which followed before the disease was virtually eliminated, it is likely that another million cases occurred. Overall, therefore, the epidemic of pellagra, which lasted from about 1906 to 1940, resulted in roughly 3 million cases, with 100,000 deaths in the reporting states. That such a huge epidemic has quietly disappeared, to the extent that most physicians today have never seen a case and are not even aware of its frequency in the early part of this century, is quite remarkable.

THE POLITICS OF THE STUDY OF THE ETIOLOGY OF PELLAGRA

Newspapers, headlining reports of the spreading epidemic, generated a public reaction referred to as "pellagraphobia" [9]. Statewide conferences were held, "Pellagra Commissions" formed, and the first hospital for the care of patients with pellagra was opened in Atlanta in October 1911. The Surgeon General of the Public Health Service, Walter Wyman, said in his annual report in 1911 that pellagra threatened to become a "national calamity." In 1912, there were urgent calls for help to the federal government from Mississippi, Tennessee, and North Carolina; state health officers, at their annual meeting that year, requested a federal commission to study pellagra. Congressman John M. Faison of North Carolina introduced a resolution in the House to appropriate $25,000 for that purpose [7].

Two philanthropists, Col. Robert M. Thompson of New York and J.H. McFadden of Philadelphia, donated $15,000 for the study of pellagra. Many physicians refused to acknowledge the severity of the problem, but the Spartanburg (South Carolina) County Medical Society voted to cooperate, and field studies were set up there. The Thompson-McFadden Pellagra Commission studied cases in the cotton mill districts, conducting a house-to-house survey. The commission concluded that there was no relationship between the disease and diet, but that its spread seemed most rapid when sanitary disposal of waste was poorest, and that the disease occurred almost exclusively in people who lived in or next to the house of another person with pellagra. The association of the disease with poverty, and thus its clustering in the poorer areas of town where housing and sanitary facilities were worst, was not mentioned. Proponents of the infection theory of the etiology of pellagra found support in the reports of the commission and quoted them for decades [10–13].

The rapidity with which the incidence of pellagra reached epidemic proportions prejudiced thinking about its etiology toward an infectious agent. It was self-evident, at that time, that all epidemic disease would be found to have a microbial cause; dramatic advances in medical science had just occurred—causative agents of the major diseases of the time were being discovered at the rate of about a disease a year, including typhoid fever, lobar pneumonia, tuberculosis, cerebrospinal meningitis, syphilis, cholera, malaria, dysentery, scarlet fever, tetanus, and diphtheria; some had clearly defined insect vectors.

Surgeon General Walter Wyman appointed a seven-man commission to study the disease, and assigned Dr. Claude H. Lavinder, a highly regarded epidemiologist, to find the etiologic agent and perhaps identify an insect vector. Lavinder toured

affected areas [14] and established a small laboratory at the South Carolina Hospital for the Insane in May 1909. Following the procedure of Italian investigators, who had reported a "Streptobacillus pellagrae," he injected rabbits, chickens, and guinea pigs with blood, spinal fluid, and spleen pulp from fatal cases of pellagra, without results. In 1911, he set up a larger laboratory at the Marine Hospital in Savannah and attempted to transmit the causative agent to monkeys. In 1912, Lavinder was elected president of the National Association for the Study of Pellagra.

Publicity continued to influence developments. An association of pellagra with poverty was clear, and emphasis on miserable economic conditions in much of the South, where the disease was concentrated, was embarrassing, an affront to delicate Southern pride, still sensitive from the "lost cause" of a half century before. As cases multiplied, embarrassment increased, and attitudes toward former heroes changed.

Babcock was forced to resign in 1914, amid a blaze of publicity and a legislative investigation of the State Hospital for the Insane. He was accused of having brought injury to the reputation and progress of South Carolina by calling attention to the prevalence of pellagra in the state [7].

