Globalizing Leprosy

A Transnational History of Production and Circulation of Medical Knowledge, 1850s-1930s

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[1] Berre, Andreas. *lepraMap; Prototyping a Tool for Modeling Historical Sources.* MA-thesis. Molde University College. 2011. Online: http://brage.bibsys.no/hsm/retrieve/1318/master_berre.pdf. My comments on the collaboration is found in Berre 2011: Appendix 1. The source code for the project is available online: https://github.com/andreasBerre/lepraMap, and parts of the project was presented in the paper "Transdisciplinary history of medicine: A 'Mode 2'-approach" at the conference "The Future of Medical History" arranged by the Wellcome Centre for the History of Medicine in London in July 2010.

Contents

1. Introduction

On August 10th, 1901, a bust of Dr. Gerhard Armauer Hansen was unveiled in the garden of Bergen Museum, and King Oscar II conferred on Hansen the distinction Commander of the Cross.[2] The initiative came from the Norwegian Medical Association, and the ceremony was headed by the German professor in dermatology Oscar Lassar on behalf of the Leprosy Committee established at the First International Leprosy Conference in Berlin four years earlier. The inscription on the bust in the town in Western Norway reads "Dr. G. Armauer Hansen, discoverer of the leprosy bacillus - a gift from physicians of all countries".[3]

Today, the bust of Hansen is easily overlooked. But in its time both bust and distinction were symbols of honor, a celebration of the Norwegian physician's status as discoverer of the leprosy bacillus. The event can also be seen as the pinnacle of the history of leprosy in Norway: National and international recognition of a successful campaign against the disease. Alarming reports on increasing numbers of leprosy sufferers in the 1840s had led to the state establishing four new leprosy institutions: The research hospital Lungegaardshospitalet (Bergen, 1849), Pleiestiftelsen No. 1 (Bergen, 1857), Reknæs Pleiestiftelse (Molde, 1861) and Reitgjerdet (Trondheim, 1861). The institutions were accompanied by a system of continuous local surveillance of every individual that had the disease, and in 1856 the world's first national patient registry was established. In 1877 and 1885 new and increasingly strict leprosy legislations were put in place. The intervention led the number of cases to decline from almost three thousand in 1856 to less than six hundred at the turn of the century. This study, however, goes beyond the Norwegian state borders and will present a different narrative.

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[2] Waaler, Erik. "Patolologi og mikrobiologi i Bergen. Det første sentrum for lepraforskning." *Nordisk Medicinhistorisk Årbok, Supplementum XI.* 1985: 43.

[3] "Dr. med. G. Armauer Hansen, leprabacillens opdager. Reist af kolleger og venner fra alle lande". All translations are by me, unless otherwise stated.

According to Lassars' speech, "With this initially little appreciated discovery, he [Hansen] alone put down the foundation for the now ruling view of the disease that is leprosy."[4] The bacillus is at the core of this thesis, which seeks to answer what this 'now ruling view of leprosy' was in 1901, and how the view changed over time. Secondly, I investigate how medical knowledge regarding leprosy travelled. How did the knowledge of the bacillus move from being an entity observed by one person in a laboratory in Bergen, to being accepted as the defining character of the disease all over the world?

Medicoscientific knowledge sets the main premises when it comes to leprosy today; both cure, care and policies. The central role of the bacillus can be illustrated by the first sentence presenting the disease on the World Health Organization website: "Leprosy is a chronic infectious disease caused by Mycobacterium leprae, an acid-fast, rod-shaped bacillus."[5] How was this scientific truth initially created? What impact did the bacillus have on diagnosis and treatment, and to what extent did the bacillus set the premises for policies towards those affected by the disease?

This thesis investigates the period between 1847 when leprosy was first clinically distinguished from other diseases and the first issue of the medical journal *International Journal of Leprosy* in 1933. The journal was the outlet of the International Leprosy Association, established two years earlier. Alongside still ongoing international leprosy conferences and the active involvement of the League of Nations (later followed up by the World Health Organization), the organization and its journal were the last elements in an international framework for the circulation of knowledge about leprosy that would remain authoritative for more than seven decades. By 1933 leprosy had become globalized.

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[4] "Allein er hat mit dieser Anfangs so wenig gewürdigten Wissensthat den Grundstein gelegt für die nunmehr universal herrschende Auffassung der Aussatz-Krankheit." Lassar, Oscar. "Gerdhard Arm. Hansen. Foredrag af O. Lassar". *Medicinsk Revue.* 1901: 196.

[5] World Health Organization. *Leprosy elimination; Leprosy: The disease*. http://www.who.int/lep/leprosy/en/. The presentation also highlights the discoverer: "When M.leprae was discovered by G.A. Hansen in 1873, it was the first bacterium to be identified as causing disease in man."

According to the established historiography, the discovery of the bacillus took place on February 28, 1873.[6] The next year, Hansen announced that he had found "in every leprous tubercle extirpated from a living individual — and I have examined a great number of them — small staff-like bodies much resembling bacteria, laying within the cells".[7] His findings were first published in a supplement to a Norwegian medical journal in 1874 and in a slightly shortened English translation the following year. The wording was careful: "Though unable to discover any difference between these bodies and true bacteria, I will not venture to declare them to be actually identical".[8] It was not until the German physician Albert Neisser published that *he* had discovered the bacillus in 1879, and Hansen's subsequent claims to the discovery in Norwegian, German, English and French in 1880, that the bacillus received any real attention. The dispute of precedence was finally settled at the first International Leprosy Conference in Berlin in 1897, when Hansen was officially recognized and honored by his peers as the discoverer of the leprosy bacillus, while Neisser was highlighted as the one who had confirmed the findings. Four years later, the bust was a material recognition of the achievement.

The event in the garden of Bergen Museum was reported both in national and international medical journals. According to an editorial in *The British Medical Journal*, Hansen's discovery had led to "that it is now practically admitted by all those engaged in the study and observation of leprosy, that the disease is contagious."[9] Although this reflected a majority view, the discussion was far from over: What was the bacillus actually? What part did it play in the disease and its causation (etiology)? What consequences should the discovery have in the fight against leprosy?

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[6] Irgens, Lorentz. "The Discovery of Mycobacterium Leprae. A medical achievement in the light of evolving scientific methods." *The American Journal of Dermatopathology*. Vol 6, 1984: 337-343.
[7] Hansen, G. Armauer. "On the Etiology of Leprosy." *British and Foreign Medico-Chirurgical Review* Vol. 55, 1875, as quoted in Irgens 1984: 349. Hansen published the Norwegian original under the title "Indberetning til det Norske medicinske Selskab i Christiania om en med understøttelse af selskabet foretagen reise for at anstille undersøgelser angaaende spedalskhedens aarsager, tildels udførte sammen med forstander Hartwig". *Norsk Magazin for Lægevidenskaben*, Vol. 3, ser. 4, no. 9. 1874: 1-88. All quotes are from the English version.
[8] Hansen 1875: 489.

In Norway and elsewhere, Hansen has had a prominent role among medical scientists and is revered with pride.[10] 'Hansen's disease' is now the accepted medical term for the disease. The legacy reflects what the leading German pathologist Rudolf Virchow stated in a letter read by Lassar in the garden of the museum in 1901: Hansen's work "had definitively cleared up a large and difficult field of pathology", and his name "is known and celebrated throughout the whole world as a benefactor of mankind."[11] According to the Norwegian Biographic Encyclopedia, Hansen is "by far the most famous Norwegian physician of all time."[12] In addition to his own autobiography, five biographies, two TV documentaries and numerous celebratory papers have been written about the discoverer.[13] However, these have been more interested in the man behind the discovery than the impact of the 'discovery' or how the knowledge travelled.

Hansen's discovery is often presented as the second scientific breakthrough in the medical understanding of leprosy. The first was Daniel Cornelius Danielssen and Carl Wilhelm Boeck's publication *Om Spedalskhed* (*On Leprosy*, 1847), which

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[9] Editorial. "The Discoverer of the Leprosy Bacillus". *The British Medical Journal,* August 24, 1901: 494. See Appendix 1.

[10] In the final stages of writing this thesis, for instance, the centenary of Hansen's death in 1912 was celebrated with a two-day international medical conference. (http: //www.uib.no/matnat/en/seminar/2012/09/armauer-hansen-100-aars-markering). The University of Bergen has also recently renovated the "Armaer Hansens hus", a building named after the discoverer (http: //nyheter.uib.no/?modus=vis_nyhet&id=52515). Outside Norway, several similar statues of Hansen have been put up to celebrate the discoverer.

[11] "Möge Ihr Herz offen bleiben für die Empfindungen der Freunde, dass Ihre Arbeit ein grosses und schweriges Gebiet definitive geklärt hat, und dass in der ganzen Welt Ihr Name als der eines Wohlthäters der Menschheit gekannt und gefeiert ist." Virchow, Rudolf. "Hochgeehrter Freund und College!" *Medicinsk Revue.* 1901: 198. Virchow was head of the committee that raised funds for the bust.

[12] "Gerhard Armauer Hansen er uten sammenligning den mest berømte norske lege gjennom tidene." Lærum, Ole Didrik. "Gerhard Armauer Hansen." *Norsk biografisk leksikon.* 2001.

[13] The biographies are: Kobro, Isak. *Gerhard Henrik Armauer Hansen (1841-1912)*. 1924; Gade, Fredrik Georg. *G. Armauer Hansen 29 juli 1841-11 februar 1912*. 1931; Vogelsang, Th. M. *G. H. Armauer Hansen*. 1968; Patrix, Johanne-Margrethe. *Armauer-Hansen.* 1976; Patrix, Johanne-Margrethe. *Gerhard Armauer Hansen: leprabasillens oppdager*. 1997. For celebratory papers see: Lie, H. P. "Armauer Hansen, Leprabasillens oppdager (1841-1912)." *Nordisk Medisin*. No. 11. 1941; Lærum, Ole Didrik. "Gerhard Henrik Armauer Hansen (1841-1912)." *Det norske Videnskaps-Akademi, Årbok 2001*. 2003. The documentaries: Sandberg, Sverre and Sandberg, Haakon. *Armauer Hansen*. Svekon Film. 1974; Herland, Ole Geir. "Gerhard Armauer Hansen – bergenslegen som oppdaget leprabasillen". Mediakey. 2012. For a studies situating Hansen's discovery in a contemporary medico-scientific context, see: Irgens 1984; Irgens, Lorenz. "Hansen, 150 Years After His Birth, the Context of a Medical Discovery". *International Journal of Leprosy.* 1992: 466-469. For a commented overview, see: Vollset, Magnus. *Fra Lidelse til Trussel. Spedalskheten i Norge på 1800-tallet*. Masteroppgave, Historisk institutt. University of Bergen 2005: 4-5.

clinically distinguished leprosy from other afflictions of the skin.[14] At the unveiling of the bust in 1901, Lassar highlighted Danielssen and Boeck as the ones who recognized the anatomical pathology of the disease and thus made leprosy an object for scientific study.[15] The third breakthrough was the first experiments with treating leprosy using the sulfone drug Promin in Carville, Louisiana (USA), in 1941.[16] Since no other breakthroughs were made, the implicit assumption is that when it came to medical research, nothing of much interest took place in the intervening seven decades.

The lack of breakthroughs stands in stark contrast to the medical research that was actually conducted. Between 1943 and 1948, *Índice Bibliográfico de lepra* was published in three volumes by the Library of the Leprosy Prevention Department, São Paulo, Brazil. The index was 1,935 pages long and contained more than 30,000 entries. The vast majority was research papers on leprosy published in the period stretching from the 1870s to the Second World War. According to the introduction, it was written "with the constant purpose of contribution to, and making easier, the study of leprosy".[17] It was also a way to save time: Prior to this, the library answered an average of 185,000 consultations by correspondence from all over the world, annually. The index is a reminder that research into leprosy was conducted in numerous sites, that medical research is more complex than a handful of

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[14] Danielssen, D. C. and Boeck, C. W. *Om Spedalskhed.* 1847; Danielssen, D. C. and Boeck, C. W. *Traité de la Spédalskhed ou Éléphantiasis des Grecs.* 1848. In 1856, a translation of Danielssen and Boeck's monograph was published in a series of eight articles under the title "On the Nature and Treatment of Leprosy" by Erasmus Wilson in *The Lancet*. (Buckingham, Jane. *Leprosy in Colonial South India*. 2002: 120; Edmond, Rod. *Leprosy and Empire. A Medical and Cultural History*. 2006: 46).

[15] Lassar 1901: 196. Similar phrases have been repeated by historians such as Sanjiv Kakar, who has stated that the book "laid the foundation for the scientific study of leprosy." (Kakar, Sanjiv. "Leprosy in British India, 1860-1940: Colonial Politics and Missionary Medicine." *Medical History.* Vol. 40, no. 2. 1996: 217.) Tony Gould goes further and argyes that the book "brought about a revolution in thinking about leprosy and marks the beginning of a modern, scientific era in the approach to – and treatment of – this disease." (Gould, Tony. *Don't fence me in. From curse to cure: Leprosy in modern times.* 2005: 37.)

[16] Faget, Q. F. Johansen, J. Dinan, B.Prejan and C. Eccles. "The promin treatment of leprosy". *Public Health Report.* No. 58, 1943: 1729-1741. See also: Lechat, Michel. "La Lèpre après Père Damien". *AMA.* No. 65, May-June 2010. (http: //www.md.ucl.ac.be/ama-ucl/Lepre65.html). Promin is a derivative of dapsone, one of the three main compounds of the Multidrug therapy (MDT) used today. The others components of the currently used medication is rifampicin, and clofazimine.

[17] Keffer, Luiza (ed.). *Índice Bibliográfico de lepra 1500-1943.* Biblioteca do Departamento de Profilaxia da Lepra do Estado de São Paulo – Brasil. Vol. 1. 1944: XIX. According to the introduction: "We have collected one of the greatest specialized bibliographies in medicine, which at present has more than 100,000 cards." (IX)

breakthroughs, and that the knowledge was relevant to other geographical locations than where it was produced. Medico-scientific knowledge went 'beyond borders'.[18]

My ambition in this thesis is not to offer a global history of leprosy, but a history of how the research into leprosy was organized on a global scale. I will argue that in the beginning of my period, there were a wide range of competing medical interpretations of 'leprosy' and disagreements on what observations medical knowledge should be based on. By the early 1930s, the chronological end-point of my discussion, the leprosy bacillus was accepted as the cause of the disease all over the world. For this to be possible, the circulation of medical knowledge regarding the disease had to be organized on a global scale. This is indeed what happened.

The thesis offers an alternative to what James Secord and several others have termed science historians' "obsession with novelty and the places in which novelty begins".[19] Instead of focusing narrowly on Hansen's discovery, I will follow Secord's suggestion of seeing science as a form of communication, a collective activity taking place in various locations – an activity that needed to be organized. For the bacillus to have an impact, people needed to be convinced that it existed and that its existence was relevant. As the title of the thesis suggests, the production and circulation of knowledge will take center stage. The aim is to explain how this extremely successful medical community established an international infrastructure for the circulation of knowledge. I will argue that an important consequence of the bacillus was that it provided a shared point of reference to a range of research efforts taking place in various sites around the world. In this perspective, how the bacillus was enacted in debates and its role as an impetus for collaboration becomes more interesting than its genesis.

[18] I have borrowed the term 'beyond borders' from Simon, Josep and Néstor Herran (eds). *Beyond Borders. Fresh Perspectives in History of Science*. 2008.
[19] Secord, James A. "Knowledge in Transit". *Isis*. 2004: 654-672, quote on p. 662.

Three debates

This thesis is informed by, and seeks to add to, three distinct research fields and traditions: The first is debates on whether bacteriology constituted a radical break with previous understandings of disease; the second is debates within the history of science regarding transnationalism, localism and the place of scientific practice; the third is the growing body of research on the history of leprosy.

Gerhard Armauer Hansen's discovery of the leprosy bacillus has been presented as an "epoch-making achievement", marking a break with previous knowledge of the disease.[20] This interpretation balances between history of medical science as an accumulation of new and increasingly accurate knowledge on the one hand, and as radical shifts where bacteriology constituted a revolution on the other. To what extent there really was such a revolution has been a source of debate among historians. Based on studying medical customs in Britain between 1870 and 1910, Michael Worboys found that bacteriology did not lead to changes in medical practices, and he has therefore argued that there was no "bacteriological revolution".[21] Andrew Cunningham, to the contrary, has argued in a study of the plague that bacteriology meant the introduction of single causes of diseases, that it heralded an epistemological transformation which lasted until the 1930s, and that the shift was so radical that we today can hardly imagine a world without diseases having a specific cause: "The identities of pre-1894 and post-1894 plague have become incommensurable. We are simply unable to say whether they were the same, since the criteria of 'sameness' have been changed".[22] There have also been studies that bypass the discussion of bacteriology as a revolution, and instead add nuances through

[20] Irgens 1984: 337.
[21] Worboys, Michael. "Was there a Bacteriological Revolution in late nineteenth-century medicine?" *Studies in History and Philosophy of Science. Part C: Studies in History and Philosophy of Biological and Biomedical Sciences.* Vol. 38, issue 1. 2007: 20-42. Elsewhere, Worboys has demonstrated that 'germs' was a flexible concept with multiple meanings, and that there was not a single bacteriological model. See: Worboys, Michael. *Spreading Germs. Disease theories and medical practice in Britain, 1865-1900.* 2000.
[22] Cunningham, Andrew. "Transforming plague. The laboratory and the identity of infectious disease". In: Cunningham, Andrew and Perry Williams. *The laboratory revolution in medicine.* [1992] 2002: 209-244; quote on p. 242.

detailing the scientific work actually conducted in the bacteriological laboratory, such as Christoph Gradmann's *Laboratory Disease* (2009), which discusses the history of medical bacteriology though the biography of Robert Koch.[23]

The question of a bacteriological revolution is part of a larger debate on the nature of scientific progress and the question of disruption versus continuity. In the seminal paper "The disappearance of the sick-man from medical cosmology, 1770-1870" (1976) Nicholas Jewson argued that medicine in the laboratory represents a transformation which "precipitated a total reconstruction of the epistemological foundations of medicine as a field of knowledge."[24] The introduction of laboratory medicine in the 1870s was the third such transformation, preceded by bedside medicine and hospital medicine. These shifts were not just theoretical. Rather, the modes of production of medical knowledge led to changes in social relations between physicians and in their relations to the individuals affected by disease: Instead of relating to patients directly, only samples made their way into the laboratories. Status from medical peers became more important than meeting the expectations of the persons suffering from disease.

Later commentaries have pointed out that Jewson's analysis was strongly influenced by Thomas Kuhn's *The Structure of Scientific Revolutions* (1962), and his model of science as 'puzzle-solving' working within successive and 'incommensurable' paradigms.[25] Kuhn in turn drew on Ludwig Fleck's *Genesis and Development of a Scientific Fact* ([1935] 1979), which presents science as a collective endeavour which not just accumulates new pieces of information, but also overthrows old 'thought-styles'.[26]

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[23] Gradmann, Christoph. *Laboratory Disease. Robert Koch's Medical Bacteriology*. 2009.

[24] Jewson, N. D. "The disappearance of the sick-man from medical cosmology, 1770-1870". *Sociology.* Vol. 10. 1976: 225-244, quote on p. 237. Jewson defines 'Medical Cosmology' as "conceptual structures which constitute the frame of reference within which all questions are posted and all answers are offered." (Op. cit.: 225.)

[25] Armstrong, David. "Indeterminate sick-men—a commentary on Jewson's 'Disappearance of the sick-man from medical cosmology'." *International Journal of Epidemiology*. No. 3. 2009: 642–645. See also: Nicolson, Malcom. "Nicholas Jewson and the disappearance of the sick man from medical cosmology, 1770–1870". *International Journal of Epidemiology*. No. 3. 2009: 639-642.

[26] Fleck, Ludwig. *Genesis and Development of a Scientific Fact*. [1935] 1979. Kuhn wrote the foreword to the first English translation of Fleck's *Genesis and Development* in 1979, edited by T. K. Trenn and R. K. Merton.

Despite endorsing Kuhn and Fleck's argument that science must be understood as a social and collective endeavor, the model of succeeding and incommensurable paradigms (or thought-styles) seems inadequate when it comes to the history of medicine. Novelties does not mean that old knowledge is suddenly no longer relevant: Bacteriology did not mean an end to clinical diagnosis, that the physicians stopped compiling medical statistics, or that that measuring the vitals of a patient had become obsolete.

To me, John V. Pickstone's framework from *Ways of Knowing* (2001) is more compelling.[27] Pickstone presents five 'ways of knowing', the first three of which correspond relatively well to Jewson's three step model: Natural history (description and classification), analysis (uncovering elements), experimentation (controlling phenomena), technoscience (creating novelties), and world readings (hermeneutics/interpretation).[28] Pickstone agrees with Jewson in that new ways of knowing were developed at specific points in time and were entangled with new ways of working. To describe and classify kinds to create 'natural history' demands a different set of activities than to learn about phenomena through experimentation. But instead of replacing the old, the new ways of knowing add to the repertoire of possible ways of knowing. They must be understood "like elements in modern chemistry and not as taxonomic boxes into which instances of STM [Science, Technology and Medicine] are to be placed, or forced."[29] Different ways of knowing and working make up 'compounds' that are given different emphasis in different contexts and at different points in time.[30]

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[27] Pickstone, John V. "Ways of Knowing: Towards a Historical Sociology of Science, Technology and Medicine." *The British Journal for the History of Science,* Vol. 26, No. 4. 1993: 433-458; Pickstone, John V. *Ways of Knowing. A New History of Science, Technology and Medicine.* 2001; Pickstone, John V. "A Brief introduction to ways of knowing and ways of working". *History of Science.* 2011: 235-245.

[28] Another important distinction between Pickstone and Jewson is that while the latter's argument concerns the history of medicine, the explicit ambition of Pickstone's 'ways of knowing' is to transcend disciplines. The analytical categories are equally relevant to all fields of knowledge, Pickstone argues. For Pickstone's comments on Jewson, see: Pickstone, John V. "From history of medicine to a general history of 'working knowledges'". *International Journal of Epidemiology.* 2009: 646-649.

[29] Pickstone 2011: 235.

[30] Lorraine Daston and Peter Galison have presented a similar line of reasoning in their study of the changing meanings of 'objectivity' in scientific atlases from the eighteenth to the twenty-first century: "Epistemic virtues

Pickstone's repeated emphasis that ways of knowing are intertwined with ways of working is a reflection of another long-standing criticism of Kuhn's perspective on science, namely that by only examining the arguments scientists make, the practical implications of science are downplayed. Arguments and dominant positions are important, but they are not the only aspects of science. In the late 1970s the Edinburgh-school argued that this perspective in effect placed science in a realm outside society: Only when the historical actors made a mistake 'outside forces' were brought in to explain the mistake. If the argument was correct, given today's standards, there was no need for further investigation.[31] To the contrary, the sociology of knowledge has convincingly shown that *all* knowledge – not just what in hindsight turned out to be false – is influenced by social and cultural factors.

The solution suggested by the Edinburgh-school's "Strong Programme" was a doctrine of causality, impartiality, symmetry and reflexivity.[32] The goal was to create a methodological antidote to teleology (a predefined history where science is nothing but a linear progression of clearing up mistakes unavoidably leading to what we today hold to be true) that would avoid relativism (that any statement about the world is subjective and thus equally valid). In practice, the programme has meant that the standards for investigating the actors who in hindsight were 'correct' and those who turned out to be were 'wrong' must be the same. Second, science cannot be explained without investigating the social and cultural context in which the production of knowledge took place. Especially the latter argument has been hugely influential for later historical investigations.

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do not replace one another like a succession of kings. Rather, they accumulate into a repertoire of possible ways of knowing." Daston, Lorraine and Peter Galison. *Objectivity*. 2007: 111-113.

[31] "When a thinker does what is rational to do, we need enquire no further into the causes of his action; whereas, when he does what is in fact irrational – even if he believes it to be rational – we require some further explanation." Laudan, Larry. *Progress and Its Problems: Towards a Theory of Scientific Growth.* 1977: 188-189. In: Enebakk, Vidar. *Vitenskapsstudier. Historie, teori, kritikk*. 2008: 99. In my opinion an equally valid criticism of this perspective is that through dismissing the blind alleys, the science that 'failed', the narratives end up framing the history of science as one of inevitable and streamlined progress, simply because of the a priori choice of dismissing everything that does not fit this picture. This might lead to unrealistic expectations to science.

[32] Bloor 1991: 7. The study most commonly highlighted as exemplary within this tradition is: Shapin, Steven and Simon Schaffer. *Leviathan and the Air-Pump.* 1985.

The vast majority of historical studies into the history of science and medicine have defined 'social and cultural context' geographically, and consequently investigated science as an intrinsically local activity. According to Peter Galison "The turn toward local explanation in the historical, sociological, and philosophical understanding of science may well be the single most important change in the last thirty years."[33] This has provided unprecedented insights into how the relation between knowledge and practice is expressed, and compared to Kuhn's emphasis on arguments it has definitively helped bring science down to earth. Even so, the perspective is not unproblematic.

A major drawback of the turn to local explanations is that it creates an artificial boundary between the geographically defined 'inside' to be studied through archival sources, and the 'outside' which either influences or is influenced by events taking place elsewhere. This leads to looking for explanations primarily in previous or concurrent events taking place in the same geographically predefined area, and makes it difficult to explain similarities in how issues were addressed in widely different contexts at more or less the same time.[34] In the entry on 'Medicine' in *The Palgrave Dictionary of Transnational History* (2009), Sanjoy Bhattacharya pointed out that the construction of medical knowledge took place in several sites: Ideas were developed in one region, tested in another and often implemented elsewhere. "Medical ideas constantly flowed in all directions."[35] I wish to fill this catchphrase with content. The first step is finding good questions to ask.

The Finnish historian and philosopher Jouni-Matti Kuukkanen has asked: If science is inherently local, how can we explain that its knowledge is universally applicable? He phrases the challenge as follows: "The problem of the globality of science challenges localism to offer an account of the mechanism through which

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[33] Galison, Peter. "Ten Problems in History and Philosophy of Science". *Isis.* Vol. 99, No. 1. 2008: 111-124, quote on p. 119.
[34] For examples of this, see for instance Vollset 2005 or Leung, Angela Ki Che. *Leprosy in China, a history*. 2009.
[35] Bhattacharya, Sanjoy. "Medicine". In: Iriye, Akira and Pierre-Yves Saunier (eds). *The Palgrave Dictionary of Transnational History: From the mid-19th century to the present day.* 2009: 708.

science can move from one locality to another."[36] Likewise, James Secord has argued that instead of looking for the genesis of new knowledge, more fruitful research questions are: "How and why does knowledge circulate? How does it cease to be the exclusive property of a single individual or group and become part of the taken-for-granted understanding of much wider groups of people?"[37] The thesis is an attempt at addressing these questions.

These are not new concerns. Already in the early 1930s, Ludwig Fleck challenged the assumption that it is possible to pinpoint scientific breakthroughs to a place and a discoverer. In his study of the relation between syphilis and the Wasserman test, Fleck pointed out:

> Very often it is impossible to find any originator for an idea generated during discussion and critique. Its meaning changes repeatedly; it is adapted and becomes common property. Accordingly, it achieves a superindividual value, and becomes an axiom, a guideline for thinking.[38]

Although Fleck underestimated the importance of physicians allocating individual honors, for instance through putting up busts or giving diseases or methods the name of their originators (a narrative he explicitly opposed), his description is valid also when it comes to medical knowledge about leprosy. Fleck's solution was to highlight the individual actors and their arguments, both proponents and opponents stepping forward to address the community of researchers. Over time these negotiations led to

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[36] Kuukkanen, Jouni-Matti. "I am knowledge. Get me out of here! On localism and the universality of science". *Studies in History and Philosophy of Science.* Vol. 42. 2011: 590-601, quote on p. 594. Kuukkanen argues that "strong localism is both historiographically and philosophically untenable", but suggest a more pragmatic localism with emphasis on extra-local inferences through studies of circulation and practices of delocalization. See also: Kuukkanen, Jouni-Matti. "Senses of localism". *History of Science.* Vol. 50, No. 4. 2012: 477-500; Livingstone, David N. *Putting Science in its place. Geographies of Scientific Knowledge.* 2003: 140-141.

[37] Secord 2004, 94: 655. Secord has argued this point since he took initiative to the 'Big Picture'-debate in 1991, which addressed disciplinary fragmentation within history, and resulted in a special issue in *The British Journal for the History of Science*, Vol. 26, No. 4, 1993. See also the *The British Journal for the History of Science*'s special issue "Transnational History of Science" (Vol. 45, No. 3. 2012: 319-442). One of the recommendations in the introduction, is to conduct studies that can explain how locally produced knowledge becomes globally accepted. (Turchetti, Simone, Néstor Herran and Sorya Boudia. "Introduction: have we ever been 'transnational'? Towards a history of science across and beyond borders". *The British Journal for the History of Science.* Vol. 45, No. 3. 2012: 331.)

[38] Fleck 1979: 121.

the establishment of a common style of thought ('Denkstil') shared by a thought collective ('Denkkollektiv'), defined as a community of individuals maintaining interactions and mutually exchanging ideas.

While I will not adapt the terminology of thought collectives and thought styles, I will in this thesis place the historical actors in the foreground; both those who 'stepped forward to address the community' and those who were involved in establishing and maintaining arenas for exchanges of ideas. My focus will be on the circulation of knowledge, understood as the communication aimed primarily at medical peers in other locations. This communication, I argue, is the backbone of any international scientific community.

In the first three chapters, my emphasis is on the content of this communication: What circulated, and what arguments were made? The last three chapters will focus on how knowledge circulated, and how this was organized. Although the cast is larger than in the typical local study, I will show that leprosy was made into a global disease by a relatively small group of individuals.[39]

Despite increasing interest in the problem that local studies do not necessarily add up to today's science as a global enterprise, there are so far surprisingly few studies which try to tackle this problem in depth. In my opinion, the most promising attempt at bridging this gap between the local and the global is the framework presented by geographer David N. Livingstone in *Putting Science in its Place* (2003). His starting point is that "Scientific rationality cannot be conceived of independently of temporal and spatial location."[40] His suggestion is therefore to put the site of knowledge production in the center: The 'venues of science'. Second, the context should be studied: The 'cultures of science'. The third part of Livingstone's model is circulation, the movements of scientific knowledge. This is what connects the sites into networks with multiple (and sometimes competing) centers.

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[39] Biographical studies have also been of much help, such as: Power, Helen Joy. *Sir Leonard Rogers Frs. (1868-1962): Tropical Medicine in the Indian Medical Service.* PhD Thesis, University College London. 1993; Gould 2005. The first is a biography of Sir Leonard Rogers, the latter presents the history of leprosy through a collective biography of lives that in different ways were shaped by the disease. I have also found obituaries useful, especially for information about the education and places of work of these historical actors.

I see the circulation of knowledge as a practice in its own right, a practice that needs to be organized. While chapter 5 will examine how circulation does not abolish local differences, the last two chapters will detail how the frameworks for circulation of knowledge related to leprosy was established, which traditions these were built on and what characterized the practice of circulation. My argument is that the circulation of knowledge is what made the medical knowledge about leprosy authoritative: It gave each individual actor access to far more knowledge than had the actor worked in isolation. The international research community is where truths about leprosy are constructed, and therefore deserves to be studied in its own right. This, I believe, also applies to other scientific activities.

In order to explain the local impacts of the circulation of knowledge, I rely heavily on the term 'appropriation'. This concept was used by the STEP-network (Science in The European Periphery) in the 1990s to challenge a framework which saw scientific communication as characterized by active transmission from a scientific 'center' and passive reception in the 'periphery'. The center/periphery-model measured the success of the communication by how fully the center was being reproduced in the periphery. The term 'appropriation' instead signifies that communication is an active process on all parts, that the outcome is *new* knowledge (not reproductions), and that all actors involved in the circulation of knowledge must be given equal epistemological status.[41] As I will show in this thesis, this leads to a de-centered model of science: The perceptions of what constituted 'centers' differed, and appropriation helps explain how local differences were maintained. At the same time, through investigating the discussions in taking place in the circulation of knowledge, it is possible to show how the dominant positions were established and changed over time.

What was known about leprosy at various times and in different places is not just a matter of theoretical interest. Especially the knowledge regarding etiology gave

[40] Livingstone 2003: 184.

rise to tangible practices with immense consequences on how those affected by the disease could live their lives. With the exception of studies of scientific breakthroughs, it is the social aspects of the disease that have been the main concern in studies of the history of leprosy so far. The attention to detail and nuance has differed. Michel Foucault, for instance, argued in the first chapter of *Madness and Civilization* (1961) that society's reaction to leprosy must be understood as an expression of confinement and social control. In the 1300s, "Leprosy disappeared, the leper vanished, or almost, from memory; these structures remained. (…) Poor vagabonds, criminals, and 'deranged minds' would take the part played by the leper."[42] Along similar lines, anthropologist Mary Douglas has proposed that leprosy became a symbol of impurity and that the consequent segregation of leprosy was "part of the successful attempt to create order that resulted in the highly structured society of the thirteenth century."[43] These studies use leprosy as a stepping stone to give insights into fundamental power-relations in society. Further studies into the history of leprosy, however, have shown that taking for granted that leprosy was a cause of universal and timeless anxiety is problematic.

Since its publication in 1989, the study *Leprosy, Racism and Public Health: Social Policy in Chronic Disease Control* by Zachary Gussow has become a shared reference point for later investigations into the history of the disease. Gussow's project was to challenge the notions of a timeless and universal stigma connected to leprosy dating back to Biblical times. Through comparing the approaches to leprosy in mainland United States with Hawaii, Norway, Britain, India and China, Gussow argued that the social construction of leprosy depended on, and changed with,

[41] Guillem-Llobat, Ximo. "Science in the Periphery". In: Simon and Herran (eds) 2008: 291-299. The STEP-network emphasizes comparative methods, but with the exception of Chapter 5 which compares leprosy policies in Norway and British India, this has not been part of my appropriation.
[42] Foucault, Michel. *Madness and Civilization*. 2007: 5. (*Folie et déraison: Histoire de la folie à l'âge classique* [1961]).
[43] Douglas, Mary. "Witchcraft and Leprosy: Two Strategies of Exclusion". *Man*, New Series, Vol. 25, No. 4). 1991: 732. The suggestion that "The initial recognition of anomaly leads to anxiety and from there to suppression or avoidance" was first put forward in: Douglas, Mary. *Purity and danger: an analysis of concept of pollution and taboo*. (1966) 2006: 4.

political forces and social concerns.[44] The disease, Gussow argued, was 'retainted' in the late 19th century, and its social practices entangled in imperialism and racism. Leprosy became a symbolic and tangible threat from the colonies, a medieval disease that if left unchecked would contaminate the 'civilized world'. He also emphasized missionaries connecting the disease with Biblical leprosy, sensationalizing the disease to raise funds and to present their work as heroic and self-sacrificing. Together, this gave leprosy the character of a metaphor, a phenomena that needed to be kept apart from the rest of society. Although irrational fear on behalf of society more often than not took place at the expense of the individuals who had the disease, the responses changed over time and varied from country to country.[45]

A drawback of Gussow's study is that his historical arguments about conditions outside the United States relied on a relatively narrow source material. His presentation of Norwegian leprosy policies as humane and scientific under the title "The Enlightened Kingdom", for instance, ignores the physicians' use of coercion, the context in which the reports he uses as sources were written, and the fact that domestically the Norwegian policies and institutions were politically contested and increasingly unpopular.[46] In this thesis I will not go into the missionary activities and their circulation of knowledge and therefore I cannot comment on their role in spreading prejudices against those affected by the disease. Still, Gussow's claim that secular medico-scientific research disenchanted the negative stereotypes connected to leprosy was not necessarily the case, at least not in the period I am studying. Rather, as I will show in this thesis, Hansen, whose status as a discoverer made him an

[44] For more on social constructivism, see: Jordanova, Ludmilla. "The Social Construction of Medical Knowledge." *Social History of Medicine.* Vol. 8, No. 3. 1995: 361-381; Hacking, Ian. *The social construction of what?* 1999.

[45] Gussow 1989. Despite acknowledging that the social responses to the disease has changed with temporal and geographical location, Gussow and most other studies in the history of leprosy take for granted that the disease today identified by the leprosy bacillus is unchanging throughout history, and judge the merits of previous studies by how well they fit the criteria today accepted to be true. For a discussion, see: Wilson, A. "On the history of disease-concepts: The case of pleurisy". *History of Science. Vol. 38.*,2000: 271-319. I will return to this in Chapter 2.

[46] Andresen, Astri. "'Patients for life': Pleiestiftelsen leprosy hospital 1850-1920s". Andresen, Astri, Tore Grønlie and Svein Atle Skålevåg (eds). *Hospitals, patients and Medicine 1800-2000. Conference Proceedings.* Bergen. 2004: 93-116; Pandya, Shubhada. "The first international leprosy conference, Berlin, 1897: the politics

international authority, was a vocal advocate for educating the population that lepers were to be feared and shunned, first in Norway and then on an international level.

Gussow's project has been seen by scholars as an open invitation for studies adding content and nuances to the overall narrative of local differences, changes over time, and that leprosy is more than 'just' a medico-scientific object. The result has been a rich historiography, including country-specific studies, the studies of institutions and studies of the experiences of those affected by the disease and society's response to it. The main finding is that the approaches to leprosy were indeed not universal, but unique on a local scale.[47] Inspired by social history, these studies offer an explicit or implicit critique of a depiction of the history of medicine "as 'conquest' of disease by great men and great ideas, independent of social and political contexts."[48]

The works of Jane Buckingham and Shubhada Pandya are prime examples of the local studies that have been important for this thesis. Buckingham has investigated the dynamics of colonial power in 19th century South India, especially the relationship between indigenous and colonial medical and legal systems, and their impacts on those suffering from leprosy. Her book is exemplary when it comes to

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of segregation". *História, Ciências, Saúde – Manguinhos.* Vol. 10 (supplement 1). 2003: 161-177; Vollset 2005.

