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Gerhard Henrik Armauer Hansen (1841-1912)—The 100th anniversary of the death of the discoverer of *Mycobacterium leprae*

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Abstract The year 2012 marked the 100th anniversary of the death of Gerhard Henrik Armauer Hansen (1841-1912), a Norwegian physician known for his identification of *Mycobacterium leprae* as the causative agent of leprosy. In the second half of the 19th century, the medical community believed leprosy had a noninfectious etiology, and it was not until Hansen uncovered the relationship between *M leprae* and the clinical manifestations of this disease did that belief begin to change. Strengthening of the late 19th and early 20th century beliefs of the infectious etiology of leprosy made it possible to develop an effective treatment for this still terrible disease.

Professional achievements of the discoverer of *Mycobacterium leprae*

The year 2012 marked the 100th anniversary of the death of Gerhard Henrik Armauer Hansen (1841-1912),[1–6] a Norwegian physician known for his identification of *M leprae* as the causative agent of leprosy (Figure 1). Leprosy is also called Hansen's disease.[7,8]

Hansen was born on July 29, 1841 in Bergen as the eighth of 15 children of a well-regarded businessman, Claus Hansen.[5] The younger Hansen finished primary and high school in Bergen. In 1866, he received his medical degree at the University of Christiania (currently University of Oslo). With his degree in hand, he went to work as a substitute for the main prosector at the university and completed his internship at Oslo's Rigs Hospital.[1] Later, he worked in the hospital on the Lofoten Islands, off the northwest coast of Norway. This work could have provided him with a prosperous life, as Lofoten, located near the Artic Circle, was an opulent marine fishery center. Hansen, however, abandoned this lucrative possibility, and 2 years after graduation returned to his hometown to face an enormous challenge, that of treating leprosy.

In 1868, he began working in St. Jørgen Hospital,[2] where lepers were cared for. This institution was established in the 14th-century hospital and had become a well-known leprosy research center.[9] The director of this hospital was a famous

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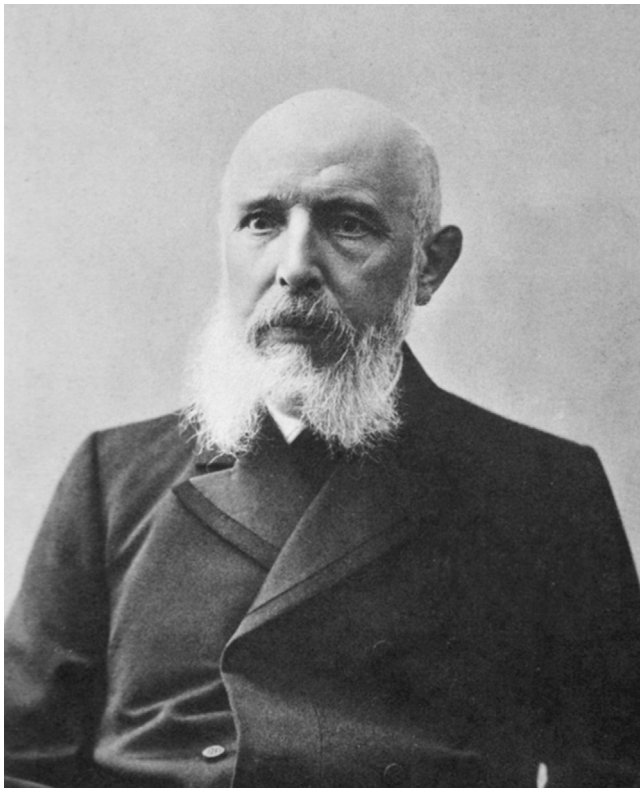

Fig. 1 Gerhard Henrik Armauer Hansen (1841-1912). (From: National Library of Medicine-National Institution of Health, Images from the History of Medicine http://www.nlm.nih.gov/hmd/ihm/).

dermatologist and leprologist, Daniel Cornelius Danielssen (1815-1894), whose major achievement was creating excellent conditions for research on leprosy. Danielssen introduced a system of postmortem and histopathologic examination for all patients who died in the course of leprosy. For covering research costs, including the founding of trips and scholarships, he gave the hospital his Monthyon Prize, which he had received for his research on leprosy from the French Academy of Medicine.[1] Hansen became Danielssen's close associate and one of his trusted disciples.

