The "Slows"
The Torment of Milk Sickness on the Midwest Frontier

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In the early nineteenth century, as white settlers flooded onto the midwestern frontier, a new and highly fatal disease appeared among them. The sickness was unknown along the eastern coast and elsewhere in the world. Settlers were forced to guess at its cause and attempt to treat it without benefit of prior experience or modern clinical research. An understanding of the disease's pathology and the discovery of a successful treatment had to wait until the advent of twentieth-century biochemistry. By that time, milk sickness had almost disappeared, and today it is nearly forgotten.

The "interior valley of North America," as Daniel Drake described it, was settled by people who moved across the mountains to an arduous and precarious life. Pioneers labored to eke out an existence, and they came into frequent contact with violence, malnutrition, and disease.

Chief among the diseases of the area and time were malaria, dysentery, cholera, typhoid fever, streptococcal infections, smallpox,
and pneumonia. These were common in the East, and pioneers possessed a level of popular understanding about the symptoms and the available treatments for each disease. Unfortunately, in the early 1800s, the popular understanding of medicine was often more medieval than modern: treatments advocated by the Greek physician Galen were still alive in Drake’s “interior valley.” Bleeding, purging, and an ancient pharmacopoeia were the rule. Both physicians and folk doctors aimed at balancing the body’s humors and knew neither the origins of their treatments nor the bases for their presumed efficacy. Diagnosis was based upon slender findings, and diseases were classified, not by etiology or pathology, but by prominent symptoms, such as fevers, fluxes, and the locations of pain. Too often efficacy was judged by improvement in individual patients rather than by the collected results of systematic trials, and the placebo effect was not recognized. Most of the sick never saw a physician.

In such a medical climate appeared the mysterious disease which came to be called milk sickness. Ordinary settlers and their doctors found it unpredictable, untreatable, and highly fatal. Milk sickness killed many, frightened more, and caused local economic crises. Villages and farms were abandoned; livestock died; entire families were killed. Migration to areas thought to be safer became common. And then the disease almost disappeared without any special preventive actions, at least without any targeted at its eradication. Its disappearance would prove to be a consequence of the progress of midwestern civilization and advances in agriculture.

THE FIRST REPORTS OF MILK SICKNESS

In 1810, Daniel Drake wrote an extensive essay on Cincinnati and its environment. In an appendix, he included an 1809 report by a Dr. Barbee of Virginia, who had visited southwestern Ohio. Barbee described a potentially new disease, with a clinical constellation of weakness, muscular pain, vomiting, severe constipation, disagreeable breath, lassitude, coma, and death. He observed a similar disorder in cows, horses, sheep, and dogs. Pioneer farmers called the sickness “the trembles,” because their animals became feeble and trembled when they exerted themselves. The condition generally occurred in oak forests and valleys. Drake reproduced Barbee’s entire report “so that physicians may determine how far it deserves the appellation of a new disease.” Drake and Barbee were the first to describe milk sickness, although neither knew of
its association with milk. The disease was first called “sick stomach,” or the “puking illness,” and later, the “slows.”

In the same year, according to a later published history, milk sickness was common in Champaign County, Ohio. Its cause was unknown, but certain localities were known to be loci of the disease. County resident Henry Basome moved three times to escape the disease but succumbed to it before he could make a fourth move. 

In 1811, the Cincinnati Liberty Hall carried a front-page article on milk sickness. It reported observations by Alexander Telford and Arthur Stewart, both of Miami County, that clearly tied “sick stomach” to milk from cows not confined to cultivated pastures. They also noted that horses allowed to graze in the woods died in numbers, and they proposed that the stomachs of animals dead from the “trembles” be examined to see what plant might have been responsible. Had their advice been followed, an understanding of the disease might have come much earlier.

In 1812, the Liberty Hall article was republished verbatim in the Medical Repository of New York, but despite the spread of this report, many early descriptions of mortality indicate that pioneer farmers and woodsmen living in their isolated cabins from the Appalachians to the Mississippi River, from lower Michigan to northern Alabama, had not yet learned the possible cause of the disease. The information published in the Cincinnati newspaper seems not to have been widely known. Physicians were rare throughout the frontier and few of them were well-educated—most had preceptorial training or had attended only a brief medical course. Medical journals were even rarer. The Medical Repository was the first American medical journal, begun in New York in 1797. In its second year its total circulation was only about 300, and it must not have had an extensive circulation in the West.