[47] For prominent examples of studies of leprosy and social control, see: Edmond 2006; Leung 2009; Moran, Michelle T. *Colonizing Leprosy. Imperialism and the Politics of Public Health in the United States*. 2007; Buckingham 2002; Robertson, Jo. *In a State of Corruption: Loathsome Disease and the Body Politic*. PhD Thesis, University of Queensland. 1997; Vollset 2005. For studies emphasizing missionaries and colonialism, see: Kipp, Rita Smith. "The Evangelical Uses of Leprosy". *Social Science & Medicine*. 1994; Vaughan, Megan. "Without the Camp: Institutions and Identities in the Colonial History of Leprosy". *Curing Their Ills: Colonial Power and African Illness.* 1991; Warwick Anderson, "Leprosy and Citizenship". *Positions*, Vol. 6, 1998; Manton, John. "Leprosy in Eastern Nigeria and the social history of colonial skin". *Leprosy Review*. Vol. 82. 2011: 124-134; Kakar 1996: 215-230; Worboys, Michael. "The Colonial World as Mission and Mandate: leprosy and Empire. 1900-1940". *Osiris.* 2001: 207-218; Marks, Shula. "What is Colonial about Colonial Medicine? And What has Happened to Imperialism and Health?" *Social History of Medicine*. Vol 10, no. 2. 1997. For studies investigating leprosy institutions, see i.e. Andresen 2004; Robertson, Jo. "The Leprosy Asylum in India: 1886-1947". *Journal of the History of Medicine and Allied Sciences,* Vol. 64, No. 4. 2009. For studies emphasizing the experiences of those who were institutionalized, see: Seng, Loh Kah. *Making and Unmaking the Asylum. Leprosy and Modernity in Singapore and Malaysia*. 2009; McMenamin, Dorothy. "Recording the experiences of leprosy sufferers in Suva, Fiji". *Oral History in New Zealand*, Vol. 17, 2005. See also: ILA's "The Oral History Project", which is part of ILA Global Project on the History of Leprosy: http://www.idealeprosydignity.org/OralHistoryWeb/OralHistory-Main.html

[48] Pandya, Shubhada S. *Leprosy in the Bombay Presidency, 1840-1897: Perceptions and approaches to its control*. Phd Thesis, Mumbai. 2001: 4.

demonstrating the importance of local negotiations in specific contexts.[49] Likewise, Pandya has in her PhD-thesis highlighted the limits of colonial power in Bombay Presidency in India. Between 1840 and 1897, also Judeo-Christian and Hindu interpretations of the disease played part in shaping the medical, sociopolitical and administrative responses to the 'leper problem'.[50] While my prime interest in this study is scientific medical knowledge, this was not the only knowledge in existence. And not only did the local conditions differ from place to place; so did what constituted relevant knowledge.

My contribution to the existing literature in the history of leprosy is primarily the investigation of the transnational circulation of knowledge. How did the dominant extralocal positions change over time, how were frameworks for the circulation of knowledge established and maintained, and what were the mechanisms involved in how knowledge from elsewhere was appropriated (or dismissed)? While a local study would not be able to answer these questions, the circulation of knowledge both originated and played back into the local. Even the actors who were in the vanguard of organizing the circulation of knowledge spent most of their efforts in local settings. In this thesis I rely heavily on previous research to grasp these local contexts. It would not have been possible for a single historian to investigate all the different contexts, and without these localized studies, my transnational project would not have been attainable. This thesis is not a critique of local studies, but an addition.

Two more works within the history of leprosy deserve special mention for charting the course for my study of the circulation of knowledge regarding leprosy: *Leprosy and Empire* (2006) by Rod Edmond, and the paper "The first international leprosy conference, Berlin, 1897: the politics of segregation" by Shubhada Pandya.[51] Edmond's study of leprosy in the British Empire relied on scientific and literary works published in Britain and the British Empire from the 1770s to the early 20th

[49] Buckingham 2002.
[50] Pandya 2001.
[51] Edmond 2006; Pandya 2003.

century. Inspired by Gussow, the goal of Edmond's study was to give historical nuances to how leprosy has been represented in colonial contexts, and "understand better the varying historical conditions in which it [stigma] has been produced."[52] My interest is not in stigma but Edmond's chapters on medical research. These convincingly show how events in one part of the British Empire, such as developing or testing new treatments, could have consequences elsewhere. Edmond has also shown how actors in the colonies took independent initiatives, and despite opposition from the establishment in London, the center/periphery-model is clearly inadequate. My thesis is an expansion of Edmond's perspectives. Pandya's paper on the first international leprosy conference in Berlin in 1897 details the competing schemes aimed at bringing individual actors from various backgrounds together to discuss and establish the first international recommendations on leprosy.[53] This paper is the starting point for my investigation of how the circulation of knowledge about leprosy was organized internationally (Chapter 6). Furthermore, Pandya's perspective was instrumental in my decision to tell the story focusing on the individual actors involved in setting up the structures for transnational circulation of knowledge, and to not overlook the attempts that in hindsight can be portrayed as 'failed'. As Fleck was the first to point out, and Pandya's paper demonstrates: Science is the outcome of debate and opposition, not the labor of individuals working in isolation.

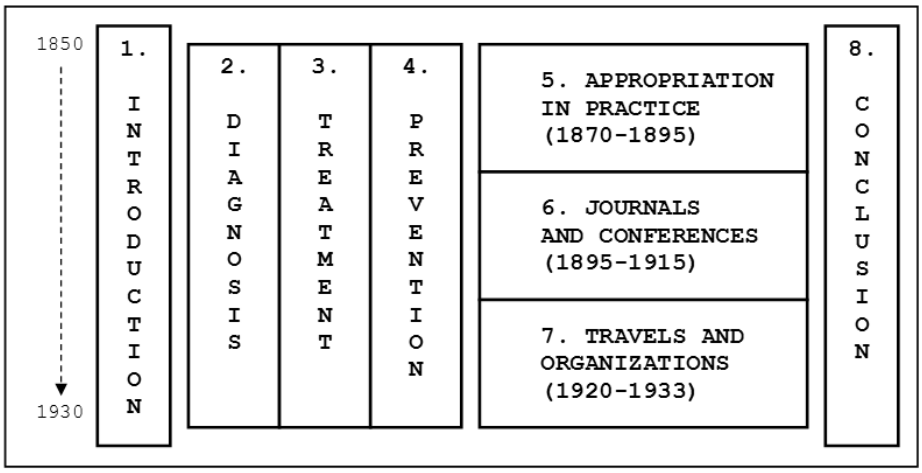
Outline and research strategy

This thesis is divided into two main parts, each with three chapters. The first part (Chapter 2-4) investigates the content of the knowledge that was circulated, and what implications the leprosy bacillus had on three traditional themes in the history of medicine: Diagnosis, treatment and prevention. Each chapter follows the discussion from the 1850s to the 1930s.

[52] Edmond 2006: 7
[53] Pandya 2003.

The second part (Chapter 5-7) problematizes the underlying assumption that science was a coherent and global conversation throughout the period, and investigates the frameworks that made the circulation of medical knowledge about leprosy possible. In each chapter I discuss the specific mechanisms involved in the circulation of knowledge: The practice of appropriation, the role of conferences and medical journals, and the tradition of travelling and establishing organizations. Furthermore, these last three chapters each emphasize specific periods of time: 1850-1895, 1895-1915 and 1920-1933. Focusing on shorter time periods allows me go more in depth on the personal relationships between the actors involved. While I have put emphasis on what I believe to be the predominant traits of each period, the mechanisms are relevant also across the periodization: Appropriation did not come to an abrupt end in the 1890s, nor did physicians start travelling only in the 1920s.

FIGURE 1: ***Structure of the thesis.***



Diagnosis, the practice of recognizing disease, is the topic for **Chapter 2: Recognizing 'the leper'**. The chapter opens with Gerhard Armauer Hansen at the first international leprosy conference in Berlin in 1897 insisting that the dominant clinically based diagnostics ('if you look like you have leprosy, you are a leper'), was

wrong. If it was impossible to demonstrate the presence of the bacillus, the patient was not suffering from leprosy, Hansen argued. In the chapter I will discuss the dominant approaches to diagnosis and training, the ambition of developing mechanically objective tests that did not rely on specialist training, and what impact the bacillus had on the debates. Finally, the chapter will show how the leprosy bacillus came to play different roles in diagnostics in different parts of the world. Most importantly, at least from the perspective of those who had the disease, the bacillus made it possible to look like a 'leper' and be paroled as 'arrested'.

Next, what did medicine have to offer those suffering from the disease? In **Chapter 3: To care or to cure** I will discusses treatment of leprosy and the various attempts at developing a cure. The chapter opens with a clinical trial of tuberculin taking place in Bergen – with disastrous results. After investigating how the news of the trial circulated and was put in context with similar experiments elsewhere, I examine the drugs and treatment regimes that dominated the medical debates. What impact did the leprosy bacillus have on medical research into treatments? To what extent did the discussions on treatments reflect local practices? Finally, I will show how the ancient Indian medicine 'chaulmoogra' was reinvented from an ailment to a possible cure. From the 1920s, chaulmoogra became the center of a global controversy which split the leprologists into two opposing camps. The dominant position was still that the disease was incurable and should be met with segregation. The challenger saw leprosy as curable, thanks to chaulmoogra, and argued for voluntary treatments and that the era of wholesale segregation must come to an end.

Until the first decade of the 20th century, the physicians generally considered leprosy to be incurable, and with devastating consequences both for the individual and society at large. Leprosy was a threat, a problem that needed to be eliminated. But how was this to be achieved? This is the topic for **Chapter 4: The question of prevention**. The chapter opens with a widely publicized letter from an American visitor to Bergen in 1852, warning that Norwegian migrants were invading America with a 'Biblical disease'. After a discussion on quarantine, I will identify the three main strategies for prevention that dominated the medical debates. In the chapter I

will show how the bacillus increasingly was entangled with the already existing argument that the disease was contagious, and how the debates on prevention were primed at finding efficient ways to keep the 'lepers' away from the healthy. Norway was not the only country to launch a campaign against the disease, but came to have a special position as the only country where the intervention had been successful: The number of 'lepers' was declining. Still, the interpretations of the cause of this success differed. In the 1920s, both those who argued for continued segregation and those arguing for voluntary treatment pointed to Norway as a case in point.

Norway and colonial India came to represent two extremes when it came to leprosy policies. The first chapter in the second part investigates the roots of this divide, namely how the view that leprosy was contagious was introduced to the debates in the decades leading up to the international leprosy conferences. **Chapter 5: Appropriating contagion** opens with a British physician working in India visiting Bergen in 1873, asking whether the leprosy he saw in Norway was the same disease as leprosy in India. This was a necessary but not self-evident premise for knowledge from other places of the world being locally relevant. The account from the visit was the first time the observation of a bacillus was mentioned in print, but the impact of the discovery differed greatly. Focusing on the years surrounding the discovery of the leprosy bacillus, what were the similarities, contacts and differences between the leprosy debates in Norway and colonial India? I will show that the period between the 1850s and the 1890s was characterized by competing, contradictory and coexisting medical models of leprosy. Personal experiences outweighed observations made by others, appropriation was selective, and not everything that went on in a local setting was reported elsewhere.

The next chapter investigates the conscious attempts at organizing the circulation of knowledge, particularly the international leprosy conferences and the specialized medical journal *Lepra Bibliotheca Internationalis* (1900-1914). **Chapter 6: Connecting the world of leprosy** opens with an announcement by an American physician that an international leprosy congress was scheduled to be held in Bergen in 1897. The goal of the conference was to establish a supra-national committee of

experts tasked with dictating a global leprosy campaign. The American's scheme never succeeded, but it shows how gathering leprologists from all 'civilized countries' was on top of the agenda at the end of the 19th century. A common trait for those involved in connecting the world of leprosy was that they all accepted the leprosy bacillus as the cause of the disease, and believed leprosy to be contagious. In the chapter I will also discuss the role of conferences and medical journals in general, before finally showing how the practice of circulation in itself could result in the production of new knowledge.

After the Great War the shared medical journal was discontinued, and several of the most prominent actors had died. How leprosy ended up as an issue for the League of Nations, their activities, and the various initiatives that came together to form the Manila meeting in 1931 when the International Leprosy Association was founded is the topic of **Chapter 7: Interwar globalization**. The chapter opens with the arrival in Bergen of a Brazilian physician who sponsored by the Rockefeller Foundation went on a global fact-finding mission for two and a half years. The goal was to reorganize the leprosy campaign in his home country in light of experiences elsewhere. During the journey he came to the conclusion that leprologists around the world needed to organize and collaborate, and he founded an international organization to this end. Although his international leprosy society never had a single meeting, it laid the foundations for how the International Leprosy Association should be organized and who should be involved. Focusing on the practices of traveling and establishing organizations, as well as the activities of the League of Nations Leprosy Commission, I will show how several initiatives aimed at organizing leprosy on a global scale came together in the interwar period and led to initiatives aimed at global standardization of medical research.

The chapter and the thesis ends in 1933 and the first issue of the quarterly *International Journal of Leprosy*. For the next 73 years this would be the main global arena for publishing and discussing knowledge regarding leprosy. The journal was the last of the four pillars that made up the leprosy world order which would last for more than seven decades. Until its final issue in 2005, the journal was where the dominant

medical discussions took place and where the latest and most authoritative research was published. The journal was published by the organization that made up the second pillar: The International Leprosy Association (ILA). ILA was established at a meeting in Manila in 1931 and still organizes leprosy researchers globally. The third component was the League of Nations Leprosy Commission, which had its first meeting in Paris in 1928. After the Second World War, its activities were continued by the World Health Organization (WHO).[54] Like its successor, the Leprosy Commission's role was to monitor the global prevalence of the disease and support governments in their leprosy campaigns. Its experts were usually handpicked from the ILA. The fourth and final component was the International Leprosy Conferences, which still is the main meeting point for the world's leprologists. The first conference was arranged in Berlin in 1897, the 18th conference in the series was arranged in Brussels in Belgium in September 2013.

In this thesis I pursue three main arguments. First, science is not just clever ways to produce knowledge about the world in specific sites. Science is also a collective activity that goes beyond the local. The circulation of knowledge is in itself a scientific practice, and must be studied as such. Those participating in the circulation of knowledge got access to more experience than had they worked in isolation, knowledge that in turn could be translated into prestige and status as experts on a local scale. In this thesis I show how this framework was organized and how the dominant positions in three of the most central debates changed over time. Second, the science of leprosy had direct and practical applications. It was never possible to fully separate the disease from those affected by it, thus to do something with the disease meant doing something with those affected by it. Since not much could be done to cure those already affected, the interventions focused on prevention. Third, a study of the circulation of knowledge builds on and does not replace the need for geographically delimited studies. These provide insight both in local practices, perspectives of other actors such as missionaries and the persons affected by the

[54] Today, the leprosy work is organized by the WHO office for Control of Neglected Tropical Diseases. See:

disease, and constraints outside the control of the medical investigators such as economic and political conditions. Only fractions of what went on in one place were made part of the circulation of knowledge.

Sources: 'Backwards' and 'sideways'

My main strategy for finding relevant source material has been to work 'backwards' and 'sideways'. My starting point was the establishment of the International Leprosy Association in 1931 and their medical journal, and I then worked backwards from there. Since the League of Nations Leprosy Commission was heavily involved in deciding who was to attend the two-week founding meeting in Manila in 1931, this brought me to the League of Nations archives in Geneva.[55] The Leprosy Commission's main ambition was to gather the most recent scientific data concerning leprosy, bring experts together, and coordinate cooperation between leprosy researchers all over the world. I was allowed to bring a camera and created a digital archive of all material related to their leprosy activities.[56]

In addition to the origins and activities of the League's Leprosy Commission, which will be detailed in Chapter 7, the material from Geneva showed that the initiative for involving the League of Nations in leprosy work came from the organizers of the Third International Leprosy Conference in Strasbourg in 1923.

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http://www.who.int/lep/en/

[55] The recommendation on the website of ILA's Global Project on the History of Leprosy (http: //www.leprosyhistory.org) was also important in this choice. The project operated between 1998 and 2007 as collaboration between ILA, WHO and the Wellcome Trust Centre for the History of Medicine at Oxford. It was funded by the Japanese Nippon Foundation. During the work with my MA-thesis I met with research officer Jo Robertson who visited Bergen twice, and I have later met with Julia Sheppard, chair of the steering group, and corresponded with Professor Emeritus Michel Lechat, former President of the International Leprosy Association and member of the steering group. I have also met with John Porter (UN advisor on questions of leprosy), interviewed Douglas Soutar, the current General Secretary of the umbrella-organization ILEP which works to coordinate the work of anti-leprosy organizations, interviewed representatives from the organization LEPRA Health in Action. I have also met with Helen Bynum who in 1991 wrote her PhD on Leonard Rogers who in 1924 founded the *British Empire Leprosy Relief Organization* (BELRA), and author Tony Gould.

[56] I am especially grateful for the help and friendship offered by archivist Neycho Iltchev, who not only helped me find the sources I was after but also where to find the best coffee and how to order pizza from the lunchroom. For a travel report, see Vollset, Magnus. "Tilbake til verdens navle". *Historikeren.* No. 2, 2010: 36-40. Available online: http: //hifo.b.uib.no/2010/06/07/tilbake-til-verdens-navle/.

There the attendees pointed at the League as a potential partner for reestablishing the international medical journal *Lepra Bibliotheca Internationalis* (1900-1915). This was the main motivation behind involving the League of Nations in leprosy work, and an obvious next step in finding relevant sources.

Consequently, I spent three and a half months at the Wellcome Trust Center for the History of Medicine in London, both because of their renowned research community and access to the Wellcome Library, which is among the few libraries that still hold a complete set of this journal. I also spent a wonderful weekend visiting author Tony Gould in his home near Newton Abbot, and was allowed to help myself to the rich archives he had collected doing research for his book *Don't Fence me in. From curse to cure: Leprosy in modern times* (2005).[57] Inspired by Pandya's paper on the first International Leprosy Conference, I also visited the Ashmead Collection at the Historical Medical Library at The College of Physicians of Philadelphia, USA. Working with my MA-thesis, a social history of leprosy in Norway in the 19th century, I familiarized myself with the Leprosy Archives in Bergen, which in 2001 was accepted to the UNESCO Memory of the World programme.[58] The archival material I have used has mainly consisted of correspondence, reports and minutes of meetings.

Although circulation of knowledge is only possible to document post factum, when a reference is made to prior research, an obvious drawback of the strategy working 'backwards' is that it is easy to fall into the trap of teleology through basing the story only on the voices of those who ended up 'winning' the arguments. I therefore supplemented this by working 'sideways'. This strategy consisted of examining the concurrent debates the original texts were part of. The most important sources for this strategy have been medical journals, reports, conference proceedings

[57] Gould's book has been a valuable starting point for finding information about the actors involved in making leprosy a global disease, and has served as a constant reminder that leprosy and the responses to the disease has shaped the lives of people in numerous and dramatic ways.

[58] Vollset 2005; Memory of the World: The Leprosy Archives of Bergen. The nomination form is available on UNESCO's website: http: //www.unesco.org/new/en/communication-and-information/flagship-project-activities/memory-of-the-world/register/full-list-of-registered-heritage/registered-heritage-page-8/the-leprosy-

and medical textbooks.

The approach of working both 'backwards' and 'sideways' reflect the original dual purposes of these publications. On the one hand they were arenas for ongoing debates; on the other hand they created an archive for later reference. As Bruno Latour has pointed out, only what was considered relevant for later researchers was referred to. Further, the act of referring to previous research is not the same as making identical copies of the original. Rather "with every new retort added to the debate, the status of the original discovery (…) *will be modified*."[59] Through the dual strategy of working backwards and sideways, it is possible to show not only what was considered vital concurrently, but the breadth and scope of the debates, and how the dominant positions changed over time.

Thanks to projects such as *The Internet Archive*, and the digitalized archives of both medical journals and newspapers, much source material has been available online.[60] This has been invaluable for my research, especially for the period prior to 1900. But the digital revolution also has its pitfalls: There might very well be biases in the selection of what to digitalize that I am not aware of. This is obviously a problem with the historical records preserved in archives and libraries as well, but since the digitalized sources are only a selection of what has been preserved, the challenge is only greater. I therefore chose to supplement my research by visiting library holdings. In addition to the Wellcome Trust Library, which holds a large collection of relevant research literature, I have spent much time at the University of Bergen Library where much of the material from the library at the Lungegaarden leprosy research hospital is found. Still, the value of convenience in the digital revolution has, for me at least, outweighed the cost of traveling to even more libraries and archives. Although it probably would have been possible to conduct this study without relying on the scanned originals available online, it would have been more costly and time

archives-of-bergen/. The archives also has an internet exhibition with digitalized sources and links to previous research: http: //digitalarkivet.uib.no/lepra-eng/

[59] Latour, Bruno. *Science in Action.* 1987: 27. Italics in the original.

[60] http: //archive.org. For other internet resources I have found useful, see the list of "Online resources" at the end of the thesis.

consuming.

After identifying the sources and getting an overview of the content, the next step was making a narrower selection and composing stories based on this material. With the exception of the sections that discuss what went on behind the scenes in the League of Nations Leprosy Commission and in the run-up to the first international leprosy conference, I have given priority to the sources that were publicly available and part of concurrent discussions within the medical community. Apart from those directly involved behind the scenes, this is what actors elsewhere based their appropriation on. Accordingly, I have given priority to medical journals and conference proceedings.

I have chosen to tell my story primarily through medical researchers and their debates. Although the number of dominant actors was limited, both in the number of persons involved in organizing frameworks for circulation and in the number of persons frequently referred to, I have tried to balance their position with focusing also on those who most vocally represented positions that were later abandoned by the research community. As Fleck pointed out, ideas are generated and accepted during discussion and critique. Ignoring the 'blind alleys' would therefore give a misleading impression of how science is actually conducted. As Pandya demonstrated in her paper on the first international leprosy conference, and as I will expand upon in Chapter 6, the first leprosy conference and its outcomes would not have been the same had it not been for the competitive scheme with a different vision for how the fight against leprosy should be organized internationally.

The choice of focusing on the medical debates in this set of sources has unavoidably led to a geographical bias. The vast majority of those affected by the disease were encountered in the colonies by physicians from Europe working for the colonial authorities. In addition to diagnosing individuals and estimating prevalence in the population, they were involved in testing drugs and formulating policies. Until the outbreak of war in 1914, most of the meetings and discussions took place in Europe and North-America, and this was where those involved in organizing the circulation of medical knowledge lived. In the interwar period physicians from Latin-

America, South Africa, India and the Phillipines also stepped forward. The geographical bias of the medical community is reflected in my thesis. This is not to say that the disease did not exist in other parts of the world, or that those affected by it did not receive attention. But in Africa and China, for instance, leprosy work was to a large extent left to the missionaries who had their own frameworks for the circulation of knowledge. Had I chosen to focus on the missionaries, the geographical bias would have been different.[61]

There were some exchanges between missionaries and physicians both on a personal level and through publications. *Lepra Bibliotheca Internationalis* (1900-1914), for instance, printed abstracts of six papers from the Mission to Lepers in India and the East's quarterly journal *Without the Camp* (1897-1971). The founding head of the Mission to Lepers, Wellesley Bailey, was invited to the first leprosy conference, and did attend both the follow-up in Berlin in 1904 and the Second International Leprosy Conference in Bergen in 1909. The replies to the League of Nations calls for information on leprosy in 1925 found in the archives in Geneva indicate that missionaries were heavily involved in leprosy work also in areas where the Western physicians had yet to arrive. Roy Porter has argued that "Leprosy provided a prism for Christian thinking about disease."[62] Pursuing the various missionary societies involved in leprosy work would undoubtedly have been fruitful, for instance using a similar transnational strategy as the one I have developed for this thesis. The circulation of missionary or religious knowledge about leprosy did tangent the medical circulation of knowledge, but it mainly existed in parallel. This is a story I have not had the capacity to pursue. My study is limited to the medical community.

[61] For studies in this direction, see for instance: Worboys 2000; Joseph, D. George. ""Essentially Christian, eminently philanthropic": The Mission to Lepers in British India." *História, Ciências, Saúde-Manguinhos*. Suppl. 1. 2003: 247-275.
[62] Porter, Roy. *The Greatest Benefit to Mankind. A medical history of humanity*. 1997: 122.

'The leper', terminology and ethical considerations

In the thesis I use quotations extensively. Due to readability I have translated the Norwegian, German and French sources into English in the main text, but the original quotes are found in the footnotes. The words used are never neutral or objective signifiers, but expressions of the perspectives and situation of those who use them. How the actors presented their arguments changed both with time and setting, and this I believe is more readily expressed through direct than indirect quotations.

The quotations show how the medical discourse especially in the 20th century became increasingly specialized, but my choice is not without its drawbacks. I have consciously tried to explain the specific meanings of the medical terms used, but after working with this material for several years I have unavoidably adapted and internalized some of the terminology. Although my advisor and readers have been helpful in pointing out what needs further elaboration, I am certain some concepts should have been explained better. All mistakes are mine.

The use of the term 'leper' requires special attention. The term has been seen as derogatory, and several organizations have argued that refraining from using the word is an important step in getting rid of the stigma connected to the disease. The first advocate for abolishing the term was Stanley Stein, a patient at the US leprosy institution in Carville, Louisiana. From 1931 to 1934 he edited the bimonthly journal *The 66 Star,* forerunner to *The Star* (1941- present). In the patient-run journal, Stein argued that 'leprosy' has Biblical connotations to sin and impurity, and advocated that the disease instead should be called "Hansen's disease".[63] The argument that labels like 'leper' is a hurtful stereotype that keeps stigma alive, has later been promoted especially by the organization IDEA, established in 1994 with 20,000 members in five

[63] Stein, Stanley. *Alone no longer. The story of a man who refused to be one of the living dead.* 1963. While the connotations to 'leprosy' in the bible were important for missionaries justifying their activities, it is beyond doubt that 'Biblical leprosy' and 'leprosy – the disease' is not the same, but can be traced to when the Bible was translated from Hebrew to Greek. As M. J. de Mallac has put it in *Hansen's disease: The shared paradigm*: "That '*Tsara'ath*' or '*lepra*' is synonymous with Hansen's disease has a semantic basis only." (Mallac 1998: 14)

continents.[64] This position has many supporters.[65]

While I agree that using the term 'leper' about individuals affected by the disease today is derogatory, when studying the history of the disease I have reached a different conclusion. Terminology and categorization is central to the creation of knowledge, and going down the alley of re-labeling would inevitably lead to anachronism. In the period I study, the word 'leper' was used both as a metaphor, a sensationalist expression to incite fear, and an unchallenged medical term. This is part of what made it so powerful. When it comes to leprosy, Sander Gilman's term about images of disease, 'disease anthropomorphized', was sadly also inherent in the language.[66] I acknowledge that terminology is a sensitive issue, but for reasons of historical precision I will stick to the terminology used in the sources.[67]

A different but related ethical consideration is that the stigma itself for some can be said to be hereditary. Discovering that the family carried a dark secret, namely that her grandmother was affected by leprosy and spent her life at "The Leper Island" Spinaloga in Greece is the theme in Victoria Hislop's novel *The Island*. The book was celebrated as 'Newcomer of the Year' at the Galaxy British Book Awards in 2007, and has later topped various bestseller lists. Although individuals' referred to in my sources are deceased, having had ancestors who were affected by the disease can for some be seen as ~~a~~ reopening wounds that need healing. While the sources often give the full names of the individuals affected by the disease, I have chosen only give the initials of the family names, or avoid using names altogether. The only exception is when the individuals themselves actively sought attention in a public arena – then I

[64] See: http: //www.idealeprosydignity.org

[65] "By refraining from using the word 'leper', either historically or in modern times, we will be challenging the stigma, restoring the humanity of those who lived hundreds of years ago, and promoting the dignity of those individuals affected by this disease today." (Law, Anwei Skinsnes. "The last leprosy communities and the people who call them home." In: Roberts, Charlotte A., Mary E. Lewis, and K. Manchester (eds.): *The Past and Present of Leprosy. Archeological, historical, palaeopathological and clinical approaches*. BAR International series 1054. 2002: 7). US president Bill Clinton voiced a similar sentiment in his opening speech at the exhibition 'Quest for Dignity' in the United Nations October 30, 1999: "An important step in our efforts to ensure that all individuals are treated equally is to permanently strike the hurtful word 'leper' from our vocabulary." (http: //www.idealeprosydignity.org/OralHistoryWeb/Terminology.html)

[66] Gilman, Sander L. *Disease and Representation. Images of Illness from Madness to AIDS.* 1988.

have used the names they have signed with.[68]

Leprosy today

In May 1991, the World Health Assembly passed a resolution to eliminate leprosy as a global public health problem by the year 2000. At the time it was estimated that 5.5 million people suffered from leprosy globally.[69] Elimination was defined as reducing the prevalence rate to less than one case per 10.000 inhabitants, first on a global, then on a national and finally on a regional scale.

That leprosy is the same disease all over the world is a necessary premise for 'global prevalence' to be a meaningful concept. But that leprosy prevalence exists as a global unit still does not make the approaches to the disease identical on a local level. There are, for instance, obvious differences between being diagnosed with leprosy in Norway and in East Timor.[70]

Today medical experts agree that the causes of leprosy are the same all over the world, namely the bacillus first observed by Hansen in 1873, *Mycobacterium leprae*, or the related *Mycobacterium lepromatosis*, which was first identified in 2008 by a team headed by the American pathologist Xiang-Yang Han.[71] However, although the bacilli are necessary, they are not alone sufficient to produce the disease. The individual affected also needs a hereditary predisposition for the bacilli to cause the

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[67] This choice, I believe, is in line with Stanley Stein's own editorial advice in *The Star:* "We dislike the word 'leprosy' intensely, but we dislike the practice of censorship even more." ("Editorial Policy on Terminology." *The Star*, all issues, p. 2.)

[68] Stanley Stein, for instance, was a pseudonym he adapted after being sent to Carville in 1931.

[69] Nordeen, S.K., Bravo L. Lopwz and T. K. Sundaresan. "Estimated number of leprosy cases in the world." *Bulletin of the World Health Organization.* Leprosy Unit, World Health Organization. 1992: 7-10.

[70] The past 20 years, nineteen new cases of the disease has been identified in Norway. The patients have been absorbed by the local health systems. Rapp, Ole Magnus. "Fortsatt tilfeller av spedalskhet". *Aftenposten.* 25. June 2012. (Online: http: //www.aftenposten.no/nyheter/iriks/Fortsatt-tilfeller-av-spedalskhet-6907004.html). In poverty-stricken East Timor, with 160 new cases detected every year, the situation for those diagnosed is radically different. Mason, Margie. "Tiny East Timor Declares War On Leprosy". *Associated Press.* 10. October 2010. (Online: http: //www.huffingtonpost.com/2010/10/10/tiny-east-timor-declares-_n_757544.html).

[71] Han, Xiang-Yang, Y. H. Seo, K. C. Sizer, T. Schoberle, G. S. May, J. S. Spencer, W. Li, and R. G. Nair. "A new Mycobacterium species causing diffuse lepromatous leprosy". *American Journal of Clinical Pathology*. 2008: 856-64.

disease, and an estimated 95 percent of the human population is immune. Third, it seems a prolonged reduction in the immune system is necessary for the disease to develop. This can be caused by insufficient diet, poor living conditions or other diseases, and explains why the disease is often linked to poverty. The disease has a long incubation time, from months to decades. The genomes of the two leprosy bacilli have been mapped, but many questions of the precise mechanisms involved in the transmission and the early development of the disease remain unanswered.

From 1995 the WHO has provided free multidrug therapy (MDT) to all relevant countries through their Ministries of Health. Cooperation between international bodies and individual countries has been necessary in order to reach the global elimination target.[72] In November 1999, as the global elimination goal was within reach, the WHO took the initiative to establish Global Alliance for Elimination of Leprosy (GAEL). They soon adopted a "Final Push" strategy, moving from focusing at the global prevalence target to reaching this elimination target at a national level. The rationale was that reaching the targeted prevalence rate would make it possible to integrate leprosy treatment into the general health services.

Over the past 20 years, more than 14 million patients have been detected and cured using MDT. At the time of writing, pockets of high prevalence still remains in areas of Brazil, Indonesia, Philippines, Democratic Republic of Congo, India, Madagascar, Mozambique, Nepal, and the United Republic of Tanzania.[73] Throughout the past decade, the global leprosy prevalence has been in steady decline (see Table 1 on the following page). According to the WHO, there were 181.941 cases worldwide at the end of 2011.

[72] In October 2010, WHO and the pharmaceutical company Novartis signed an agreement to continue to provide free MDT free of charge to all countries where the disease is endemic until 2015. See: http://www.who.int/lep/resources/press_releases/en/index.html

[73] WHO. *Leprosy Fact Sheet*. Available online: http: //www.who.int/mediacentre/factsheets/fs101/en/index.html

Table 1. Detection of new cases of leprosy, 2001-2010 (excluding European Region)

2001	2002	2003	2004	2005	2006	2007	2008	2010
763,262	620,638	514,718	407,791	299,036	265,661	258,133	249,007	228,474

Sources: WHO. *Weekly epidemiological record*, No. 33, 2009, 84: 33-340; Nr. 32, 2006, 81: 309-316; http: //www.who.int/lep/situation/

The decline of leprosy prevalence has had consequences for the campaign against the disease. In 2005, the International Leprosy Association (ILA) decided to close down the *International Journal of Leprosy* after 73 years.[74] The organization LEPRA – Health in Action, established as British Empire Leprosy Relief Association (BELRA) in 1924 with the aim "to eradicate the disease altogether"[75], continues to publish the medical journal *Leprosy Review* in its 83rd volume. However, the organization itself is moving away from a single-disease focus into the wider area of health-related development work.

The fight against leprosy has been highly successful in its goal of reducing leprosy to "just another straightforward, curable disease".[76] However, the WHO leprosy elimination program has been, and still is, contested. The "Final Push"-strategy gave attention and resources to the fight against leprosy, but has also been met with fears that the disease will be neglected, including persons who have disabilities resulting from having had the disease. Declaring leprosy eliminated by reaching the 1:10,000 target, some fear, will lead to lack of competence or active efforts in detecting new cases, and that the prevalence consequently will rise in the "post-elimination" era.[77]

[74] Scollard, David. "Elimination of (the International Journal of) Leprosy." *International Journal of Leprosy.* Vol. 73, 2005: 303.
[75] Oldrieve, Frank. "The Work of the Association." *Leprosy Notes,* No. 1, 1928: 2.
[76] World Health Organization (WHO). 2003. *The Final Push Strategy to Eliminate Leprosy as a Public Health Problem. Questions and Answers, Second Edition.* World Health Organization, Geneva. Available online: http: //www.who.int/entity/lep/resources/Final_Push_%20QA.pdf
[77] See i.e.: Ji, Baohong. "Comments on WHO/AFRO's 'Post-Elimination' Strategy Paper: A New Bottle with Old Wine of the 'Final Push'". *International Journal of Leprosy*, vol. 73. 2005: 216-218; Khaitan, Binod K. et.

A transnational approach is never disconnected from the local context. Since I as a historian have been situated in Bergen, an emphasis on the role of the leprosy bacillus within these medical networks seems given. Had the thesis been written elsewhere, the starting point would probably have differed. In July 1998, for instance, 127 former leprosy patients filed a lawsuit in Kumamoto District Court against the Japanese government seeking compensation for being locked up for decades "under an unconstitutional quarantine policy".[78] Japan's Leprosy Prevention Law, enacted in 1907, set a standard for forced segregation policies which were abolished only in 1996.[79] On May 11th, 2001, the court ruled in favor of the plaintiffs, who received in total $15 million in compensations; pointing to international scientific recommendations which from the 1960 has favored outpatient treatment. The story was widely reported in media all over the world, and led to increased interest in the history of leprosy from scholars and general audiences alike. Had I been situated in Japan, this recent event would undoubtedly have influenced the research, both questions and strategies.

Likewise, had my starting point been in Belgium, Father Damien's departure for Hawaii in 1873 could have been a likely starting point.[80] In 1889 the news that the missionary had caught the disease and died caused headlines all over the world. And had I been situated in Ireland, Wellesley Bailey and the establishment of the Mission to Lepers in Dublin in 1874 would have been a likely point of departure.[81] By the 1920s the mission ran the majority of all leprosy institutions in India and had already expanded elsewhere in Asia. Studying how the missionaries established frameworks for circulation of knowledge would indeed be interesting, but that would have been a different project.

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al. "Final push of leprosy: It is prudent to pause before declaration!" *Indian Journal of Dermatology, Venereology and Leprology*. Vol 72, No. 2. 2006: 151-153. For more on this debate, see Gould 2005: 362-365.

[78] "State Blamed for Leprosy Misery." *Mainichi Daily News*. May 12, 2001. "Japan's Lepers Win Long-Awaited Compensation." *International Herald Tribune.* May 12, 2001.

[79] Ohtani, Fujio. *The walls Crumble. The Emancipation of Persons Affected by Hansen's Disease in Japan.* 1998.

[80] Lechat, Michel. "La Lèpre après Père Damien". *AMA.* No. 65, May-June 2010. http: //www.md.ucl.ac.be/ama-ucl/Lepre65.html.

This thesis is written in Bergen. Although I as a researcher have travelled to archives and met with colleagues at conferences elsewhere, opening with the unveiling of Hansen's bust in 1901 seemed more fitting. Alongside the Second International Leprosy conference in Bergen five years later, this was the pinnacle of Bergen as a renowned international center for medical research into the disease. As I will return to in Chapter 7, by the late 1920s, this was no longer the case.

[81] Davey, Cyril J. *Caring Comes First: Story of the Leprosy Mission.* 1987.

2. Recognizing 'the leper'

Four years before the bust of Gerhard Armauer Hansen was unveiled in Bergen, in the spring of 1897, Hansen received a message from a district physician asking him to investigate a man suspected of being affected by tubercular leprosy. After examining the man in his home, Hansen was convinced that the diagnosis was correct. But when he returned to the laboratory and failed to find the bacillus in the tissue samples he had taken, he was forced to reconsider. "I cut out nodules and examined them, but could not find any bacillus. Naturally I had to give up my diagnosis." A month and a half later he repeated the examination with the same result: "Again I did not find it [the bacillus], and I was obviously compelled to conclude: This is not leprosy."[82]

At the leprosy conference in Berlin later that year, Hansen argued that demonstrating the leprosy bacillus was *compulsory* in order to make the diagnosis, and that the presence of the bacillus should be given precedence over the clinical symptoms. For Hansen, the bacillus (or lack thereof) made it possible to look like a 'leper' and still be considered a non-leper.