One year after starting work in St. Jörgen, based on the analysis of epidemiologic data provided by Danielssen, Hansen realized that leprosy was a disease caused by an unidentified exogenous (probably infectious) factor.[1,10] In 1870, to improve his medical knowledge in histopathology and to prove this hypothesis on leprosy, he went first to Bonn and then to Vienna through a scholarship provided by Danielssen. After returning to Bergen, he undertook intensive studies to prove his hypothesis. Soon, examining tissue fluids obtained from lepers, Hansen observed acid-alcohol–resistant small rods.[1,4] Hansen also observed these rods in other tissue samples taken from patients diagnosed with leprosy. (Gonzalez Prendes points to 1871 as the date when Hansen first observed *M leprae*,[1] despite the fact that 1873 is widely accepted as the year of this discovery.) In 1874, for the first time, Hansen published the reports on the observed pathogens and postulated a casual relationship between their existence in histopathologic preparations and signs of leprosy.[11,12] After identifying the stained and unstained cells and acid-alcohol–resistant small rods, Hansen attempted their cultivation and inoculation under laboratory conditions. He delayed having his discovery published due to multiple unsuccessful attempts to produce the pathogen and failure to inoculate it into experimental animal organisms. Ineffective inoculation of experimental animals was the main reason for the medical community to reject Hansen's hypothesis on infectious properties of leprosy.

The difficulty in experimental confirmation of the relationship between the discovered pathogens and the signs of leprosy resulted in Hansen's controversial decision to inoculate a patient without her consent. On November 3, 1879, Hansen attempted to inoculate the pathogen of leprosy into a patient who had been treated for this disease for 17 years. Using a cataract knife, Hansen inoculated the eye of a woman suffering from the maculoanasthetic form of the disease with material drawn from a leprous nodule of a patient suffering from the tuberous form. Because the inoculation was performed without the patient's knowledge of the nature and purpose of the procedure, Hansen was accused of acting to her detriment. During the May 1880 hearing held before the municipal court in Bergen, Hansen lost his position as resident physician of the Bergen leprosy hospitals[13]; however, he maintained the position as leprosy medical officer for the entire country of Norway—a position he held until his death. Additionally, he still conducted research on the discovered leprosy bacillus.

Despite prior criticism of his achievements, he managed to gain recognition from the medical community. Additionally, the First International Congress of Leprology, held in 1897 in Berlin, accepted Hansen's hypothesis on the infectious nature of leprosy and the possibility of transmitting the disease from person to person. The Second International Congress of Leprology, which took place in Bergen in 1909, also referenced Hansen's achievements.[1] The results of his research led to passing a resolution on the necessity of prophylactics and conducting precise epidemiologic analyses, although Hansen himself was still overlooked in some publications as late as the 1930s as the discoverer of *M leprae*.[14]

Biographical details

In January 1873, Hansen married Danielssen's daughter, Stephanie. The marriage, however, would last only a few months because on October 25 of the same year, she died due to complications from pulmonary tuberculosis. In August 1875, 2 years after the death of his first wife, he married Johanne Margarethe Gran, and on May 21, 1876, their first son, named Daniel Cornelius was born. The child was named in honor of Hansen's first father-in-law. Daniel would follow in his father's footsteps as a physician.[1,5]

In 1875, Hansen was appointed the Chief of the Norwegian Leprosy Service. Not only did he become involved in the search for the cause of leprosy and the treatment of patients suffering from this disease, he also made efforts to ensure

adequate protection against being stigmatized by the society. Thanks to his involvement, in 1877, people treated for leprosy became legally protected.[1]

In 1895, Hansen, in collaboration with Carl Looft (1863-1943), published a monograph devoted to the pathophysiology and clinical aspects of leprosy.[15] In this work, he provided a definition of leprosy as a chronic disease caused by the "leprosy bacillus." He also emphasized the inability to grow this pathogen in laboratory conditions. By the end of his life, Hansen suffered from complications of syphilis. He died of a heart attack on February 12, 1912, in Florø, Norway at the age of 70.

Conclusions

Hansen discovered the acid-alcohol–resistant small rods, which appeared later to be *M leprae*. Hansen's discovery negated an age-old tradition of considering leprosy as a noninfectious disease brought on the diseased by him or herself (contingent on poor lifestyle, ie, improper moral behavior) or as a hereditary matter. He had to overcome a great deal of adversity before gaining proper recognition and before the medical community accepted his discovery.

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