When the epidemiologic studies of Dr. Joseph Goldberger, a physician who was both a Northerner and an immigrant, pointed to social and economic factors as being responsible for the occurrence of pellagra, because of the effect they had on the diet of the poor, southern sensitivities were further riled. Such conclusions were unacceptable, and a severe setback to the expectation that the "South would rise again," at least economically. Editorial pages and speeches by congressmen criticized and condemned such insulting inferences concerning the contentment of the people of the South. Furthermore, the suggested remedy of improving the diet of impoverished citizens was clearly impractical.

At one point, a letter from Joseph Goldberger to the Surgeon General reached the press, describing the extent of pellagra and its relationship to poverty and poor diet; it stimulated the newly inaugurated president, Warren Harding, to write the Surgeon General asking for a complete report. Harding suggested that the Red Cross provide aid, and offered to ask for a congressional appropriation. The attendant publicity was too much for southern pride. Congressman James F. Byrnes (later U.S. Senator, Secretary of State, and Supreme Court Justice) called the news reports of "famine and plague" in South Carolina an "utter absurdity," calling for rejection of offers of aid from the Red Cross. A Georgia city wired one of their senators, Tom Watson, "When this part of Georgia suffers from famine, the rest of the world will be dead!" [7]. The United Daughters of the Confederacy at first voted to thank President Harding for his concern, but a month later it sent him a letter of protest. "Famine does not exist anywhere in the South," their letter stated, "and we fail to find a general increase in pellagra"[7].

Pellagra was a "social stigma," but its existence was undeniable; thus it was more acceptable for it to be considered infectious than a direct result of poverty. Since pellagra was known to be common in Italy, an influx of Italian immigrants into the South could be blamed for the outbreak of the disease. No one seemed to notice that the Italians living in the southeastern U.S. did not have pellagra, since they did not favor the classic diet that led to the disease in that part of the country—cornbread and molasses.

GOLDBERGER'S STUDIES

When Lavinder asked to be reassigned in 1914, Surgeon General Rupert Blue substituted another officer experienced in the study of epidemic infectious disease, Dr. Joseph G. Goldberger, then aged 40. Goldberger's earlier epidemiological studies had determined the nature of several disease outbreaks, and he had acquired and survived many of them. His experience and acquired immunity made him the ideal investigator of the cause of the latest epidemic of infectious disease, pellagra.

Goldberger had immigrated to the U.S. from Hungary at the age of six, and his parents had managed to send him to Bellevue (New York University) School of Medicine. He graduated before he was 21 and served an 18-month internship at that hospital during 1896 and 1897. After unsuccessful attempts to earn a living in private practice and to join the Navy during the Spanish-American War, he took a competitive examination for the Public Health Service. He scored highest in the country and spent the rest of his career in that service [15].

When Goldberger began his assignment, the epidemic was still growing, and national concern was increasing. Starting with a three-week tour of southern institutions, including a hospital for pellagrins in Spartanburg, South Carolina, the Georgia State Asylum for the Insane in Milledgeville, and orphanages in Jackson, Mississippi, Goldberger confirmed the earlier observations reported by Searcy [4,5] that staff members who were in intimate contact with the inmates never got pellagra, and he immediately dismissed the concept that pellagra was communicable. His certainty that the disease was not infectious was reinforced by the studies of Dr. Edward Francis, who, working with Lavinder in Savannah, had failed to transmit the disease to monkeys [16], and Francis, who had caught every other disease he had worked on, had not caught pellagra.

Struck by the limited, monotonous diet served at those institutions, Goldberger immediately focused on it as a likely cause of the disease. He had difficulty explaining the absence of the disease in staff members, since they usually ate the same meals in the same dining rooms. He noticed, however, that the staff ate first, taking the leanest portions of the meat and whatever variety was available, and, of course, they could also supplement their diet with food obtained outside the institution. At the Georgia State Sanitarium, milk was occasionally served to patients, but attendants, who never got pellagra, drank it twice a day. Inmates were left with cornmeal mush, cane syrup or molasses, gravy, and biscuits—the ubiquitous diet of the poor southerner. This diet was called the "Three M's," meaning meat, meal, and molasses; the meat was mostly very fatty pork, such as "fatback," and the meal was cornmeal.