Few historians have examined the impact of bacteriology on diagnosis. Still, comparing for instance Michael Worboys' investigation of the impact of bacteriology on disease theories and medical practices in Britain in the 19th century and Andrew Cunningham's study of how laboratory medicine transformed the identity of plague would indicate that a radical shift took place during the first decades of the 20th century. In *Spreading Germs* (2000), Worboys argues that: "Given doctor's confidence in their existing methods of diagnosis, bacteriological methods were

[82] "Ich schnitt Knotten heraus, konnte in diesen keine Leprabacillen finden und musste da natürlich meine Diagnose aufgeben. 1 ½ Monate später sah ich die Patientin wieder, schnitt aufs neue einen Knoten aus, härtete ihn in Alkohol und machte Schnitte, um erneut auf Bacillen zu suchen. Ich habe sie auch da nicht gefunden, muss also ganz bestimmt sagen: das ist keine Lepra." Hansen, Gerhard Armauer. "Inwieweit ist man berechtigt, den Leprabacillus als die Ursache der Krankheit anzunehmen?" (Discussion). *Mittheilungen und Verhandlungen der internationalen wissenschaftlichen Lepra-Conferenz zu Berlin im October 1897.* II. Abtheilung. 1897: 43-44. (Hereafter: '*Mittheilungen 1897*') See also: Neisser, Albert. "Inwieweit ist man berechtigt, den Leprabacillus als die Ursache der Krankheit anzunehmen?". *Mittheilungen 1897.* I. Abtheilung. 1897: 1-10; Looft, Carl. "Die anästhetischen Formen der Lepra". *Mittheilungen 1897.* II. Abtheilung. 1897: 99-101.

largely used to confirm clinical judgments or used in doubtful cases."[83] Worboys has later argued that there was indeed no bacteriological revolution between the 1870 and 1910, at least not in Britain. Instead, the period was characterized by richer uncertainties and possibilities regarding etiology and pathology, a growing repertoire of techniques and technologies, and new possibilities for analogical and analytical reasoning.[84] Cunningham, on the other hand, argues that the discovery of bacteria meant the introduction of single causes of diseases, and that laboratory medicine transformed the identity of disease. Clinical diagnosis could only *suspect*. "The only way a suspicion of plague can be confirmed or *established* is by bacteriological methods; in other words, by a laboratory."[85] Cunningham then goes on to argue that by accepting that a disease has a specific cause, we have all in essence become bacteriologists. "To oppose the claims of bacteriology is now not a rival view, nor an alternative view, nor even a dissident view. It is now a lunatic view."[86]

Identifying the disease and thereby deciding who was to be considered a 'leper', was the first step in dealing with leprosy. As Hansen's example in the discussion in Berlin shows, the results from diagnosing clinical signs and diagnosing in the laboratory were not necessarily overlapping. This chapter will investigate what diagnosis was based on prior to the bacillus, and to what extent Hansen's insistence that demonstrating the bacillus was compulsory for diagnosis was accepted by colleagues elsewhere. To what extent did the bacillus lead to global standards for diagnosis of leprosy? What new research on diagnosis did the bacillus inspire? What practical implications did the bacillus have when it came to recognizing the leper?

[83] Worboys 2000: 214.
[84] Worboys 2007: 20-42.
[85] Cunningham 2002: 209-244; quote on p. 213.
[86] Cunningham 2002: 239. For a related discussion on the assumed conflict that arose between the laboratory and the clinic, see: Sturdy, Steve, Morten Hammerborg, Rosemary Wall and Mirjam Stuij. "Bedside and Bench Revisited". *Social History of Medicine,* Vol. 24, issue 4, 2011: 739-812.

Backwards from the corpse

Establishing how to clinically recognize leprosy and separate it from other illnesses was the main ambition of Daniel Cornelius Danielssen and Carl Wilhelm Boeck's monograph *Om Spedalskhed* ('*On Leprosy*') published in 1847.[87] For the two Norwegian physicians, leprosy was a unique illness that slowly made its presence known. As they summarized their presentation of the diagnosis: "We hope that we in our description of leprosy have portrayed this as a distinctive disease which, fully developed, cannot be mistaken for any other."[88]

Leprosy manifest was the end point; the dead body. By opening the corpse, observing how the disease had affected the internal organs, the disease was no longer obscured. Only when observing the internal organs, the pathological changes could be contrasted with the complaints made while the sufferer was still alive. Death was thus the starting point for unraveling the mysteries of the disease. In the monograph, the disease was traced backwards from the corpse, via the symptoms to the early, non-specific signs. As I will expand upon in Chapter 5, in the majority of the cases the authors argued that the disease could even be traced further back in the lineage.

In *On Leprosy* the presentation of the disease was in chronological order, starting with the prodromes – the stage where the disease was lingering but had not yet made itself firmly known. Then the progress of the disease was presented both in general terms and with 70 detailed case stories from St. Jørgen's Hospital in Bergen observed by Danielssen, most of which ended in autopsies conducted between five and forty hours after death. The observations were supplemented by chemical analysis and microscopy. Emphasizing that the disease was the same in other places, there were also eleven 'foreign' cases, patients observed by Boeck in Varazze (6), Athens (2), Hamburg (1), Turin (1) and Rognac (1).

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[87] The monograph was published in Norwegian in 1847 with the title *Om Spedalskhed*, and the following year in a French translation with the title *Traité de la Spédalskhed ou Éléphantiasis des Grecs*. In 1855, the book won the Prix Monthyon from the Academy of Sciences in Paris, and in 1856 the findings were summarized in English under the title "On the Nature and Treatment of Leprosy", a series of eight papers by Erasmus Wilson in *The Lancet*. (Edmond 2006: 46.)

[88] Danielssen and Boeck 1847: 269.

Learning to recognize the disease as early as possible was a goal in itself. The logic was that in order to find a cure, treatment must start early. Finding a cure was the objective for the research hospital which was being built while the book was finalized (Lungegaardshospitalet). Although the reaction to medication given to the patients was documented as part of the progress of the disease, treatment as such was not an ambition of the work.

Danielssen and Boeck separated leprosy into two distinct forms based on their symptoms, the nodular ('Den knudede Form') and the anesthetic ('Den anæsthetiske Form'). The prodromes were identical: Fatigue, stiffness, sleepiness, occasional light chills, passing pain, lack of appetite and nausea sometimes leading to vomiting. More often than not, the patient would then develop symptoms from both forms, sometimes to the extent that neither stood out as the dominant. These sufferers would be classified as 'mixed'. The descriptions reveal how the act of producing the accounts demanded close observation over time and the patient explaining the sensations accompanying the visible changes in their bodies.

For the nodular form, the specific symptoms consisted of slightly elevated spots in the face and on arms and legs, scarlet to dark brown in color, sizes ranging from that of a coin to the palm of a hand. The spots were less sensitive to touch than the rest of the skin; they sometimes itched and were made more visible when exposed to extreme cold or extreme heat. After a few days, weeks or months the spots would disappear, only to reoccur at a later point in time. At some point, the spots would become permanent and swollen, usually first on the back of the hands and in the face, leading to the eyebrows falling out. Often the eyes were attacked leading to blindness. Over time, the spots would turn into nodules, accompanied by deep pain. While the nodules themselves were dry, the rest of the body would sweat, making the skin appear smeared and greasy. Next, spots would appear on the inside of the mouth and throat, leading to a distinct hoarse voice, trouble with breathing and sometimes fatal suffocation. The nodules would continue to grow and gain a brown color. Sometimes the nodules would burst with liquid floating out. The whole process could take years, but the disease could also enter more active periods, usually lasting up to two weeks,

producing fevers, a feeling of heaviness, headache, delirium, thirst, and high pulse. "The disease can now in few weeks produce the destruction that it in the chronic phase takes years to achieve."[89] While some patients would experience periods of apparent recovery, these were with but a few rare exceptions all passing stages – the disease would inevitably return. In the end leprosy would produce diarrhea and dramatic loss of body weight (marasmus). Finally, sometimes after decades of suffering, death would come as a relief.

The anesthetic form had a more chronic and covert development. Often the one affected would experience chills, making the whole body stiffen and cause "an unexplainable indisposition, sometimes forcing him to solitude".[90] This stage could last for months or years. Then, with no prior warning, blisters the size of walnuts or hens eggs would form on the arms and legs, filled with a yellow-green, sometimes milky, viscous (thick and sticky) fluid. After bursting and then healing like a normal wound, the affected areas would more often than not be less sensitive to touch than the rest of the skin. The phase of producing blisters could last up to five years. The next step would be intense and lasting pain, often located in deeper and previously unaffected parts of the body, producing hypersensitivity where even the slightest touch or movement would cause stabbing anguish. Then spots of anesthesia set in. The skin would go pale, dry and with no production of sweat, often leaving the skin tough and with no elasticity. Over time the anesthetic spots would spread to larger parts of the body, the face would take on a pale yellow color with hints of purple. Like the nodular form, the disease would attack the eyes causing blindness, and produce lesions inside the mouth and in the gums. As the disease progressed, the tissues and bones would begin a 'necrotic' phase, causing the attacked areas to be insensible to the extent that they could be cut without causing pain. Muscles in the face would stop holding the facial expressions up, leading the sick to drool; palms and feet would contract. The loss of sensibility would usually be combined with a deep

[89] "Sygdommen kan nu I faae Uger frembringe alle de Ödelæggelser, hvortil behöves Aar under den chroniske Gang." Danielssen and Boeck 1847: 152.

pain that felt like originating in the bones. This was in turn followed by strong fever, violent shakes, headache, and delirium, burning thirst, pressure in the stomach, frequent vomiting and an 'annihilating powerlessness'.

> Under too frequent and harsh suffering, years go by while the disease slowly and surely progresses. Anesthesia takes over the whole body, the breath finally fades, and one can with good reason claim that the body is dead long before the sick end their days. It is like all parts diminish, and life goes out unnoticed.[91]

The last part of the monograph was a colored atlas with 24 plates made by the Norwegian lithographer Johan Ludvig Losting, *Atlas Colorié de Spedalskhed (Elephantiasis des Grecs).* The plates depicted faces, eyes, body parts, magnified skin lesions and internal organs, set against a neutral white background. The images were realistic but slightly exaggerated, representing the archetypes of the disease as it appeared in different parts of the body. Frequent references in the text make it clear that the atlas was to serve as a visual guide to the disease, exemplifying what to look for when making a diagnosis. In Lorraine Daston and Peter Galison's vocabulary, the atlas can undoubtedly be classified as 'truth-to-nature'; idealized, selecting the essential and leaving out the accidental.[92] Again, the atlas played into making the monograph decidedly useful in making the diagnosis. It equipped the physician facing a patient in different stages of the disease not only the tools to decide if it really was leprosy or not, it also gave insights into the probable case history, how the patient was likely to describe his or her symptoms, and predictions on what would happen next.

The usefulness of *On Leprosy* would ensure its status as a standard reference work on diagnosis for more than half a century. Apart from less emphasis on communicating with the sufferer, not much would change in clinical diagnosis until

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[90] "(…) sætte den Syge I et for ham uforklarligt Ildebefindende, der undertiden tvinger ham til at söge Eensomhed." Danielssen and Boeck 1847: 204.
[91] "Under alt for hyppige og haarde Lidelser hengaae mangfoldige Aar, hvori Sygdommen gaaer sin sikkre Gang fremad. Anæsthesien indtager hele Legemet, Aanden slöves omsider, og man kan med god Grund sige, at Legemet er dödt længe förend den Syge ender sine Dage. Det er ligesom om alle Dele svinde hen og Livet udslukkes fast umærkeligt." Danielssen and Boeck 1847: 213.
[92] Daston and Galison 2007: Chapter 2.

the advent of the leprosy bacillus.[93] In short, leprosy was a horrible disease identified through clinical diagnosis. It was the appearances and progression associated with leprosy that decided if you had the disease or not.[94]

Of pathogens and expertise

When Gerhard Armauer Hansen and Carl Looft wrote the next major textbook on leprosy in Bergen almost half a century later, leprosy was no longer described as a disease slowly overtaking the healthy body causing suffering and pain, but as the effect of the leprosy bacillus acting on the body. In *Die Lepra vom klinischen und pathologish-anatomischen Standpunkte* (1894), the two Norwegian physicians described the disease not as a mystery to be unraveled in collaboration with the sufferer, but as an object to be discussed among experts.[95]

Hansen and Looft also separated the disease into two main forms, but used a different nomenclature than Danielssen and Boeck. The form producing nodules they named *Lepra tuberose* (*Lepra tuberculosa*), while skin reactions and nerve affections were the distinguishing features of *Lepra maculo-anæsthetica*. What bound the two forms together was not that they could appear as 'mixed' forms in the same patient, but that both forms were caused by the presence of the leprosy bacillus: "We must

[93] The feeling and localization of pain was not something that could be observed, but relied on what the patient could tell about his or her experience. For Danielssen, the patient was not (only) an object containing disease, but a partner in unraveling its mysteries. This emphasis on the patients' experiences was also reflected in Danielssen's tuberculin trials, which I will investigate in Chapter 3.

[94] Danielssen and Boeck's monograph was but one of several descriptions of the disease. Most accounts divided the disease into several categories based on its clinical signs. In 1852 Dr. W. S. Oke at the Royal South Hants Hospital in Southhampton, for instance, reported in *The British Medical Journal* that the disease had "three principal species – the albida, nigricans and candida." Albida was "characterized by elevated patches of squamous matter and of a dull white colour", candida by its "bright whiteness […] the denomination has a complete agreement with that emphatically given to it in several parts of the Old Testament, such as 'leprous as snow'." The last form was not found in Britain and was therefore not described. According to Oke, the disease caused "considerable local irritation; but although it continues for a long time, [leprosy] does not produce any great amount of constitutional disturbance." (Oke, W. S. "Notes on the Treatment of Curable Diseases". *The British Medical Journal.* 1852: 138.)

[95] The book was translated to English by Norman Walker from Edinburgh in 1895 and given the title *Leprosy: In its Clinical & Pathological Aspects.* Two years later Hansen and Looft authors published a Norwegian

regard them as the same disease, only with varied intensity in the action of the bacilli."[96] Again, the descriptions reveal what activities went into producing the accounts: Observation over time was still indispensable, but the experiences of the patients were supplanted by what could only be observed either by the naked eye or through a microscope. Leprosy was becoming an object, not a subject.

Lepra tuberose was characterized by massive amounts of bacilli producing oblong nodules, from one to two millimeters in diameter; first of red color, later yellow. The nodules initially appeared on the backs of hands, on top of the wrists and in the face. Eyebrows, eyelids, eyes and the soft parts of the nose were especially prone to attack. This, they speculated, was probably due to being exposed to climate, and possibly connected with peculiarities in the structures of the tissues making them more fertile to the bacilli.[97] Next, nodules would appear near the joints of the limbs before spreading to other parts of the body. In addition, the lymphatic glands were always swollen.

This nodular form always advanced through outbreaks or eruptions accompanied by fever, repeating at longer or shorter intervals. For some the eruptions lasted only a few days with almost unnoticeable rise in body temperatures and almost no growth of the nodules. For others the fever could reach 40° C, last for weeks or months and lead to dramatic development of new nodules. Stronger and more frequent eruptions meant a quicker course and a worse outlook. Whether the individual differences were caused by the virulence of the bacilli or the structural conditions of the individual was impossible to say, but either way the consequence was that the fate of each patient could differ greatly. Intermittently all affections could spontaneously disappear and the patient would heal, but in general the life of the patient would end after eight or nine years. Death was not a direct effect of the

edition: *Lepra (spedalskhed): klinisk og pathologisk-anatomisk fremstillet* (1897). All quotes are all from the English translation.

[96] Hansen and Looft 1895: 52. Here, the discovery of the bacillus was dated to 1871 and all observations made before this point were labeled 'pre-bacillary era'.

[97] Climate was also put forward as a possible explanation for which form of the disease a patient developed. Geographically the nodular form dominated along the moist coast of western Norway, while maculo-anæsthetic

bacillus proper, but a consequence of the internal organs degenerating under fatigue caused by ulceration (the inflammatory response). Most patients suffering from Lepra tuberose died before the disease had run its full course.

The maculo-anæsthetic form of leprosy was more chronic and could last ten, twenty or even forty years or more. Its distinguishing feature was that the bacilli were fewer in number and attacked mainly the nerves and skin causing anesthesia and spots. The marks were observed first in the face, on the back and on the extremities; shapes and sizes varied, the colors ranged from bluish red to yellowish or brown and were made more visible with changes of temperature or by friction. Some spots were gone in days, others could last for years. If the macula (the discolored spots) remained, the affected skin would become thin, shiny, slightly scaly, and hinder the secretion of sweat. Usually the spots only affected the surface of the skin, but it could sometimes affect the tissue as deep as the bone. Initially the spots would be hypersensitive, but over time anesthesia (the loss of sensation) set in. The anesthesia would often lead the patients to accidentally injure themselves, and the resulting wound was prone to infection. Accompanying the spots were painful, tender and stiff joints that often appeared swollen. Often the face was attacked by anesthesia, causing blindness as the eyelids could not be closed due to paralysis. In most cases, however, the disease would at some point cease to progress.

> Thus the specific leprous affections gradually disappear, and only their results remain – in other words, *the leprosy is healed.* Most maculo-anæsthetic patients become in time purely anæsthetic; they no longer suffer from leprosy, *but only from its results.*[98]

This was the greatest implication of Hansen and Looft's bacteriological disease model: Leprosy was now a disease you could get well from. While clinical diagnosis meant that if you looked like a leper you had the disease, bacteriological diagnosis

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cases were more frequent in the dry climate in the east of the country and in the more sheltered villages along the western fjords.

[98] Hansen and Looft 1895: 67. Italics in the original.

meant that you could have *had* leprosy some point in the past and now suffered from the results of the disease – not the disease proper.

On the other hand, Hansen and Looft were quick to point out that the disease could reappear at a later point in time, indicating that the bacilli could remain hidden inside the body. And even if the disease never returned the results of having had the disease were devastating, possibly a fate worse than death:

> We have occasionally a complete subject with vigour and good health, but usually only a miserable rudiment of a human being, with more or less paralysed and deformed hands and feet, with unclosable eyes, of which the lower part of the cornea is opaque, and from which the tears run down over the cheeks, and with paralysed facial muscles unable to close the mouth, so that the saliva constantly dribbles from it. Such cases may, however, live long and reach great ages, if under such circumstances this can be looked upon as any advantage. They die usually from some intercurrent disease.[99]

Determining the exact genesis of the disease, Hansen and Looft considered impossible. This would rely on the patients' own observations. Unlike Danielssen and Boeck, Hansen and Looft found the patients not to be trusted, since "the patients either observe themselves insufficiently, as may frequently be noted, or they conceal many facts".[100] When Danielssen and Boeck saw leprosy as a disease that slowly took over the whole body, the experiences of the person suffering from the disease were relevant in uncovering its mysteries. In comparison, Hansen and Looft perceived the observations made by the sufferers to be of little interest. Consequently, *Leprosy in its Clinical & Pathological Aspects* contained no detailed patient stories. The purpose of

[99] Hansen and Looft 1895: 85.

[100] "If at the commencement only the extremities are affected the patients may conceal their condition for years, and through this concealment become so accustomed to lie, that later it is impossible to receive from them correct information." (Hansen and Looft 1895: 17). That lepers were considered untrustworthy was also reflected in their discussion on surgical removal of necrotic (dead) tissue as part of the treatment. "In such operations it is a frequent experience that the bone is reached before the patient feels anything, but he immediately feels pain when the periosteum [the membrane covering the bone] is scraped or the bone attacked with forceps or saw. We believe, however, that it is only nervous individuals who complain of *pain;* though it is certain that when the bone is meddled with, something is *felt.* Probably in this connection may be explained by the statement of the patients, that when walking they *feel* the ground." (Hansen and Looft 1895: 64, italics in the original.)

the book was to uncover the underlying mechanisms governing the development of the disease. With the disease being juxtaposed with the bacillus, the experience of having the disease was simply not relevant. The cause of the disease, the bacillus, was an object found *within* the sick body. Exposing the true nature of the disease required special expertise, not merely the experience of being host to the bacillus.

Expertise meant accuracy and impartial observation. In the text, with but one exception, analogies to other objects were replaced by metric measurements.[101] The use of illustrations further underline the authors' emphasis on the pathogen rather than clinical signs: In addition to five photographs in the English edition, selected not to highlight the typical but to produce unedited and accurate accounts of the disease, were eight plates containing forty chromo-lithographs (colored prints) based on microscopy, the position of the bacilli in the tissue and the cells. Instead of Danielssen and Boeck's idealized highlighting of the typical, this was 'nature speaking for itself' in all its complexity. In the terminology of Daston and Galison, the scientific ethos was governed not by 'truth-to-nature' but by 'mechanical objectivity'.[102] The major discussions revolved around where in the body the bacillus was to be found, commenting observations made by other experts. Did the bacilli reside inside or between the cells? What was the nature and role of the characteristic 'globi', the brown collections of what appeared to be dead bacilli broken down into granules (small compact particles)? These were questions for experts familiar with ongoing debates, not an introduction to the disease for laymen – and definitively not a reflection of the experiences of those being hosts to the bacillus.

Compared to Danielssen and Boeck, Hansen and Looft presented a new and different way of knowing leprosy. The patients were still 'lepers', suffering from the

[101] The only exception not using actual measurements was a reference to the French dermatologist Henri Leloir's description of the ulcerating blisters that maculo-anæsthetic cases sometimes developed: "They vary in size – they may be small, from the size of a pea to that of a bean, or as large as the palm of the hand." Hansen and Looft 1895: 59. Leloir's study, *Traité pratique et théoretique de la lèpre* (1886), was based on direct study of 900 cases, many of them observed during a prolonged stay in Norway in 1884. See: Beeson, B. Barker. "Henri Leloir, M. D." *Archives of Dermatology and Syphilology*. Vol, 23, No. 3. 1931: 532-533; Sandmo, Sigurd. "Portretter av en sykdom. Fotohistoriske perspektiver på det norske lepraarbeidet". In: Irgens, Lorentz M, Yngve Nedrebø, Sigurd Sandmo and Arne Skivenes. *Lepra.* 2006: 51-64.
[102] Daston and Galison 2007: Chapter 3.

same disease, but how they were to be studied, and what made the observations interesting, differed. When what the experts see as relevant observations to produce knowledge change, so do the activities necessary to produce the observations. As John V. Pickstone has argued, each way of knowing is associated with a way of working.[103] For Hansen and Looft recognizing leprosy was no longer primarily a question of looking for clinical signs, asking the patients about their experiences and family background, or uncovering patterns in how the disease progressed over time, but a matter of gathering tissue samples, mixing them with chemicals and observe them through a microscope. For leprosy, as the opening example from Hansen illustrates, the two approaches could ultimately lead to different conclusions in deciding if a person was in fact a 'leper'.

New ways of knowing are not like Kuhninan paradigms, replacing the old. Instead they add to the repertoire of possible ways of knowing.[104] This was also the case for leprosy. Hansen and Looft's study did not replace clinical diagnosis, it was an addition. Despite the emphasis on the pathogen, the first act in making a diagnosis was based on recognizing the characteristic clinical signs, especially nodules, local anesthesia and swollen lymph glands. Despite presenting the bacillus as the ultimate cause of the disease, actually detecting it was a backup. If in doubt, "a piece of skin may be removed and examined for the presence of bacilli, which, at least in the nodular form of the disease, are never absent."[105] Compared to *On Leprosy* (1847), which aimed at teaching others to recognize the disease,[106] Hansen and Looft's book was not as practically useful in making a diagnosis. Hence, Danielssen and Boeck's monograph would remain the celebrated go-to reference for clinical diagnosis also after the publication of Hansen and Looft's textbook.

[103] Pickstone 2011: 235-245.
[104] Op. cit.; Pickstone 2001; Pickstone 1993: 433-458.
[105] Hansen and Looft 1895: 13-14.
[106] The same can be said about Leloir's richly illustrated monograph from 1886, which contained 12 photographs of patients and 10 colored prints of dermatological details. For an analysis of the use of photography in leprosy research, see: Sandmo 2006.

A nose for the ultimate proof

At the international leprosy conference in Berlin in 1897, the bacillus was one of the highlights. In the widely published report of the conference the bacillus was the first topic that was addressed: "As might be expected, a considerable portion of the discussion has related to the bacillus Leprae, which the Conference accepts as the Virus of Leprosy, and which for upwards of 25 years has been known to the scientific world through the important discovery of Hansen and the able investigations of Neisser."[107] What did the bacillus entail when it came to diagnosis?

The claim that the leprosy bacterium should impact diagnosis was first made by the German physician Albert Neisser in 1884 in his chapter on chronic infectious skin-diseases in the 16th volume of Hugo Wilhelm von Ziemssen's edited series *Handbuch der speciellen Pathologie und Therapie.*[108] Drawing on Danielssen and Boeck's distinction between tubercular and anesthetic forms, Neisser stressed that diagnosis primarily meant looking for clinical signs. The role of the bacillus was to confirm the clinical diagnosis: Instead of having to wait (sometimes for years) to see if the disease continued the path towards overtaking the body, a simple detection of the bacillus was enough to remove all doubt. The bacillus was thus not only the cause of the disease, but the ultimate proof that the disease was present.

In tubercular cases, finding the bacillus in samples from the affected areas was relatively straightforward, Neisser withheld. "The diagnosis presents no difficulty in the tubercular form. But it is the demonstration of bacilli which facilitates the diagnosis of tubercular leprosy in the same way as it places each individual case beyond doubt."[109] Diagnosing the anesthetic variety was more complicated: The bacilli were fewer, and only found in the nerves and not in the surface on the skin. Therefore, diagnosis had to be made based on clinical signs alone:

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[107] "Report of the general conclusions". *Mittheilungen 1897.* II. Abtheilung. 1897: 190-192. The full text of the reports and resolutions from the international leprosy conferences are found in Appendix 2.
[108] The volume on *Hautkrankheiten* was translated to English by William Wood & Co in 1885, and published in the United States as *Handbook of Diseases of the Skin*. All quotes are from the English translation.

> An accurate knowledge of the symptomatology is all the more requisite for these cases because the criterion valid for the tubercular form, the presence of the bacilli, cannot be utilized during life, of course, owing to the localization of the leprous process in the peripheral nerves.[110]

At the conference in Berlin thirteen years later most physicians limited the importance of the bacillus to confirming the symptom-based diagnosis in early or doubtful cases. As for instance the Hungarian dermatologist Moritz Kaposi, professor at the dermatological clinic at the University in Vienna, argued: Revealing the presence of the bacillus was an ideal, but in practice not always possible. While Hansen argued that without the bacillus there was no leprosy, Kaposi argued that if it was not possible to detect the bacillus, clinical symptoms, especially local anesthesia, should take priority.[111] Judged by the discussions published in the proceedings of the conference, published less than three months later, this was the majority view.

The main problem with basing diagnosis on clinical symptoms was that it relied on judgment, and the judgement could differ. When American physician J. G. McDougal presented two sisters at the Ohio State Medical Society in 1895 and asked for help in making the diagnosis, for instance, the colleagues only hesitantly agreed the sisters were suffering from leprosy.[112] Some saw the clinical symptoms and progress of the disease as a textbook example of leprosy, others pointed at the lack of nodules and that the progress, color and location of the symptoms were strange. The legs and hands were most severly affected, and several of the digits had been utterly destroyed. The anesthetic patches were black. Although the disease appeared to progress in stages, the fevers were not as disabling as usually the case with leprosy. According to McDougal "Even though they have not true leprosy, they suffer from

[109] Neisser 1885: 327.
[110] Neisser 1885: 327.
[111] Kaposi, M. "Allgemeine Bemerkungen". *Mittheilungen 1897.* I. Abtheilung. 1897: 182-183.
[112] McDougal, J. G. "Is this leprosy?" *Transactions of the Semi-centennial meeting of the Ohio State Medical Society held at Columbus, May 15, 16 and 17, 1895.* 1895: 232-259. The conclusion of the debate is found on page 25. The presentation and discussion, including a photograph, was reprinted in *Journal of the American Medical Association* on November 2, 1895: 762-765.

some disease so nearly like it in all its apparel of symptom as to afford me ample apology for choosing the inquiry I did as the subject of my paper."[113]

Further complicating the matter, the bacterial diagnosis pointed in a different direction. Some months before the presentation, the left hand of the elder sister was amputated and sent to the Military Health Service in Washington for bacteriological examination. Surgeon M. J. Rosenau who conducted the bacteriological work could not find a single bacillus and therefore concluded that the two were suffering from syphilis.[114] This, in turn, was countered by the argument that since the two sisters probably suffered from *anesthetic* leprosy, detecting the bacillus was not always possible and the lack of bacteriological evidence was irrelevant.[115]

After the proceedings were published, the discussion continued in the columns of the *Journal of the American Medical Association*. New York-based physician Albert S. Ashmead insisted that the sisters were probably syphilitic, pointing at the unusual clinical signs, the missing leprosy bacillus and the fact that although the father had experienced similar symptoms the mother was not affected.[116] Physician D. C. Newman, however, argued that no two cases of leprosy were exactly alike, but that the two sisters were 'close enough' to the textbook examples for a definitive clinical diagnosis. "I am sure that if Dr. McDougal's two patients were to go before any

[113] McDougal 1895: 239.

[114] "All the sections were stained for lepra bacilli, with negative results. Those sections containing giant cells were also stained for tubercle bacilli. None were found. The disease, therefore, in my opinion is neither leprosy nor tuberculosis, which, by exclusion, throws some weight upon the suspicion of syphilis." Letter from Assistant Surgeon M. J. Rosenau, dated Washington, D. C, March 28, 1895. (Op. cit: 241.)

[115] As physician J. B. Keber, who attended the meeting, put it: "I should be inclined to doubt the accuracy of the diagnosis of tubercular leprosy if the diagnosis could not be confirmed by the discovery of the bacillus, but I personally would not feel it necessary to modify my diagnosis of anesthetic leprosy one iota if a bacteriological investigation failed to demonstrate the characteristic bacteria. That is the rule and not the exception. The clinical symptoms are amply sufficient for accurate diagnosis." (Op. cit: 258-259.) When J. G. McDougal again presented the cases at the Section of Cutaneous Medicine and Surgery of the Fiftieth Annual Meeting of the American Medical Association in Ohio in 1899, he had reached the same conclusion: "The fact that on examination no lepra bacilla were found in the amputated hand can not mitigate as has been said, against the diagnosis of anesthetic leprosy, because in this form of the disease their presence can but exceptionally be demonstrated." (McDougal, J. G. "Some Questions Relative to the Diagnosis of Anesthetic Leprosy". *The Journal of the American Medical Association*, January 27, 1900: 211.)

[116] Ashmead, Albert S. "The Ohio Cases not Leprosy". *The Journal of the American Medical Association*, November 16, 1895: 872.

medical examining board at Honolulu, they would be sent to Molokai without a dissenting voice."[117]

In the columns of the medical journal *Lepra Bibliotheca Internationalis*, papers on diagnosis made up about a sixth of the 833 items published throughout the lifetime of the journal (1900-1915). In general, clinical judgment took precedence over laboratory findings.[118] The case-studies typically began with describing the clinical symptoms in the patient, often combined with the history of the patient's travels and speculations on where he or she could have caught the disease. The bacillus was often highlighted as the decisive argument for settling the diagnosis, but usually mentioned only after the clinical symptoms were exposed.[119] In cases where the bacillus was not found, this was emphasized as an abnormality but I have found no cases where this had any impact on the diagnostic verdict.[120]

In principle, demonstrating the bacillus was an increasingly easy task. New staining techniques were continuously being developed, making the bacteriological examination both faster and more precise. In addition to being demonstrated at conferences, the methods were frequently referred to in both textbooks and medical

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[117] Newman, D. C. "Was It Leprosy?" *The Journal of the American medical Association.* November 30, 1895: 965. From the late 1860s Molokai was used as Hawaii's main leper colony. More on this in Chapter 4. See also: Tayman, John. *The Colony: The Harrowing True Story of the Exiles of Molokai.* 2006.

[118] The journal referred to 64 studies on diagnosis, in addition to 69 case studies which put more emphasis on the patient's prehistory in an attempt to find out where and when they caught the contagion but also described the symptoms of the disease. There were also abstracts of sixteen studies which described smaller groups of patients, ranging from two to forty cases. For more on *Lepra Bibliotheca Internationalis* and the role of medical journals, see Chapter 6.

[119] [119] See i.e: Fabio, G. "Un caso di lepra tubercolare: herpes zoster in un lebroso". *Lepra Bibliotheca Internationalis*. 1903: Fasc. 3, originally published in: *Gazzetta Medica Italiana* 1902, No. 16; Dekeyser, L. "Cas de lèpre anesthésique mutilante", and Asselberg "Cas de lèpre maculeuse anesthésique".*Lepra Bibliotheca Internationalis*. 1903: Fasc. 3, reprinted from *Bulletin de la Société Belge de Dermatologie et de Syphiligraphie*. March 1903, and *Journal des Maladies Cutanées et Syphilitiques*, October 1903: 743; Matagne. "La lèpre à Bruxelles" *Lepra Bibliotheca Internationalis*. 1904: Fasc. 4, originally published in *Annales de la Sociéte Scientifique de Bruxelles.* 1904; Grön, Kristian. "Leprafälle in Christiania in 1903". *Lepra Bibliotheca Internationalis*. 1905: Fasc 1, originally presented as a lecture in Christiania Medical Association, 18. Mai 1904; Beurmann and Gougerot "Troubles sensitifs des lépromes". *Lepra Bibliotheca Internationalis*, 1907: Fasc. 4, originally presented as a lecture in Société francaise de dermatologie, 7. December 1907.

[120] See for instance Poirier's presentation of two leprosy cases diagnosed in Antwerp in 1908. Poirier. "Deux cas de la lèpre". *Lepra Bibliotheca Internationalis*, 1908: Fasc. 4, originally published in: *Societé Belge de dermatologie et de syphiligraphie*. 1908, No. 3.

journals.[121] But staining was not the only laboratory technique necessary in order to find the bacillus. At the First International Leprosy Conference in Berlin in 1897, the American bacteriologist Luis F. Alvarez, in charge of leprosy research in Hawaii, argued that especially in the early stages of the disease, the elusive bacillus could hide *inside* the tissue samples. Failure to detect the bacillus thus only proved that the physicians sometimes were unlucky when selecting what piece of the body to look at under the microscope. The solution, Alvarez argued, was a new technique for preparing the sample prior to staining. This consisted of washing the tissue in saline solution (and if necessary boiling it), and then grinding it in a mortar before staining. This would ensure that no bacilli avoided detection by hiding inside the sample. This was not the only solution to the problem, but unlike its alternatives, it was fast and required no special equipment, like a centrifugal machine.[122] In the following decades, varieties of Alvarez' method were adapted in several places, for instance Calcutta.[123]

Grinding, boiling and staining did not assist in bacteriological diagnosis if the physician chose a sample which contained no bacilli in the first place. Another hot topic at the conference in Berlin was *where* in the body the bacillus was to be found. In addition to skin snippets of leprous lesions, especially in the tubercular cases, the nasal mucus, sputum and nasal excretion were long considered the most promising sites for early detection. In *Die Lepra Auf Madeira* (1891) the German physician and medical superintendent at the Lazaretto Hospital in Funchal, Julius Goldschmidt, argued that he had found the bacillus in the nasal cavities in several of his patients, and suggested that this might be the primary lesion of the disease. Leading up to the conference in Berlin, several studies confirmed that the bacillus was detected in the

[121] See i.e. Fick, James. "Zur Färbung der Leprabazillen in dünnen Gewebsschnitten". *Lepra Bibliotheca Internationalis,* 1907: Fasc. 4, originally published in: *St.Petersburger Medicinische Wochenschrift*. 1907, Nr. 27; Unna, Paul Gerson. "Über eine neue Doppel-färbung normaler und abgestorbener Bacillen im Lepragewebe". *Lepra Bibliotheca Internationalis,* 1907: Fasc. 4, originally published in: *Monatsschrift für Praktische Dermatologie*. 15. February 1907.
[122] Alvarez, Luis F. "A New Method of Bacteriological Diagnosis of Leprosy". *Mittheilungen 1897.* II. Abtheilung. 1897: 123-124.
[123] For a slightly updated modification of the procedure, see: Rogers, Sir Leonard and Ernest Muir. *Leprosy.* [1925] 1940: 210-213.

patients' nasal secretion.[124] At the conference proper, the French physician Edouard Jeanselme argued that the nasal membranes were the best spot to examine in order to make early diagnosis, and in addition to being the initial lesion of the disease this might be a pathway for contagion.[125] In the concluding report from the conference, examination of the tissue and excreta originating inside the nose was highlighted as a promising avenue for further research.[126] In Norway, it inspired Hansen to organize a collection of nasal excretions from family members of home-living patients, not necessarily to develop an 'early warning system', but with the hope that this could bring light on the initial lesion of the disease.[127] In several leprosy institutions, the physicians began systematic testing of the noses of the patients. The results from the nasal test did, however, differ greatly. While Georg Sticker found the bacillus in 127 of 153 cases in Germany (83%),[128] Wilhelm Kolle detected the bacillus in 88 of 137 cases in South Africa (64%).[129] At the Pleiestiftelsen leprosy asylum in Bergen, Norway, H. P. Lie discovered the bacillus in only 35% of the patients' nasal mucus.[130]

In the medical textbooks published after the Berlin conference in 1897, the leprosy bacillus was elevated as the decisive cause of the disease.[131] There were also

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[124] For details, and also an overview of studies aimed at detecting the bacillus in saliva, urine, faeces, sweat and lachrymal (eye) secretions, see: Hollmann, Harry T. "The presence of acid-fast bacilli in secretions and excretions of lepers." *Public Health Bulletin.* No. 61. 1913: 15-17. For a list of 30 studies examining the presence of the leprosy bacillus in the nose, see: Hollmann, Harry T. "A statistical study of the nasal lesions in leprosy". *Public Health Bulletin* No. 50. 1911: 21-23

[125] Jeanselme, E. and Laurens. "Des localisations de la lèpre sur le nez, la gorge et le larynx." *Mittheilungen 1897.* II. Abtheilung. 1897: 18-48. The argument was backed by 31 cases.

[126] 'Report of the general conclusions'. *Mittheilungen 1897.* II. Abtheilung. 1897: 190-192. See: Appendix 2.

[127] Hansen, G. Armauer. "Nogle leprasprgsmaal". *Medicinsk Revue,* 1897: 324-325. A year later, having received only two replies, he repeated the request. Hansen, G. Armauer. "Spedalskhedens overførelse". *Medicinsk Revue.* 1898: 297. The project was later abandoned.

[128] Sticker, G. "Untersuchen über die Lepra." *Arbeiten aus dem Kaiserliche Gesundheits-Amte*, Band 16, 1899: 38.

[129] Kolle, W. "Mittheilungen über Lepra nach Beobachtungen in Südafrica." *Deutsche Medizinische Wochenschrift*, 1899: 647.

[130] Lie, H. P. *Beretning fra Plejestiftelsen for spedalske No. 1 i Bergen for årene 1895 til 1898*. 1899. Referred to in *Norsk Magazin for Lægevidenskaben*. 1899: 1217 and *Lepra Bibliotheca Internationalis.* 1900. For more results from nasal tests, see: Rogers and Muir [1925] 1940: 153-155.

