Prefácio / Preface
Adolpho Lutz, a life dedicated to leprosy

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It is an honor for me to write the preface to a book about Adolpho Lutz. I learned to admire this physician and other illustrious Brazilians bit by bit, over time. Then, when I became familiar with more information, I realized I had only a vague notion of who Lutz really was. I had never imagined him to be such a complete scientist.

Lutz was born on 18 December 1855 in Rio de Janeiro and received a thorough education in Switzerland and other European countries. He distinguished himself in several fields of medicine and biology, such as clinical medicine, dermatology, therapeutics, veterinary medicine, helminthology, bacteriology, protozoology, entomology and mycology. His studies of ancylostomiasis and *Ancylostoma duodenale* are memorable. In 1908, he was the first to describe paracoccidioidomycosis, in two patients in São Paulo, and to isolate its etiological agent in culture.

Throughout his lifetime as physician and scientist, he was always interested in leprosy. In 1881, when he finished his studies, he returned to Rio de Janeiro, where he revalidated his diploma. The following year, he moved to Limeira, in the interior of the state of São Paulo, and that is probably where, at the beginning of his career, he encountered a considerable number of leprosy patients. In 1885 he left Limeira temporarily and worked for about a year at a German clinic founded by Paul Gerson Unna, a famous dermatologist who was very interested in Hansen’s disease, and who had already proposed several treatments for the illness and was well versed in bacteriological studies of its etiological agent. Unna greatly influenced the young Brazilian doctor, whose first published paper, dating from 1886, was on the subject of the bacteria that cause leprosy.
On his return to Brazil, Lutz moved from Limeira to São Paulo and continued his research on the disease. In 1887 he spent a short period at the Hospital dos Lázaros, linked to the Sisterhood of the Holy Sacrament of the Candelária, in Rio de Janeiro. It was one of the main Hansen’s disease treatment centers in Brazil at the time.

Then, Adolpho Lutz’s medical and scientific life changed considerably.

Far away, in the Pacific Ocean, an archipelago called the Sandwich Islands on its discovery, and then the Archipelago of Hawaii, is composed of eight principal islands: Niihau, Kauai, Molokai, Lanai, Kahoolawe, the largest, Hawaii and Oahu where Honolulu, the capital, is located. Hansen’s disease victims began to be mandatorily hospitalized on the island of Molokai. This isolated area became legendary to scholars of the disease because of the work of Father Damien, a lay brother of the Catholic Church who dedicated his life to leprosy patients and then caught the disease. Adolpho Lutz’s destiny was to go to Molokai.

In 1887 the Health Council of the Kingdom of Hawaii asked the famous Dr. Unna to recommend a physician to work at the leprosarium on Molokai. Unna invited Lutz, who accepted. After some contractual negotiations, Lutz traveled to Hawaii, arriving in Honolulu in 1889.

He worked at the Kahili reception station, a makeshift hospital located some miles from Honolulu where the patients were examined; those with confirmed cases of leprosy were sent to Molokai. Before departure, Lutz had asked to maintain a private clinic in Honolulu. The accounts of Dr. J. H. Kimball, president of the Health Council, indicate that from the beginning, only months after Lutz took up his post at the hospital, difficulties arose from bad working conditions, shortage of instruments, medicines and support personnel and because of the distance from other hospitals to which patients could be sent for emergency treatment. Improvement came and his life was changed when the English nurse Amy Marie Gertrude Fowler arrived at the hospital. She was very devoted to the patients and contributed a great deal. They married in 1881.

Lutz worked for less than a year at the station in Kalihi. After an incident with an employee that worked there, mishandled by the Health Council, Lutz and Amy resigned. In 1892 the couple left Hawaii for San Francisco, returning to Brazil at the beginning of 1893.

During his stay in Hawaii, Lutz continued his studies of biology and worked on his theory of transmission of leprosy by mosquito, his hypothesis since his first contact with the disease.
Lutz was a respected leprologist when he arrived in Honolulu, quite experienced with clinical aspects of the disease; in 1888 he said he had treated about 250 patients, besides those he studied in Hawaii. His wide experience and his characteristic meticulousness made him one of the great specialists of his time.

Lutz did not say much about the island of Molokai or the colony of patients confined there: how they lived, the medical care they received, their living conditions and the discipline to which they were subjected – if any – and the characteristics of the island as a whole. In his lecture about the archipelago, he spoke of the beauty of each island, its vegetation, the volcanos and many other details, but about Molokai he spoke very little. It was almost as if he had never been there. The leprosarium was located on a peninsula on the north side of the island, confined by the ocean in front and both sides and by the mountains behind. This wall, known as Pali, could only be crossed by a path that led to the house of a German who was the agent of the Health Council on the island and superintendent of the colony.

Adolpho Lutz knew everything to be known about leprosy at the time and tried to work in all possible fields of research. His bacteriological, clinical and epidemiological studies were outstanding.

Hansen described the bacillus that causes leprosy in 1873 and ten years later Koch discovered the tuberculosis bacillus. It was during this interval that Lutz studied in Europe, went to Lister’s lectures, assimilated Pasteur’s teachings and, influenced by Unna’s work on hanseniasis, returned to Brazil.

At the time, the giants of bacteriology – Hansen, Neisser, Unna and others – were wrangling over the definition of the agent that causes hanseniasis and its surrounding environment.