MILK SICKNESS ON THE MIDWEST FRONTIER

On their own, the pioneers learned that the sick stomach was different from the usual fatal diseases characterized by fluxes, fevers, or

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1Daniel Drake, Notes Concerning Cincinnati (Quarterly Publication of the Historical and Philosophical Society of Ohio, vol. 3; Cincinnati, 1908), Part 1, appendix 1.


3Cincinnati Liberty Hall, January 16, 1811.

4Medical Repository (New York), 1812, 15:92-94.
rashes. They learned to look to the symptoms of their farm animals first. The sickness occurred where grazing animals or their suckling offspring developed the “trembles,” characterized by weakness, shaking, and finally death.

One of the pioneer families who learned about milk sickness through direct experience was the Thomas Lincoln family of southern Indiana. In the summer of 1816, Thomas and Nancy Hanks Lincoln, with their two children, a cousin, and later Mrs. Lincoln’s uncle and aunt, settled in the woods near Pigeon Creek, in what is now Spencer County. At first, they subsisted on what they could hunt, gather, and fish. Their first dwelling was a lean-to enclosed on three sides with skins to cover the open south side. A log cabin was built some months later. In the autumn of 1818, a cluster of neighbors died—all of the same disease. Among the dead were Nancy’s aunt and uncle, and then Nancy Lincoln herself. She left behind a husband and children, including Abraham, not quite ten years old.

There are no contemporaneous accounts of Nancy Lincoln’s illness, but in the 1870s, her cousin, Dennis Hanks, who had lived in the Lincoln cabin for many years, confirmed that she died of milk sickness. His diagnosis, of course, was based only on pioneer medical lore and his memory of the disease. He also recalled that the family left Indiana for Illinois to escape the endemic illness. It is quite likely that in 1818, the Lincoln family did not know what had killed Abraham’s mother, as fatal mini-epidemics often occurred without clearly established cause.6

Long after the Lincolns departed for Illinois, milk sickness persisted in Indiana. In the southern region of the state, the disease remained endemic along the road from Louisville to Vincennes, especially in the area of French Lick, where the chronic form was called the “slows.” In 1832 in Fountain County, Mary Hovey wrote to her sister in New York that the disease prevailed in that part of the state, but never in the prairies or groves.7 In 1838, sixty people died of the illness in Hendricks


7In the twentieth century, Indiana established a memorial at the site of Lincoln’s boyhood. It was made part of the National Park Service in 1962.

8Mary Hover to Sister, March 13, 1832, Hovey Collection, Folder III, 175 (Indiana State Library, Indianapolis).
County; the next year, fifty died in Danville. From 1830 to 1840 Dr. W. Bunnel of Milton reported treating as many as five to six cases of milk sickness at any one time. In 1841, Benjamin Russell of Vermillion County wrote to an Indianapolis acquaintance that the disease was well developed on both sides of the Wabash River from its mouth upwards for one hundred miles. Russell wrote that he planned to move elsewhere to protect his family. In DuBois County, more than half of all of the recorded deaths in the early nineteenth century were due to milk sickness. Other affected states had similar experiences.8

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8Edwin Lincoln Moseley, *Milk Sickness Caused by White Snakeroot* (Bowling Green, Ohio, 1841); G. W. H. Kemper, *A Medical History of the State of Indiana* (Chicago, 1911), 133; E Russell Benjamin to William H. English, July 5, 1841, English Collection, Box 1, Folder 3 (Indiana Historical Society, Indianapolis).
Over the first half of the nineteenth century, numerous publications described the disease, and the “sick stomach” evolved into milk sickness. There were still more questions than answers: what was its cause? How could it be prevented? How could it be treated? In all, a period of 150 years would pass while research scientists and physicians discovered the answers to every question.

Milk sickness is a vegetable poisoning caused by tremetol, an alcohol found in the white snakeroot plant. Grazing animals, allowed to feed in the woods, ate the plant. Humans then acquired the disease by drinking the milk or eating the meat of affected animals. Tremetol poisoning produces a biochemical defect, the suppression of citric synthetase, an intracellular enzyme that introduces certain acid food fragments into one of the vital energy producing cycles. Patients fatally affected with tremetol poisoning accumulate the acid substances normally metabolized for energy. The ensuing excess acid produces an acidosis much like diabetic acidosis, formerly a common, highly fatal complication of severe diabetes mellitus.