[131] The opening statement on the entry on leprosy in *An Introduction to Dermatology* (1899) by Norman Walker (who translated Hansen and Looft into English four years earlier) was that: "Leprosy is a chronic disease caused by the presence of the lepra bacillus." (Walker, Norman. *An Introduction to Dermatology.* [1899] 1902: 257.) With some differences in emphasis, identifying the disease with the bacillus soon became a standard formula, both in general textbooks and books on dermatology and tropical diseases. Some examples: "A chronic infective granulomatous disease produced by a specific bacterium, and characterized by lesions of the skin, nerves and viacera eventuating in local anæsthesia, ulceration, and a great variety of trophic lesions. After a

frequent references to the nasal test. For Richard L. Sutton "the quickest and most certain method of diagnosing the disease is by the recognition of the bacilli in the affected tissues, in serum or blood obtained from the lepromatous nodules, or in the nasal discharges."[132] Ludwig Török argued that changes in pigmentation and anesthetic lesions were the first signs to look for; the bacillus in the lesions and nasal discharges was the second.[133] For W. Hamilton Jefferys and James L. Maxwell early diagnosis should focus on whether the patient experienced loss of temperature reaction and absence of sweating from the affected areas. Detecting bacilli in the nasal secretion was not strictly necessary, but offered "considerable assistance" in the diagnosis.[134] Sir Leonard Rogers and Ernest Muir argued in *Leprosy* (1925) that: "The examination of the nasal mucous membrane is almost as important as that of the skin. It occasionally gives positive bacteriological findings when the skin is negative."[135] The nasal test was also made part of the medical education. In two lectures to students at the St. Thomas Hospital in London in 1907 referred to in *Lepra Bibliotheca Internationalis*, for instance, British epidemiologist Fleming Sandwith stressed that the nose should *always* be tested.[136]

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long course it is almost invariably fatal." (Manson, Patrick. *Tropical Diseases. A Manual of the Diseases of Warm Climates.* [1898] 1903: 478); "Leprosy is a chronic, infectious malady, due to the bacillus leprae of Hansen, characterized by alteration and destructive changes in the cutaneous, nervous or bony structures of the body." (Sutton, Richard L. *Diseases of the skin.* 1919: 803); "Leprosy is a general disease with a chronic and paroxysmal course, due to the Bacillus leprae of Hansen, and characterised by lesions in the skin, macous membranes, nerves and viscera. It is transmissible from man to man, but the method of infection is unknown." (Macleod, J. M. K. *Diseases of the Skin. A Text-Book for Students and Practitioners*. 1920: 496.)

[132] Sutton 1919: 814.

[133] Török, Ludwig. *Spezielle Diagnostik der Hautkrankheiten für Praktische Ärtze und Studierende*. 1906: 319-325.

[134] Jefferys, W. Hamilton and James L. Maxwell. *The Diseases of China. Including Formosa and Korea.* 1911: 100. Interestingly, Jefferys and Maxwell also included a third form of leprosy in addition to the nodular and nerve (their term for anaesthetic) leprosy, namely the 'macular' variety. The aacular form produced small, lightly raised patches less sensitive to pain and recognizing hot and cold, but it never produced mutilations. Their argument for this being a variety of leprosy was that it was only found in areas where the disease was endemic; that the loss of temperature sensation was rare in other diseases; that it often mixed with the other forms; and finally "Because occasionally lepra bacilli are to be found in the nasal secretion of these patients." (Op. cit.: 97.) Unlike the other textbooks, Jefferys and Maxwell underlined that "the discovery of the bacillus has hardly increased our knowledge of the pathology and treatment of the disease." (Op. cit.: 92.)

[135] Rogers and Muir [1925] 1940: 211.

[136] Sandwith, F. M. "Two Lectures on Leprosy". *Lepra Bibliotheca Internationalis.* 1907: Fasc. 4, originally published in *Clinical Journal*, 1907: 782-3.

But did the nose test settle diagnosis? Several physicians were critical. In Lisbon in 1906, at the fifteenth International Medical Congress, the Portuguese dermatologist Zeferino Falcão pointed out that occasionally family members of lepers also tested positively on the nose-smear test, without ever producing clinical symptoms of the disease.[137] In his view, the test was unreliable because it detected too many cases. Jonathan T. MacDonald at the leprosy investigation station in Hawaii argued, to the contrary, that the nasal examinations were worthless because they did not detect *enough* cases. After studying 150 cases in 1903, he concluded: "After many faithful trials in every stage of the disease I have found them so seldom that I now attach but little importance to it as a means of diagnosis."[138] Seven years later, leprologists Walter Ramson Brinkerhoff and William J. Moore, also working on Hawaii, agreed that the nasal test alone was secondary to clinical diagnosis but that it could detect 'the most dangerous' cases:

> When it is not practicable, to make a complete physical examination of all individuals of a class suspected of leprosy, the examination of the nasal septum and the bacteriological examination of the nasal secretions will prove of value by permitting the recognition of the most dangerous type of the disease, and is therefore worthwhile, even if it does not reveal all cases of the disease in those who come under observation.[139]

By the 1930s, however, the premise for the nasal test had changed. Subsequent studies of 250 leprous children in the Philippines showed that the bacillus was detected in the skin before it reached the nose, and that instead of being a primary

[137] Zeferino Falcao. "La rhinite lépreuse. Communication au Congrès internat. De Lisbonne 1906." *Lepra Bibliotheca Internationalis*. 1907: Fasc. 1, originally published in: *Presse de médecine* 1906.
[138] MacDonald, J. T. "A Diagnostic Examination of One Hundred and Fifty Cases of Leprosy." *The Journal of the American Medical Association*. 1903: 1567.
[139] Brinckerhoff, W. R. and W. L. Moore. "The Utility of the Examination of the Nose and the Nasal Secretions for the Detection of Incipient Cases of Leprosy". *Lepra Bibliotheca Internationalis*, 1910: 174, also referred to in Pinkerton, F. J. "Leprosy of the Ear, Nose and Throat. Observations on more than two hundred cases in Hawaii." *Archives of Otolaryngology*, No. 4. October 1932: 469-487.

lesion, the nasal mucous membrane was one of the last places the bacillus persisted in those who reacted positively to treatment.[140]

Despite questions of its reliability, the nasal examination would be used not only to detect new (or dangerous) cases; it was also employed in leprosy institutions to decide if a patient could be released. In South Africa, where the passing of the Leprosy Repression Act in 1892 made segregation compulsory, the leper asylums was a final destination. Once admitted, the patients were imprisoned for life. This changed in 1908, when a national Leprosy Commission began periodic visits to the institutions to look for misdiagnosis and 'arrested' (bacteria-free) cases. On Robben Island leprosy colony, which had hosted leprosy sufferers since 1846 and was massively expanded after 1892, the Commission's first visit in March 1908 led to four patients being discharged. In September, one more patient was released and ten more subject to further testing at their next visit. At the nearby Emjanyana Leper Asylum, which opened in 1897, six patients were discharged at the Commission's first visit.[141]

From around 1915 similar procedures were introduced on Hawaii. An absence of the leprosy bacillus in three successive microscopic examinations at intervals of three months was grounds for 'parole'. After four years of out-patient treatment, a final examination could result in a release.[142] Between 1915 and 1925, 104 patients from the Kalihi Hospital received the 'final release', while in 102 cases the disease relapsed during parole.[143] The nasal test was thus redefined from initial diagnosis to a test employed in deciding if someone could be released. As physician Forrest Joy Pinkerton put it in 1932:

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[140] Solis and Wade's study of Philippine children was published in *Journal of the Philippine Island Medical Association*, Vol. 5, December 1925, the follow-up in the same journal in 1927 (No. 1). Referred to in: Burnet, Et. "Provisional report presented at the Leprosy Commission at its meeting at Tokyo in April 1930: Consideration of urgent matters to which the commission drew attention at its May session, 1928". League of Nations, C. H./Leprosy/7a, Geneva, January 1930: 5

[141] Gregory, John A. "Report of the Medical Officer of Health for the Colony of Cape of Good Hope (1908). *Lepra Bibliotheca Internationalis*. 1909: Fasc. 3.

[142] A similar system was set up at the leprosarium in Carville, Louisianna, after it came under Federal control in 1921. Patients who tested negatively on a bacteriological test were moved to a 'non-contagious section' and held there for two years before being submitted to a final examination. After release, they were subject to examinations every six months for three years before receiving a definite discharge as 'arrested' and no longer a danger to public health. De Souza-Araújo, Heráclides César. *Leprosy Survey made in Fourty Countries (1924-1927)*. Instituto Oswaldo Cruz, Rio de Janeiro. 1929: 32.

> In Hawaii, so much do we depend on the nasal examination that no patient is recommended for release as having a quiescent case until his nasal 'snippings' have been bacteriologically negative for a period of three months, or even longer, even though the disease is otherwise clinically inactive before the nasal investigation is begun.[144]

On the Philippines, a similar system was introduced in March 1922. By September 1925, 545 'lepers' at the leprosarium on Culion were reported as 'bacteriologically negative'; 238 had been discharged.[145] The introduction of the bacillus as the cause of the disease thus made it possible to develop the terms 'quiescent', 'arrested' or 'bacteriologically negative'. These terms were introduced by the end of the first decade of the 20th century (followed by the term 'burnt-out case'), and ultimately made it possible to look like a 'leper' and still be released from confinement.[146] For the individuals concerned, this could have dramatic consequences. The bacillus, or more precisely the lack of the bacillus, was what set them free.

At the same time, bacteriologically negative did not mean that mutilations healed. In the Philippines, for instance, several of those discharged from Culion returned to live in the colony. As physician Heráclides César de Souza-Araùjo from Brazil reported after a visit in the mid-1920s: "…either they were not welcomed by their families or by the society, having been considered lepers on account of their scars or mutilations. Here is a new economic-social problem to be solved."[147]

Showcasing the living body

Identifying the bacillus was an addition to, not a replacement for, clinical diagnosis. Besides opening for possible disagreements, a problem with symptom-based

[143] De Souza-Araújo 1929: 75-78.
[144] Pinkerton, F. J. "Leprosy of the Ear, Nose and Throat. Observations on more than two hundred cases in Hawaii." *Archives of Otolaryngology*, No. 4. October 1932: 475.
[145] De Souza-Araújo 1929: 152; 205-206.
[146] The term 'burnt-out' seems to be in use from the late 1920s, and was made famous by Graham Greene's novel with the same title published in 1960, set in a leprosy colony in Congo.

diagnosis was that it required experience and specialist training.[148] Reading descriptions or looking at images and photographs was helpful, but not sufficient when learning how to recognize the disease. The solution was learning from living patients.

From the published proceedings of the medical meetings, often printed in abstracts in the medical journals, it seems the practice of showcasing living patients was widespread. There were mainly two motivations behind bringing patients to medical meetings: To teach others what clinical signs to look for, and to determine diagnosis in uncertain cases – sometimes with lifelong consequences for the individual put under scrutiny.

The most prominent exhibition was arranged by the British physician Jonathan Hutchinson at the Seventh International Medical Congress in London in 1881. Beneath the chandeliers of the boardroom of the Geological Society at the Burlington House, seven patients with leprosy, six diagnosed with myxedema and several cases of lupus were exhibited in a makeshift medical ward.[149] The exhibition was, however, a victim of its own success: As Hutchinson noted: "confusion and crowding (…). Our exhibition was more popular than we had expected: every morning at the hour announced, the room filled. The weather chanced to be very hot, and as the room looked into Piccadilly, it was exceedingly noisy."[150] Still, already the next

[147] De Souza-Araújo 1929: 152. More on de Souza-Araújo's travels in Chapter 7.
[148] The same could be argued for bacteriological diagnosis. In 1909, in the case of John Early, a former soldier and suspected 'leper', samples of skin tissues was sent to Armauer Hansen in Norway for a final verdict. Johnson, Felix S. S., American Consul to Norway. Letter to The Honorable Assistant Secretary of State, dated Bergen August 20, 1909. The National Archives, Washington, USA. Five years later, John Early caused a public outcry when traveling to Washington to meet with politicians demanding that the government provided better care for people in his situation. See: Moran 2007: Chapter 1.
[149] Schupbach, William. "The Exhibition of Living Patients, 1881" *Wellcome Library Blog*. 2009. (http://libraryblog.wellcome.ac.uk/libraryblog/2009/10/the-exhibition-of-living-patients-1881/). In the adjoining exhibition, among more than 700 items, there were also twelve pictures of leprosy patients and two microscopical demonstrations. See: *International Medical Congress. Catalogue of Temporary Museum held at the Geological Society, Burlington House*. 1881.
[150] *Transactions of the International Medical Congress*, 7th session, London 1881, vol. 1, pp. 109-132, referred to in Schupbach 2009.

International Medical Congress in Copenhagen in 1884 saw a similar exhibition, though with fewer patients and more wax-models and images.[151]

Showcasing patients neither started, nor ended, at the International Medical Congresses.[152] But instead of arranging exhibitions, displaying local cases in smaller sessions seems to have been more common. After J. G. McDougal again had presented the two Ohio-sisters suspected of having leprosy at the Fiftieth Annual Meeting of the American Medical Association in 1899, Dr. Bishop of New York City argued that showcasing living patients was necessary in order to teach physicians how to recognize the disease. The rationale was not primarily that the individuals could be treated, but because it would promote early segregation and thus prevent new cases:

> The general practitioner is very much interested in leprosy, always, and when he gets hold of a case he generally shows it around. Last winter, I borrowed some cases from Dr. Fox and showed them at the session of the General Academy, and they excited much interest. General practitioners, myself included, do not know these conditions until they are brought before them. I think many of these cases abound and many might be saved from infection if these patients were more generally recognized.[153]

In addition to showcasing patients at meetings, leprosy institutions opened their doors to visiting physicians. As Ashburton Thompson, head of the leprosy services in New South Wales (Australia), stressed: "Every opportunity has been given to members of

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[151] "Of the many diseases under consideration, leprosy claimed a large share of notice. Four living examples were shown – two adults by Mr. Hutchinson, and two boys aged respectively eight and nine years by Dr. Radcliffe Crocker. All these had contracted the disease abroad, and the chief points of interest were the gradual dying out of the disease in an elderly woman (…) Many wax models, photographs and portraits of leprosy were on view in the museum from the collection of Dr. E. de Wahl of Dorpat, and Guy's Hospital, &c." Editorial. "Disease of the Skin at the Congress in Copenhagen." *The Lancet*. 13. August, 1881: 307.

[152] Although no living patients were showcased at the international leprosy conference in Berlin, several pictures and microscopic preparations were exhibited. A. Grünfeld from Rostow brought one hundred photographs, while Phineas S. Abraham from London brought 18 lantern slides. Abraham also brought an installation showing the leprosy bacillus stained with different techniques and at different levels of magnification, prepared by Dr. Lawrence Herman, also from London.

[153] McDougal, J. G. "Some questions relative to the diagnosis of anæsthetic leprosy." *Lepra Bibliotheca Internationalis*. 1900: 246, originally published in: *The Journal of the American medical Association*, Jan. 17, 1900: 210. The discussion led to no firm conclusion on whether the disease the sisters suffered from actually was leprosy. As McDougal concluded: "I hope that the character of these cases may yet be positively ascertained, and if it is, I hope it may be found to be some other disease than leprosy, for it would seem a

the medical profession to visit the lazaret for the purpose of seeing such patients as were formerly under their care, and for study of the disease."[154] As I will return to in Chapter 7, the practice of traveling to visit leprosy institutions was a common practice. Visiting institutions also included investigating patients.

The patients in the institutions had already been diagnosed. The verdict leading to institutionalization was often organized in health boards, which too involved inspecting living patients. The procedures were often detailed in books and medical journals, and indicate that in practice clinical and laboratory techniques were complimentary, not mutually excluding. In the Philippines, for instance, former Director of Health Victor G. Heiser described:

> For the clinical examination of the anesthetic form the suspect was blindfolded. Then his skin was touched with a cotton swab, a feather, a camel's hair brush or a paper spill, and he was asked to indicate where he had been touch. The head and the point of a pin were pressed alternately against suspected spots, and the patient was asked which caused the more pain. Test tubes, one filled with hot water and the other with cold, were held against his skin, and he was asked to tell which was warm and which was cold. Finally, a scraping was taken from the septum of the nose with a blunt, narrow-bladed scalpel, and put under the microscope.[155]

In Calcutta, Sir Leonard Rogers and Ernest Muir described a similar procedure:

> The patient is stripped as far as possible and blindfolded. Sensation is tested by touching different points on the skin surface with a light object such as a feather or camel-hair brush, the patient being instructed to place his index finger exactly on the points touched. (…) The most conclusive proof of leprosy is finding Hansen's bacillus in the tissues. But, as we have shown, it may be difficult or impossible to find bacilli in lesions of the neural type, and in these we must rely chiefly on clinical signs.[156]

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menacing disclosure to learn that the bacillus of leprosy can be carried from a distant place to a fertile soil in the interior of the United States." (Op. cit: 248-249.)

[154] Thompson, Ashburton. "On leprosy in New South Wales, for the year 1904". *Lepra Bibliotheca Internationalis* 1906: 125.

[155] Heiser, Victor. *An American Doctor's Odyssey: Adventures in Fourty-Five Countries.* 1936: 232.

[156] Rogers and Muir [1925] 1940: 207-208.

In the columns of *Lepra Bibliotheca Internationalis,* there were those that argued that the leprosy bacillus had stolen the limelight from much needed clinical research. In 1914 American physician James Albert Honeij published a study based on examining fifteen patients at the Massachusetts leprosy colony on the island of Penikese.[157] His main finding was that the patients all had a very high pulse, and that recognizing this sign should be part of the diagnostic toolbox. Had the physicians spent more time at the bedside, and less time in the laboratory, he argued, this would have been noticed much earlier.[158] In general, however, there does not seem to have been much conflict between the clinic and the laboratory. Identifying the leprosy bacillus in the laboratory was by most considered an addition to clinical judgment and not a replacement.

The elusive test

Diagnosis could sometimes lead to disagreements among the experts, and much research was driven by an aspiration of finding a test which quickly and decisively could determine whether a person was affected by leprosy or not. The tests built on different strategies, but all had one thing in common: They failed to achieve the objective. Some of the tests initially designed to decide whether a person was affected by leprosy would instead be reinterpreted and used in classification of cases, or used in the hope that they could help predict the progression and outcome of treatments.

The first test which ended up as part of the diagnostic toolkit was uncovered by mistake when Danielssen, probably in the 1860s, tried using potassium iodide to treat patients. Administering the chemical compound orally did not lead to improvement, rather the opposite: The patients consistently reacted with a strong fever. Instead of

[157] The Massachusetts leprosy colony was established in 1905, and closed down in 1921. The thirteen remaining patients were then transferred to the U.S. National Leprosarium in Carville, Louisiana.
[158] Honeij, J. A. "Leprosy. Some Notes on Symptoms" and "Leprosy. The Pulse as a possible Indicator of the Progress of the Disease". *Lepra Bibliotheca Internationalis* 1914, Fasc. 2, originally published in *Boston Medical and Surgical Journal*, 15. January and 22. February 1914, and also printed in *Tropical Disease Bulletin*, May 1914. The paper also shows how the same observations could be presented as new discoveries several times. In 1908, Japanese dermatologist Takekichi Sugai had discussed the exact same symptoms. Sugai, T. "Über die Erweichung und Vereiterung der Lepraknoten". *Lepra Bibliotheca Internationalis.* 1908: Fasc. 3.

putting the drug on the shelf, this became Danielssen's final test in deciding if a patient could be released as 'cured' from the Lungegaarden research hospital in Bergen: If the compound did not produce a fever, the patient was cured.[159] At the Berlin conference in 1897, where Jeanselme advocated the nose as a place to look for the leprosy bacillus, administering potassium iodide was suggested as a way to incite the bacillus so that it did not avoid detection. When Etienne Burnet, secretary of the League of Nations Leprosy Commission, in the late 1920s took stock of the numerous diagnostics methods in use, he referred to potassium iodide as the best known method for making a 'biological diagnose'.[160]

While potassium iodide was quietly adopted in numerous locations, the Bordet-Wassermann-test led to far greater attention. This method was developed in Berlin in 1906 by August Wassermann, Carl Bruck and Albert Neisser to detect syphilis, and drew on Jules Bordet and Octave Gengou's research on complement-fixation that had begun five years prior.[161] The test consisted of collecting blood serum from a suspected case and mixing it with an antigen consisting of alcohol-treated muscle extract from a bulls' heart in a glass cylinder. The intensity of the reaction correlated with the intensity of the infection. Ludwig Fleck has argued that the test in fact redefined syphilis from a display of symptoms to testing positively on the Wassermann-test.[162]

For leprosy patients, like the nasal test (and unlike potassium iodide), the results of the Wassermann-test were highly inconsistent. When the three Swedish physicians J. Jundell, Johan Almkvist and F. Sandmann in 1908 tested 26 'lepers' in

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[159] Opposed to Hansen and Looft's bacteriological diagnosis, it seems Danielssen's demanded the patients to be symptom-free before using the label 'cured'. When discharging patients at their request, he often used the term 'improving'. For a review of potassium and other compounds producing fevers, subcutaneous haemorrhage and other side effects, see Siebert, C and V. F. K. Klingmüller. *"*Beiträge zue Kenntnis der Jodreaktion der Leprösen*." Lepra Bibliotheca Internationalis*. 1904: Fasc 4; Pautrier, L.-M. "Le Diagnostic de la Lèpre par les Méthodes de Laboratoire." *Lepra Bibliotheca Internationalis*. 1915: Fasc 1, originally published in: *La Presse Medicale*, 14. March, 1914.

[160] Burnet, Et. "Provisional report presented at the Leprosy Commission at its meeting at Tokyo in April 1930: Consideration of urgent matters to which the commission drew attention at its May session, 1928". League of Nations, C. H./Leprosy/7a, Geneva, January 1930: 5.

[161] Wassermann, A. P., A. Neisser and C. Bruck. "Eine seriodiagnotische Reaktion bei Syphilis. " *Deutsche medizinische Wochenschrift*. No. 19, 1906: 475-476.

[162] Fleck [1935] 1979.

Sweden's only specialized leprosy institution in Järvsö, they found 4 positive reactions, 4 partial reactions and 16 negative reactions. The positive reaction, they speculated might indicate that some of the lepers had in fact syphilis.[163] Divergences were noticed also in other studies, and led to different strategies for classifying the cases in order to explain the results. Two years after the Swedish study, German physicians Carl Bruck and E. Gessner found that five out of seven of their tubercular patients reacted positively to the test, but none of the anaesthetic cases.[164] That the reaction was more common in tubercular cases was also argued by the Berlin-based bacteriologist (and later Robert Koch-biographer) Bernhard Möllers. Not having access to any patients of his own, he received 32 serum samples from Memel, Southern Karpaten in Romania, Egypt, Bosnia and Tokyo and found that all but seven reacted positively. The reaction was stronger in the more tubercular cases.[165] In 1923 the Calcutta based researchers R. B. Lloyd, Ernest Muir and G. C. Mitra found that of 286 cases of 'undoubted leprosy', 41,7% of all cases tested positive, with a higher proportion in nodular cases and "very much higher figures" in children than in adults.[166]

The Wasserman test was also used in differential diagnosis. In 1913, American bacteriologist Moses Tran Clegg found that 11 of 24 cases at the United States Leprosy Investigation Station in Hawaii tested positively to the test, and suggested using lutein as a tool to decide whether the positive results were caused by syphilis or leprosy. Lutein was an inactive solution of the syphilis spirochete which Clegg argued reacted with syphilis but not with leprosy.[167]

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[163] Jundell, J., Johan Almkvist and F. Sandmann. "Wassermanns Syphilisreaktion bei Lepra". *Lepra Bibliotheca Internationalis*. 1909: Fasc. 4, originally published in: *Centralblatt für Innerren Medicin*, 28. November 1908. Järvsö hospital in Hälsingland was the only state leprosy institution in Sweden. It was established in 1867, and closed in 1943.

[164] Bruck, Carl and E. Gessner. "Über Serumuntersuchungen bei Lepra." *Lepra Bibliotheca Internationalis.* 1910: Fasc. 1, originally published in: *Berliner Klininische Wochenschrift*, 29. March 1909.

[165] Möllers, Bernhard. "Serologische Untersuchingen bei Leprösen." *Lepra Bibliotheca Internationalis*. 1913: Fasc. 3, originally published in: *Deutsche Medizinische Wochenschrift*. 1913, no. 13.

[166] Lloyd, R. B; Muir, E; Mitra, G. C. "The Wasserman Reaction in Leprosy". *Indian Journal of Medical Research.* Vol. 11, No. 1, July 1923: 229-233.

[167] M. T. Clegg. "Absence of luetin reactions on lepers showing a positive Wassermann reaction." *Lepra Bibliotheca Internationalis.* 1915: Fasc. 1, originally published in: *Public Health Bulletin* no. 61, July 1913.

There were also attempts at changing the doses, administering the test using different temperatures, as well as using other sources than bovine heart or muscle-extracts to produce the antigens used in the test.[168] After analyzing 75 papers on the Wasserman test in the mid-1920s, H. C. de Souza-Araùjo found that the proportion of cases reacting positively varied between 30 and 90 percent, and that "in cold and temperate climates the percentage of positive reactions nearly doubles."[169] This was not the only interpretation. Did the results prove that leprosy was closely related to syphilis, or was it an indication that many had both diseases? Did the stronger reaction in children prove that disease caused the patient's antibodies to change over time? Could the reaction (or lack thereof) be used to predict the progress of the disease? At the end of the 1920s, Dr. Etienne Burnet concluded on behalf of the League of Nations Leprosy Commission that "the value of this test for the diagnosis of leprosy would seem to be very small", and recommended that the researchers instead focused their efforts elsewhere.[170]

One of the attempts at finding an alternative to the Wassermann-test did have lasting consequences. Inspired by Clemens von Pirquet and Charles Mantoux' skin-test which assessed the immune response to tuberculosis, developed in 1906, Japanese leprologist Kensuke Mitsuda published a paper on Lepromin in 1919 which argued that the substance could be used in a similar way to decide if someone had leprosy.[171] The research was presented at the International Leprosy Conference in Strasbourg in 1923 but received little attention outside Japan. Ten years later it was reworked by Fumio Hayashi and featured as one of the major pieces in the first issue of the *International Journal of Leprosy*.[172] By then it was clear that the Mitsuda's test had failed to achieve the objective of making a decisive diagnosis, but it remained in use

[168] See i.e. Bruck and Gessner 1909.
[169] De Souza-Araújo 1929: 29.
[170] Burnet 1930: 9.
[171] Mitsuda, K. "On the value of skin reaction with emulsion of leproma". *Japanese Journal of Dermatology and Urology*. No. 19. 1919: 697-708. In 1931, Mitsuda was appointed to be the first Vice-Chairman of the Eastern Section of the International Leprosy Association. ("Association News." *International Journal of Leprosy*. No. 1, 1933: 95-96.)
[172] Hayashi, F. "Mitsuda's skin reaction in leprosy". *International Journal of Leprosy*. No. 1, 1933: 31-38. Thanks to Bjørnar Mortensen Vik for scanning this for me.

for decades to distinguish between different types of the disease, and to monitor the impacts of treatment.

Diagnostic pluralism and local variations

While the presence of the leprosy bacillus from around 1900 had dominated as a decisive proof when making a diagnosis, the 1920s saw a new dawn for disease models arguing that the bacillus alone was not sufficient to produce the disease. Some predisposing cause was also necessary. Originating at the medical community at the School of Tropical Medicine and Hygiene in Calcutta, the main argument was that if the bacillus alone had been enough to produce leprosy, more people would have caught the disease. As Sir Leonard Rogers and Ernest Muir put it; "We consider that it is highly probable that in an endemic area less than half of those who are inoculated with the germs of leprosy develop the disease."[173] The same objection, that developing the disease took more than just the bacillus, had also been raised in the decades surrounding the turn of the century but was not pursued as a line of research.[174]

Emphasis on predisposition was closely linked to early diagnosis and the argument that treatment should begin as early as possible, ideally before the clinical signs became severe.[175] In the 30-page booklet *Leprosy: Diagnosis, Treatment and Prevention* (1924), head researcher in Calcutta, Ernest Muir, argued that "a relative cure can in the large majority of cases be obtained if the disease is taken in hand in

[173] Rogers and Muir [1925] 1940: 220.
[174] One of the strongest opponents of predisposition was Gerhard Armauer Hansen. In his review of Leopold Glück's *Die Lepra in knapper Darstellung* (1900), for instance, he argued: "We are too ignorant to know what confers the predisposition. (…) To say that poor people are more predisposed to these diseases than the well to do is in my opinion only to clothe our ignorance in high sounding words which tell us nothing. While it seems to me tolerably certain that the greater the overcrowding and intercourse among the poorer classes the greater the chances of infection; and in consequence I prefer this interpretation, for as far as I know leprosy everywhere prevails where the chances of infection are increased. Well to do people often get the disease when they expose themselves to such conditions." (Hansen, G. A. "Die Lepra in knapper Darstellung". *Lepra Bibliotheca Internationalis.* 1900: 217.)
[175] I will discuss in more details the Calcutta school's line of reason in Chapter 7.

the first stage; but this becomes increasingly difficult and requires an increasingly long course of treatment as the disease advances."[176] The publication was distributed by The British Empire Leprosy Relief Association (BELRA), both within the British Empire and Mission to Lepers.[177]

The two cardinal points in diagnosis, according to Muir, was "finding of lepra bacilli and the presence of anæsthesia, the former in the skin type and the latter in the nerve type."[178] In absence of the bacillus or anesthesia, a combination of two of more of the following signs would arouse a strong suspicion of leprosy: Deep analgesia (insensitivity to the prick of a pin or the cut of a knife), loss of the sense of heat and cold in the areas around a patch of anesthesia, hyperanesthesia (an abnormal increase in sensitivity to stimuli), depigmentation, erythematous patches (redness of the skin), thickening of superficial nerve trunks, parakeratosis (scaly, dry, thickened and glossy skin), anhydrosis (the inability to sweat), interfollicular swelling (enlargement of the lymph nodes), dry rhitintis with ulceration (infection in the nostrils), irregular pyrexia (fluctuating body temperature), tropic blisters and later tropic ulcers in the midst of anesthetic areas.

> The presence of one or more of these subsidiary signs should be considered a sufficient reason for keeping a patient under observation. As a rule, if the case is one of leprosy, one of the two important signs will soon establish itself and clear up the diagnosis.[179]

Two points separated the Calcutta-school from the bacteriological school of thought that had dominated before the Great War: A firm belief that leprosy in most cases could be cured, and an emphasis on predisposing causes.[180] Or, as Muir put it: "Some predisposing cause or loss of natural resistance must also be present to change the

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[176] Muir, Ernest. *Leprosy: Diagnosis, Treatment and Prevention*. *Illustrated.* Cuttack: Orissa Mission Press. 1924: 2. The arguments were repeated in Rogers and Muir 1925.
[177] Correspondence between the League of Nations and BELRA, 1925. (LNHO: 12B-R898/2942/42641). Muir's work was also reprinted in Chinese medical journals.
[178] Muir 1924: 2.
[179] Muir 1924: 8.
[180] The specific treatments will be addressed in Chapter 3.

tissues of the human body into a medium fit for the growth of the lepra bacillus."[181] In the monograph *Leprosy* published in 1925, he expanded: "When a case has been established beyond all doubt as one of leprosy, only half the diagnosis has been made; it is no less necessary to find out what is the predisposing cause."[182] The predisposing cause was what lowered the resistance of the body and made the attack of the lepra bacillus successful. The main predisposing causes were temporary diseases such as influenza, chronic ailments such as bowel diseases, syphilis, malaria or hookworm; climatic conditions, unhealthy and insanitary surroundings; lack of sufficient exercise or unsuitable diet. Early treatment did not primarily address the bacillus but the predisposing causes: "Failure in treatment is frequently due to failure to diagnose and eliminate the predisposing cause."[183]

Although this led Muir to suggest a classification based on the numbers of bacilli found under the skin of the patients, it is possible to argue that Muir's emphasis on predisposing causes led to a more holistic disease model. While Hansen emphasized that the leper was to blame for catching the disease, the Calcutta school saw the disease as the result of unfortunate circumstances for which the 'leper' himself could not be blamed, such as poverty or poor sanitary conditions. While BELRA did work on a specific cure for leprosy, they also emphasized elevating the sufferer's circumstances through education and sanitary improvements.

This was not the only interpretation of predisposition. The researchers in Calcutta cooperated closely with Philippine researchers at the Culion leprosy colony and in Manila, and were a clear inspiration when the Philippines in the 1920s abandoned their segregationist line which had relied solely on compulsory segregation. The replacement, the "Dual Plan", added two more layers to the leprosy approach: Voluntary outpatient treatment for those in the early stages of the disease, and hospitalization for those in need of extensive treatment. Segregation at Culion should, at least in theory, be reserved for those who did not respond to the

[181] Muir 1924: 9.
[182] Rogers and Muir [1925] 1940: 220.
[183] Muir 1924: 9.

treatment.[184] The policies were made possible by a disease model which did not require the presence of the leprosy bacillus in order to establish the diagnosis. As the American pathologist Herbert Windsor Wade and colleague Jose N. Rodriguez put it:

> While the finding of acid-fast organisms that are sufficiently characteristic to permit their recognition as *M. leprae* settles the diagnosis, it must be realized that *negative bacteriological findings do not necessarily rule out leprosy*.[185]

The Manila researchers agreed with the Calcutta school that local anesthesia was a 'cardinal sign', but their classification saw cutaneous (skin) lesions as second necessary determinant for confirming diagnosis and not the bacillus. From this premise they reached a classification with wide-reaching consequences for the individual sufferer. Those who had at least one of these signs (anesthesia or skin lesions) were diagnosed 'clinically positive', and were to be offered regular treatment locally, without segregation. If the leprosy bacterium was detected, the patient was labeled 'bacteriologically positive' and was to be treated in regional stations open to visits from friends and family. "Third, cases that prove not satisfactorily to treatment will be removed to the Culion Leper Colony. It is important that such patients do not accumulate in the stations, for they tend to discourage others."[186]

Instead of seeing predisposition as something found in society that could be combated by raising the general health of the population, the Manila school focused on 'individual resistance'. This was not linked to poverty or unfortunate circumstances, but improper feeding or weakening disease, sexual over-activity, age (those between ages 10 and 20 were particularly susceptible), and race. Individual resistance was one of five conditions involved in contagion, the others revolved

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[184] Despite being the world's largest leprosy camp, with 5,257 lepers registered at the end of 1925, Culion did not have enough capacity to segregate *all* of the estimated 20,000 suffering from the disease in the Philippines. The change was also influenced by political turmoil and increasing public criticisms of the vast expenses connected with the leprosy program (see Chapter 7). The meager impact the segregationist policies had on reducing the number of new cases was repeatedly put forward as case in point by the Calcutta-school, arguing that the main effect of segregation was that the lepers went into hiding to avoid detention.

[185] Wade, H. W. and J. N. Rodriguez. *A description of leprosy: Its etiology, pathology, diagnosis and treatment*. Manila. 1928: 52, emphasis in the original.

around the bacillus: The release of bacilli in contact with other lepers, type of contact, the number of bacilli involved in the contact and its portal of entry.

While the diagnosis in Calcutta and on Manila in most cases overlapped, the systems were not identical. In borderline cases, diagnosis could differ depending on where the suspected case was diagnosed.

When Etienne Burnet, the secretary of the League of Nations Leprosy Commission, in the late 1920s was asked to prepare a memorandum on diagnosis, he was painfully aware that diagnosis varied from place to place. Instead of focusing on one method, he cautiously outlined five concurrent strategies in use or being researched in different configurations in different locations: Clinical diagnosis, bacteriological diagnosis, biological diagnosis, serological diagnosis, and chemical- and physiochemical diagnosis – all with different strengths and weaknesses.[187]

Clinical diagnosis meant looking for symptoms. The problem was that it required training by specialists to know what discolored spots ('maculae') to look for. There was, according to Burnet, a desperate need for "a collection of colored photographs which could be printed off in a suitable number of copies. (...) Nothing is more instructive than to see the real thing side by side with the picture. We remember with reverence the plates published by Danielssen, the pioneer of the modern study of leprosy."[188] Burnet did not accept 'predisposition' as part of the disease model, but was closer to Danielssen and Boeck's 'prodromes' when he argued that physicians, especially in areas where leprosy was endemic, should pay more attention to general symptoms: Fever, lack of appetite, sensation of chill, skin irritation, fatigue, headache, pains of rheumatic type, vasomotor troubles (changes in the blood vessels) and anemia should all rise suspicion of leprosy.

Bacteriological diagnosis consisted of detecting the presence of the leprosy bacillus in samples taken from the skin, the nasal mucous membrane, the lymph

[186] Wade and Rodriguez 1928: 7.
[187] Burnet, Et. "Provisional report presented at the Leprosy Commission at its meeting at Tokyo in April 1930: Consideration of urgent matters to which the commission drew attention at its May session, 1928". League of Nations, C. H./Leprosy/7a, Geneva, January 1930: 1-20.
[188] Burnet 1930: 2.

glands and other organs, such as the testicles. Burnet was, however, critical of using the bacillus as an ultimate proof: Referring to four independent studies from Brazil and the Philippines, he repeated the argument made by Zeferino Falcão in 1906, that: "Persons in whom it (the bacillus) was found did not always develop leprosy".[189] In other words: Leprosy was not merely the presence of the bacillus, but the symptoms caused by this presence.

Biological diagnosis consisted of influencing the organism by compounds that produced a reaction, with Danielssen's potassium iodide producing fever as the best known and most widely used. In addition, injections of Koch's Tuberculin, Babes' Leprin, Rost's Leprolin, Mitsuda's Leproma and Deycke's Nastin were all used or tested to determine specific reactions in lepers.[190] Reacting to leprosy-specific stimuli indicated the presence of the disease.

Serological diagnosis meant producing serum from blood samples and testing them with fixation techniques in test-tubes. While the Wasserman-test had received most attention, Burnet concluded that this research had been of limited value.[191] This did not mean that the numerous attempts at modifying the test to give it a higher degree of specificity to leprosy should be abandoned. Using de-fatted *streptothrix leproides* as antigen, a procedure that was extensively tested in São Paulo, was highlighted as a promising alternative. Some potential was also found in precipitation tests consisting of mixing serum and distilled water or alcohol to produce a deposit in the bottom of the small test tubes, a technique researched in the Philippines and in the Netherlands respectively. A related line of research focused on measuring not the intensity of the reaction, but the speed of sedimentation of the red blood corpuscles, the hypothesis being that the blood of lepers would sediment faster than 'healthy' blood.