At orphanages, the pellagra occurred among children who were between six and 12, but rarely affected infants or older children. Such gaps in the distribution of the disease would not occur if the disease were infectious. Goldberger noted that infants were given whatever milk was available, and older children were given work to do and fed a little better. In Jackson, at the Methodist Orphanage, 68 of 211 children (32 percent) had pellagra on July 1, 1914; at the Baptist Orphanage, he found 136 among 226 (60 percent), plus 24 suspected cases [17,18].

Goldberger estimated that it would cost $700 a month to provide adequate food to each orphanage; beginning in September 1914, using federal funds to test his hypothesis that dietary inadequacy was the cause of the disease, Goldberger provided the orphans at the two institutions in Jackson a more varied diet, including fresh meat once a week and abundant milk. He also began a similar study among the

inmates in one building of the asylum at Milledgeville. Cases of pellagra in these institutions cleared, and, in the spring of 1915, when new cases were expected, none occurred, and no relapses were observed [19,20].

Goldberger's report to the Public Health Service revealed his elation with the results and his optimism that the disease could be controlled. But, as soon as the federal funds ran out, the old diet was resumed and pellagra returned; during the spring of 1916, 40 percent of the children in the orphanages had the disease.

In the fall of 1914, still in the first year of his studies, Goldberger was asked to give the Cutter lecture on preventive medicine at Harvard, where he was well received and congratulated. He was also invited to speak at the Southern Medical Association and the Association for the Study of Pellagra in 1915. Proponents of the theory of an infectious etiology dominated those meetings, and Goldberger was attacked and vilified. His conclusion that a more varied diet was needed, one which included other sources of vegetable protein, especially beans, was considered absurd.

To convince the skeptics, Goldberger decided to try inducing pellagra with an experimental diet. Earl Brewer, the Governor of Mississippi, cooperated by offering pardons to inmates of the Rankin Prison Farm who participated in the experiment. There had never been a case at the 3,200-acre prison. Twelve members of a "pellagra squad" were housed separately in clean quarters, to isolate them from possible spread of infectious material from other inmates; they were fed biscuits, gravy, cornbread, grits, rice, syrup, collard greens, and yams, while the rest of the prisoners were given a more varied diet.

The experiment began on February 4, 1915, and was terminated on October 31, when manifestations of pellagra were present in six of the 11 volunteers [21,22]. Several were so miserable that they tried to end the experiment prematurely, one saying, "I have been through a thousand hells!" while others pleaded for a quick death from a bullet. Goldberger was accused of torture, and Governor Brewer was accused of having arranged the whole experiment in order to be able to pardon two of the prisoners who were his friends, convicted of embezzlement. Skeptics remained unconvinced that pellagra was not infectious [7].

Goldberger had not mentioned the Rankin prison farm experiment during the Columbia meeting in 1915, since it was being conducted secretly, but two weeks later the information appeared in the newspapers. Physicians who had attended the meeting and supported Goldberger were now loudly critical.

In a further attempt to demonstrate that the disease could not be transmitted in the fashion of known infections, Goldberger set up "filth parties." He had himself, his wife, and his associates injected with blood from pellagrins, and then he and his male colleagues wrapped skin scales, feces, dried urine, and other dirt from pellagrins in dough and swallowed repeated doses. None of them got pellagra, or anything else except nausea and mild diarrhea [23] (*mirabile dictu*), but these studies were also dismissed, since pellagra was more common in women, and thus his staff must have been "constitutionally resistant" to the disease.