Milk sickness had a spotty distribution; occasionally the afflicted areas were quite small (40 to 50 acres). Forested areas, especially hill slopes and valleys, were especially dangerous. The sickness occurred most often in spring and summer. Dry summers, with thinning and browning pasture grasses, were particularly troublesome. The primary animals affected were grazers: cattle, sheep, horses, and deer. When pastures browned, unfenced herbivores ate what they could find. When these animals became affected, the carnivores who fed on their carcasses could also contract the disease. Humans could then be sickened by ingesting tainted milk, butter, cheese, or meat.

Three to ten days after ingesting infected animal materials, humans began to show symptoms: vomiting, abdominal pain, severe constipation, and fatigue. Over the next few days the patient would fall into a stupor, and perhaps from there into a coma and death. One of the most characteristic symptoms of the sickness was an offensive odor of the patient’s breath, often so strong that it could be detected on entering a frontier cabin (doctors would discover much later that the odor was produced by acetone.) Treatments were varied and widely proclaimed, but none was specific and none effective. Purging the bowels with calomel, sedation with opium and/or alcohol, and bleeding were common. Those who did not die quickly usually recovered, albeit slowly. Some had protracted weakness and/or relapse precipitated by exercise or unrelated, otherwise mild diseases. Others were disabled with fatigue and
weakness for weeks or even months. This extended form of the disease was called "the slows."

FINDING THE CAUSE AND THE TREATMENT

After Telford and Stewart reported their findings in an 1811 Cincinnati newspaper, there were other early, partially successful attempts to understand and contain milk sickness. Settlers in Franklin County, Tennessee, observed that when they confined their cattle they also reduced the incidence of milk sickness. One group fenced off an entire slope of a local extension of the Cumberland Mountains, and in 1821, the Tennessee legislature responded to their success by obligating local authorities to maintain the fences. In 1827 Kentucky also took some action at the state level, offering a reward to anyone who could prove a cause for the disease. By 1840, however, Drake was able to note that the reward had not been claimed.

Sometime after 1828 an Illinois pioneer physician named Anna Pierce Hobbs recorded that she was told by an Indian woman that a creeping plant known as white snakeroot, *Eupatorium ageratoides*, was poisonous. The doctor educated her neighbors about the problem, even growing a garden of snakeroot so that people could identify the plant. Hobbs and most others apparently did not realize, however, that she had found the cause of milk sickness.

In 1840 W. J. Barbee, "late of Marshall, Illinois," published an outstanding description of the setting and mortality of the disease. He reported that Dr. David Dale Owen, the Indiana state geologist and a medical school graduate, had been given by a Vigo County farmer a plant that was said to be the cause of milk sickness. Owen identified the plant at *Eupatorium ageratoides* and gave a decoction of the plant to a calf that died of the trembles shortly thereafter. Despite the publication of Barbee's description, it was not until later that the connection between the plant and milk sickness was fully realized.

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of these facts, other medical experts were conducting their own research and arriving at other theories.\textsuperscript{12}

Up to the mid-nineteenth century, there were three principal thoughts as to the cause of milk sickness: miasma, metallic poison, or vegetable poison. The seasonal nature of the disease argued against a metallic cause. Later, bacterial infection was strongly considered, and some scientists even cultured bacteria which they named \textit{Bacillus lactomorbus}. But by 1840, the vegetable origin for the disease was fairly well accepted. Several plants were suspect: poison oak, white snakeroot, wild parsnip, and others. In 1841, Drake argued for poison oak as the cause.\textsuperscript{13}

More than one early scientist conducted tests by feeding animals: white snakeroot appeared to produce the disease in sheep, but poison oak could also produce the same symptoms. George Graff, of Edgar County, Illinois, also writing in 1841, expressed the frustration of many physicians and scientists. He argued that the disease had been known since the early eighteenth century, citing the writings of a French missionary who described it in cattle, but that

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\textit{[O]ccurring as the milk sickness universally does in our frontier settlements, where medicine as a science is in its infancy, and its practice too much in the hands of the ignorant, we have imperfect accounts of the affection, either as regards its history, symptoms, or methods of cure.}\textsuperscript{14}
\end{quote}

Curiously, given the number of individual physicians writing about the disease, milk sickness almost escaped attention in most nineteenth-century American medical textbooks. One early text from 1845 did not mention it; another from 1848 cited Drake's comments.\textsuperscript{15} For the rest of the century, almost all medical texts (written, not coincidentally, east of the Alleghenies where milk sickness was unknown) ignored the

\textsuperscript{12}W. J. Barbee, "Facts Relating to the Endemic Disease Called by the People of the West, Milk-sickness," \textit{Western Journal of Medicine and Surgery} (1840), 2:178-90.