Finally, *chemical and physiochemical diagnosis* consisted of analyzing the proportions of different substances in the blood, such as calcium, the reserve of alkali

[189] Burnet 1930: 5.
[190] I will discuss these drugs in detail in the following chapter.
[191] Burnet 1930: 9.

and cholesterol. There were also more specialized tests, such as the proportion of albumin in the nasal mucus. Increasingly, these tests were seen as useful not in deciding if someone was a leper, but in classification of cases and a way to monitor and ideally predict the effects of treatment.

Despite local variations, by the 1920s the discussion on diagnosis had moved from setting standards for deciding if someone was a leper or not, to finding a common classification of the different varieties of the disease. Since local diagnostic practices differed, classification had consequences for how reports from afar could be interpreted and to what extent it was possible to compile and compare the results. At the 1931 Leonard Wood Memorial Conference on Leprosy in Manila, which I will discuss in more detail in Chapter 7, establishing a global standard for classification was high on the agenda. The outcome was the "Memorial Conference classification", soon renamed "the Manila classification". This divided leprosy into two main types based on clinical features: the Neural and the Cutaneous. The Neural resembled Danielssen and Boeck's "Anesthetic" form, and was defined by alteration in the nerves causing anesthesia, contractures (hardening of tissue) and ulcerations but no skin lesions. The Cutaneous form referred to "All cases showing leprotic lesions in the skin."[192] These were in turn divided into three subtypes each based on the degree of severity of these clinical signs.

Within months after being published, the Manila classification was criticized. In the columns of the quarterly journal *Leprosy in India* (started by Ernest Muir in 1929), Indian physician S. N. Chatterji working at Muir's clinic in Calcutta argued that all cases in reality had mixed clinical signs, and that "any classification on anatomical or pathological grounds (...) is but arbitrary".[193] Instead of symptoms, Chatterji argued, classification should rest on bacteriological findings. Hansen's bacillus was, after all, the cause of the disease.

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[192] Wade, H. W. "A proposed revision of the Memorial conference classification of leprosy". *The American Journal of Tropical Medicine*, Vol. 17, No. 6. 1937: 773-801, quote on p. 775. The term 'leprotic' was in turn defined as: "those changes which present clinical or microscopic evidence of inflammatory processes, typically of granulomatous nature, which are apparently caused by *Mycobacteria leprae* in them." (Op. cit.: 776.)

The debate continued in the columns of the *International Journal of Leprosy* and other medical journals. When the founding editor of the journal, the American pathologist Herbert Windsor Wade working on Culion in the Philippines, in 1937 suggested a compromise at least seven different alternatives had been laid on the table.[194] At the Fourth International Leprosy conference held in Cairo the following year, classification, and thus indirectly diagnosis, would be one of the main discussions.[195]

Conclusion

Danielssen and Boeck's clinical diagnosis from the middle of the 19th century meant that if you look like a leper, you have leprosy. Diagnosis was made in the meeting between the individual suspected of having the disease and an expert who knew what signs to look for. Hansen and Looft's bacteriological diagnosis from the 1890s focused on detecting the bacillus in tissue samples, removed from the person suffering from the disease. What defined the disease was the bacillus, and what the physicians could observe in the clinic was merely a consequence of its effect on the human body.

In theory, establishing the bacillus as the cause of the disease made the clinical symptoms secondary. From the 1890s, several bacteriologists argued that the bacillus reduced the clinical signs from a decisive argument to a suspicion that needed

[193] Chatterji, S. N. "Critical review of the Leonard Wood Memorial Conference report." *Leprosy in India*, No. 3. 1931: 142-146, as referred to in Wade 1937: 777.
[194] Wade 1937: 778-782.
[195] The discussion in Cairo concluded with a new classification, but already the next international leprosy conference in Habana, Cuba, in April 1948, this was overturned by a new standard. See i.e. Canizares, Orlando. "New Official Classification of Leprosy". *Archives of Dermatology.* No. 5. 1949: 584-586. Discussions on classification continue to this day, for instance on Dr. Salvatore Noto's "Leprosy Mailing List" (LML). [http://www.aifo.it/english/resources/online/lml-archives/index.htm]. Medical anthropologist Annemarie Mol's astute observation based on studying atherosclerosis in a Dutch hospital, that parallel (and sometimes competing) ontologies are at work and that diagnosis and interventions are decided on a case to case basis, seems to be the case also for leprosy. (Mol, Annemarie. *The body multiple: ontology in medical practice.* 2002.)

confirmation in the laboratory.[196] From around 1910, several leprosy institutions introduced bacteriological tests in deciding whether a person could be released from segregation. This was the most dramatic consequence of the bacillus: It made the disease something you could get well from.

When establishing whether or not a suspect was indeed 'a leper', however, most saw the bacillus as a welcome confirmation of uncertain cases, but not as a necessity. Hansen's attempt at redefining leprosy as based not on its clinical signs but on the presence of the bacillus alone failed. Bacteriogical diagnosis was was a supplement to clinical diagnosis but never a replacement.

The introduction of the laboratory in diagnosis was not limited to detecting the bacillus. There were numerous attempts at finding a decisive test to decide if a patient was suffering from leprosy or not and thus leave the question of clinical judgement out of the picture. After failing to meet the objective of conclusively separating 'lepers' from 'non-lepers', some of the tests were reinterpreted and used to create new subcategories of the disease and to monitor the effects of treatments. But instead of giving birth to unifying diagnostic practices globally, these techniques played different roles in different locations. In borderline cases, the final verdict depended on where the suspected 'leper' was diagnosed.

This diagnostic pluralism was challenged in the early 1930s as part of initiatives to organize leprologists around the world. This is hardly coincidental. As I will expand upon in the following chapter, in order to compare the efficacy of treatments, a common set of definitions was perceived to be a necessity. Instead of addressing diagnosis directly, the approach was to establish global standards for classifying the forms and varieties of leprosy so that the results of local studies could be disentangled from their site-specific origins and thus be more useful for researchers elsewhere.

So did bacteriology lead to a revolution when it came to diagnosis? Worboys is probably right that the changes read into the 1880s took longer time, and the 1880s

[196] The same argument has been made by Andrew Cunningham regarding the plague. See: Cunningham [1992]

was certainly not a decade of a rapid and universal reductionist trajectory in medicine. Rather, the immediate consequence of bacteriology was that it opened for a plethora of new ways of conceptualizing disease. Likewise, Cunningham is correct in pointing out that by the 1930s medical understanding of disease was transformed. Diseases now had a clear cause.

The introduction and interpretations of bacteriology changed over time and differed from place to place and from actor to actor. Not only did it take time before the new ways of knowing were made available, simply reading about new theories was not enough for everyone to be convinced. As I will expand upon later, it does seem that the medical elite in Britain were slower and more reluctant in accepting the bacteriology than similar elites in continental Europe and in the colonies, at least when it came to leprosy. At the same time, even those who accepted the bacillus as relevant only later dated its genesis not to when it was introduced locally, but to the first time it was observed. Agreeing on a common genesis seems to have been a shared trait in the medical community.

To the physicians in the 1920s, the late 1870s and early 1880s were indeed seen as a time of medical revolution. The explanation lays not only in the numerous new medical techniques or the rise of new specialties, but in the practice of backdating. When Hansen and Looft in 1895 dated the discovery of the leprosy bacillus to 1871, they labeled all medical observations made earlier as belonging to the 'pre-bacillary era'. Likewise, as the presence of the leprosy bacillus from turn of the 1890s was increasingly accepted as a more definite proof of disease than diagnosis based on clinical judgment alone, the genesis of this new way of understanding disease was backdated it to its origins in the 1870s and 1880s.

The 'revolution' happened over decades. Despite the development of new staining techniques continuing into the 1900s, this too was seen as part of the changes in the late 1870s. In hindsight 'bacteriology' and the 'bacteriological revolution' became the catch-all concept to give meaning to the change that had taken place.

2002: 209-244.

Already from the first decade of the 20th century, the understanding that there had been a bacteriological revolution, starting in the 1880s, had commenced.

3. Treating leprosy: Care or cure?

A quarter past nine in the morning on Tuesday January 16, 1891, 26 year old Johan P. S., affected with 'tuberculo-anesthetic' (mixed) leprosy, received an injection of one milligram tuberculin at Lungegaardshospitalet in Bergen.[197] This was the beginning of what became the world's largest tuberculin-trial on leprosy patients in 1891. Over the next three months fourteen patients received injections. The results were disastrous.

This chapter will investigate the medical discussions regarding the treatment of leprosy. What did medicine have to offer those who suffered from the disease? What efforts were made to find new medications, and what impact did the leprosy bacillus have on treatment?

In hindsight the short answer to what medicine had to offer is 'not much'. Most leprosy histories discussing treatment take the successful trial of Promine at Culion in 1941 as their starting point.[198] This was the first time dapsone drugs were used against leprosy, and dapsone is part of the Multidrug therapy that the World Health Organization (WHO) has offered free of charge to all countries where leprosy is endemic since 1995.[199] Since the drugs used earlier followed radically different rationales and thus were not steps on the way to a cure, prior treatments are explicitly or implicitly seen as 'mistakes' or 'failed science'.[200] I will argue for the contrary: This period was one of the most active when it came to developing and testing new

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[197] Danielssen, D. C. "Tuberkulinen (Kochs lymfe) anvendt paa spedalske i Lungegaardshospitalet". *Medicinsk Revue.* 1891: 185.
[198] Faget, Q. Johansen, F. Dinan, J. Prejan, B. and C. Eccles. "The promin treatment of leprosy". *Public Health Report.* No. 58, 1943: 1729-1741.
[199] World Health Organization. "Leprosy elimination". [Online: http: //www.who.int/lep/mdt/en/index.html]
[200] Ironically, the sulfones that Promin was based on were first synthesized in 1908 and published in a chemical journal. (Fromm, E. and J. Wittmann. "Derivate des p-nitrophenols." *Berichte der Deutschen Chemischen Gesellschaft.* Vol. 41. 1908: 2264-2273.) Sulphones were not picked up by medicine until the 1930s. (Gottfried, Wozel. "The Story of Sulphones in Tropical Medicine and Dermatology." *International Journal of Dermatology.* 1989: 17-21; Zhu, Y. Isabel and Matthew J. Stiller. "Dapsone and sulfones in dermatology: Overview and update." *The Journal of the American Academy of Dermatology.* Vol. 45, No. 3. 2001: 420-434.) Since the dapsone drugs were not discussed among leprologists in the period I am interested in, it will not be not part of the story I will tell in this thesis.

remedies. Concurrently, medicine had much to offer – but whether or not the treatments actually worked was contested.

In this chapter I will first unpack the tuberculin trial in Bergen and investigate what happened as the findings were circulated in medical journals. I will then outline the most discussed remedies developed specifically for leprosy both prior and after the bacillus became a target for new medications, and ask how the rationale behind the treatments changed over time. Next, did the treatments work, and to what extent were the medical trials representative of local treatment practices?

Finally, I will investigate how chaulmoogra, an ancient wood-oil from India, around the turn of the 20th century was reinterpreted as a cure instead of a temporary relief. The efficacy of chaulmoogra was at the core of the controversy where leprologists at the end of the 1920s could be separated in two opposing camps: One saw leprosy as curable and argued for voluntary treatment, the other saw the disease as incurable and argued for segregation and prosecution.

I will also show how the 'lepers' were not passive recipients, but active partners in the search for a way to get rid of the disease. Some traveled far in search for treatments that promised a cure, others convinced their physicians to conduct experiments or threatened with violence if treatment was not offered. Yet others ran away when it turned out that the treatments were both painful and failed to make them better.

The tuberculin trials

In 1891 tuberculin was tested on leprosy patients in at least twelve different locations around the globe. The trials were promptly compiled and compared, and neither the rationale nor the outcomes of the trials can be explained based on studying only the sites in which each individual trial took place. The trials will therefore be investigated not primarily as a series of local events, but as expressions of debates which went on in conferences and medical journals. How was the news of the alleged 'miracle cure' spread? How were the experimental observations disseminated, and how did the trial

in Bergen relate to tuberculin trials elsewhere? What happened with how the studies were represented as they were circulated?

The genesis of the tuberculin trials was not the injections in Bergen in 1891, but a presentation at the Tenth International Medical Congress in Berlin five months prior. Arranged the first week of August, the congress was the largest event for medical science in 1890. In total, 5,737 people attended the six day conference; there were eighteen parallel sessions, daily excursions to Berlin hospitals, and daily journals publishing highlights and changes to the program in German, French and English.[201] Afterwards, the proceedings were published in five comprehensive volumes.[202]

The highlight of the conference was the presentation by bacteriologist Robert Koch. Nine years earlier, at the Seventh International Congress in London, he had made his fame by demonstrating a method for cultivating microbes on solid medium. His paper at the first plenary session of the Berlin conference lived up to the expectations. After first presenting the history of bacteriology as fifteen years of progress, he concluded with the ultimate breakthrough: Tuberculin. This was a new substance that had brought the disease process in tuberculosis to a complete standstill both in the Petri dish and in guinea pigs. This was a proof, Koch argued, that it was possible to render disease-producing (pathogenic) bacteria harmless in living organisms without harming the host.[203]

Although Koch refused to disclose how the drug was manufactured, the lecture was an immediate success. This is perhaps not surprising given that tuberculosis was estimated to be the cause of one in seven deaths globally.[204] "Koch's miracle cure"

[201] Hanssen, Klaus. "X. internationale medicinske kongres i Berlin". *Medicinsk Revue.* Vol. 7, No. 9. 1890: 273.
[202] *Verhandlungen des X. Internationalen medicinischen congresses, Berlin, 4.-9. august 1890*.
[203] Koch, Robert. "Ueber bakteriologische Forschung." *Verhandlungen des X. Internationalen medicinischen congresses, Berlin, 4.-9. august 1890*. Bd. 1. 1891: 35-47.
[204] This estimate was made by Koch some eight years earlier, and was widely accepted. Koch, Robert. "Die Ätiologie der Tuberkulose." *Berliner klinische Wochenschrift.* No. 15, 1882: 221-230.

was widely reported in both medical journals and the popular press.[205] Tuberculin showed that bacteriology held the promise of a cure.

Known among physicians as "Koch's lymph" or "tuberculin", the drug was put up for sale on November 13, 1890.[206] Danielssen was among those who sent an order, leading to the clinical trials which started in January 1891. According to Danielssen, the initiative for testing tuberculin at the Lungegaardshospitalet research hospital in Bergen came from the patients themselves who had read about it in the local newspaper.[207]

The stories of Koch's lecture were not the only thing that needed to travel for the trial to take place, so did the drug itself. Just like the news moved through several channels, so did access to tuberculin. Although Koch was the only producer, it was also possible to get hold of his drug via proxy. On December 20, 1890, the Medical Secretary of the National Leprosy Fund in Britain and editor of the *Journal of the Leprosy Investigation Committee* (1890-1891), Phineas S. Abraham, published a small note in the *British Medical Journal* informing that he had received a supply of tuberculin from Robert Koch and was inviting physicians to try it.

> I shall be glad to hear from any medical man who has a case of the kind at present in England, and willing to be treated. A definite effect has, I believe, already been obtained in Vienna by Professor Kaposi in a leper, and I understand that the remedy is now being tried by Dr. Koch's advice, for leprosy in Germany.[208]

Danielssen made two basic assumptions to justify acquiring tuberculin and testing it on his patients, reflecting how clinical and bacteriological arguments were in practice

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[205] Various medical journals printed full translations, abstracts and reprints within weeks, such as *The British Medical Journal* (1890: 380-383), *Deutsche medizinische Wochenschrift* 1890: 756-757, *Medicinsk Revue* (1890: 280-284). For an extensive review of the reception and the following tuberculin-trials on patients suffering from tuberculosis, see Gradman 2009: Chapters III and IV.

[206] Gradman 2009: 126.

[207] Already on August 13, 1890, nine days after the lecture in Berlin, the local newspaper *Bergens Tidende* published a short note about the lecture (1890: 2). Danielssen himself did not attend the meeting in Berlin, but several of his colleagues from Bergen were among the 57 Norwegian physicians who participated and it is not unlikely that they told Danielssen about Koch's lecture. (Hanssen 1890: 273-276.)

[208] Abraham, Phineas S. "Leprosy and Professor Koch's Treatment". *The British Medical Journal.* December 20, 1890: 1438.

entangled. First, tuberculosis and leprosy had "remarkably similar clinical expressions". Second, the tubercle bacillus and the leprosy bacillus seemed identical in the microscope, "a similarity so great, that even Dr. G. Armauer Hansen, the discoverer of the leprosy bacillus who for many years has concerned himself with bacteriological research, no longer is able to separate them".[209] The first argument relied on experience tending to patients suffering from the disease, the second on what had been observed through the microscope. Both the clinic and the laboratory suggested that the mysterious drug developed for tuberculosis would work equally well on leprosy patients. To my knowledge, this was the first time the leprosy bacillus itself was presented as a rationale for a clinical trial, and the first time Danielssen publicly endorsed Hansen as its discoverer.

When the trial started, 75 year old Danielssen was "the grand old man" of leprosy research. For five decades he had tried – and failed – to find a cure for the disease.[210] Danielssen and Boeck's monograph *On Leprosy* from 1847 was still seen as a leading reference work, and was regularly referred to all over the world.[211]

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[209] "(…) en lighed, der er saa stor, at selv dr. G. Armauer Hansen, som er leprabacillens opdager, og som i mange aar har beskjæftiget sig med bakteriologiske undersøgelser ikke nu længer i stand til at kunne adskille dem." Danielssen 1891: 177.

[210] Already in *Om Spedalskhed* (1844) Daniellsen proclaimed that "From now on one of the author's principal activities shall be devoted to leprosy therapy; this is his goal in life, which he with the healthiest of hopes will respond to." (Danielssen and Boeck 1847: 288.) In 1891 Danielssen's colleagues in Norway published a Festschrift, a memorial volume celebrating his 50 year anniversary as a civil servant: *Festskrift i Anlædning af Overlæge Dr. Med. D. C. Danielssen's 50aarige Embetdsjubilæum. Udgivet af Medicinsk Revue.* 1891. Available online: http: //www.nb.no/utlevering/nb/4ce01326e33d24956f3ef1cf91b11ff6. For a list of the honors bestowed upon Danielssen, see: Brunchorst, J. *Bergen Museums Aarbog 1893. D. C. Danielssen. A biographical sketch.* 1894. Karen Helle is currently working on a biography of Danielssen.

[211] While a common reference, the interpretations of its importance differed. In *Treatise on Diseases of the Skin for the use of Advanced Students and Practitioners* by Philadelphia dermatologist Henry Weightman Stelwagon, for instance, Danielssen and Boeck was presented the primary reference work on diagnosis of leprosy i until at least its 7th edition. (1914: 914). In *A Handbook on Leprosy* (1896: 19), medical superintendent Samuel Patton Impey at Robben Island Leper and Lunatic Asylum referred to Danielssen and Boeck as the authority when it came to deciding the relative frequency of anæsthetic, tubercular and mixed cases. This distinction was introduced by the two Norwegian physicians in 1847, and was and in general use until the 1920s. Despite the fact that Danielssen at this point had acknowledged the leprosy bacillus as the cause of the disease, *The British Leprosy Commission in India* (1893: 204ff), put Danielssen and Boeck forward as the main proponents of the theory of heredity. Later Danielssen and Boeck's theory of heredity was highlighted and ridiculed by Leonard Rogers and Ernest Muir as "strongly supported on very inconclusive evidence by the Norwegian authorities Danielssen and Boeck in their work of 1848". (Rogers and Muir 1940: 64.) After the entry on leprosy was rewritten for the eleventh edition of *The Encyclopedia Britannica*, Danielssen and Boeck's monograph was highlighted as one of the "primary treatises on modern leprosy in particular locations." (*Encyclopedia Britannica.* Volume XVI, 1911: 481.)

The trial at the Lungegaarden research hospital consisted of injections every morning, starting at 1 milligram per dose and increasing over time to a maximum of 320 milligrams. Temperature and pulse were measured three times daily for as long as the trial ran. These observations and the patients' own experiences made up the main bulk of Danielssen's study.

Johan P. S., who had been admitted to the hospital only three months prior and was among those advocating that tuberculin should be tested, received his second injection on January 17. The dose was doubled to two milligrams, and the drug caused a reaction: A strong headache and pain in the neck was accompanied by a fever rising to 39.5° Celsius (103.1° Fahrenheit) mid-day. The following day, the temperature rose to 40.4° C (104.7° F). The nodules in the face turned red with inflammation, some of them burst with seropurulent liquid oozing out.[212] When his part in the trial was terminated at his request five weeks later, Johan could no longer walk; the leprosy nodules on the face, arms and legs had increased both in number and size.[213]

One by one, the trials were aborted, either by Danielssen or the patients themselves, in several cases to avoid the loss of lives. In the case of a 20 year old woman, Ingeborg A. T., the trial was terminated by Danielssen in March. "Continuing the treatment was out of the question; I am convinced that continued treatment would have led to a fatal result."[214] For 31 year old Inga A. H., who had interrupted her other medication to be part of the tuberculin-trials, the first reaction came after five weeks when the doses had reached 15mg: Heat spells followed by chills, nausea and red-brown patches spreading over the face and arms. Over time, the symptoms grew only worse. "The hope I once had regarding her cure is extinguished. Worse, this is thanks to tuberculin", Danielssen concluded.[215]

The last trial was stopped April 15, 1891. By then it was clear that the effects of the drug were immediate, generalizable and relatively consistent: Between four and

[212] Danielssen 1891: 180. Seropurulent: Liquid consisting of both serum and pus.
[213] Danielssen 1891: 185.
[214] "Der kunde ikke være tale om at fortsætte med behandlingen; thii det er min overbevisning, at en fortsættelse vilde have ført til en dødelig udgang." (Danielssen 1891: 196).

six hours after the injection (occasionally delayed up to three days), patients would develop a strong fever, new eruptions of leprosy would appear, and over time the health of the patient would dramatically deteriorate. In some cases, the patient would develop a resistance to the drug, but this seemed to have no impact on the progress of the disease.

Danielssen's conclusion did not question the premise that leprosy was caused by a bacillus, nor that 'Koch's lymph' reacted with this bacillus. Rather, he strongly warned against using tuberculin *because* of this connection.

> The tuberculin does not kill the leprosy bacillus, but it seems to provide good nutrition to the bacillus, so that it with increased vigor enters the bloodstream, travels around in the body and with larger productivity spreads its destruction faster and with greater intensity than had the disease been left to itself. (…) After a time, a sort of immunity to tuberculin can be developed, but this does in no regards bring a stop to the disease. No, the leprosy bacillus is very well under this apparent immunity. It quietly produces its destructive poison, and the effects are visible in the increase of disease phenomena.[216]

By accepting the bacillus as the cause of the disease, it was also the correlating explanation for the clinical outcomes of the use of Koch's drug.

Spreading the results

Danielssen's study was initially published as a 63-page report in the Norwegian journal *Medicinsk Revue*, and then in a two-part series the German *Monatshefte für*

[215] "Det haab, jeg engang kunde have om hendes helbredelse, er ganske udslukt. Tuberkulinen har desto verre sørget derfor."(Danielssen 1891: 222).

[216] "Af de ovenanførte i Lungegaardshospitalet anstillede forsøg af prof. Kochs lymfe fremgaar: (...)
At tuberkulinen ikke dræber leprabacillen, men at den synes at være et godt fodringsmiddel (eller om man vil et godt livsmedium) for bacillen, saa at denne med vigør og større produktivitet vandrer ved blodinokulationen omkring i legemet og anretter sine ødelæggelser hurtigere og med større intensitet, end om sygdommen overlades til sig selv.
At der, efter at tuberkulinen har været benyttet en tid, kan oppstaa en slags immunitet, men at denne ingenlunde bringer stans i sygdommen, eller er en følge af, at leprabacillen er tilitetgjort. Nei, leprabacillen

praktische Dermatologie.[217] Both publications focused on Danielssen's bedside experiences during the trial, detailing observations of each individual patient; body temperature, pulse and clinical signs, as well as how the patient's outlooks for cure changed over time. The conclusion was phrased as a warning to other physicians stating that the drug was dangerous and should not be used.

Danielssen's choice of publishing the results of the trial in both the Norwegian and the German journal was not arbitrary. The Norwegian journal was founded by the research community in Bergen that Danielssen himself had been a key figure in establishing, and its main asset was the 148 different medical journals that the library at the research hospital subscribed to.[218] Not everybody read Norwegian. While French was the 'international' language of choice in around 1850, German had by the 1890s taken over, at least for the physicians in Bergen. 86 out of the journals in the library catalogue were in this language.[219] The *Monatsheft* (established in 1882) was not the only journal publishing clinical and laboratory experiments on skin diseases, but it followed tuberculin especially close. In spring 1891 the German journal published 35 studies on the effects of the drug in various diseases. In addition, Danielssen knew some of the editors personally.[220]

Outside the medical conferences, medical journals were the fastest way to reach colleagues domestically as well as abroad. First, they reached the immediate subscribers. Second, the studies were referred to in other journals. Most of the 35 reports on tuberculin in the *Monatsheft* were abstracts of papers published in other

befinder sig meget vel under denne tilsyneladende immunitet, og producerer i al stilhed sin ødelæggende gift, hvis virkninger viser sig i den stadige tiltagen af sygdomsfænomenerne." (Danielssen 1891: 240).

[217] Danielssen, D. C. "Tuberculin im Lungegaardshospital gegen Lepra angewendet." *Monatshefte für praktische Dermatologie.* Vol. XIII, No. 3 and 4. 1891.

[218] Dethloff, H. G. *Katalog over Lungegaardshospitalets bibliothek ved udgangen af aaret 1904.* Bergen. 1905. More on *Medicinsk Revue* and circulation of knowledge through medical journals in Chapter 6.

[219] The second largest category was "French, Italian and other" journals (23 titles), followed by "Scandinavian, Danish and Finish" (22), and "English and American" (17).

[220] The main editor, Paul Gerson Unna, visited Danielssen in Bergen in 1884. (Romiti, Ney. *Anais Brasilieros de Dermatologia.* Vol. 80, No. 1. 2005: 89.) While it is unclear if co-editor Hans von Hebra ever visited Bergen, his father Ferdinan Ritter traveled there in 1852, when Hans von Hebra was five years old (http://www.whonamedit.com/doctor.cfm/708.html). The third editor, Oscar Lassar, was the one unveiling the memorial for Danielssen after his death in 1894. (Lassar, Oscar. "Zur Erinnerung an Daniel Danielssen". *Dermatologische Zeitschrift.* Vol 2, No. 5. 1894: 534-544.)

journals. Also for Danielssen's report, this was how the results were disseminated – through abstracts in other journals.

The first English-language reference to the tuberculin trials in Bergen was published as a 200-word note in *Weekly Epitome of Current Medical Literature,* a supplement to *The British Medical Journal*, in the end of August 1891.[221] In December, a 6-point conclusion was published by Dr. P. Ferrari in the short-lived London-based *Journal of the Leprosy Investigation Committee*.[222] The following spring the result of the tuberculin trial in Bergen was referred to in *The Lancet* and put in context with other trials, book reviews, official reports, and abstracts from discussions on the use of tuberculin against leprosy in various medical meetings.[223] Danielssen's trial was also highlighted in Paul von Baumgarten's *Jahresberichte über die Fortschritte in der Lehre von den pathogenen Organismen* for 1891, published two years later.[224] As the distance in time increased, the abstracts were increasingly void of all traces of local context. The names or daily experiences of the individuals taking part in the trials never travelled past the original publication. The clinical gaze, local particularities, or the voices of the sufferers were not mentioned; only the generalized findings remained: 14 'lepers'; 0,001-0,320 gram doses; reactions after 12, 48 and 72 hours; bacillus detected in the blood.[225]

While Danielssen's trial was the largest, he was by far the only one testing tuberculin on leprosy patients. In 1891 alone, tuberculin-trials on leprosy patients were performed in at least twelve different locations on three continents: Bergen,

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[221] Editorial. "Tuberculin in Leprosy". *Supplement to the British Medical Journal.* August 29, 1891: 69-70. A 120-word abstract of the second part of Danielssen's German paper was referred to in the same supplement on November 28, 1891: 174.
[222] Ferrari, P. "Koch's Tuberculin in Leprosy". *Journal of the Leprosy Investigation Committee.* No. 4, December 1891: 46-47.
[223] Editorial. "The tuberculin treatment of leprosy". *The Lancet*. April 16, 1892: 877-879.
[224] Baumgarten, Paul von. *Jahresbericht über die Fortschritte in der Lehre von den pathogenen Mikroorganismen umfassend Bakterien, Pilze und Protozoën.* Vol. 7. 1893: 284-285.
[225] Currie, Donald H., Moses T. Clegg and Harry T. Hollmann. "XV. Attempts at specific therapy in leprosy." *Public Health Bulletin No. 47: Studies upon Leprosy.* 1911: 25. Removing traces of local contexts in concurrent summaries was characteristic for abstracts as a genre, see: Chapter 5. That the tuberculin trials were revisited by researchers on Hawaii twenty years later shows how published research did not merely have concurrent interest, but also created an archive for later reference. On the other hand, only what was considered relevant to the research question at hand was referred to – in this case attempts at developing a specific immunity to leprosy.

Bucharest, Hamburg, Hong Kong, London, Madeira, Madras, Mauritius, New York, Tallinn, Tokyo, and Tunisia. Initially the results differed greatly.

Reporting in *Deutsche medizinische Wochenschrift,* Dr. Truhart in Tallinn noted that he had tested tuberculin on six cases, and found that leprous ulcers healed promptly using tuberculin, more so in tubercular cases than in mixed or nerve cases.[226] In an attempt to use tuberculin reaction as a tool for differential diagnosis, Eduard Arning tested the drug on two cases of anaesthetic leprosy and one case of lepra tuberosa in Hamburg. The anaesthetic cases had no reaction to the drug, while the man suffering from lepra tuberosa developed a strong fever.[227] Publishing in the same journal, physicians Babes and Kalindro in Bucharest tested in total eleven cases with mixed results. In two cases the reaction was so severe that it threatened the life of the patients.[228] For the nine others, local reactions about nodules were interpreted as signs of improvement, but none of the patients progressed to a complete cure.

At the Lazzaretto Hospital in Madeira, Medical Superintendent Julius Goldschmidt first tested five cases in May 1891, and was hopeful that it would result in a cure. After a second trial on eleven more cases, he concluded in the columns of *Berliner klinische Wochenschrift* that the best possible outcome was that the disease remained stationary. In most cases the treatment made the health of the patient deteriorate.[229] In Hong Kong, James Cantlie found bacilli in the blood of the patients

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[226] Truhart. "Ein Beitrag zur Leprabehandlung mittels Tuberkulin." *Deutsche medizinische Wochenschrift.* 1891, No. 36-38.

[227] Arning, Eduard. "Über die wirkung des Kochschen Heilmittel bei Lepra und Lupus erythatiodes". *Deutsche medizinische Wochenschrift*, 1890, no. 50; Arning, Ed. "Mittheilungen über Versuche mit der Koch'schen Injectionsflüssiggkeit bei Lepra und Lupus erythematodes." *Robert Koch's Heilmittel gegen die Tuberculose*. 2. Heft, 1890:63-70. The conclusions were summarized in in: *Monatshefte für praktische Dermatologie* 1891: 101-102.

[228] Babes and Kalindro. "Resultate, erhalten durch Injectionen mit Koch'scher Lymphe bei den verschiedenen Formen der Lepra." *Revue de Médecine*, October 1891; Babes and Kalindro. "Ueber die Wirkung des Koch'schen Heilmittels bei Lepra." *Deutsche medisinsche Wochenschrift.* 1891, No. 3; Babes and Kalindro. "Zwei Fälle von mehrere Wochen lang anauernder Allgemeinureaction bei Leprösen nach einmaliger Einspritzung von 0,8 mg Tuberkulin." *Deutsche medisinsche Wochenschrift.* 1891, No. 14. See also: *Monatshefte für praktische Dermatologie* 1891: 243-244, 499.

[229] Goldschmidt, J. "Wirkung des Tuberkulins auf Lepra". *Berliner klinische Wochenschrift.* 1891, No. 15; Editorial. "The tuberculin treatment of leprosy". *The Lancet*. April 16, 1892: 878.

after administering injections to seven patients, and dryly noted that this was not a promising sign.[230]

Tuberculin was also tested at several sites in India. The largest trial was conducted by Surgeon-Major H. D. Cook at the Government Leper Hospital in Madras at the request of the Leprosy Commission to India. He treated twelve patients beginning June 27, 1891, and concluding after a month that "'Tuberculin' for leprosy is a decided failure."[231] At the same time, reports referred to in *The Madras Times* and Calcutta *Daily News* in the end of October 1891 stated that trials elsewhere in India had produced no negative effects, and a slight improvement in health.[232] The reports were picked up by physicians in London, where tuberculin was tested on one patient at King's college Hospital and one patient at University College Hospital.[233]

In Barkly Asylum Public Hospital on Mauritius, Charles Poupinel de Valencé reported to the British Colonial Office that he had tested tuberculin on five lepers, and that three of them had experienced nodules shrinking, improved sense of well-being, increased weight and general improvements in health.[234] This, he argued, was partly due to the treatment being carried out in alteration with large doses of chaulmoogra oil. The report was sent by Lord Knutsfor (Henry Thurstan Holland) to *The Lancet*, where the editor promptly put it in context with other tuberculin trials, including one case at His Imperial Majesty's Army Hospital in Tokyo and two cases treated separately in New York, both previously referred to in *The British Medical Journal*.

The editorial in *The Lancet* stressed that these cases "do not exhaust the list of experiments". Still, it shows how it was only when the results of a wide range of experiments were compiled and compared that larger conclusions could be drawn.

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[230] Seven years later Chantlie was the co-founder, and for the following 23 years main editor, of the *Journal of Tropical Medicine and Hygiene* which was to become the most-referred to journal in *Lepra Bibliotheca Internationalis.* His trial with tuberculin was referred to in: Editorial. "The tuberculin treatment of leprosy". *The Lancet*. April 16, 1892: 879.

[231] "Surgeon-Major H. D. Cook's Report on Tuberculin". *Leprosy in India. Report of the Leprosy Commission in India 1890-91.* Appendix II to Chapter VII. 1893: 371.

[232] According to the editorial in *The Lancet* on April 16, 1892*,* these improvements were soon reinterpreted as the result of improved hygiene for the patients taking part in the trial.

[233] Referred to in: Editorial. "The tuberculin treatment of leprosy". *The Lancet*. April 16, 1892: 878.

[234] The two other cases had refused to carry on after the first injection. The report from Mauritius was what sparked the editorial in *The Lancet* in 1892.

And this was exactly what the *Lancet* did. In an editorial published in April 1892, they argued that the results "are sufficient to show that tuberculin is very uncertain in its immediate effects on leprosy". Relying heavily on Danielssen, their conclusion was to recommend the trials be stopped: "the fact of its action being to set free bacilli rather than to destroy them should make us seek in another direction for remedial agents for the relief of victims of this much-dreaded disease."[235]

While each of these tuberculin-experiments took place in specific locations, neither the rationale nor the outcomes of the trials can be reduced to the local. For the physicians, the medical trials were understood and discussed in context of similar experiments conducted in other locations. The premises for the trials came from a presentation by Robert Koch in Berlin in 1890, and the news were spread through both medical journals and the popular press. The drugs necessary to conduct the trials were dispatched from a single German supplier but could also be acquired indirectly, for instance through Abraham in London. In turn, the outcomes of the experiments were published and referred to in medical journals around the world. Finally, the results were compiled and compared.

Rubbing lotion on the skin

Tuberculin was neither the first nor the last attempt at finding a remedy for leprosy, but most attempts remained local. It was the claim that a cure that was found that got attention. The first claim to receive global attention was a treatment regime developed in in the late 1860s in Cumana, Venezuela, by the French physician Louis Daniel Beuperthy. The three main features of Beuperthy's cure were good hygiene (fresh air, nourishing food including fresh meat and vegetables and abstaining from salted meat or fish), external application of oils (coconut oil, olive oil and cashew nut oil massaged into the skin twice a day followed by baths) and internal medicine (either

[235] Editorial. "The tuberculin treatment of leprosy". *The Lancet*. 1892: 879.

perchloride of mercury or sodium carbonate, both fairly well-known antiseptics).[236] The British physician Dr. Bakewell reported favorable on the cure to both houses of the British parliament in 1871, and Gavin Milroy from the Royal College of Physicians was sent to investigate.[237] When Bakewell in a letter a month later offered Daniel Danielssen to visit Bergen and teach him techniques involved "against a negligible compensation", Danielssen welcomed the visit but refused to offer any payment.[238] From what he had read about Beuperthy's cure, the only real novelty not yet tested in Bergen was the cashew nut-oil, and since Danielssen at this point still believed the disease was caused by an imbalance in the blood (a dyscrasia), rubbing lotion on the skin was at best palliative.

> …none of the numerous irritant agents I have used against leprosy over the years have been able to elicit any changes in the dyscratic conditions which underlie it, and I do not believe – based on my understanding of the disease – that any external remedy will ever be found. The case must differ for Dr. *Beauperthy*, who believes leprosy to be a local disease caused by parasites, and with this understanding pursuing a treatment partly with corrosive and irritant agents, is quite natural.[239]

Beauperthy died shortly after Milroy arrived in Venezuela, and although his alleged cure was tested in various locations and was reported to ameliorate some of the

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[236] *Report of the Leprosy Commission in India, 1890-1891*. 1893: 333-334.

[237] Milroy's work on the Royal College of Physicians' report on leprosy in 1867 had made him an authority on the disease, at least among the British medical and political elites. I will return to this in more detail in Chapter 5.

[238] "Skulde den norske Regjering ønske at se Dr. *Beauperthy's* Behandling forsøgt, vilde jeg være fuldkommen rede til at komme over mod en meget ubetydelig Godtgjørelse for at vise Dem Metoden." Letter from Bakewell to Danielssen, dated Leicester, England, June 17, 1871. In: Danielssen, D. "Lungegaardshospitalets Virksomhed i Treaaret 1871-73". *Norsk Magazin for Lægevidenskaben*. 1874: 320-321. The correspondence also included a letter of introduction from the British physician Jonathan Hutchinson.

[239] "Men hverken det ene eller andet irriterende Middel af de mangfoldige, jeg i Tidernes Løb har anvendt mod Spedalskheden, har været istand til at fremlokke væsentlige Forandringer I de dyskrastiske Forholde, som underholder den, og heller ikke vil, efter den Opfatning af Sygdommen jeg har, et saadant udvortes Middel nogenside findes. Anderledes maa Sagen stille sig for Dr. *Beauperthy*, der antager den spedalske Sygdom for local og foraarsaget ved Parasiter, og at han derfor forfølger den dels med Caustica, dels med lettere Irritamenter, er jo ganske naturligt." Danielssen 1874: 332. Despite the skepticism, based on Bakewell's descriptions from Trinidad and cashew nut oil from an apothecary in Kristiania, Danielssen did indeed test the cure on five patients in Bergen. Beyond causing irritation, he found it to have no apparent effects.

symptoms, the consensus was soon that it fell far short of a complete cure.[240] Still, the premise was made: News of alleged cures travelled fast, and many were eager to try. Beuperthy's cure was to be the first of many.