Goldberger eventually stopped trying to convince the stubborn believers in an infectious etiology for the disease and devoted the remainder of his life to an attempt to identify the factor which prevented or cured pellagra. Various names were suggested for the factor, including vitamin G for Goldberger, but he called it "PPF," pellagra preventive factor. His dietary approaches cured thousands of patients before PPF was eventually identified as nicotinic acid.

An animal model of pellagra was sought, since the human studies were laborious and dangerous. A dog model came to Goldberger's attention almost accidentally. In 1916, Russell H. Chittenden and Frank P. Underhill, studying malnutrition at Yale, fed dogs boiled peas, cracker meal, and cottonseed oil [24]. The dogs developed sores and hemorrhages in the mouth; the dried, crusted blood led to the name "black tongue."

In the South, a disease in dogs, "canine plague," was thought to be related to human pellagra, since they occurred in the same localities, reinforcing thinking that pellagra was infectious. Goldberger did not study the disease in dogs until he learned that black tongue had developed in hunting dogs of an employee at the research facility at the Georgia State Asylum in Milledgeville, when the dogs were fed half their usual ration, supplemented with cornbread, supposedly to improve their hunting avidity. Autopsy revealed black tongue [25]. The sore mouth made the dogs unwilling to swallow (as occurred in human cases), and yeast was generally used to stimulate appetites. The Public Health Service investigators soon realized that yeast contained the "pellagra preventive factor" and then found it to be the most potent source of this factor [26].

Yeast then began to be used in the treatment of human pellagra, where it was particularly valuable for patients whose painful mouth lesions prevented them from swallowing, and it saved many lives before parenteral treatment became available. Yeast also saved many lives when Goldberger arranged to have the Red Cross ship large quantities of it into flood-stricken areas along the Mississippi River in 1927 [27]. The expected outbreak of pellagra did not occur; instead, the disease fell below its usual seasonal incidence. After the emergency, yeast was no longer provided, and the usual level of pellagra returned to the area.

Liver was known to be a rich source of pellagra preventive factor and, later, when George Minot's liver extract began to be used to treat pernicious anemia, Goldberger arranged through Minot to obtain some from the Eli Lilly Company, which was beginning commercial production. The extract was effective in black tongue in dogs [28] and was valuable in treating humans who could not swallow. (This study was the last organized by Goldberger before his death from renal cancer on January 17, 1929.)

The final, virtually complete elimination of pellagra dates to the 1940s. Elvehjem and Koehn showed that nicotinic acid was the specific micronutrient deficiency [29], and Sydenstricker and others showed that most patients with pellagra actually had deficiencies of several vitamins, particularly of the B group, including riboflavin [30]. The availability of cheap, synthetic forms of the various vitamins led to legislation in many states requiring their addition to commonly used foods [31]. Several nutritional deficiency syndromes were virtually eliminated, almost simultaneously.

WHY DID THE EPIDEMIC OF PELLAGRA OCCUR?

The reason for the appearance, or at least the marked increase in frequency, of pellagra in the U.S. in the early years of the twentieth century has not been thoroughly studied. A phenomenon which caused epidemics of another nutritional deficiency disease, the outbreak of beriberi in the Far East, which had appeared about two decades earlier, seems to have been the cause.

The "Three M" diet of meat, meal, and molasses had long been a favorite, especially among the poor, in the South. This diet led to pellagra, and it still does if

sufficient amounts of other foods are not combined with it. The long-standing popularity of this ubiquitous diet was one of the reasons Goldberger had difficulty convincing southerners that pellagra was due to diet and was not infectious. Maize had been a dietary staple of the American Indians, and no change had occurred in the genetics of the corn used, or in the method of planting and harvesting, to account for the appearance of an epidemic of pellagra in the U.S. early in the twentieth century. The Indians ground their corn into meal using lime water, and alkali has been demonstrated to increase the extraction of the nicotinic acid which is present in corn, increasing its nutritional value. This practice may account for the absence of any known pellagra among the Indians, but it is more likely that a varied diet was the most important factor.