From the start, Hansen and Looft argued that the microbe causing the disease was a rod, a bacillus that accumulated in large numbers within cells of different sizes and found in the tuberous forms of the disease. On the other hand, Unna and Lutz did not believe that the microbe was a rod, or a bacillus. The staining methods developed by Unna and Lutz showed, inside what others considered as a bacillus, spherules lying side by side and in a way that sometimes recalled rods. Lutz thought these spherules divided and pushed on each other laterally and that they were enveloped in a mucous substance that formed a gelatinous layer around them. The accumulation of these formations gave the

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impression that the granulations, in a variety of forms, were part of an amorphous mass that he called zooglea.

Lutz and Unna were against the idea that these gelatinous masses were cell cytoplasm and argued that the cell nuclei were not visible. Hansen had already tried to show that the germs were really inside the cells. But often only vacuoles were observed, interpreted as the disappearance of a nucleus by some degenerative process.

It was an arduous task to defend these different points of view, because researchers used staining methods that were improved only gradually and the resolution power of microscopes was limited at the time.

Lutz always defended his view that the germs causing leprosy were not bacilli but coccus and chose the genus *Coccothrix* for them. Many people thought that this name should be given priority over the genus *Mycobacterium*, created later by Lehmann and Neumann, but actually Lutz’s name could not be maintained because the germ causing Hansen’s disease is a bacillus and not a coccus.

An interesting fact that emerges from our bacteriological observations is Lutz’s supposition that the granulations were the active elements and that many of the forms observed inside the zooglea were dead. Hansen, on the other hand, thought the highly colored bacilli were alive, and that others, fragmented, with irregular coloration, or forming granules, were degenerated forms of the bacteria.

The special granules found by Lutz in the Hansen’s bacillus were always made visible by the staining he and Unna used, based on the Gram method. In 1910 Much also showed Gram-positive granulations in the tuberculosis bacillus, called Much granulations; like Lutz, he supposed they were part of the evolutionary cycle of the micobacteria. When Antônio Cardoso Fontes studied these granulations at Instituto Oswaldo Cruz in 1910, he filtered tubercular pus and verified that its inoculation in guinea pigs reproduced an atypical tuberculosis. These observations were not confirmed by other authors. Although the nature of these granules has not been completely explained, today they are of secondary importance.2

Adolpho Lutz, as we have seen, had become a great leprologist thanks to his experience over the years with patients, whom he continued to treat. The

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careful descriptions of the forms of the disease and his intuition about the real significance of the lesions led him to describe the illness so well that today not much can be added to what he said about its various clinical aspects.

Lutz observed the erythema nodosum as a form of nodular leprosy without the same cell content and considered fever, in these cases, to be a sign of bacteremia. He also thought that these were the contagious phases of the disease. We now know that torose erythema is the expression, in humoral immunity, of the organism’s response to bacilli that are being destroyed by the immune system. Lutz had already observed that in macular form there was little or no bacillus and that this was a benign form, and in this he agreed with what Hansen thought. Lutz had very firm ideas about the disease and thought it was not very contagious, much less so than tuberculosis, and that contagion did not depend on heredity.

He attributed contagion to stings by insects of the *Culex* genus. Throughout his life, when he was director of the Bacteriological Institute of São Paulo and afterwards when he moved to Rio de Janeiro to work at Instituto Oswaldo Cruz until his death in 1940, he emphasized his belief in this mode of transmission of leprosy.

From his first studies of the disease he had developed the theory of transmission of the microbe by mosquito. This was coherent with knowledge at the time, when it was proven that great endemic diseases like yellow fever and malaria were related to mosquitoes. Lutz thought the *Culex* genus was responsible because transmission could not be effected by other orders, like lice, fleas and bedbugs: they were as common in countries free of Hansen’s disease as in those that were not. When he was in Hawaii, he noticed that culicidians were a real pest there. They had probably proliferated because of extensive rice and taro cultivation and seemed to have been present only relatively recently, like leprosy – such that there were no words in the archipelago’s language for the illness or the mosquito. Lutz attributed the difficulty of direct contagion to the fact that most of the bacilli of the zooglea were dead, which would explain the negative results of experimental inoculation. On the other hand, bacilli that were eliminated in large quantity by the ulcerated skin and mucosae had been viable, the number of patients would be far higher. According to Lutz, no one had yet described the development of the bacillus

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inside the mosquito, but some transformations were necessary. It was not enough for the bacillus to remain within the insect or be eliminated with its feces for transmission to occur. One might also take into account the moment it stung the patient, the lesion that was the sting’s target and the conditions the bacillus required to be able to transmit the disease. Lutz thought that the mosquito had to sting the patient during a fever and that it stung mainly patients whose lesions were richly bacilliferous. He thought the germs transmitted the disease through their granulations, that are not always acid resistant and appeared either free or lined up. Lutz defended this opinion until the end of his life. He thought compulsory hospitalization was absurd and noted that doors were closed to patients but windows were open to mosquitoes, the great disseminators of the disease.

Adolpho Lutz was always up to date on progress in leprosy research and it is strange that he made no reference to the Strasbourg Conference of 1923, when Mitsuda presented the first results of his test and Darier took the first steps towards identifying tuberculoid leprosy.4

The use of immunity to explain different clinical forms began to be developed at that time.

Later studies of the bacillus, done with new methodologies like the electronic microscope, explained many of its characteristics, including its degenerative aspects, and the theory of disease transmission by insects did not resist the various new arguments raised against it.

Lutz had great influence on the study of Hansen’s disease by his firm ideas about contagion, procedures to be adopted regarding the disease and his solid knowledge of it, which he tried to disseminate through books, articles and lectures. By his example as a scientist, Lutz made an enormous contribution to the study of the disease and should be included in the pantheon of the world’s great leprologists.

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