In 1873, Surgeon-major J. Dougall at Port Blair on the Andaman Islands in the Indian Ocean began experimenting with gurjun-oil made from a local tree, mixed with lime water. Inspired by Beauperthy, the treatment regime consisted of massage and oil applied both internally and externally. Every morning at the break of dawn, 24 patients would be supplied with dry earth for use as soap and sent to a stream to wash. They would then be placed in a row and served a drink consisting of gurjun-oil and lime water in equal proportions.

> Having seen this swallowed by all of them, the apothecary sees that each man has some small vessel (usually half of a coconut shell) into which the compounder puts a quantity of the gurjun ointment, and with this they proceed to rub themselves all over, not merely by smearing it on, but thorough and continuous friction is kept up for two hours, and the compounder all the while is walking up and down the line armed with a tin of the ointment and a spatula for distributing more of it as it is required amongst the patients, and no limit is placed upon the amount given further than whatever is given must be well rubbed in.[241]

The same prolonged rubbing was repeated for two hours every afternoon. Although Dougall explicitly considered the patients malnourished, he made no changes to the diet "in order to avoid complications and to test the gurjon oil in its merits". After testing the treatment for six months, he concluded that the treatment should be recommended to others:

> The time has been long enough to show that leprosy, both tubercular and anæsthetic, can not only be arrested, but the condition of the lepers can be greatly ameliorated; and men here

[240] Buckingham 2002: 135-140; Edmond 2006: 61-62.
[241] "Dr Dougall's Treatment with Gurjun Oil." In: *Leprosy in Foreign Countries.* 1886: 75. The report was first published in Calcutta in 1875. See also *Report of the Leprosy Commission in India, 1890-1891*, 1893: 335-338.

> who have not for years been able to do more than drag out a miserable helpless existence are now able and willing to work, and every sore is quite healed.[242]

Being both inexpensive, readily available and apparently of great use, gurjun oil was soon tested in Madras and other parts of India, as well as in other parts of the world.[243] Surgeon C. T. Peters at Belgaum near Goa in Western India, for instance, tested first Beauperthy's cashewnut oil and then Dougall's gurjun on 29 cases. He concluded that while consuming gurjun produced indigestion in some, diarrhea in others, it had value when applied to the skin externally. His assessment of the treatment indicates how its effect on the disease was measured under clinical diagnosis, namely by the ability to produce visible changes in the skin: Chronic leprous ulcers rapidly healed, and although the gurjun did not stop fresh ulcers from appearing elsewhere on the body, these too could be contained and forced to retract using the oil. Finally, the treatment regime introduced much-desired order and cleanliness.

> This emulsion also keeps the skin in a soft condition, and so prevents cracking, which is often the commencement of an inveterate ulcer. It has, moreover, the advantage of keeping away flies which infest leprous patients, and in the absence of cleanliness, give rise to maggots, which increase the extent of the ulcer, but under its use the lepers, as well as their rooms, presented a very clean appearance.[244]

It did not take many years before the immediate optimism waned. While many reported that more or less relief had been obtained by the use of gurjun, none were cured. When The Leprosy Commission to India summarized the trials in the early 1890s, they concluded that gurjon oil could produce some temporary relief, but found

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[242] "Dr. Dougall's Treatment with Gurjun Oil." In: *Leprosy in Foreign Countries.* 1886: 76.
[243] For instance, two 500-pound barrels of gurjon were shipped to Bergen in 1875, in the hope that the Norwegians would give the oil a trial. (Buckingham 2002: 141.) For more on Dougall's treatment, see Buckingham 2002: 88-91.
[244] Peters, C. T. "Extracts from report on cases of leprosy treated". In: *Leprosy in Foreign Countries.* 1886: 69-70.

it doubtful that this was more than could be obtained by skin-massage and cleanliness alone.

> Dr Dougall's success at Port Blair was probably due to the fact that he was dealing with a colony of convict lepers under prison discipline, and was, therefore, able to carry out his plan of treatment far more rigorously than in an asylum where, as a rule, patients are adverse to any therapeutic measures which involve the least exertion. It is quite possible that the prolonged frictions with dry earth, and the regular sea-bathing, were quite as potent in affording relief as the use of the gurjun oil.[245]

This was also the verdict for cashew nut, gurjun, kowti, chaulmoogra and other plant oils that were tested, as well as German dermatologist Paul Gerson Unna's treatment introduced in 1886, consisting of ichtyol and resorcin. Medical treatment gave some relief, but "the incurability of leprosy by any means yet known is unfortunately certain."[246] The same could be said about the treatment introduced in 1890 by physician Adolpho Lutz at Hawaii, which consisted of oral administration of a mix of the antipyretic (feber-reducing) salicylate and the newly developed chemical compound salol, and rubbing the organic powders chrysarobin and pyrogallic acid on the skin. The treatments reportedly did some good in ameliorating the disease for some, but did not produce cures.

Still, the Leprosy Commission to India firmly concluded that oils, hygiene, ventilation, sanitation, as well as improved diet made the disease run a milder course, compared to no intervention at all. Since all these measures would be easier to administer in asylums, this was used as an argument for establishing voluntary asylums throughout India where treatment could be administered under careful supervision. Although no cure was (yet) available, and the prospect of segregating 100,000 individuals was simply not realistic, voluntary institutions could provide the necessary discipline and patient motivation to test new drugs and treatments.

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[245] *Report of the Leprosy Commission in India, 1890-1891*, 1893: 337.
[246] Op. cit: 331.

Serum failure and a new benchmark

The serum therapy developed by physician Juan de Dios Carrasquilla in Bogotá, Columbia, would follow the same pattern as the other proposed cures. First, the drug was developed and tested locally. Then, it was published in a medical journal and picked up by physicians elsewhere. Next, the drug was distributed either directly to other locations, or manufactured based on the initial descriptions. The drug was then tested, the experiences compared and discussed – and finally, it turned out that the drug did not live up to the expectations. Still, the failure was important in that it would introduce a new benchmark for future treatments.

While lotions and orally administered drugs combined work in the clinic and chemical laboratories, serum therapy was an outcome of bacteriology. Introduced in the early 1890s, the ruling principle of serum-therapy was that bacteria produced poisons and these in turn produced disease. By injecting processed serum under the skin or directly into the bloodstream, the bacilli's ability to produce the poison could be stopped. German physiologist Emil Adolf von Behring's discovery of diphtheria- and tetanus antitoxins in 1890, which quickly led to the development of a serum-therapy that cured the diseases, led to widespread optimism that similar strategies could be applied to other illnesses.[247]

Carrasquilla's serum was tested by himself and five medical students at the newly opened government-sponsored Serotherapeutic Institute in Bogotá in 1896.[248] The first results presented in the Columbian medical journal *Revisita Médica* in 1896, including information both on how the drug was manufactured, how it was administered and the results of treating first fifteen, and then one hundred cases. The drug was based on serum from blood from leprosy patients, ideally tapped during a fever attack. The serum was then injected under the skin (subcutaneously) of horses,

[247] In 1901 von Behring would be the first recipient of the Nobel Prize in Medicine for his this research.
[248] Obregón, Diana. "Building National Medicine: Leprosy and Power in Colombia, 1870-1910". *Social History of Medicine.* Vol. 15, No. 1. 2002: 102. See also: Obregón, Diana. "Lepra e investigación bacteriológica en Colombia: los casos de Carrasquilla y de Lleras." *Biomedica : Revista del Instituto Nacional de Salud.* 2000: 181-189.

mules or asses, causing a fever that would last for up to five days. After repeating the injections at least three times in ten-day intervals, the animals were bled and serum again separated from the blood. This serum was then injected subcutaneously into the persons suffering from leprosy. Two to six hours after each injection, the patient would develop a fever with chills, anxiety, headache, thirst and 'general malaise'. After two hours the fever would enter a 'hot stage' with hot and cold flushes, stronger fever, increased pulse and breath, anorexia (loss of appetite), fatigue and occasional delirium. This would in turn be followed by a phase of sweat, and the patient would usually recover within a day. The injections were to be injected every three to five days, with doses increasing from 1 ccm to 5 ccm or more.

The results of the first hundred cases under treatment, Carrasquilla reported, were wonderful. Tubercles would soften, flatten and finally be absorbed and heal like normal wounds. Leprous patches would be replaced by normal skin, skin color would return, eyebrows and other hair would grow back. Anaesthesia would vanish, nerves return to normal, sight restored to those previously blind, the sense of taste and smell would be regained. Appetite, digestion, sleep, health – all would improve. And once the treatment had begun, no new manifestations of the disease would ever occur. In August 1896, *Revisita Médica* reported that the announcement had led to "Lepers flocking to Bogota" in order to receive the treatment.[249]

In addition to the reports of astounding success, and serum-therapy being in vogue thanks to the promising results in diphtheria and tetanus, it was active promotion by the New York-based physician Albert S. Ashmead that helped spread the news of the new therapy.[250] In January 1897 Ashmead received a shipment of seventy-two bottles of the serum from Bogotá, and quickly forwarded portions to Hansen in Bergen, Oscar Petersen in St. Petersburg, and the President of the Board of

[249] As reported in: "Lepers Flocking to Bogota". *The Journal of the American Medical Association.* December 26, 1896: 1356.

[250] Ashmead was told about the experiments already in 1895 by the U.S. Minister Luther F. McKinney when investigating the prehistory of leprosy in the Americas. McKinney, in turn, had been informed of the experiments by a private physician who had contacted Carrasquilla after reading about it in a local newspaper in Bogota. See: Ashmead, Albert S. "Serum-Therapy in Leprosy" *The Journal of the American Medical Association.* December 7, 1895: 1008.

Health on Hawaii for them to experiment with and report at the upcoming leprosy congress.[251] Carrasquilla also shipped the drug to Walter Herschel Atherstone and Robert Sinclair Black at Robben Island; A. C. Smith in New Brunswick; Ludwig Brieger and Fausto Buzzi in Berlin; Phineas Abraham and Lawrence Herman in London and Eduard Arning in Hamburg, who all tested it on up to ten cases each. In Paris, a committee consisting of Jean Alfred Fournier, Ernest Henri Besnier, Pierre Paul Émile Roux and François Henri Hallopeau was set down to treat nine cases and evaluate the results. In Dorpat, Karl Gottfried Konstantin Dehio manufactured the serum using a horse and tested it on eighteen cases. Also using a horse and the directions Carrasquilla had published, Frank Tidswell in New South Wales, Australia, tested the serum on two cases. In Algiers, Louis Barillion used an ass to create serum and tested it on two men suffering from leprosy.[252]

At the first international leprosy conference in Berlin in 1897, the treatment was generally considered a failure.[253] In South Africa, there were no results, nor in Algiers, Berlin, Dorpat or New South Wales. In the Paris-trial four of the nine patients saw some improvements but this was considered nothing more than the usual fluctuation of the disease. This was also the case in Hawaii, where Louis F. Alvarez saw some improvements in two out of fourteen cases. In a special correspondence from "The Berlin Leprosy Conference" in *New Orleans Medical and Surgical Journal* for December 1897, Isadore Dyer summarized:

> Excepting his own countrymen, no results were reported from the use of his discovery, and the Germans, the English and others came down in full force to deny not the usefulness but

[251] Ashmead, Albert S. "Antileprous serum". *The Journal of the American Medical Association.* January 27, 1897: 181. See Chapter 6 for more on Ashmead's role in preparing the 1897 leprosy congress.
[252] Carrasquilla, Juan de Dios. "Memoria sobre la Lepra Griega en Colombia." *Mitteilungen 1897*. Bd. 1, Abt. IV. 1897: 81-124; Discussion. "Die Therapie, insbesondere Serotherapie". *Ibid*. 1897: 145-160; Thumpson, Ashburn. "Serum treatment in Leprosy". *Lepra Bibliotheca Internationalis.* 1900: 141-148. Smith, A. C. "Report on the Lazaretto Tracadie, New Brunswick." *Lepra Bibliotheca Internationalis.* 1900: 232.
[253] The final resolution from the Berlin Conference simply stated: "The treatment of Leprosy has only had palliative results up to the present time. Serum therapy has so far been unsuccessful." *Mitteilungen 1897*. Bd. 2. 1897: 191-192.

> to argue dangers from its employment (…) Poor Carrasquilla, to have travelled so far and yet found so little at the end of his journey.[254]

The main objection to Carrasquilla's serum would however mark a new standard for justifying the treatments: The problem with the serum was not just that it failed to produce the promised cure, but that there was no reason why it should work at all. This was first argued by Ludwig Brieger who tested the serum on two cases in Berlin and found that the injections had no impact what so ever. Taking serum from a human and incubating it in a horse for a month, Brieger argued, did not make any immunological sense: All experience pointed at antitoxins injected in animals quickly diminished. And since it had been impossible for anyone to detect toxins produced by the leprosy bacillus, how could one assume that the serum that was injected in the animals had contained any antitoxins in the first place?[255] Thus, in line with other bacteriological knowledge, the ability to act directly on the leprosy bacillus was introduced as a new benchmark for future treatments.

Summarizing the experiments in the columns of *Lepra Bibliotheca Internationalis* in 1900, John Ashburton Thompson, president of the Board of Health in New South Wales, had two explanations for why Carrasquilla's serum quickly lost interest. First, Carrasquilla appeared to have had no knowledge about proper serum-production and consequently had taken no precautions against possible contamination when producing the serum. Second, even if the leprosy bacillus did produce a poison which so far had eluded detection, nothing indicated that it could interact with bodies of other animals. In short, failure was to be expected: If the treatment actually worked, it would be a breakthrough in a range of fields at once.[256]

It was the lack of specialist skills and an explanation for how the drug theoretically interacted with the leprosy bacillus that made the physicians declare the

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[254] As quoted by Ashmead, Albert S. "Poor Carrasquilla." *The Journal of the American Medical Association.* February 5, 1898: 330. Ashmead, the one who had introduced Carrasquilla to many of the physicians who tested the drug, appears to have been alone in publicly defend the Columbian from the mocking reception of his alleged cure.

[255] Brieger, Ludwig. "Die Therapie, insbesondere Serotherapie", *Mitteilungen 1897*. Bd. 2. 1897: 155-156.

serum therapy a definite failure. As one would expect, the justification for the next set of anti-leprosy drugs was that they interacted directly with the leprosy bacillus.

Leprolin: Tuberculin for leprosy

The first drug aimed specifically at the leprosy bacillus was 'Leprolin', developed by Captain E. R. Rost working in Rangoon, the capital of British Burma, in 1904. The drug was inspired by Robert Koch's tuberculin, and a direct outcome of experiments with cultivating the leprosy bacillus.

Experiments aimed at growing the leprosy bacillus outside the human body had begun in the 1880s. Bacilli were taken from leprosy sufferers, both fresh and old cases. The bacilli were then put in a dish containing nutrient media, ranging from agar to blood serum, from boiled flesh to potatoes, and then stored under different temperatures for varying amounts of time.[257] Despite occasional claims that the experiments were successful, these proved hard to reproduce. Koch's four postulates for proving that a bacillus was causing a disease were never explicitly discussed. Instead, the case for the bacillus being the cause of the disease (and not an outcome) was in the 1880s and 1890s made by frequent analogies to the tuberculosis bacillus, which shared many of the same properties and was made visible through the same techniques for staining.

During its existence, the medical journal *Lepra Bibliotheca Internationalis* published 29 papers on cultivation, about 3.5 percent of the total number of papers. Again the occasional reports of successful cultivations proved difficult – if not impossible – to reproduce. Likewise, the results of attempts at inoculating animals with the cultivations were inconsistent.

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[256] Thompson, Ashburton. "Serum Treatment of Leprosy". *Lepra Bibliotheca Internationalis.* 1900, Fasc. 3: 141-147.

[257] For an extensive overview of the early cultivation attempts, the cultivation medias used, as well as the rationales and outcomes of the experiments, see: Scholtz, W. and Viktor Klingmüller. "Über Züchtungs-Versuche des Leprabacillus und über sogenanntes 'Leprin'." *Lepra Bibliotheca Internationalis.* 1900: 93ff.

In 1902, lecturer at the pathological department of the University of Moscow W. J. Kedrowsky introduced the argument that the leprosy bacillus had morphology of its own.[258] The acid-fast bacilli made visible in the microscope through stained samples from leprosy sufferers were but a certain stage in the development of the bacillus, Kedrowsky withheld. This explained the inconsistencies in the attempts at cultivating the bacillus outside the human body: Even if the samples selected for cultivation contained the end-stage of the bacilli, it was not given that they contained the bacilli in its fertile stages. As physician H. Bayon later summarized: "Once it is admitted that Hansen's 'bacillus' is but the end phase in the tissues of a filamentary and branching organism, a quantity of bacteriological and also clinical observations finds an easy and satisfactory explanation."[259] This debate continued into the 1920s.

Rost had a different explanation: The reason why most cultivation attempts failed was that the most common growth mediums contained chloride salts. This inhibited the growth of the bacillus, Rost claimed. By instead using a medium made by distilled and superheated beef extract, the leprosy bacillus would grow in three to five days "as a white, and later as a yellow or brick red, curly, thick growth on the surface of the agar, very much like the bacillus tuberculosus [sic] grows on the surface of glycerinized nutrient agar."[260] After six weeks, an extract was suspended in glycerin in the same fashion as when producing tuberculin from cultures of the tuberculosis bacillus. Rost named the outcome Leprolin.

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[258] Kedrowsky's first paper was published in Robert Koch and Carl Flügge's *Zeitschrift für Hygiene und Infectionskrankheiten,* No. 1, 1901, and was reprinted under the title "Über die Kultur des Leprabacillus" in *Lepra Bibliotheca Internationalis.* 1901: Fasc. 4.

[259] Bayon, H. "The Present Position of Leprosy Research (Paper read before the Meeting of the British Medical Association at Cape Town October 24, 1912)". *Lepra Bibliotheca Internationalis.* 1913: 54. For a summary of the cultivation attempts in the first decade of the 20th century, see: Bayon, H. "The Micro-Organism of Leprosy, has it been cultivated?" *Lepra Bibliotheca Internationalis.* 1913: Fasc 4.

[260] Rost, E. R. "On the Pathology and Treatment of Leprosy". *The British Medical Journal.* February 11, 1905: 295; Rost, E. R. "The Cultivation of the Bacullis lepræ". *Lepra Bibliotheca Internationalis.* 1905: Fasc 1. The paper was first read before the Burmah Branch of the British Medical Association and reported in the *Indian Medical Gazette* in May and June 1904, before being reprinted in *Journal of Tropical Medicine*, June 15, 1904. The first results of the treatment were printed in: E. R. Rost. "Further notes on the treatment of leprosy by injections of leprolin". *Indian Medical Gazette*, December, 1904, and reprinted in *Lepra Bibliotheca Internationalis.* 1905: Fasc 2. For details on the production of Leprolin, see also: Rost, E. R. "The Cultivation of the Bacillus of Leprosy and the Treatment of Cases by means of a Vaccine prepared from the Cultivations". *Lepra Bibliotheca Internationalis.* 1911: 125-130, originally published in: *Scientific Memoirs by Officers of the Medical and Sanitary Departments of the Government of India.* No. 42. 1911.

The drug was injected either in the arms or the buttocks of leprosy sufferers, and like Carrasquilla's serum it caused a strong fever and high pulse. This, Rost withheld, proved that the drug was working as intended.

> A case of leprosy, after the first injection of Leprolin, will get a severe reaction, which may come on very soon after the injection, the temperature running up to 104° F. [40° C] or higher, the anaesthetic areas becoming red, hot, and swollen, and the pulse and respiration rate going up very soon after injection. (…) As a general rule, the severity of the reaction may be taken as an index of the benefit that is likely to occur, and this in itself is a good sign of the beneficial action of this material in the disease.[261]

After three or four days, the fever would subside and previously anesthetic areas would regain sensation. Over time ulcers would heal, cramps in hands or feet would loosen up, the patients would become more energized; the pain and heavy sensation associated with the disease would disappear.[262] Like most concurrent bacteriological research, exactly how the drug worked in the body was open for speculation. Rost had two competing theories: Either the Leprolin made the body produce antitoxins which in turn acted as an antidote (the 'antautoxic' view), or the Leprolin poisoned the bacteria directly by exposing it to their own excreta (the 'autotoxic' view).

In addition to testing it on 120 patients in Rangoon, where a majority showed signs of improvement and four patients were cured within months, the drug was distributed to thirty different sites in India. But the results were not promising. T. C. Rutherford concluded after treating 32 patients in Bilaspur District in eastern central India with Rost's Leprolin that all but two cases had gotten worse, and that "in at least

[261] Rost 1905: 295-296.

[262] "The most remarkable effect of leprolin on lepers is the suddenness with which the sensation returns in the anaesthetic patches. I know that it is hard sometimes to say that some low-class Indians have anaesthesia or not, but the majority of my patients have been intelligent Burmans, and some Eurasians, and only the minority jungle folk. I have made absolutely certain before injection that the patient could not feel the prick of a pin in certain areas, and I am glad to say that in almost every case injected with this material there has at least been some obvious return of sensation in some anaesthetic area." (Rost 1905: 295-296.)

one of the cases the downward progress was so very marked that it is difficult not to hold the treatment as at least partially responsible."[263]

At the time Leprolin was introduced, several physicians had again resumed experimenting with tuberculin. In 1902, for instance, physician M. Sée in Paris deviced an experiment which would decide if the tuberculin reaction was brought about simply because the leprosy patients had also been infected with tuberculosis.[264] Even in Bergen, the failure of other remedies and occasional positive reports on tuberculin over time leading to improvements in patients suffering both from leprosy and other diseases, led to renewed interest for new tuberculin trials. Three years after the death of Daniel Danielssen in 1894, the Lungegaardshospitalet was closed and both patients and research activities were transferred to the neighboring Pleiestiftelsen leprosy asylum. From the turn of the century, director and physician H. P. Lie at Pleiestiftelsen would repeatedly argue that increased knowledge about doses and possible positive long term effects justified new trials:

> It may perhaps seem useless to make a fresh trial of tuberculin, when both Dr. Danielssen and so many other observers have tried it with such bad results. There are a few, however, whose verdict is not altogether unfavorable; and the experience that has of later years been gained with regard to the employment of tuberculin in tuberculosis, might prove useful in the application of the remedy to lepra.[265]

But, as Lie argued in the annual report for Pleiestiftelsen which in turn was printed in *Lepra Bibliotheca Internationalis*, the trials would have to take place elsewhere: "The tuberculin injections were still too fresh in the recollection of the patients who had

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[263] Rutherfoord, T. C. "Report of Cases of Leprosy treated with Leproline during 1911-12 in Bilaspur District (Mirror)". *Lepra Bibliotheca Internationalis.* 1913: 180, originally published in: *Indian Medical Gazette*. February, 1913, and then *Tropical Diseases Bulletin*. 1913: 564.

[264] Sée, M. "Les traitements de la lèpre". *Gazette des hòpitaux.* Vol. 25. No. 60. 1903: 559-606, as referred to in *Baumgartens Jahresbericht*. Vol. 18, 1905: 373. The claim was contested, see: Satinéano, A and D. Daniélopolu. "Sur les reactions des lépreux à la tuberculine." *Comptes redus de la Société de Biologique.* Vol. 66, No. 25. 1909, as referred to in *Centralblatt für Bakteriologie.* Vol. 45. 1909: 467 ; Babes, H. "Au sujet de la reaction des lépreux à la tuberculine." *Comptes redus de la Société de Biologique.* Vol. 67. 1909 : 411, as referred to in *Centralblatt für Bakteriologie.* Vol. 45. 1909: 555.

[265] Lie, H. P. "Report of the Leper Hospital (Pleiestiftelsen no. 1) in Bergen for the 3 years 1899-1901". *Lepra Bibliotheca Internationalis*. 1903: 19.

come from the Lungegaard Hospital to allow me venturing upon them again."[266] In other words, the same patient body which in the early 1890s had insisted that tuberculin should be tried now, in light of experience, refused further experiments. Lie, who had been appointed doctor at the hospitals in Bergen three years after the trial, tried to convince the patients but was unsuccessful. This is but one of many examples of how the lepers themselves could influence what medical trials to be conducted: By refusal to participate.[267]

The renewed interest for tuberculin helped promote Rost's Leprolin. But despite receiving much attention for the potential cure developed specifically for leprosy, with no other official manufacturer than Rost himself, the availability of the drug was severely limited.[268] The claim that Rost had succeeded in cultivating the bacillus was also controversial.[269] In early 1905, for instance, Rost was invited by director David Semple to the newly opened Pasteur institute in Kasauli in northern India to oversee an attempt at producing Leprolin. Despite letting Rost choose the material and supervise the process, the attempt failed. As Semple concluded:

> …we have ended in proving a negative (…). It is unpleasant, no doubt, to produce a piece of work and find it to be wrong, but that the publication is useless is by no means true. Mistakes may benefit the man who makes them, and profit to many other men who take up

[266] Op. Cit: 20. Lie himself had no direct experience with the drug, since he was employed only in May 1893, after Danielssen's trial had been stopped. In 1904 Lie repeated the call for recommenced experiments with tuberculin as a remedy in *Deutsche medizinische Wochenschrift*. (Lie, H. P. "Die Therapie der Lepra". *Deutsche medizinische Wochenschrift*, vol. 34. 1904 : 1381.)

[267] In 1930, the secretary of the League of Nations Leprosy Commission, Etienne Burnet, summarized: "The majority of the lepers want to be treated and simply ask that the treatment shall not be too painful; they read the papers, never miss any item dealing with leprosy and are on the look-out for the newest remedies. If revolts occur when the lepers are dissatisfied at their compulsory segregation, again cases could be quoted of lepers who have objected because they were not given sufficient energetic treatment (even threatening to kill doctors who were opposed to treatment.)" Burnet, E. *League of Nations Health Organisation. Report on the Study Tour of the Secretary of the Leprosy Commission in Europe, South America and the Far East. January 1929 - June 1930*. (C. H. 887). Geneva, 1930: 43.

[268] In 1910, for instance, the Spanish physicians Juan de Azua and Jose S. Corvisa explained that the reason why they had not tried Leprolin in treating the two hundred known leprosy sufferers around Madrid was that they had not reached the point where they could cultivate the bacillus and thereby produce the drug themselves. (Azua, Juan de and Jose S. Corvisa. "Traitement de la lèpre." *Lepra Bibliotheca Internationalis*. 1910: 148-9.)

[269] Already in 1905, Frank Tidswell from Sydney concluded that they were unable to verify Rost's reported cultivation of leprosy bacilli, and that the premise for producing Leprolin was therefore dubious. Tidswell, Frank. "Note on Rost's 'Achloretic' Culture Medium (Report of the Board of Health on Leprosy in N. S. Wales 1905)". *Lepra Bibliotheca Internationalis.* 1906: 197.

> the same line of enquiry. By the mistakes of ourselves and others we learn correct methods and arrive at accurate conclusions, be they positive or negative, and, from a scientific if not from a remedial point of view, the one is almost as important as the other.[270]

At the second international leprosy conference in Bergen in 1909, physicians Beurmann and Gougerot working in Hôpital Saint-Louis in Paris reported that their initial tests were promising.[271] And should the treatments eventually fail, it was still useful: Since Leprolin was specifically tailored to leprosy, only those actually suffering from the disease would react. Hence, it could be used in diagnosis.[272]

Rost was not the only one trying to produce a tuberculin from cultivated leprosy bacilli. In the fall of 1907, the director at the Pasteur Institute in Tunis, Charles Nicolle, reported in a lecture at the Medical Academy in Paris that their experiments had failed: "our ignorance for what conditions the leprosy bacillus needs to thrive is almost complete."[273] Some years later, three physicians at the Leprosy Investigation Station at Molokai, Hawaii, referred to previous tuberculin trials to justify further experiments into a "vaccine and tuberculin-like extract therapy".[274] Eventually, this attempt failed too.

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[270] Semple, D. "Rost's Leprolin." *Lepra Bibliotheca Internationalis.* 1906: 257, originally published in *Journal of Tropical Medicine*. September 1. 1905.
[271] Beurman, M. de and Gougerot. "Traitement de la lèpre par la lèproline de Rost". *Mittheilungen und Verhandlungen der Internationalen wissenschartliche Lepra-Konferenz zu Bergen, 1909* (Hereafter '*Mittheilungen 1909'*) and *Lepra Bibliotheca Internationalis.* 1910: 301.
[272] Beurman and Gougerot. "Le lèproline diagnostic". *Mittheilungen 1909* and *Lepra Bibliotheca Internationalis*. 1910: 191.
[273] Nicolle, Charles. "Réaction à la tuberculine dans la lèpre (inoculations sous-cutanée, dermique et conjonctivale". *Lepra Bibliotheca Internationalis.* 1908: Fasc. 3, first presented at Acadademie de Médecine, August 12, 1907. Failing to produce a leprosy-tuberculin, Nicolle resorted to the tuberculosis-based tuberculin administered through subcutaneous inoculation, skin-smears and smears on the skin under the eyelids – and found that varying the delivery had no impact on the effects.
[274] Currie, Clegg and Hollman 1911: 23-62.

Nastin over the counter

Collaboration with a professional manufacturer was the secret to success for the German researcher Georg Deycke and his drug 'Nastin',[275] which was produced by the company Kalle & Co in Biebrich on the Rhine from 1907.[276] Deycke's Nastin was developed in parallel to Rost's Leprolin, and was also sparked by attempts at cultivating the leprosy bacillus. Unlike Rost, Deycke concluded that the organism he managed to cultivate was not the leprosy bacillus proper but a characteristic microorganism found in leprous ulcers that he named *Streptothrix leproides*. This microorganism could be cultured in a normal salt solution.[277]

Determining that the organism could be developed into a drug was serendipitous. In an attempt to ascertain the relationship between the culture and the leprosy bacillus, he injected it into a patient suffering from leprosy to see what happened.

> Irrespective of a slight induration [hardening] at the place of injection no result was observed; the patient, however, obstinately maintained that the injection had had a beneficial effect on him, and he was not satisfied until I repeated the injections at weekly intervals. Under this extraordinary treatment the patient, whose disease was severe and accompanied by high fever became completely free from fever, and his extensive leprous symptoms retrogressed so rapidly and in so remarkable a manner that after two months he

[275] Franz Burghardt Georg Deycke from Hamburg developed the drug at the imperial ottoman hospital Gülhane in Constantinople where he worked from 1898 to 1907, from 1903 as its director. Deycke returned to Hamburg in 1907. At the end of 1908 Deycke visited the Public Leper Asylum at Mahaica in British Guiana for six months in order to direct a trial of Nastin on 135 cases, before returning to continue his research in Hamburg. (Editorial. "The Nastin Treatment of Leprosy". *The Lancet.* July 22, 1911: 237-238). The initial research was made in collaboration with Reschad Beyer and published in *Deutsche Medizinische Wochenschrift* in 1905, nos. 12 and 14. See: Deycke, G. "Zur Theorie une Praxis der immunisierenden Behandlung der Lepra mit Nastin." *Lepra Bibliotheca Internationalis.* 1907, Fasc 1.

[276] Deycke, G. "A Lecture on a Specific Treatment of Leprosy". *The British Medical Journal.* April 4, 1908: 802-806.

[277] In the years that followed, several physicians referred to Kedrowsky's model of the leprosy bacillus having its own morphology, and argued that the streptothrix was in fact the leprosy bacillus proper but in a different stage of its growth. As A. G. Foulerton put it in a discussion following Deycke's presentation of Nastin in London in 1912; "the acid-fast forms of a streptothrix represent only a single phase in the life of the organism. Young cultures are not acid-fast; the property is one which is acquired in later growth, and even then is, under the conditions of culture on artificial media, of very variable occurence. In a sense, the acid-fast phase of a streptothrix is an accidental one; the acid-fast type probably representing more resistent forms of the organism." ("Discussion". *Transactions of the Royal Society of Tropical Medicine and Hygiene.* March, 1912: 196.)

> considered himself to be thoroughly cured and left the hospital. Seeing that similar results were also obtained in other cases of leprosy, we naturally made it our task to discover the curative principle contained in the cultures.[278]

The unexplained success meant taking the substance back into the laboratory for further investigations: Testing, filtering and chemical experimentation. The outcome was a substance Deycke named 'Nastin', a fatty acid in a state suitable for injections just beneath the skin (hypodermic injections). Further tests on patients revealed that the responses differed. Some had violent reactions 'dangerous to life'; others showed no reaction at all. Applying the drug to a culture of tuberculosis under the microscope did however give insights into how the drug worked: The Nastin would mix with the fat contained in the bacillus, dissolve it and then leave the bacillus vulnerable. This could also explain why the drug did not work in all cases: It needed a substance to activate the fat-dissolving reaction. After testing several benzoic acids in vitero, again using cultures of the tubercle bacillus to see what best dissolved the fat surrounding the bacteria, he settled on benzoylchloride.

> The nastin is carried to the lepra bacilli, to which, owing to its near chemical and physical relation, it attaches itself, and then benzoyl can fully display its antibacterial action in the fat-removing sense. When deprived of fat the lepra bacilli seem to be doomed; the human organism then effects with comparative ease the further dissolution and ultimately the complete destruction of the bacteria nuclei.[279]

The drug was put up for sale in three varieties containing different mixtures of benzoyl and Nastin, named Nastin-B_0, Nastin-B_1 and Nastin-B_2, in addition to the drug "Lösnung K" which contained pure Benzoylchlorid. The drug was administered by injecting it under the skin of the arms, the hips and the back, producing a burning but passing pain. The injections were initially to be given once a week, gradually increasing to five injections per week. In direct contrast with Rost's Leprolin, a strong

[278] Deycke 1908: 802.
[279] Deycke 1908: 805.

fever reaction was perceived to be a bad sign, and the proscribed procedure was to replace the injections with Lösnung K.

Due to increased availability, Deycke avoiding the controversies regarding culture, positive reports from the first independent trials, being relatively free from ill effects, and the modest insistence that Nastin was not a magical remedy that always worked but that it frequently produced very good results, the drug was tested in numerous locations. In *Lepra Bibliotheca Internationalis* between 1907 and 1915, Nastin was the topic of twenty papers. When including all research on treatment from 1900 to 1915, this made up about a fifth of the total number of papers and almost four times as many as Rost's Leprolin. That failed treatments could be ascribed to the physician neglecting to individualize the drugs correctly, and not a failure of the drug itself, probably also played part in the initial optimism.

In the invitation to the second International Leprosy Conference in Bergen in 1909, Nastin was highlighted as one of the new means which science could offer governments in order to achieve the goal of exterminating leprosy, much like Carrasquilla's serum had been a topic in Berlin twelve years earlier.[280] The session on treatment was opened by Deycke himself, who claimed that Nastin was a specific drug that targeted the pathogen directly; "a significant weapon in the battle against the leprosy epidemic".[281]

Among the first to send in an order for Nastin was Captain T. S. B. Williams of the Indian Medical Service, who worked with leprosy sufferers in Bushire by the Persian Gulf (now Bushehr, Iran). After testing Nastin on twelve cases for up to a year, he reported that seven of the patients had improved dramatically, while the rest had no relapses. In all but one case, ulcers and lepromata (leprous nodules) had steadily improved and the number of leprosy bacillus in skin and nose samples had

[280] "Programme provisoire de la Deuxième Conférence Internationale Scientifique contre la Lèpre devant avoir lieu du 16 au 19 Août 1909 à Bergen, Norvège." *Lepra bibliotheca Internationalis.* 1908, Fasc 2: 125.
[281] "Das Nastin B ist ein spezifisches, d. h. direct die Leprarreger angreifendes Mittel. (…) Mit einem so gearteten Mittel ist uns aber, wie ich überzeugt bin, eine nicht zu verachtende waffe im kampf gegen die lepra als volksseuche in die hand gegeben." . Deycke, G. "Über die Therapie der Lepra". *Lepra Bibliotheca Internationalis.* 1910: 211-222, quote on p. 222. For Deycke, Nastin did not substitute the need for forced

diminished. “Deycke’s brilliant researches have, I am sure, given us at last a specific foundation on which to base our treatment.”[282]

Despite some reservations voiced in the discussion, the final resolution of the Bergen conference rejected the conclusion from Berlin that the disease was incurable.[283] Reflecting the cautious optimism regarding the new drugs, from 1909 the official consensus among the leprologists was that: “The clinical study of Leprosy induces the belief that it is not incurable. We do not at present possess a certain remedy. It is desirable, therefore, to continue the search for a specific remedy.”[284] A winning argument was what the German dermatologist Paul Gerson Unna first had voiced at the Fifteenth International Medical Congress in Lisbon in 1906: If leprosy was considered incurable, further research into finding a cure was per definition meaningless.[285]

Further tests with Nastin, however, produced confusing and conflicting results. In the capital of German East Africa, Bagamojo (in what is now Tanzania), physician Lenz tested Nastin on five patients in 1907, and then twelve more in 1908. Failing to obtain any results led him to the conclusion that the drug might have been damaged during transportation.[286] Likewise, after one year of testing the drug on six patients at the leprosy investigation station at Honolulu and seeing only slight improvement in two of them, Walter R. Brunckerhoff and James T. Wayson sent the drug back to the manufacturer in Europe, asking them if the drug was inactive. The drug was returned

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isolation, but being able to offer treatment and the hope of a cure, he argued, would make such policies easier to implement.

[282] Williams, T. S. B. “Nastin treatment of Leprosy”. *Lepra Bibliotheca Internationalis.* 1910: 261. The paper was submitted to the Bergen Conference, but was not read as Williams was unable to attend.

[283] See Appendix 2.

[284] Lie, H. P. “Internationale Wissenschaftlige Lepra-Konferenz. Abgehalten vom 16. bis 19. August 1909 in Bergen (Norwegen). Mitteilung und Verhandlungen.” *Lepra Bibliotheca Internationalis.* 1910, Fasc 3: 418-419. In addition to Nastin and Leprolin, the German physician Franz Engel-Bey working in Cairo presented his drug Antileprol, a derivate of chaulmoogra oil which from 1908 sold via the German company Beyer & Co from 1908. (Engel-Bey. “Zur Behandlung der Lepra mit Antileprol”. *Lepra Bibliotheca Internationalis.* 1910: 274-290.) More on chaulmoogra and its derivates later in this chapter.

[285] Unna, P. G. “Sur la pathologie et la thérapeutique de la lèpre”. *Lepra Bibliotheca Internationalis.* 1906: Fasc. 3. According to Unna, the prevalent view that leprosy was incurable created a self-fulfilling prophecy which needed to be rejected.