\textsuperscript{14}G. B. Graff, "On the Milk Sickness of the West," \textit{American Journal of the Medical Society} 1 (1841), 351-69.

disease. An important exception was George B. Wood’s textbook issued in Philadelphia in several editions beginning in the 1840s. In his fifth edition, published in 1858, Wood stated that he had never seen a case of milk sickness, but he included seven pages of discussion on the disease and quoted important authors on the subject. In 1892, William Osler included the disease in his textbook, although he probably never saw a patient who had it. Osler’s influence was so great that milk sickness was included in textbooks for the next seventy-five years. In retrospect, it seems strange that early texts omitted milk sickness at a time when the disease was common. After Osler, the disease commonly appeared in American medical texts but had become rare in the real world.

In 1852, the Indiana State Medical Society formed a committee to develop survey data, based on county-by-county physician surveys, about milk sickness. The next year, Sutton reported that the committee had begun its study, but it seems not to have issued a report. An 1858 report of the Ohio State Board of Agriculture gave a detailed description of white snakeroot’s role in causing the trembles and milk sickness. The report included an illustration and description of the plant (now referred to as Eupatorium urticaefolium). The report also contained a letter from W. J. Vermilya of Ashland County, who outlined feeding experiments which demonstrated the poisonous nature of white snakeroot. One year later, Richard Owen, professor of geology at Indiana University (and later first president of Purdue University), traveled through southern Indiana to conduct his own survey. He reported that “milk sickness prevails in Brookville, near New Castle and Greencastle.”

Owen’s survey confirmed that, despite increasing knowledge of the cause of milk sickness, many rural families were still being affected by

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17William Osler, The Principles and Practice of Medicine (1st ed., New York, 1892), 266.
19“Milk Sickness,” Annual Report of the Ohio State Board of Agriculture (1858), 670-75. Vermilya also cited an earlier 1839 trial in which John Rowe of Fayette County was able to kill cattle by feeding them Eupatorium ageratoides.
20R. Owen, Survey of Indiana, Second Notebook, Fall 1859, Owens Collection, S1036 (Indiana State Library).
the disease. In 1859 three children of the Charles Crist family in Randolph County died of milk sickness. In 1862, the disease was apparently still endemic along portions of the Wabash River. In a letter to her grandparents, Mary Bell Wilson of Lockport, in Carroll County, wrote that “a disease called milk sickness carried off a good many people last fall. Three buried from one family in a single week.” In 1869, one case of direct snakeroot poisoning of a human was reported. A farmer who gathered nettles for a pot of “greens” included white snakeroot among them. He became ill with milk sickness within a few hours. His wife, who did not like greens and refused to eat them, remained well.

THE DECLINE OF MILK SICKNESS

During the period from 1850 to 1900, milk sickness became less common. The degree to which this change was driven by the spread of information is impossible to determine. The trend, however, paralleled the organization of midwestern farmlands into defined fields and fenced pastures, as well as the emerging industrialization of agriculture. The illness also disappeared much more quickly in the towns and cities where milk was coming from larger, “tamer” farm pastures. Perhaps, as well, dairy practices that pooled milk from many individual farms tended to dilute the poison.

By 1900, milk sickness was uncommon, although reports of individual cases continued to appear in medical journals. In 1917 C. Dwight Marsh reported the results of his extensive toxicity study, confirming the toxicity of white snakeroot. Ten years later, J. F. Couch identified the toxic component of white snakeroot as an alcohol that he named tremetol, to reflect its role in causing trembles. Doctors had also become more confident about treating the disease in humans, based on their knowledge that milk sickness produced ketoacidosis, a severe metabolic acidosis much like the acidosis of severe, complicated diabetes mellitus. W. E. Walsh of Morris County, Illinois, reported in 1909 that he had

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38 INDIANA MAGAZINE OF HISTORY

