**Leprosy**

*Hereditary or contagious? How the quest for answers led to Minnesota*

*BY RICHARD M. CAPLAN, MD*

It was 1873. Armauer Hansen's colleagues at the leprosy hospital in Bergen, Norway, thought well of him. Even his predecessor as hospital director—who was also his father-in-law and a leader in the academic world of leprosy—liked him well enough to excuse his fixed idea that leprosy was not a hereditary disease but rather an infection caused by a specific type of bacteria. If that was true, then leprosy might be a contagious disease. The prevailing opinion was that it was a hereditary affliction—passed down in families like baldness or (as "everyone knows") tuberculosis or, perhaps, a moral or sexual taint resulting from divine punishment.

But G. H. Armauer Hansen, MD, born in 1841, could not be dissuaded by the opposition. He had taken biopsies repeatedly from his leprosy patients who had multiple cutaneous nodules; with careful study, using his fairly primitive microscope and with hardly any success at staining, he always found great numbers of macrophages engorged with tiny particles that he felt certain, by their size and shape, must be bacteria. After diligent study of so many patients, most of them poor fisherfolk from the western coast of Norway, he was ready by 1873 to publish a report of his findings and conclusions—considered "outrageous" by a medical establishment that was convinced otherwise. By that date, scientists had observed many kinds of bacteria, and some experts had strong suspicions that particular identified organisms might be the specific cause of a corresponding disease; but no one yet had convincing arguments for this, certainly nothing that could be considered "proof" worth endangering one's reputation.

Hansen's article about the intracellular particles he had seen and studied since 1871 became the first report in medical history to claim for any disease that it was caused by the specific microscopic particles that he saw and that they were bacteria; thus, leprosy was an infection that might be transmitted from one person to another. The World Health Organization in 1990 urged that this infection be renamed Hansen's Disease, not only to honor Hansen, but also to purge the world's vocabulary of the hugely stigmatizing words "leprosy" and "leper." That change in vocabulary and the progress in wiping out the illness have been slow.

**A tenacious struggle**

Hansen battled with such tenacity that he has reminded some observers of Herman Melville's character, Captain Ahab, and his obsessive struggle to kill Moby Dick, the great white whale. Hansen's struggle to persuade the medical and scientific world that he was correct was aided by the later development of improved microscopy and more effective histological staining techniques. An important step forward occurred in 1879 when the German scientist Albert Neisser visited Hansen in Bergen. Neisser, whose various contributions to the science of bacteriology are memorialized in the genus *Neisseria*, had become interested in the Norwegian's work and Hansen generously demonstrated his patients and their biopsy slides. He also provided Neisser with tissue specimens to take home to Breslau, where microscopes and staining methods were more advanced. Neisser was able to use newer techniques to confirm what Hansen had claimed. Hansen's generosity backfired, however, when Neisser published his own work as an original finding, without crediting Hansen's prior claims or assistance. That led to a long unpleasant tussle for priority called the "Hansen-Neisser struggle," a debate that
As late as 1906, however, the great Victorian medical polymath, Sir Jonathan Hutchinson, published an extensive monograph claiming that although *Bacillus lepra* caused the illness, it arose not via transmission from an infected patient but from eating rotten or contaminated fish. His mass of information and speculation was impressive—although wrong—and indicated the persistence of uncertainty in the medical world. Hutchinson clearly held his view even earlier than 1895, because the monograph of that year by Hansen and Looft argues against Hutchinson in these words:

“Against Hutchinson’s hypothesis there is in the first place the fact that we have never succeeded in cultivating the bacillus, which, if the bacillus lived as a saprophyte on decaying fish, would be a very simple matter. And there are, secondly, places where the inhabitants certainly and frequently enjoy decaying fish without the disease appearing. And thirdly, there are many places authoritatively indicated where leprosy is present, and where no fish is ever eaten.”

In 1888, two years before Koch’s Postulates were proclaimed, Hansen still sought further ammunition in his battle to persuade the scientific world that his career-long claims were correct. He heard reports from Norwegian emigrants living in Minnesota since as early as the 1830s that although some of them developed leprosy after their emigration, none of their children showed signs of it. Such reports, if indeed true, contrasted markedly with the experience in western Norway, where so many afflicted parents also had one or more children with the illness. In order to study the curious assertion personally, Hansen applied to the Norwegian government for a travel grant to visit Minnesota. He planned to examine families in which this seeming non-inheritance of leprosy was reported. His request was denied. A financial “angel” appeared, however, in the form of a St. Paul physician of Norwegian descent, Edward Bockman, who offered travel funds plus the hospitality of his own home. This led to Hansen’s visit. He departed Norway in January 1888, for a six-month stay in St. Paul, where he lived in Bockman’s home. Hansen’s examination of the families in which the illness was known, and especially his scrupulous study of the children, disclosed no instance of an affected child. This information naturally contributed to the power of the argument against hereditary transmission that he had been urging since 1873.

The generally accepted reasons why Hansen found no infected children in Minnesota, as he enumerated them, were the improvement there in nutrition, far better cleanliness and sanitation, and less crowding, as well as more successful isolation of infected persons. Experts now believe that a genetic component or “susceptibility” to Hansen’s Disease may exist in some families, a mechanism likely operating through heredity’s genesis of the immune system.

The work in perspective

Hansen’s fight against traditional belief and powerful opposition is, of course, not unique in the history of science—witness the romantic struggles exemplified in the lives of Vesalius, Copernicus, Harvey, Galileo or Darwin. In our own time, many can recall the delays and arguments before infection was accepted as the cause of peptic ulcer disease, or Legionnaire’s Disease, or even AIDS. But the obverse can also...
Hansen's life, which ended in 1912, was then and yet recognized for his diligent effort in science and public policy to improve the health and care of patients who suffered from an ancient scourge, and potentially even to eliminate it entirely.

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REFERENCES