[286] Lenz. “Berich über die Behandlung Aussätziger mit Nastin und Chaulmoograöl”. *Lepra Bibliotheca Internationalis*. 1910: Fasc. 1. In comparison he found chaulmoogra oil to be more efficient.

with a note saying that the Nastin was indeed active and ought to work – given the right doses.[287] At the leprosarium in Kuuda in Estonia, physician Arthur Kupffer recommended using Nastin injections as a supplement only if chaulmoogra oil alone did not produce results.[288]

In many cases the initial impressions were positive, but faith in the drug declined as the trials progressed. Visiting medical officer to the Pretoria Leper Asylum in Transvaal (now South Africa), Gordon B. Messum, put in an order for monthly shipments of Nastin in 1908 and tested it on twenty patients for between six and eighteen months. Initially the results were promising, but after a while doubt set in. Messum's report was one of few occasions where also statements made by the patients themselves were part of the argument:

> It is curious to note that after the first 10 or 12 injections, so many of the patients declared themselves much benefited, and greatly improved in general health. Expressions were used, such as "I now feel much stronger", "I can now use my hands with more grasping power", "I can now walk without getting so easily tired". Whether these feelings were due or suggested by anticipatory expectations of the reputed curative property of Nastin or not, is a question, because many of the patients had been told and had read of some wonderful cures as the result of this treatment, and they may thus have been impressed suggestively. It was clearly noticeable that there was much more cheerfulness amongst them, due to hopeful anticipations, and they were so positive in their statements that I was myself at first carried away on the wave of optimism. In order to test the improvements thus reported, they were at this time thoroughly examined, and I was myself somewhat astonished and taken back to find no definite difference or improvement in them. I did not let them know, however, that such had been my finding, but I was beginning to feel that most were inclined to be impressionists, whose imagination created more than I could discover.[289]

[287] Brinckerhoff and Wayson. "Report upon the treatment of six cases of leprosy with Nastin (Deycke)". *Lepra Bibliotheca Internationalis*. 1910: 228.
[288] Kupffer, A. "Ein Beitrag zur Behandlung der Lepra mit Chaulmoograöl und Nastin". *Lepra Bibliotheca Internationalis*. 1909: Fasc. 3.
[289] Messum, Gordon. "Twenty cases of Leprosy treated simultaneously with Deycke's Nastin". *Lepra Bibliotheca Internationalis*. 1910: 239-240, originally published in *The Transvaal Medical Journal*. May. 1910. See also: Editorial. "The treatment of leprosy by Nastin." *The Lancet*. July 30. 1910: 325-326.

Messum's misgivings only grew stronger when one patient who secretly received saline injections instead of Nastin reported that he too benefitted from the cure. As months went by the early confidence in the cure was replaced by indignation and anger that the drug had left them worse off than before. After a year and a half Messum concluded: "In the treatment of the cases which I have here recorded, I am sorry to say that Nastin has not shown itself capable of producing any definite remarkable results."[290]

Interestingly, the interpretations of a single trial could also differ, such as in the Nastin trial at the Public Leper Asylum at Mahaica in British Guiana between 1908 and 1912 where 135 voluntary patients received injections. The first six months of the trials were done in collaboration with Deycke, who had been invited by the Governor, Sir Frederick Mitchell Hodgson. While Deycke concluded that the injections in almost all cases arrested the progress of the disease, the local physician K. S. Wise concluded that only three were cured, some had gotten worse, and the majority remained in status quo. Finally, in the letter accompanying Wise's report Governor Hodgson claimed that

> (…) there are 56 cases in which no leprosy bacilli can be found, and of this number it is hoped that 75 percent will be fit to be discharged within the next three months. Never before in the history of the Leper Asylum of the British Guiana has leprosy been successful fought, and the results now obtained will doubtless lead to further research and to a more complete conquest of this most terrible of diseases.[291]

After Deycke had left, Wise in collaboration with E. P. Minett continued the trial. Over four years, 244 individuals suffering from leprosy received injections. At first the results were encouraging, but by September 1912 they concluded that Nastin, at best, only offered a slight temporary check to the course of the disease. Ultimately, a

[290] Messum 1910: 240.
[291] Editorial. "The Nastin Treatment of Leprosy". *The Lancet.* July 22. 1911: 237.

control group was given injections of only benzoyl-chloride (Lösnung K designed to activating the Nastin) and ended up producing identical results.[292]

Only months earlier, at the February meeting of the Society of Tropical Medicine in London in 1912, Deycke had enthusiastically referred to the drug being tested on 529 cases in 69 different locations, not mentioning the results from British Guiana as he had been personally involved: 13 were reported as cured, 34 as nearly cured, 131 considerably improved and 154 as improved. Instead of highlighting that only 13 out of 529 cases had resulted in a cure, Deycke stressed that beneficial results were attributed to the treatment in 332 cases (62.76 %).[293] By then, however, Deycke was fighting a losing battle. *The Lancet*, which followed the Nastin-trials closely, concluded in an editorial in July 1910 that the jury was still out: "In our opinion further experience of its use on a large and extended scale in leper establishments is needed in order to supply the evidence on which its acceptance or rejection by the medical profession must ultimately depend."[294] Two years later, they were not convinced: "With all this conflicting evidence before us it is not easy to arrive at a definite conclusion, but in the circumstances we think we are justified in saying that up to the present the evidence put forward in favour of Nastin is hardly convincing enough to lead to its general acceptance."[295]

Nastin did remain in use in certain locations, depending on the local experiences, but most physicians were deterred by the dubious results and high cost.[296] Like Beauperthy's cure, Tuberculin, Carrasquilla's serum and Leprolin before it, the general consensus was that Nastin did not live up to the expectations. Instead, it

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[292] Wise, K. S. and E. P. Minett. "Treatment of Leprosy by Nastin". *Journal of Tropical Medicine and Hygiene*. September 2. 1912: 259.
[293] Deycke, Georg. "Treatment of leprosy by nastin, and results so far obtained by this treatment." *Transactions of the Royal Society of Tropical Medicine and Hygiene.* March. 1912: 168-174, 195-204; Editorial. "The treatment of leprosy by nastin." *The Lancet.* March 30. 1912: 881-883.
[294] Editorial. "The treatment of leprosy by nastin." *The Lancet.* July 30. 1910: 325.
[295] Editorial. "The treatment of leprosy by nastin." *The Lancet.* March 30. 1912: 883.
[296] H. P. Lie, who tested the drug on six patients in Bergen between 1907 and 1911, seems representative when he concluded that Nastin might be of some use in isolated cases, and this made it interesting, but that the drug was too unpredictable to in any way revolutionize the campaign against the disease. (Lie, H. P. "Om nastin og nastinbehandling i lepra." *Medicinsk Revue.* 1911: 353-362.)

was an old herbal medicine that from the 1920s dominated discussions on treatment of leprosy worldwide.

Did the treatments work?

Before investigating the chaulmoogra treatment, it is necessary to take a step back and discuss whether the treatments worked. In the strictest sense, all the drugs mentioned so far failed to achieve consistent cures in other locations than the one where it was first tried. On the other hand, there were several reports of at least initial improvements in those treated. In the following I will show how the criteria for answering the question 'did the treatments work?' depended on the yardstick used, and that the measure for success changed between the 1850s and the 1930s. I will also discuss to what extent these medical trials reflected actual local practices.

Up to the 1870s, a common explanation for why the occasional report of successful leprosy-cures proved impossible to reproduce was that the label 'leprosy' was used on a variety of skin diseases. If a treatment succeeded, it was often dismissed as a case of initial misdiagnosis. And even if the diagnosis was accepted as 'correct', each individual case was unique. Leprosy, like any other diseases, was the result of multiple causes working together, and many argued that attempts at finding a single drug or a standardized treatment plan which would work equally well in all cases was doomed to fail. In 1871, Robert Welbank Macaulay with the Bengal Medical Service summarized in a medical dictionary aimed at a wider audience:

> The insignificant remedies which have been recommended for the cure of leprosy are very numerous, probably arising from the circumstance, that there is no single application or plan of treatment that will be uniformly successful, even where we are attentive to the nature of the disease, and the circumstances of each case; but where we merely take up the name, and

> indiscriminately apply to every case we call leprosy, some one medicine or plan, we may be certain of being repeatedly altogether disappointed.[297]

Many of the treatments did reportedly do some good. Explaining why the effects of the treatments differed was a recurring topic for debate. Gordon Messum in Pretoria was but one of many who argued that every treatment initially did some good based on anticipation alone. Another often used explanation was that each individual differed in their resistance to the disease and that this inherent resistance could overcome the disease, regardless of any medication. In the case of a British soldier successfully treated with strychnine, gurjon and chaulmoogra in Sydney, for instance, John Ashburton Thompson explained that the drugs probably had been of little consequence: "on the whole I feel obliged to express the opinion that it would not have produced the excellent results recorded in this case, unless the patient had possessed an unusual inherent power of combating the infection."[298] Yet others argued that the drugs worked, but that a successful treatment required a persistence that most 'lepers' lacked.

In general, the smaller trials were reportedly more successful than the larger ones. In the discussions on treatments, several pointed out that even Danielssen had occasionally discharged patients as cured, and it was well accepted that a small proportion of those affected by the disease spontaneously recovered, independent of any treatment. This undermined the confidence in the value of the smaller trials. Was reporting a success simply more attractive than describing treatments that failed? Did this create a bias? Was the initial diagnosis wrong?

Taking stock of the state of leprosy treatments at The Fifteenth International Medical Congress in Lisbon in 1906, German dermatologist Paul Gerson Unna pointed out that many of the reported successes were made by physicians with only

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[297] Macaulay, Robert Welbank. *A dictionary of medicine and surgery designed for popular use containing an account of diseases and their treatment, including those most frequent in warm climates.* 1871: 437. This was, in turn, a direct quote from the first edition by his father, Alexander Macaulay, published in 1828. My thanks to Åsne Hagen for this.

[298] Thompson, J. Ashburton. "A Case of Lepra Tuberosa; Approximate Recovery." *Lepra Bibliotheca Internationalis.* 1907: 23.

limited experience with the disease. Judged for instance by the number of different authors publishing in *Lepra Bibliotheca Interationalis*, it seems there was some merit to this criticism: Of 104 papers on treatment published in the journal's fifteen years of existence, 92 authors published only once. According to Unna, papers presenting single cases were naïve, misleading and contributed to the difficulties in getting an overview. Instead, large studies conducted by experts with extensive experience should be the norm.[299] Large studies required access to large numbers of cases and the ability to monitor the patients over time. This meant excluding the general physician meeting occasional patients on the bedside, in favor of those working in leprosaria and other specialist institutions with hospital wards.

However, measuring the efficacy of a drug in larger groups instead of in individuals was not enough to avoid bias. In a discussion in London in 1912, Messum's predecessor as medical Medical Superintendent at the Pretoria Leper Asylum, George Turner, suggested giving half the patients a mixture of lime juice, syrup and tincture of ginger.[300] In his experience, "the glorified ginger beer" always produced equally good results when compared to proposed cures, proving that the drugs used were in themselves insignificant: "Unless, as I said before, the cures or arrests very greatly exceed 2 or 3 per cent., the figures obtained without control mean absolutely nothing."[301] In the 1920s, some argued that the percentages of expected cures produced independent of any treatments could be as high as 20 percent.

While Unna simply argued that the experts working with groups of patients would produce *better* knowledge than general practitioners seeing at most a handful of cases, it is important to note that they also produced a *different kind of* knowledge.

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[299] Unna, P. G. "Sur la pathologie et la thérapeutique de la lèpre". *Lepra Bibliotheca Internationalis.* 1906: Fasc. 3.

[300] George Turner worked as Medical Officer of Health in the Cape Colony from 1895, and was Medical Officer at the Pretoria Leper Asylum from the early 1900s until retiring and returning to England in 1908. In 1913 he was knighted, and it became publicly known that he himself was a victim of leprosy. In the popular press, he was immediately portrayed as the "Father Damien of the British Empire", a term that was repeated in the obituaries when he died two years later. See i.e. "Doctor, Knighted, A Leprosy Victim. Story of Turner's Self-Sacrifice among Lepers at Pretoria Moved the King." *The New York Times.* January 19, 1913. "England's Father Damien. A Victim of Leprosy." *The Advertiser*. Adelaide. February 24, 1913: 16; "Sir George Turner Dies From Leprosy. Sacrificed His Life in His Efforts to Find a Cure for the Dread Disease." *The New York Times*. March 14, 1915.

In Pickstone's vocabulary, walking the wards was a different way of working than meeting single patients, and this was entangled with a different way of knowing. This is reflected in the reports produced: The case-stories generally contained far more information that only the person suffering from the disease could provide compared to the quantitative studies produced in institutions, such as details of the life the (often named) sufferer led prior to being diagnosed. In some cases, they contained details of where, how and why the physician and leprosy sufferer met. The descriptions of lesions and the rationale for the diagnosis were also much more elaborate. Although reports on relatively large trials could emphasize the individual patients taking part, such as Danielssen's tuberculin trial, and while working in institutions generally meant having the sufferers under continuous scrutiny over longer periods of time, increasingly the individualizing details were seen as less important. After all, when referred to by others these details were almost consistently ignored.

By the 1920s, studies of individuals had not completely disappeared, but the genre of case studies was mainly withheld for cases explicitly out of the ordinary. Increasingly, studies that produced conclusions which could be presented in numeric ratios took over. In the interwar period the large scale trials, where the success of treatments could be measured in numbers and proportions rather than minute descriptions, began to dominate. When Solis and Wade examined several hundred children in Manila in the mid-1920s, for instance, it is obvious that any details on the individuals had to be omitted.[302] Textual elaborations on what changes had occurred in the individual patients were slowly replaced by statistics separating the patients into groups. Producing knowledge that could be quantified became a goal in itself. When Etienne Burnet in the late 1920s argued on behalf of the League of Nations Leprosy Commission that time was ripe for coordinated large-scale experiments, this was a

[301] Discussion. *Transactions of the Royal Society of Tropical Medicine and Hygiene.* March, 1912: 200.
[302] Solis and Wade's study of Philippine children was published in *Journal of the Philippine Island Medical Association* (vol. 5, December 1925), the follow-up in the same journal in 1927 (vol. 1). Referred to in: Burnet, Et. "Provisional report presented at the Leprosy Commission at its meeting at Tokyo in April 1930: Consideration of urgent matters to which the commission drew attention at its May session, 1928". League of Nations, C. H./Leprosy/7a, Geneva, January 1930: 5.

reflection both of the ideology of the League, and new emerging medical virtues which put emphasis on the production of comparable results. [303]

According to historian Theodore M. Porter, quantitative and procedural forms of accountability must be understood as 'technologies of distance', developed to provide a check on subjectivity and combat distrust. Quantification, Porter has argued, is a communication strategy that goes beyond the boundaries of locality and community, and helps produce knowledge independent of the particular people who made it. Calls for quantification came mainly from outside pressure on weak research communities and was employed as a way to increase authority.

> The advances of statistics in medicine must be understood as responses to problems of trust, which have been most acute in the context of regulatory and disciplinary confrontations. This, and not any inherently statistical character of clinical medicine, explains why inferential statistics entered medicine through therapeutics.[304]

While the international leprosy research community can be perceived as relatively weak, the rationale for quantification was not a response to demands from 'outside' forces. Instead, it reflected the assumption that leprosy was the same disease regardless of location, and that the disease was governed by the same general laws. These laws were not deterministic on an individual level; instead they were made visible only in larger numbers. Hansen's argument for contagion in the early 1870s, as I will return to in the following chapters, was based on a quantitative comparison of prevalence in different districts in Norway. The statistics showed that the number of new cases declined proportional to those already having the disease being 'evacuated'

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[303] "Time has come to organize large-scale experiments; the comparative study, on groups of similar cases (…) These experiments should be world-wide; they call for universal co-operation, that is to say, continual exchange between all leprosy countries. This appears to be one of the most important items of the Commission's programme." (Burnet 1930: 8-9.) The ensuing attempts at creating a global system for classifying cases (see Chapter 5) were a seen as a necessity to facilitate globally comparative large-scale trials.

[304] Porter, Theodore M. *Trust in Numbers. The Pursuit of Objectivity in Science and Public Life.* 1995: 208-209. For a review discussing the reception of the book, see: Hagendijk, Rob. "An Agenda for STS: Porter on Trust and Quantification in Science, Politics and Society." *Social Studies of Science.* Vol. 29, No. 4. 1999: 629-637.

to institutions.[305] The same rationale applied to testing the efficacy of treatments. Quantification was not a matter of distrust, but a tool to go beyond the local particularities and to make a general argument applicable to other contexts than where the numbers were first produced.

The question of what was to be observed, whether counted or described, was contested. This was connected to competing disease models. In the late 1860s, summarizing his experiences of treating sixteen lepers at the Hospital of San Lazaro in Lisbon, James J. L. Donnet argued that "In the treatment of this disease the hygienic, the dietetic, and the palliative had more influence than the therapeutic."[306] Later, especially British physicians would hold on to the argument that improved diet and a change in climate would produce better results than any drugs. The prominent physician Jonathan Hutchinson, for instance, argued that the disease was caused by eating badly cured fish, and was cured by moving sufferers to a different climate, making them do physical exercise, administering small doses of arsenic, "and a resolute abstinence from all fish."[307] While I have focused on observations of patients, many argued that observations on climate and diet were equally relevant to a discussion on treatment. Only in the 1920s, climate and diet were displaced by attention to hygienic conditions.

Making the bacillus the target for medications did not put an end to yet another rationale for treatment strategies: Analogies with other diseases. Following The Danish physician Niels Ryberg Finsen's Nobel Prize in Medicine in 1903, for instance, the light radiation treatment developed especially for lupus vulgaris (tubercular skin infections) was employed extensively on leprosy patients. This was but one of many analogies. The de facto main editor of *Lepra Bibliotheca Internationalis*, Danish physician Edvard Ehlers, argued for instance that leprosy,

[305] For more details on the Norwegian debates, see Chapter 5.
[306] Donnet, James J. L. "Clinical Notes on Leprosy." *British Medical Journal.* August 10, 1889: 301-304. Donnet saw the disease as ultimately incurable; "it is one that follows a determined course, rapid in some, more dilatory and seemingly stationary in others, but never retrogressing, always advancing." (op.cit.)
[307] Hutchinson, Jonathan. "On cases of recovery from leprosy." *Lepra Bibliotheca Internationalis.* 1901: 53.

tuberculosis and syphilis belonged to the same family of diseases.[308] Others argued that leprosy was the fourth stage of syphilis, yet others that it was an expression of Morvan's disease (syringomyelia), linked to racial degeneration, goitre (struma), and dwarfism. While making analogies to other diseases was an important rationale for trying drugs such as mercury and salvarsan (both known to work against syphilis), this did not invite to debates on finding a common ground for assessing results. Analogies remained important for justifying new and creative treatments, but also added to the complexity of the debates.

Agreeing that the disease was connected to the leprosy bacillus was a step in the direction of global convergence of treatment regimes, but the exact nature of the bacillus remained contested. The research aimed at cultivating the leprosy bacillus outside the human body did not succeed in producing an entity which was identical in different parts of the world. Strains of both Kedrowsky's lepra-cultures from Moscow and Deycke's *Streptothrix leproides* from Hamburg were shipped and cultivated elsewhere, but problems reproducing the results, not to mention interpreting the outcomes of the procedures, made it difficult to stabilize the leprosy bacillus as a unifying entity outside its mere presence in skin samples. By the 1920s most agreed that the attempts at demonstrating the bacillus in the same way as Koch had demonstrated the pathogen of tuberculosis had all failed. With a possible exception of studies into 'rat leprosy', there were no animal models. Medical trials testing new treatments had to be conducted on those suffering from leprosy directly.

Finally, there were different standards for measuring success. For some increased energy, the ability to work, increased sex drive and a feeling of optimism among the patients was in itself enough to justify declaring the treatment a success. Others seem to have been content if treatment introduced cleanliness or order. Some focused on improvements in the clinical signs of the disease, mainly ulcers healing and anesthetic spots regaining sensation; some focused on stopping the progress of the disease. From around the First World War, demanding that no bacilli were found

[308] See i.e. Ehlers, Edvard. "Le traitement mercureil de la lepre." *Lepra Bibliotheca Internationalis.* 1900: 45.

in tissue samples over a period of several years was increasingly adapted as a standard, but some still withheld that a cure meant that all symptoms of the disease ever having been present must disappear.

The best case scenario

In 1893, the Leprosy Commission to India concluded that leprosy was incurable, at least with the drugs available. This was also the consensus at the first international leprosy conference in Berlin. But incurable did not mean untreatable. The Commission to India, for instance, still recommended ointment and other treatments, as the palliatives made the disease progress more slowly. While the progress of the disease could never be stopped completely, it could be slowed down.

At the second international leprosy congress in Bergen in 1909, the final resolutions stressed that despite science not possessing a specific remedy, the disease was not incurable.[309] After all, occasionally some patients did get well. But although the increasingly dominant goal of finding a specific cure led to even more attention to developing and testing new drugs, only a minority of the physicians working with leprosy on a daily basis seems to have been very optimistic. Etienne Burnet's report on behalf of the League of Nations on the status of treatment in 1928 also applies to the preceding decades. The report opened as follows:

> For a long time, lepers were left to themselves; this is true at any rate of leprosy if not of lepers. Leprosaria were homes for incurables. Intercurrent diseases were treated, and symptomatic treatment was applied if the means were available. There was no idea of curing leprosy. The enquiry shows that the cause of treatment has not yet completely triumphed. Treatment is frequently given from a sense of duty to do something.[310]

[309] Lie, H. P. *Internationale Wissenschaftlige Lepra-Konferenz. Abgehalten vom 16. bis 19. August 1909 in Bergen (Norwegen). Mitteilung und Verhandlungen*. Bd. 3: 418-419. (Distributed in *Lepra Bibibliotheca Internationalis*. 1910.)

[310] Burnet 1930: 7.

Generally, treatments for which the best case scenario was keeping the disease at bay, or at least temporarily alleviating some of the suffering caused by the disease, were not published in the medical journals. Palliative treatments were mentioned in medical textbooks, but seldom much elaborated. To some extent, however, local practices were reflected in reports and testimonies made by visitors. Souza-Arùja's *Leprosy Survey made in Fourty Countries* (1929) and Etienne Burnet's *Report on the Study Tour of the Secretary of the Leprosy Commission in Europe, South America and the Far East* (1930) suggest that only when local practices could be compared and differences in daily routines identified, local customs were seen as something worth highlighting. In addition, some of the papers on the prevalence of leprosy in specific countries presented at the international leprosy conferences discussed daily routines within and outside the institutions. Together with the papers published in *Lepra Bibliotheca Internationalis* under the heading 'Geographia', they often gave a very different picture of the situation on the ground than the impression gathered from reading just the presentations of proposed cures and medical trials. There seems to have been a clear mismatch between the printed discussions on treatments, which represented something new and out of the ordinary, and the most common local practices, which mainly consisted of routines and palliative treatment.

The use of drugs was not the only strategy for pain relief. From the late 1870s, nerve stretching was used as a palliative, especially in anesthetic cases.[311] The treatment consisted of cutting open the leg or lower back of patients to reach the main nerve going into the lower limbs (the sciatic nerve), and then massage it using fingers or the back of a surgical knife. One of the foremost proponents was the British physician Beaven Neave Rake, medical superintendent at the leprosy asylum in Trinidad. In 1888, after one hundred operations on sixty patients, he concluded in the columns of *The British Medical Journal* that the procedure had led to "more or less relief in forty-seven cases, no relief in forty-nine cases, and a doubtful result in four

[311] According to historian Jane Buckingham, the first procedure of nerve stretching was reported in the *Indian Medical Gazette* in July 1877. (Buckingham 2002: 87-88).

cases."[312] Nerve stretching, he argued, provided relief and was in many cases an alternative to amputations.[313] While it is unclear how widespread this procedure actually was, general palliative treatments and daily care received little attention in medical journals such as *Lepra Bibliotheca Internationalis* (1900-1915). Despite being relatively easy to administer, nerve stretching was only mentioned once in a summary paper throughout the journal's fifteen years existence.[314] In comparison, despite demanding expensive equipment and a steady electrical supply, seven papers discussed the use of X-rays at length.[315] As late as 1940, Rogers and Muir explicitly warned against the ill effects of nerve-stretching, which suggests that the procedure was still being practiced.[316] This, again, indicates that not all medical practices were reflected in the circulation of knowledge. The production of *new* knowledge was more interesting than routines; the search for a cure was perceived to be more

[312] Rake, Beaven. "The value of nerve stretching in leprosy: based on one hundred cases." *The British Medical Journal.* December 22. 1888: 1373-1378, quote on p. 1378.

[313] For tubercular cases, Rake recommended surgery. To treat an ulcer under the foot, he recommended using a surgical knife (bistoury) and cutting through the foot, starting with the ulcer, and cutting straight forwards until the knife could be brought out between the toes of the patient. The foot was then stuffed to arrest hemorrhage. (Rake, Beaven. "The treatment of perforating ulcer in leprosy". *The British Medical Journal.* November 8, 1890: 1059-1060. See also: Rake, Beaven. "On the treatment of tubercular leprosy by excision". *The British Medical Journal.* June 9, 1888: 1214-1215.) Surgical interventions were widespread. In Bergen, for instance, Eduard Boeckmann used surgery to create scars in an attempt to contain the disease and stop blindness. (Boeckmann, Eduard. "Om de Spedalskes Øienlidelser." *Medicinsk Revue*. 1886: 257-267, 289-296.)

[314] "Arsenic internally, guryonoil externally and internally, carbolic acid externally and internally, appeared to do the most good. Among other remedies employed were perchloride of mercury, ichtyol, resorcin, thyroid extract, zinc sulphate etc etc. In some cases nerve stretching was practiced." (Simpson. "410 Cases of Leprosy treated in the North-West Province and Oudh in India." *The Polyclinic.* March 1902: 159ff; *Lepra Bibliotheca Internationalis.* Vol. 3, Fasc. 3. 1902: 184.)

[315] Oudin. "Lèpre tuberculeuse traitée par les rayons X". *Annales d'électrobiologie et de radiologie* 1903: No. 1, in *Lepra Bibliotheca Internationalis,* 1903; Belot. "Les rayons-X dans la lèpre." (Extract from: "La Radiothérapie dans les affections cutanées". *Lepra Bibliotheca Internationalis.* 1904, originally published in *Annales de Dermatologie et de Syphiligraphie*. 1904: 533; Wilkinson, H. B. "Leprosy in the Philippines with an Account of its Treatment with the X-rays." *Lepra Bibliotheca Internationalis,* 1906: Fasc 3, originally published in *The Journal of the American Medical Association*. February 3, 1906; O. Lassar, A. Siegfried and Urbanowitz. "Versuche mit der Behandlung Leprakranker mit Röntgenstrahlen." *Lepra Bibliotheca Internationalis* 1907, originally published in *Klinische Jahrbuch*, 1905: Heft 1; Matthews. "Treatment of Leprosy with X-rays and high Frequency." *Lepra Bibliotheca Internationalis.* 1908: Fasc 3, originally published in *Indian Medical Gazette*, August 1908; Heiser, Victor. "Leprosy in the Philippine Islands". *Lepra Bibliotheca Internationalis.* 1910: 38-39, originally published in *Treasury Department Public Health Report.* No. 35, 13. August. 1909; Heiser, Victor. "Case of Leprosy apparently cured with the X-rays." *Lepra Bibliotheca Internationalis.* 1910: Fasc. 1, originally published in *Medical Record*. New York, October 31. 1908.

[316] "Nerve-stretching is always harmful." (Rogers and Muir 1940: 243)

interesting than daily care. In other words: Although routines differed from place to place, this was generally not reflected in the papers that were circulated.

Despite impressive numbers, such as Nastin over a five year period being tested on close to a thousand individuals in more than seventy different locations, this was but a fraction of the 'lepers' that met with physicians globally. And this number was in turn but a fraction of the 'lepers' reported in the statistics presented for instance at the international leprosy conferences. For the vast majority of those affected by the disease, the medical trials – or Westernized medical treatment at all – was never part of the experience.

The answer to the question 'did the treatments work?' depended on who was asked, their own personal experiences and what reports they had access to. Both language skills and access to reports from afar differed. With the advent of medical trials, several journals published review-articles aimed at giving a comprehensive and updated picture of the status of the possible cures. But despite editorials and numerous new and specialized medical journals aimed at improved communication, which I will discuss in Chapter 6, not everything was published in more languages than one, or in more places that one. Not everyone read every language, had access to every report, read every issue of every medical journal, attended every meeting or read every proceeding.

There were dominating trends, though. Until the first decade of the 20th century, leprosy was widely believed to be incurable. What medicine could offer was palliative at best. From the second international leprosy conference in Bergen, the occasional cases of individuals' spontaneously healing disease convincingly proved the disease was curable, although the mechanisms involved were not known, nor how they could be reproduced. Various remedies were proposed, and in general the physicians (at least those who actively published) agreed that new drugs would eventually offer a medical solution.

The introductions of new remedies were all surrounded by optimism and reports of success. The news was spread at conferences and through medical journals, increasingly containing explanations for why the suggested treatments ought to work.

The new drugs were then tested in various locations, and the results of the trials were also circulated mainly through journals and conferences. Although some reported that the trials were successful, compiling the results showed that the few glimmers of hope were overshadowed by numerous reports of the treatments having failed.

Over time the standards for success changed. Despite most experience pointing to the opposite, the faith that medical research eventually would produce a solution in terms of a drug which could cure leprosy only grew. Linking the disease to the bacillus probably contributed: Instead of expecting the clinical signs of the disease (mainly ulcers and anesthesia) to disappear, the lack of bacillus was slowly introduced a new standard for success. The best case scenario changed from providing palliatives which could slow the unstoppable progress of the disease, to the eradication of the bacillus in the body being introduced as proof that a cure had indeed taken place.

Reinventing Chaulmoogra

In the medical discussions there were frequent references to the long list of medications used to treat leprosy, which in the words of Gerhard Armauer Hansen included "all possible and, we may say, impossible remedies".[317] Such lists included potassium iodide, arsenic, antimony, copper, vaccines, aniline dyes, mercury, gold, iodine, thymol, trychnine, sodium salicylate, carbolic acid, collargol, creosote, phosphorus, ichthyol, and surgical procedures such as nerve-stretching, bleeding and surgical removal of ulcers; baths and steam baths of various kinds, X-rays, radium, electrical currents, snake-poison and other venoms, proteins, serum, various herbs, plants and derivates; rest, exercise, change of climate, diet, prayer, ivory, and other parts of exotic animals. All were reportedly employed in more places than one for shorter or longer periods of time, but were seldom, if at all, the subjects of organized research like the medical trials detailed earlier in this chapter. From the 1870s,

[317] Hansen and Looft 1895: 105.

chaulmoogra would be at the top of this list: Extensively used but never the subject of coordinated medical trials.

When chaulmoogra was introduced to Western medicine in the middle of the 19th century, it had been in use in parts of Asia for half a millennium or more.[318] In 1854 the British physician Fredric John Mouat, working in Calcutta, reported that he had had 'remarkable and indisputable' success on one case and encouraged others to test it.[319] Based on oil from seeds in the fruits of trees locally available in India and other parts of Asia, its use slowly proliferated, first in Asia and later in Europe. The honor for introducing chaulmoogra to French and German-speaking audiences, respectively, was usually attributed to Ernest Henri Besnier and Paul Gerson Unna who made chaulmoogra part of the leprosy treatment at their hospitals in Paris and Hamburg.

While some physicians saw improvements in their patients and recommended it to others, chaulmoogra was until the first decade of the 20th century generally considered belonging in the category of palliatives. In 1894, the Leprosy Commission to India compared it favorably to gurjon oil, but did not go as far as considering it a cure. The expectation of the drugs was relief, at least temporarily:

> (…) it seems that the action of chaulmoogra oil in leprosy, though at the best palliative, is nevertheless more marked than that of gurjun oil. Indeed it is probable that a prolonged and regular use of this oil may in some cases arrest the progress of the disease, though for how long must still be doubtful.[320]

Since chaulmoogra was mainly perceived to be an additive to other treatments, and since the friction produced through rubbing it on the skin was considered more important than the drug itself, the actual content was for decades deemed of little importance. 'Chaulmoogra' was a catch-all category of oils produced from a range of plants. In 1879, testing chaulmoogra at Blackfriars hospital for diseases of the skin in London physician Wyndham

[318] Parascandola, John. "Chaulmoogra Oil and the Treatment of Leprosy." *Pharmacy in History.* Vol. 45, No. 2, 2003: 47-48. In China it was introduced in the thirteenth century under the name Ta Feng Tzu, see: Leung 2009: 56.
[319] Parascandola 2003: 48-49.

Cottle explained that "Chaulmoogra is the oil expressed from the seeds of the *Gynocardia odorata*."[321] Others highlighted *Taraktogenos kurzii* and other evergreen trees from the Hydnocarpus family found in central Asia. Most referred to it simply as what they got when buying 'chaulmoogra' at the local market and gave no further details. In 1900 the French botanist Georges Desprez tried to clear up some of the vagueness in the monograph *Le Chaulmoogra, Huile de Chaulmoogra, Acide Gynocardique* (1900) by describing and differentiating plant species and their oils. At the beginning of the 20th century Frederick B. Power and his colleagues at the Wellcome Chemical Research Laboratory in London tested the content of the various oils chemically, and concluded that proper chaulmoogra oil could only be produced from three species of the *Hydnocarpus* family and not *Gynocardia odorata*.[322] It did however take several years before there was any common agreement on the actual content of chaulmoogra. While chaulmoogra was mentioned as part of the treatment in almost two out of five papers on treatment (37 out of 106) published in *Lepra Bibliotheca Internationalis*, the compounds making up the content of chaulmoogra was never discussed. Still, transforming chaulmoogra from a generic to a distinct substance was the first step in redefining chaulmoogra into a specific medication for leprosy.

Chaulmoogra was not only administered as oil to be massaged, it was also given orally as drops or in capsules mixed with Vaseline. This was not unproblematic. Chaulmoogra was frequently referred to as having a distinct and unpleasant bitter taste, and it had severe side effects. For some it produced nausea, for most severe indigestion. In order to have an effect when taken orally, resistance had to be built up over time to reach the necessary high doses. Then treatment had to be continued for so long that most patients gave up. Failure to produce cures was in many instances blamed on patients' lack of determination to get well. Apparent cures were often explained by unprecedented resolution in those who managed to follow through with the chaulmoogra-treatment. What should be considered proper dosage, the use of

[320] *Report of the Leprosy Commission in India, 1890-91*. Calcutta. 1893: 340.
[321] Cottle, Wyndham. "Chaulmoogra oil in leprosy". *The British Medical Journal*. June 28, 1879: 969.
[322] Parascandola 2003: 50; Editorial. "Chaulmoogra Oil Therapy in Leprosy". *California State Journal of Medicine*. February 1922: 64-65.

additives and different regimes to develop resistance in the patients was an ongoing and lasting discussion.[323]

Finally, chaulmoogra was given by subcutaneous and intramuscular injections. This method was pioneered by Tourtoulis-Bey in Cairo, who in 1899 reported on four years of experiments. But also the injections were problematic. When demonstrating the procedure at the Academy of medicine in Paris in 1901, François Henri Hallopeau summarized some of the drawbacks:

> Subcutaneous injections of Chaulmoogra Oil give rise to marked inflammatory reaction of the cellular tissues. They lead to obstinate indurations. The injections are painful and in nearly all cases, it is impossible to administer them in a continuous manner. Moreover, they give rise to pulmonary fatty emboli. (…) On the whole, the treatment must be employed only in exceptional cases, and not in a routine manner.[324]

In addition to causing inflammation and blocking the blood flow, the oil solidified at room temperatures and needed to be heated before injections. This made the injections even more painful to endure.

The numerous drawbacks led to several reports of chaulmoogra treatment being given up. In Algerie, J. Brault concluded in 1908 that the disadvantages outweighed any alleged benefit, and concluded that the best a physician could do was to ensure hygienic surroundings and care.[325] In the German colony of Kamerun (Cameroon), Hans Ziemann gave up on chaulmoogra in 1909, the moment he

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[323] See i.e.: Brousse, A. and Vires. "Sur un cas de Lèpre tuberculeuse. Traitement par l'huile de Chaulmoogra. Amélioration trés rapide." *Lepra Bibliotheca Internationalis.* 1900: Fasc. 4; Crocker, Radcliffe. "On Leprosy as seen in London". *Lepra Bibliotheca Internationalis.* 1901: 49, originally published in *The Polyclinic.* October 1900: 243; Espanda, Joaquin Petron. "Quelques considérations sur la lèpre, son traitement curatif." *Lepra Bibliotheca Internationalis.* 1901: 185; Danlos. "Lèpre anestésique, relief considérable des taches, dyspepsie, traitement par l'huile de chaulmoogra en lavements." *Lepra Bibliotheca Internationalis.* 1904: Fasc 1; Alfonso, Manuel F. "Les traitements de la lèpre". *Lepra Bibliotheca Internationalis.* 1904: Fasc 1, originally published in *Revisita medica Cubana.* July. 1903, *La Caducée.* September 26. 1903; Lie, H. P. "Die Therapie der Lepra". *Lepra Bibliotheca Internationalis.* 1905: Fasc 4, originally published in *Deutsche Medizinsche Wochenschrift.* No. 38, 1903.