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21 E. Tucker, History of Randolph County, Indiana (Chicago, 1882), 370; Mary Bell Wilson letter, January 15, 1862, Pierce-Kruell Collection, L180 (Indiana State Library); William Jerrey, A Treatise on the Cause and Cure of the Milk Sickness (St. Louis, 1869).

treated more than thirty-eight cases of milk sickness during his practice. Most of the infected dairy products came from two small, local farms—one forty acres, one less. His successful treatments had come to include clysters of sodium chloride and sodium “carbonate.” In 1914 Dr. Arthur Clay reported that he had “had it personally” and related that sodium carbonate administered rectally every 2 hours was an effective treatment. Walsh wrote another report on milk sickness in 1930, confirming that cases still occurred in his home of Morris County, and noting that acetone and diacetic acid were now regularly found in the urine of milk sickness patients and reporting the use of sodium bicarbonate in large doses as his preferred treatment. Both Clay’s and Walsh’s treatments were ideal for the correction of acidosis before intravenous therapy was possible or common.

In 1963, the last reported humans with milk sickness were treated in St. Louis, Missouri. Both of the patients were infants. Terribly ill at admission, they were rapidly found to have a severe metabolic acidosis whose cause was unknown. They responded slowly to large quantities of intravenous bicarbonate. The rapidity of the diagnosis of the metabolic derangement and its successful treatment would have bewildered the pioneers and their physicians. Although those who treated the children did not immediately know the cause of the metabolic derangement, they successfully treated the condition based upon the abnormal laboratory tests. When an older physician recalled a patient with milk sickness from his earlier years of practice, careful inquiry disclosed that the children had been drinking specially obtained milk, provided by a farmer who allowed his cows to graze in a nearby woods where white snakeroot grew in abundance. This set of concurrences led to a convincing diagnosis.

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24 A. F. Hartman, Sr., et al., “Tremetol Poisoning Not Yet Extinct,” Journal of the American Medical Association 185 (1963), 706-709. Other cases have probably occurred but have either gone unreported or were not recognized.

25 Each child had become quite ill late in the season, after the first hard frost, an unusual time for milk sickness. Each illness had started with an intercurrent infection, one respiratory and one diarrheal. Reawakening of milk sickness was common and probably accounted for most “off season” episodes.
Biochemists had been working since the 1950s to understand the chemical basis of tremetol poisoning. In 1963, synthesis of a tremetol ketone was reported. This tremetone lacked toxicity though the related crude tremetol remained toxic. Finally in 1973, using chickens, C. H. Wu demonstrated that tremetol poisoning caused selective suppression of the activity of the enzyme citric synthetase, and consequent inability to process food fragments presented for energy production. The consequence was chemical starvation and accumulation of acid materials producing overall body acid excess.26

In 1983, four horses in Greene County, Ohio, died of tremetol poisoning, known to cause the trembles in cattle and horses. Researchers at the Ohio State University College of Veterinary Medicine studied the animals and found that their pasture had large amounts of heavily grazed white snakeroot.27

At the beginning of the twenty-first century, milk sickness seems to be long gone. Yet the white snakeroot plant is still growing in the Midwest, especially at the edges of wooded areas.28 The 1960s St. Louis case illustrates the hazard of ignoring the risks to be found in "natural" milk.

The early settlers of the midwestern woods learned, through long and torturous experience, both the source of milk sickness and the method of its prevention, long before physicians and researchers discovered the specific poison. But new medical information spread slowly in nineteenth-century cities, towns, and farms. Milk sickness shaped the lives of many generations of Hoosiers and other midwesterners before it began to disappear late in the nineteenth century. The "slows" are just one example of the challenges of pioneer life before organized research, biochemistry, and molecular biology were available to solve medical mysteries.

26C. H. Wu, “Metabolic Changes Induced in Chickens by the Administration of Tremetol,” Biochemical Pharmacology 22 (1973), 2835-41.
27C. T. Olson, et al., “Suspected Tremetol Poisoning in Horses,” Journal of the American Veterinary Medical Association 185 (November 1984), 1001-03. In the southwestern United States, the rayless goldenrod, Apolopappus heterophyllus, also containing tremetol, has been incriminated in trembles fatalities in horses and cattle.
28White snakeroot still grows freely throughout the southern Indiana woods, even within twenty feet of the putative grave of Nancy Lincoln. A beautiful stand of white snakeroot is visible within one hundred feet of the porch at the Inn at McCormick's Creek State Park, Spencer, Indiana.