Casimir Funk, who helped elucidate the role of thiamin in the etiology of beriberi, was an early investigator of the problem of pellagra. He thought that micronutrients in addition to the "anti-neuritic factor" would be found to be necessary for life. Beriberi had developed as an epidemic in the Far East in the 1880s after a change occurred in the method of milling rice. Funk suggested that the same mechanism was responsible for the outbreak of pellagra [32]—but no attention was paid to his article on this subject.

Changes in the methods of milling corn might have resulted in the outbreak of pellagra, since the germ or embryo in the corn kernel contains a high proportion of the lipid, enzymes, and co-factors, including nicotinic acid. Degerminating corn during milling removes the embryo with the lipid and enzymes, and the resulting cornmeal is more stable and can be stored and shipped without enzymatic decay; this type of change is exactly the same phenomenon which occurs when rice is finely milled.

A question relevant to the epidemic of pellagra, therefore, is, when did degermination of corn begin? The epidemic began shortly after the turn of the century. Degermination of corn began after the Beall degerminator was patented in 1900 and 1901. That information provides an explanation of some puzzling observations of the epidemiology of pellagra. The disease was most prevalent in institutions in which people existed on a restricted, monotonous diet for long periods. In the South, cornmeal was a main source of calories in these institutional diets, but, despite cornmeal's restricted nutritional value, pellagra was rare or non-existent until after 1900. After degermination began, the food used for the same diet was less nutritionally adequate [33].

Epidemiologic investigations, including those conducted by Goldberger, pointed to an especially high incidence of pellagra in the cotton-growing areas near the textile mill towns, along the railroad lines in the rural South, and an inverse relation to income in those areas [34]. In retrospect, we can see that, in the textile mill towns, surrounded by cotton fields, food was shipped in by railroad, and the cornmeal that could be purchased in the company stores was processed in the Midwest, where it had been degerminated. More rural areas had less pellagra; being away from the railroads, they ground their own corn, mostly in old, water-driven stone mills. The tradition still exists in the South that "stone-ground" corn is healthier. When the new method of degerminating the corn was introduced, the precarious diet of the poor in mill towns and institutions became even worse, and pellagra appeared.

If changes in food technology caused outbreaks of deficiency disease, could the same process happen again? Beriberi and pellagra may not be the only possible

examples of changes in food technology which might be introduced because of commercial advantages, but which can result in outbreaks of disease not recognized for long periods as nutritional in origin.

REFERENCES

1. Casal G: The natural and medical history of the principality of the Asturias. In Classic Descriptions of Disease. Edited by RH Major. Springfield, IL, CC Thomas Publishers, 1932
2. Roe DA: A Plague of Corn. The Social History of Pellagra. Ithaca, NY, Cornell University Press, 1973, 217 pp
3. Harris HF: A case of ankylostomiasis presenting the symptoms of pellagra. Trans Med Assoc of Georgia :220–227, 1902
4. Searcy GH: An epidemic of acute pellagra. Trans Med Assoc State of Alabama :387–392, 1907
5. Searcy GH: An epidemic of acute pellagra. JAMA 49:37, 1907
6. Babcock JW: How long has pellagra existed in South Carolina? Am J of Insanity 69:185–200, 1912
7. Etheridge EW: The Butterfly Caste. A Social History of Pellagra in the South. Westport, CT, Greenwood Press, 1972, 278 pp
8. Hall WS: Psychiatrist, humanitarian and scholar: James Woods Babcock, M.D. J South Carolina Medical Assoc :366, 1970
9. Niles GM: Pellagraphobia: A word of caution. JAMA 58:1341, 1912
10. Siler JF, Garrison PE: Pellagra. First Progress Report of the Thompson-McFadden Pellagra Commission. New York, New York Post-Graduate Medical School, 1913
11. Siler JF, Garrison PE, MacNeal WJ: Second Progress Report of the Thompson-McFadden Pellagra Commission. Arch Int Med 14:289–293, 1914
12. Siler JF, Garrison PE, MacNeal WJ: A statistical study of the relation of pellagra to the use of certain foods and to the location of domicile in six selected industrial communities. Arch Int Med 14:294–373, 1914
13. Siler JF, Garrison PE, MacNeal WJ: The relation of methods of disposal of sewage to the spread of pellagra. Arch Int Med 14:453–466, 1914
14. Lavinder CH: The prevalence and geographic distribution of pellagra in the United States. Public Health Rep 27:2076–2081, 1912
15. Terris M (ed): Introduction. In Goldberger on Pellagra. Baton Rouge, LA, Louisiana State University Press, 1964, pp 3–16
16. Lavinder CH, Francis E: Attempts to transmit pellagra to monkeys. JAMA 63:1093–1094, 1914
17. Goldberger JG: The etiology of pellagra. The significance of certain epidemiological observations with respect thereto. Public Health Rep 29:1683–1686, 1914
18. Goldberger JG: The cause and prevention of pellagra. Public Health Rep 29:2354–2357, 1914
19. Goldberger JG, Waring CH, Willets DG: The prevention of pellagra. A test of diet among institutionalized inmates. Public Health Rep 30:3117–3131, 1915
20. Goldberger JG, Waring CH, Tanner WF: Pellagra prevention by diet among institutionalized inmates. Public Health Rep 38:2361–2368, 1923
21. Goldberger JG, Wheeler GA: The experimental production of pellagra in human subjects by means of diet. Hygienic Laboratory Bulletin 120:7–116, 1920
22. Goldberger JG, Wheeler GA: Experimental pellagra in the human subject brought about by a restricted diet. Public Health Rep 30:3336–3339, 1915
23. Goldberger JG: The transmissibility of pellagra. Experimental attempts at transmission to the human subject. Public Health Rep 31:3159–3173, 1916
24. Chittenden RH, Underhill FP: The production in dogs of a pathological condition which closely resembles human pellagra. Am J Physiol 44:13–66, 1917
25. Wheeler GA, Goldberger JG, Blackstock MR: On the probable identity of the Chittenden-Underhill pellagra-like syndrome in dogs and "black tongue." Public Health Rep 37:1063–1069, 1922
26. Goldberger JG, Wheeler GA, Lillie RD, Rogers LM: A further study of butter, fresh beef and yeast as pellagra preventives, with consideration of factor P-P of pellagra (and black tongue of dogs) to vitamin B1. Public Health Rep 41:297–318, 1926
27. Goldberger JG, Sydenstricker E: Pellagra in the Mississippi flood area. Public Health Rep 42:2706–2725, 1927
28. Goldberger JG, Sebrell WH: The blacktongue preventive value of Minot's liver extract. Public Health Rep 45:3064–3070, 1930

29. Elvehjem CA, Koehn CJ Jr: Studies on vitamin B_2 (G): The non-identidy of vitamin B_2 and the flavins. J Biol Chem 108:709–728, 1935
30. Sydenstricker VP: The history of pellagra, its recognition as a disorder of nutrition and its conquest. Am J Clinical Nutrition 6:409, 1958
31. Wilder RM: A brief history of the enrichment of bread and flour. JAMA 162:1539–1540, 1956
32. Funk C: Studies on pellagra. The influence of the milling of maize on the chemical composition and nutritive value of the meal. J Physiol 47:389–392, 1913
33. Carpenter KJ: Effects of different methods of processing maize on its pellagragenic activity. Fed Proc 40:1531, 1981
34. Goldberger JG, Wheeler GA, Sydenstricker E: A study of the relation of family income and other economic factors to pellagra incidence in seven cotton-mill villages of South Carolina in 1916. Public Health Rep 35:2673–2714, 1920