[324] Hallopeau, François Henri. "Traitement de la lèpre par l'injection sous-cutanée d'huile de chaulmoogra". *Lepra Bibliotheca Internationalis.* 1901: 115. 'Obstinate indurations' meant untreatable hardening of the tissue; pulmonary fatty emboli meant that lumps of chaulmoogra entered the bloodstream and could block the circulation.

acquired a batch of Nastin.[326] With frequent reports on at least moderate results through the use of chaulmoogra, 'conversion' could also go the other way. When Ziemann changed from chaulmoogra to Nastin, his colleague Dr. Lenz in Bagamojo, the capital of neighboring German East Africa (now Tanzania), gave up on Nastin and switched to chaulmoogra.[327] In Lazaretto Tracadie in New Brunswick, Canada, physician A. C. Smith justified testing chaulmoogra on his patients with "favourable reports on the use of chaulmoogra oil and creolin in foreign leper hospitals."[328] In his summary of treatments presented at the international medical congress in Lisbon in 1906, Paul Gerson Unna listed 35 researchers who had tested chaulmoogra, situated in North, Central and South America, Russia, Japan, England, Germany, Austria, Egypt and Greece.[329]

As the uses of chaulmoogra proliferated, so did the attempts at overcoming its drawbacks. The easiest and initially most widespread methods was mixing it with olive oil or, as Lenz did, have the patients wash it down with palm wine or other alcohols. A less common, but eventually more influential, solution was creating derivatives in collaboration with chemists. The medical superintendent at Robben Island, Robert Sinclair Black, was among the first to collaborate with Burroughs, Wellcome & Co in London. From 1903 he started testing capsules and injections of various mixtures of fatty acids produced by Power and colleagues in London.[330] Two years later, Isadore Dyer started collaborating with Parke, Davis & Company, then the world's largest pharmaceutical company situated in Detroit, Michigan. Parke-Davis produced acids based on crude chaulmoogra, and Dyer tested them on patients at the

[325] Brault, J. "Les lépreux en Algerie". *Lepra Bibliotheca Internationalis.* 1908: 107, originally published in *Archiv für Schiffs- und Tropenhygiene.* 1908: No. 7.
[326] Ziemann, H. "Berichte über den gegenwärtigen Stand der Lepra in Kamerun, Westafrika, mit Beitrag zur Nastintherapie." *Lepra Bibliotheca Internationalis.* 1909: Fasc. 1.
[327] Lenz. "Bericht über die Behandlung Aussätziger mit Nastin und Chaulmoograöl." *Lepra Bibliotheca Internationalis.* 1909: Fasc. 1.
[328] Smith, A. C. "Report of the Lazaretto Tracadie, New Brunswick". *Lepra Bibliotheca Internationalis.* 1900: 324.
[329] Unna, P. G. "Sur la pathologie et la thérapeutique de la lèpre". *Lepra Bibliotheca Internationalis.* 1906: Fasc. 3.
[330] Black, R. Sinclair. "Leprosy and its treatment by Claulmoogra Oil." *Lepra Bibliotheca Internationalis.* 1904: 140, originally published in *South African Medical Record*. June 15. 1903 and *Journal of Tropical Medicine*. September 15. 1903.

Louisiana Leper Home in Carville, Louisiana.[331] The first commercially available derivate was developed by the German physician Friedrich Engel Bey working in Cairo, Egypt, in collaboration with the pharmaceutical Bayer & Co in Elberfeld, Germany. "Antileprol" was put on the market in 1908.[332]

The formula that would do the most to inspire new research into chaulmoogra treatments was developed in the Philippines by Victor Heiser and Eliodoro Mercado between 1909 and 1914. Chaulmoogra had been in use in Manila since the 1890s, but after the US occupation in 1899 it was abandoned due to severe side effects and limited success. The use was resumed after Heiser visited Louisiana Leper home in 1908 and was impressed by Isadore Dyer's experimental results. Back in Manila, oral administration led to nausea, indigestion and consequent patient resistance. Heiser and Mercado therefore started experimenting with injections. In an attempt at finding a formula which would lead to increased absorption of the injections, Heiser contacted the pharmasutical and chemical company Merck & Co in New York. Among their suggestions was the use of camphor or ether. The final recipe, which was initially named the 'Heiser-Mercado-formula' and later just 'Mercado-formula', consisted of equal proportions of chaulmoogra and camphor with 4 percent resorcin as a disinfectant.[333] The drug was to be injected weekly, and in 1913 and 1914 Heiser reported that treatment "produces apparent cures in some cases, causes great improvement in many others, and arrests the progress of the disease in almost every instance."[334]

The advantages of the Heiser-Mercado-formula were that it was easy to produce using standard laboratory equipment and had fewer side effects than pure

[331] Dyer, Isadore. "The cure of leprosy". *Lepra Bibliotheca Internationalis.* 1906: 51-55, originally published in *The Medical News.* July 29. 1905.
[332] Engel-Bey, Franz. "Zur Behandlung der Lepra mit Antileprol." *Monatshefte für praktische Dermatologie.* 1909: 290-293. An extended version of the paper was distributed at the conference in Bergen in 1909 and translated to English in 1911 under the title *The treatment of Leprosy Especially by Antileprol.*
[333] Parascandola 2003: 51-52.
[334] Heiser, Victor. "Leprosy. Its treatment in the Philippine Islands By the Hypodermic use of a Chaulmoogra Oil Mixture." *Public Health Report.* Vol. 29, suppl. 20, 1914: 25; as quoted in Parascandola 2003: 52.

chaulmoogra.[335] Among those who tested the recipe was the Brazilian physician and leprologist Heráclides César de Souza-Araújo, who after treating 900 patients reported that 11.1 % of the cases had achieved radical cures. In further 44.4 % of the patients, the signs of the disease had retracted ('clinical cure'), while 33.3 % had experienced considerable improvements.[336] On his three year trip around the world in the mid-1920s, which I will return to in Chapter 7, de Souza-Araújo documented the use of chaulmoogra also in the United States, Japan, Korea, China, Philippines, Federated Malay States, India, Burma, Iraq and Palestine.

But more than being useful in itself the research at Manila inspired others. From 1915, chemistry teacher Alice Ball at Hawaii College's chemistry department in Honolulu pioneered a method of producing a fluid of the fatty acids found in chaulmoogra oil that was thinner, easier to absorb and thus more suitable for intramuscular injections. After Ball's death at the end of 1916, the head of the department, Arthur Dean, continued the work. In the 1920s 'Dean's derivatives' was widely used around the world.[337] When the leprosy workers at Culion on the Philippines in 1921 extended the treatment from voluntary experiments on selected cases to a routine procedure, they used the esters and not the Mercado-Heiser formula, which by then had proved both painful and prone to produce tissue damage.[338]

Honolulu was not the only site for developing new drugs based on chaulmoogra. Just before Heiser in 1915 retired from his position as Director of Health in the Philippines to begin working for the Rockefeller Foundation, he visited Leonard Rogers in Calcutta and convinced him to continue this line of research.[339] In

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[335] For instructions on the production of the most commonly used derivates, see i.e. de Souza-Araújo 1929: 192-198; Muir 1924: 12-14; Rogers and Muir 1940: 246-247.
[336] De Souza-Araújo 1929: 31.
[337] Parascandola 2003: 53.
[338] In 1921, the treatment was expanded first to 500 cases, and then doubled by the end of the year. By the end of 1922, 4,458 lepers received treatment with chaulmoogra products at Culion. (Lara, C. B. *Progress of Leprosy Treatment at the Culion Leper Colony.* 1929: 5; Rodrigues, Jose N. "Brief Review of the Medical Work at Culion Leper Colony". *Journal of the Philippine Islands Medical Asociation*, February, 1926; Wade, W. and J. N. Rodriguez. *A Description of Leprosy. Its etiology, pathology, diagnosis and treatment.* 1928: 61)
[339] Power, Helen Joy. *Sir Leonard Rogers Frs. (1868-1962): Tropical Medicine in the Indian Medical Service.* PhD Thesis, University College London. 1993: 195; Rogers, Leonard. "Recent Advances in the Treatment and Prophylaxis of Leprosy". *Edinburgh Medical Journal.* 1930: 11. The paper was a reprint of the Cameron Prize Lectured delivered at the University of Edinburgh on October 18, 1929.

the years that followed Rogers collaborated with chemists at Calcutta Medical College and, after moving to London in 1920, with Burroughs, Wellcome & Co. Wellcome quickly launched a derivate similar to Bayer's Antileprol, branded 'Moogrol'. This was followed by 'Alepol', a compound of sodium salts of selected fractions of hydnocarpus oil.[340] In 1926 they devised a formulation of mercury in hydnocarpus oil marketed as 'Avenyl', soon followed by compound 'Avenyl-Moogrol'.[341]

The main differences between esters and hydnocarpus oil were price and consistency. The esters were less viscid and thus easier to inject and more rapidly absorbed in the tissue; the hydnocarpus oils were thicker, cheaper to produce and more stable for long-term storage. Having a slower absorption rate was occasionally highlighted as a selling point for the hydnocarpus oils, but whether this actually was beneficial to the patients was contested. Most institutions that could afford it used multiple chaulmoogra varieties. Throughout the 1920s, the different formulas were discussed under the same heading: Chaulmoogra. When Muir's continued experiments at refining chaulmoogra at the end of the 1920s led to the compound 'ECCO', consisting of ethyl ester chaulmoogra, olive oil free from fatty acids and creosote, the division between the hydnocarpus oil and the esters was in effect bridged.[342]

One reason why the use of chaulmoogra spread in the 1920s was organized distribution. Rogers, himself a devote Christian, collaborated especially close with medical missionaries. In February 1920 he represented the Indian government in a conference of missionary superintendents of leper asylums arranged by the Mission to Lepers.[343] When Rogers left India for England a month later, he appointed Ernest Muir, former medical missionary to Bengal, to be his successor as head of the

[340] All the chaulmoogra derivatives stemmed from the fatty acids, and were differentiated by their melting points. Those with a low melting point were termed gynocardic acid, those with highest melting point chaulmoogric acid – with hydnocarpic acid in between. (Power 1993: 197)
[341] Greenwood, David. *Antimicrobial Drugs: Chronicle of a twentieth century medical triumph*. 2008: 192-193.
[342] Power 1993: 221.
[343] Oldrieve, Frank. *Report of a Conference of Leper Asylum Superintendents and others on the Leper Problem in India*. 1920.

Leprosy Section at the Calcutta School of Tropical Medicine. In 1924, Rogers formed the British Empire Leprosy Relief Organization (BELRA) in collaboration with Frank Oldrieve, who went from the position as Mission to Lepers' secretary in India to being secretary for the new organization.

BELRA's main objective was to "stamp out leprosy"; their method was to spread the gospel of chaulmoogra treatment, both through education and through the distribution of drugs. From 1924 BELRA sponsored four courses on leprosy in Calcutta every year, and by the end of 1928 the courses had been attended by 404 doctors who in turn had trained at least 600 more.[344] In 1928 alone, the organization distributed 500,000 doses of Alepol to various clinics and colonies throughout the British Empire.[345] By 1930, their medical journal *Leprosy Review* had reached a circulation of 2000,[346] and Mission to Lepers had adapted the slogan "Faith, Oil and Work".[347]

Even for BELRA, chaulmoogra medication was never more than part of the treatment. As Ernest Muir put it in 1924; "No drug has yet been discovered which can be relied upon of itself alone to cure leprosy."[348] In addition to the injections, efforts should be made at improving the sanitary surroundings where leprosy occurred. Also, the patients' natural resistance needed to be restored through a strict diet of fresh food, especially vegetables, fruits and dairy products. Alcohol, milled rice and grains should be absolutely avoided, and tea, coffee and tobacco should be taken only in moderation. Furthermore, the patients should exercise, preferably through walking or working in the fields, and bathe every day. Other diseases should be tended to. Finally, "The mind should always be kept fully occupied and all brooding on his own

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[344] Burnet, Et. League of Nations Health Organisation. Report on the Study Tour of the Secretary of the Leprosy Commission in Europe, South America and the Far East. January 1929-June 1930. LNHO: C. H. 887. Geneva, 1930: 11.
[345] Rogers, Sir Leonard. "Recent Advances in the Treatment and Prophylaxis of Leprosy". *Edinburgh Medical Journal*. 1930: 24.
[346] *Leprosy Review* was first published as the quarterly *Leprosy Notes* in 1928, and was renamed in 1930. (Rogers 1930: 10.)
[347] *A report of the fifty-seventh year's work in India of The Mission to Lepers.* September 1930-August 1931: 4.
[348] Muir, Ernest. *Leprosy. Diagnosis, Treatment and Prevention.* 1924: 22. See also: Muir, Ernest. *Popular lecture on leprosy, illustrated with lantern slides.* Lahore. 1925.

condition should be carefully avoided by the patient."[349] The result of the treatment program ranged from 10 to 40 percent cures, and up to 60 percent in new cases.[350]

Not everyone agreed that chaulmoogra worked. In Hawaii chaulmoogra was used systematically from 1914, changing to Dean's ethyl esters in 1918. By 1925, 414 patients were paroled as cured, but in 104 of them the disease returned. "Spontaneous improvement or quiescence and even the arrest of the disease, are observed in 8 to 10 per cent of clinically diagnosed cases – *i.e.,* precisely the proportion of released patients who remain negative for some years after their discharge."[351] According to James Thomas Wayson of the Board of Health at Hawaii, chaulmoogra could be used to hold leprosy in check, but the disease could never be cured. Therefore, all registered lepers should remain under medical supervision indefinitely. In Japan, chaulmoogra was given to three-fourths of the patients, but even their foremost expert, Kensuke Mitsuda, argued that "*all* lepers who are rendered negative are bound to be positive again."[352] Secretary of the League of Nations Leprosy Commission, Etienne Burnet, fittingly termed this position "Once a leper, always a leper."[353]

In 1930 Burnet, published a report from his travels in Europe, South America and Asia. The use of chaulmoogra was his main focus.

> Is it really efficacious? (…) The technical reviews give no idea of the spirit with which the question is being discussed by enthusiasts and sceptics alike. Some go so far as to say that all this excitement about cure is mere humbug, while others declare that to be lukewarm about treatment is as great a crime as to refuse quinine to a malaria patient or arsenobenzol to a person suffering from syphilis.[354]

[349] Muir 1924: 24.
[350] Rogers 1930: 18-20.
[351] Burnet, E. "League of Nations Health Organisation. Report on the Study Tour of the Secretary of the Leprosy Commission in Europe, South America and the Far East. January 1929 - June 1930." (C. H. 887). Geneva, 1930: 24.
[352] "Rendered negative" meant that no bacilli were detected in any tissue samples. (Burnet 1930: 13-14, italics in the original.)
[353] Burnet 1930: 24; Wayson, N. E. *Public Health Reports.* Vol, 44, No. 51, December 20. 1929.
[354] Burnet 1930: 39.

The actual value of the drug was impossible to ascertain, as even those most skeptical offered chaulmoogra as part of their treatment due to pressure from the patients.[355] Especially the American Surgeon General Hugh Cumming repeatedly called for the League of Nations to coordinate comparative studies so that what he considered myths surrounding the alleged efficacy of chaulmoogra could be debunked.[356] Even Burnet, who held chaulmoogra in high regards, had to admit that "No conclusive evidence exists of the efficacy of chaulmoogra as such. The crucial test of a comparison of two like groups of patients – one being treated by chaulmoogra and the other not – has, it seems, never been carried out."[357]

The peak of chaulmoogra was in the 1920s and early 1930s. By the end of the 1930s, however, even the medical advisors to the League of Nations, such as Earl Baldwin McKinley from Leonard Wood Memorial, reported that "those who advocate chemotherapy [using chaulmoogra] in leprosy have the burden of proof upon them".[358] If chaulmoogra did not work in for tuberculosis, which was also caused by an acid-fast bacillus, there was no reason why it should work on leprosy, he argued. In McKinley's view, the institutional care alone was enough to explain the apparent cures: Chaulmoogra had no effect.

The core of the debate was not only the dubious efficacy of chaulmoogra, but also disagreements regarding how treatment should be organized. The foremost proponents of chaulmoogra, namely BELRA and the Calcutta-school, argued that having a treatment available meant that the whole treatment regime should be revolutionized. Chaulmoogra was a carrot that would attract people looking for a cure. Therefore, compulsory segregation should be abandoned in favor of voluntary treatment. Compulsion only led to the hiding of cases, Rogers and Muir argued,

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[355] Burnet 1930: 43.
[356] In a letter to the Director of the Health Section of the League of Nations, Ludwik Rajchman, dated September 25, 1931, for instance, Cumming argued that "chaulmoogra oil is being stressed too greatly as a therapeutic specific and that too little attention is being given by leprologists to other factors of perhaps equal or greater importance." He therefore suggested "that a selected group of patients in an existing institution or in an area where leprosy is prevalent might be subjected to a modified sanitarium regime without chaulmoogra oil, and be productive of suggestive, and perhaps startling, results." (LNHO: 8A/R5892/32219-6651).
[357] Burnet 1930: 40.

ensuring that the disease was spread before the lepers were segregated.[359] While chaulmoogra did not cure all cases, it provided hope and could be used in prevention. The treatment was most efficient in the early stages of the disease, but this was also when they considered the disease to be most contagious.

The strategy BELRA promoted consisted of propaganda, treatment and survey (the 'PTS-model'), and was aimed at winning the confidence and cooperation of villages through offering voluntary out-patient treatment. First, a survey was carried out and the results used as "special propaganda" in explaining the prevalence of the disease, how it had spread and what signs to look for. Then, the public was informed what measures should be taken to avoid contagion, and that leprosy could be cured. Treatment was administered in out-patient clinics where the lepers were free to come and go as they pleased. While the treatment admittedly was of limited curative value, out-patient treatment would encourage the lepers to volunteer for proper institutional care. Releasing and returning those who were cured would in turn encourage other sufferers to seek institutional treatment at an early stage.[360] The primary goal of treatment was thus not necessarily to cure all lepers, but to fight new infections. Spreading the PTS-model was an integral part BELRA's training program and publications.

While BELRA in the 1920s became the foremost proponents of the voluntary model, they did not invent this. In 1919, Dr. Léon Stevenel in French Guiana was the first to argue that chaulmoogra treatment was the key to solving 'the leprosy problem':

> "For many reasons there can no longer be any question of isolating every leper of the colony. The public authorities are mistaken if they think the spread of leprosy can be

[358] McKinley, E. B. "Note on LEPROSY". *Report to the League of Nations.* Geneva, November 10, 1937. (LNHO: 8A-R6071/31395/1681).

[359] "Before we had any effective treatment even of early cases, compulsory segregation was the most generally recognized method for reducing leprosy, but it has the serious drawback that it inevitably leads to all early cases being hidden until past the most curable stage, and usually until they have infected others." Rogers 1930: 22. See also: Rogers and Muir 1940: 121.

> prevented by sequestrating a small number of sufferers while thousands of lepers are left free to carry on trades of all kinds in various parts of the colonies. (…) I am in favor of the establishment in the more populous centers of anti-leprosy or preferably mixed preventoria to prevent 'free' lepers. (…) would come with their families for treatment, medicaments and advice on how to prevent the disease from spreading."[361]

An important advantage of the out-patient treatment model was that it was cheap. While the official census for 1921 showed 102,513 lepers in India, Muir believed the actual numbers to be at least seven times as high.[362] Rogers estimated in 1930 that there were at least three million lepers globally.[363] The scale of the problem made wholesale segregation unattainable, it was simply too costly. Economically, out-patient treatment was far more efficient: "The cost of the drug for a year's treatment per patient is one-sixtieth of that of isolating a single leper in India, while the sum of £200 expended in New South Wales for each segregated leper will supply the drugs for treating 1600 lepers for a year."[364]

The faith in chaulmoogra was closely correlated with the faith in the out-patient treatment model. Japan and the United States both justified compulsory segregation by pointing out that treatment did not provide reliable cures. In Bergen, chaulmoogra was hardly used at all, because the system of home-segregation alone made it possible to stamp out the disease.[365] The Philippines adapted a middle way by retaining Culion as an 'in-patient' leprosy colony for segregation, and adding outpatient clinics. According to Herbert Windsor Wade, in charge of research at the Culion leper colony and first Medical Director of the Leonard Wood Memorial, treatment was an addition – not a replacement for segregation. "To eliminate all segregation would be as dangerous as to employ this system alone. (…) Thanks to

[360] Rogers and Muir 1940: 135. See also: Rogers, Leonard. "Memorandum on the prevalence and prophylaxis against leprosy in the British Empire, based on replies to the questionnaire of the British Empire Leprosy Relief Association; with suggestions for dealing with the problem". 1925: 19-22. (LNHO: 12B-R898/4264/29272.)
[361] Quoted in Burnet 1930: 9-10.
[362] Burnet 1930: 15-16.
[363] Rogers 1930: 1.
[364] Rogers 1930: 22.
[365] Burnet 1930: 8, 12-14, 24.

treatment, the utility of segregation is increased."[366] The first outpatient clinics opened at Pilapilan and Baldad in 1922.[367]

Also Rogers stressed that in countries where much money had been spent on segregation, out-patient treatments should be an addition, not a replacement for already existing institutions.[368] This did little to convince the segregationists. After all, why offer out-patient treatment when the treatments did not work? In the next chapter I will adress the question of prevention.

Conclusion

"The number of drugs which have been used in the treatment of leprosy almost embraces the whole pharmacopædia."[369] While this exact expression was coined in 1903 by the medical superintendent at Robben Island, Robert Sinclair Black, similar statements were repeated in almost all surveys of treatments published in the period I have studied. It reflected both a hope that leprosy could be cured given the correct drug and a disappointment that no cure had yet been found.

Whether it actually was possible to cure a 'leper' remained contested. Until around the turn of the 20th century, a majority of the leprologists believed the disease was incurable. At best, the progress of the disease could be slowed down, and some of the pain alleviated. This changed at the second international leprosy conference in Bergen in 1909. Based on the observation that some people did in fact get well, a majority of the attendees agreed that the disease in theory could be treated but that no

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[366] Wade, quoted in Burnet 1930: 21.
[367] Rodrigues, Jose N. "Brief Review of the Medical Work at Culion Leper Colony". *Journal of the Philippine Islands Medical Association*. February. 1926: 5. (reprint)
[368] As Rogers put it in *The Practitioner* in April 1928: "In countries where much money has been expended in segregating lepers compulsory, I do not advise that this plan should be abandoned for the present, but that it should be modified (…) to prevent it doing great harm by preventing the patients coming forward for treatment in the earliest stages." (Quoted in Burnet 1930: 16.)
[369] Black, R. Sinclair. "Leprosy and its treatment by Claulmoogra Oil." *Lepra Bibliotheca Internationalis.* 1904: 140, originally published in *South African Medical Record*. June 15. 1903 and *Journal of Tropical Medicine*. September 15. 1903. The same sentiment was expressed both earlier and later in time. See i.e. Parascandola 2003: 47; Rogers and Muir 1925: 245-254; Rogers and Muir 1940: 226-245; Hansen and Looft 1895: 105-125; Danielssen and Boeck 1847: 115-133.

specific cure yet existed. In the 1920s and 1930s, however, several influential experts still argued that ‘once a leper, always a leper’.

From the 1880s, numerous new medical journals, and the increasingly widespread practice of publishing abstracts of reports printed elsewhere, provided a fundament for increased coordination in the testing of new treatments. The tuberculin-experiments exemplify how medical trials no longer were a matter of individual or local experience. Instead, treatments were tested in numerous locations and the results compiled and compared before any definite conclusions could be drawn. In general, the numerous drugs developed and tested to cure leprosy all suffered the same fate: Initial optimism, distribution of the active ingredient (or the methods for producing it), extensive testing, publishing results, compilation and comparison of various studies – and finally a declaration of defeat as the drugs failed to live up to the hype. If a drug was perceived to be successful in one place, however, reports from other places were not alone enough for a treatment regime to be discontinued. Just as with diagnosis, treatment regimes were never identical around the world.

Increasingly, it was not enough to present the method for producing a compound, or anecdotal claims that the drug had worked in specific cases. Reports that could be quantified were progressively given more weight than reports on treatments of individual cases. This was both an effect of journals and conferences leading to an increased globalization of the medical community, and a reflection of the increasingly shared assumption that the disease was governed by the same general laws regardless of location. These laws were not deterministic on an individual level, but could be observed as statistical proportions in larger groups. While collaboration with chemists and professional manufacturers from around 1900 led to increasing standardization of the compounds used, quantification led to a standardization of the collective leper body. The rationalization for medical statistics was that they enabled the physicians to see beyond individual particularities and thus get unbiased access to the underlying regularities governing the disease and the efficacy of any given medical treatment.

From around the turn of the 20th century, new proposed treatments also required a rationalization justifying the claims to an effect based on previous scientific medical knowledge. Possibly, the belief in incremental steps instead of radical breakthroughs was due to the numerous sensationalist announcements of radical cures that all turned out to be impossible to reproduce. A consequence of the increased desire for quantification and theoretical medico-scientific contextualization, however, was that the general practitioners who encountered only a handful of cases were excluded in favor of experts working in specialized leprosy institutions.

As the chapter on diagnosis showed, more and more leprosy meant the leprosy bacillus acting in the body, not the presence of clinical signs. Also when it came to treatments, the bacillus was increasingly singled out as a target. The first medical trial that used the leprosy bacillus as part of the rationale was the tuberculin trials in the early 1890s. The failure to cultivate the bacillus in artificial media was a problem. Despite numerous claims that the bacillus had been cultivated, these were all surrounded by controversy and proved hard to reproduce or interpret.

With no animal models, the treatments had to be tested on those affected by the disease directly. Danielssen's patients taking part in the tuberculin trial in Bergen is but one of numerous examples of leprosy sufferers asking or being asked to take part in the medical trials. There are also several reports of leprosy sufferers who after failing to experience any benefit from the drugs refused to take part in further experiments.

Some of the treatments were explicitly intrusive. For Leprolin, for instance, provoking fever, nausea and pain was seen as proof that the drug was efficient. For Nastin, in comparison, a fever was seen as a sign of deterioration in health and the treatment therefore should be suspended. However, this did not change the fact that also Nastin in the great majority of cases failed to produce lasting benefits.

Chaulmoogra was an exception to the rule of breakthroughs and subsequent failures. For the critics, the lack of a specific effect on other acid fast bacilli and the fact that chaulmoogra had never been subject of coordinated medical trials proved the

drug to be nothing but a placebo. For the proponents, its efficacy was based on experience.

By the time chaulmoogra was made known in Europe from the 1850s, it had been in use in Asia for centuries. The redefinition of chaulmoogra from a palliative to a specific (but highly contested) drug against leprosy underwent several phases. Around the turn of the 20th century, the chemical content of 'chaulmoogra' was defined. Some ten years later, experiments with derivatives began, consisting of chemically removing impurities from the herbal remedy and mixing it with other substances in order to dampen its severe side effects. Methods for producing treatments according to well-defined formulas were distributed widely, and chaulmoogra products were rebranded and put up for sale by medical companies.

The primary advocate of chaulmoogra was The British Empire Leprosy Relief Association (BELRA). The drug admittedly did not cure everyone, but it was good enough to provide a carrot to get leprosy sufferers to seek early treatment and thus avoid spreading the contagion. The stick offered by compulsory segregation, BELRA argued, only led to people hiding their disease. While some physicians firmly believed chaulmoogra would produce consistent cures, given that the treatment started early enough and the patients had the necessary determination to go through with the prolonged and often painful treatment, others saw it as a mostly harmless drug that could be used in order to have at least *something* to offer the patients. Yet others pointed out that the chaulmoogra derivates were essentially the same drug that for decades had been perceived to be a mere palliative, and called out for comparative large-scale trials.

Finally, the coordinated medical trials expose the discrepancy that existed between the publications and the actual local practices. Instead of describing daily routines and day-to-day care, the published medical trials focused on what was new and out of the ordinary. Reports by visitors who could compare local practices in different locations provide glimpses into how the daily routines differed, but studying the circulation of knowledge certainly does not replace the need for local studies. The published reports only tell part of the story.

It was not the ability to provide a cure for the individuals suffering from leprosy that gave the medical experts their authority. But although the individual sufferer could not be cured, leprosy could be stopped by protecting the healthy majority through segregation. The following chapter will investigate the medical discussions on prevention of leprosy (prophylaxis). There, too, quantification and arguments based not on individual experiences but on producing numbers that could be used to manage populations would play a pivotal role.

4. The question of prevention

On September 20, 1852, after visiting the recently opened Lungegaardshospitalet in Bergen, the American Episcopalian Reverend James C. Richmond wrote an alarmed letter to his friend Bishop Jonathan Mayhew Wainwright in New York. "It will probably surprise you to learn that the Oriental leprosy, as described by Moses and healed by our Saviour, exists at this moment in Norway. It is not an hour since I have seen over a hundred cases of this frightful and loathsome disease, which is here exactly the same that you found at Nablous and elsewhere in Palestine." While impressed by Danielssen's recently opened leprosy research hospital, the main purpose of the letter was to sound the alarm: Among the increasing number of Norwegian emigrants arriving in the United States there were lepers, and unless something was done the dreaded disease was bound to become permanent also in the United States. The solution, Richmond argued, was to stop the lepers at the border and send them back.

> Many vessels with emigrants now sail annually from Norway to the United States. They land chiefly in New York. Let the City or the State enact a law, and make it known in Norway, appointing a physician to enquire if the disease exists among the emigrants who arrive, and if such be found, let them have their choice between being transferred to a hospital or returned to their own country. The remedy may act harshly in some individual cases, but it is by no means more tyrannical than the Quarantine laws that already exist. It will tend to secure future generations against one of the most fearful calamities that can become permanent among a people.[370]

As I showed in the previous chapter, medicine could not offer a cure to those affected by the disease. With the exception of those who from the 1920s had faith in chaulmoogra, this was as true in 1850 as in 1930. Increasingly throughout this period, leprosy was perceived as a problem not only to the sick but also to the healthy. In a

[370] Richmond, James C. "Norwegian Leprosy". *The New York Times.* December 27, 1852 (Appendix 4).

context where the health of the population was increasingly seen as a state matter in the West, this brought the question of prevention to the forefront. Instead of curing those already affected, prevention was about stopping new individuals from catching the disease. Prevention was an integral part of the medical debate, and more and more a matter for medical experts alone. Stopping those affected by leprosy at the border was but one of several strategies.

In this chapter I will first present the debate on quarantine. Next, I will investigate the debates on etiology and prevention leading up to the recommendations from 1897 which stressed the need for segregation. Then I will inspect the actual preventive measures taken, with a focus on on the period between the late 1890s and early 1920s, and present the three main strategies for prevention pursued around the world. Finally, I will expand upon the Calcutta-school's new rationale for prevention.

As numerous studies into the history of leprosy have shown, the immediate context for prevention of leprosy being raised to the national agenda differed from country to country. My intention in this chapter is not to pursue all local specificities, but to focus primarily on the rationales presented in the most widely distributed reports, medical textbooks, *Lepra Bibliotheca Internationalis* and the international medical conferences. These sources reflect perceptions of the situation on the ground and the preventive measures taken, and were used by others looking for advice. What arguments were available to those looking for advice on leprosy prevention in the transnational medical debates? Which strategies were suggested and how did the dominant positions change over time? What was the role of the bacillus when it came to prevention?

Quarantine: Stopping leprosy at the border

Reverend James C. Richmond's letter in the fall of 1852 was reprinted in numerous newspapers and periodicals, and provoked several responses.[371] Some argued that to be on the safe side all Norwegians should be barred from entering the United States, others pointed out that since the disease was hereditary it posed no danger to anyone but the unfortunate sufferers and their families. The Norwegian businessman Peder Anderson in Boston, who had left Bergen in 1830 and was among the first promoters of Norwegian emigration, argued that given the improved diet and milder climate in the United States, the disease would probably completely die out. Therefore, there was no need to take any action.[372]

When Richmond wrote his letter, quarantine was already discussed in relation to other diseases, primarily cholera, plague and yellow fever. Accelerating transportation, with steam engines in trains and ships, exploding international trade and the increasing scale of colonialism was perceived not just as progress, but as opening up to diseases originating outside the Western hemisphere. In 1851 the first in a series of International Sanitary Conferences was held in Paris, the impetus being the cholera epidemics that haunted Europe from the 1830s. Arguably, these conferences were the first effort to regulate health on an international scale.[373]

For leprosy, quarantine measures can be traced back to the Bible and Leviticus'

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[371] The letter was first published in the monthly *Evangelical Catholic* (1851-1853) in New York, edited by William Augustus Muhlenberg, an early leader in the Angelical Christian community in the United States. In addition to *The New York Times*, re-prints made the front pages of *The Daily Times* (December 22, 1852), *New York Evening Express* (December 24, 1852), *New York daily Tribune* (December 24, 1852) and *The Daily Gazette* (Thursday January 6, 1853, adding a report on leprosy in Jerusalem). In addition, the report on Norwegian leprosy was reprinted in *The Religious Observer* and *Northern Christian Advocate*, indicating the wide interest produced by the report.

[372] Haugen, Eva L. "The Story of Peder Anderson. Anderson's autobiography translated and edited." *Studies and Records.* The Norwegian-American Historical Association. Vol. 26, 1974: 43. (Available online: http: //www.naha.stolaf.edu/pubs/nas/volume26/vol26_2.htm). Between 1836 and 1930, 852,142 Norwegians emigrated to the United States - no country except Ireland had a higher rate of emigration. Haugen, Einar. "Norwegian Migration to America". *Studies and Records.* The Norwegian-American Historical Association. Vol. 18, 1966: 1-23. (Available online: http: //www.naha.stolaf.edu/pubs/nas/volume18/vol18_1.htm).

[373] Huber, Valeska. "The Unification of the globe by disease? The International Sanitary Conferences on Cholera, 1851-1894". *The Historical Journal.* Vol. 49, No. 2. 2006: 454-476.

instructions for declaring lepers 'unclean' and belonging outside the camp.[374] Experts such as Danielssen and Boeck, who firmly believed that the disease they saw concurrently was the same as the Biblical disease, warned that this exact line of reasoning had led to inhumane prosecution in Medieval Europe.[375] As medical historian Erwin H. Ackerknecht has pointed out: The mechanisms of how a disease originated determined the principles for how it could be stopped. Whether etiology or policies were the prime mover was seldom easy to untangle. Rather, cause and prevention were always connected, making etiology an inherently ideological question.[376] No disease model fully explained why some persons developed leprosy and others not, and different schools of thought disagreed both on what observations should be given weight, how these should be interpreted and what preventive measures they prompted.

Despite widespread attention, Richmond's report had no practical consequences. When the first big surge of Norwegian emigration hit the United States between 1866 and 1873, the worry that Norwegians could bring leprosy seems to have faded from public attention.[377] This is fairly representative also for the debate among

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[374] "And the leper in whom the plague is, his clothes shall be rent, and his head bare, and he shall put a covering upon his upper lip, and shall cry, Unclean, unclean. All the days wherein the plague shall be in him he shall be defiled; he is unclean: he shall dwell alone; without the camp shall his habitation be." (Leviticus 13: 45-46, King James Version)

[375] Danielssen and Boeck. 1847: 90-92.

[376] Ackerknecht, Erwin H. "Anticontagionism between 1821 and 1867. The Fielding H. Garrison Lecture". *Bulletin of the History of Medicine.* 1948: 562-593, reprinted in *International Journal of Epidemiology.* 2009: 7-21. As Chrostopher Hamlin summarized in a commentary: "This is not a world in which the enterprise of etiological hypothesis-testing is suddenly and surprisingly found to be rife with political implications and complications, which, in the absence of adequate data and analytical methodologies come to dominate. On the contrary, the medical was inherent to the political." Hamlin, Christopher. "Commentary: Ackerkecht and 'Anticontagionism': a tale of two dichotomies." *International Journal of Epidemiology.* 2009: 22-27, quote on page 23. See also: Baldwin, Peter. *Contagion and the State in Europe, 1830-1930.* 1999.

[377] About 110,000 Norwegians emigrated during the eight-year period. In Iowa it was not until the Swedish physician Fredrik Eklund contacted the State Board of Health in 1883 asking for information on their number of lepers that they were made aware that the disease existed in the State at all. (Kennedy, J. F. "Extract of the State Board of Health of Iowa on Leprosy". *Leprosy in Foreign Countries*. Honolulu, *1886: 198ff).* Further, Eklund informed the Board of Health of the two previous expeditions made by Norwegian physicians investigating leprosy among Norwegian emigrants: Jens Andreas Holmboe in 1863 and Carl Wilhelm Boeck in 1869-70. In 1889 Gerhard Armauer Hansen would argue that 160 Scandinavians with leprosy had immigrated to the United States, that only 13 had survived until the time of his survey, and that these had not infected any new cases. This confirmed, Hansen argued, that the disease was contagious (and not hereditary) but that hygiene and improved living conditions had stopped its spread. In a letter to state public health officials he therefore recommended that proposed rigid segregation would be unnecessary. Moran 2007: 20; Hansen, G. Armauer. "Spedalskhedens arvelighed". *Nordiskt Medicinskt Arkiv.* No. 4, 1889: 1-13. See also: Lie, H. P. "Norwegian Lepers in the

physicians: As long as the disease mainly was framed as a disaster to individuals or families, leprosy remained a matter for the individual sufferer, their families and philanthropy. This changed in the 1890s, when leprosy increasingly was believed to be contagious. More and more frequently, the presence of the bacillus was used as proof that the disease was contagious, and that prevention meant interrupting the path of infection.[378]

The rationale for quarantine was that disease could be stopped at a geographical border. Quarantine was especially relevant where leprosy was seen as a foreign disease imported through migration. In the United States, leprosy was made part of the federal quarantine regulations in 1894: "Vessels arriving at quarantine with leprosy on board shall not be granted pratique until the leper with his or her baggage has been removed from the vessel to the quarantine station."[379] Persons affected by leprosy were to be detained until the ship was outbound, and returned at the expense of the vessel.[380] This was an extention of the National Quarantine Act passed one year earlier, initially aimed at cholera, yellow fever, smallpox, plague and typhoid fever. Letting the shipping companies take the financial burden of harboring lepers was to serve as an incentive for them to screen their passengers at departure. Canada

United States: The investigations of Holmboe, Boeck and Hansen". *International Journal of Leprosy*. No. 3. 1938: 351-356; Gussow 1989: 81-82; Davidsen, Bjørn. ""Forskerstafett" til Amerika 1863-1888." *Bergensposten*, no. 4. 2001: 29-39.

[378] The bacillus was not a required component for this line of reasoning. As I will return to later in this chapter, Hawaii pursued segregation from 1866, based on the observation that the prevalence of the disease was increasing.

[379] White, James C. "Leprosy in the United States and Canada". *Mittheilungen 1897*. Bd. 1, Abt. IV. 1897: 29; Kerr, J. W. "Communicable diseases: An analysis of the laws and regulations for the control thereof in force in the United States." *Public Health Bulletin No. 62.* 1914: 22-23. See also: Moran 2007: 22. Some states had established quarantine-regulations prior to the federalisation of the US quarantine system in 1890. From August 1883, for instance, any vessels carrying persons affected with leprosy or elephantiasis within the Bay of San Francisco, were to deliver them to the local lazaretto. (Meares, J. L. "San Francisco, California". *Leprosy in Foreign Countries*. Honolulu, *1886: 216).* For a discussion arguing that the discourse linking leprosy with fear of immigration has lately reignited in the United States, see: White, Cassandra. "Déjà Vu: Leprosy and Immigration Discourse in the Twenty-First Century United States". *Leprosy Reveiw. 2010: 17-26.*

[380] For more on the role of shipping interests in US migration policies, see: Feys, Torsten. "The visible hand of shipping interests in American migration policies 1815-1914". *Tijdschrift voor sociale en economische geschiedenis*, vol 7, nr 1, 2010: 38-62, and Sebak, Per Kristian: *A Transatlantic Migratory Bypass – Scandinavian shipping companies and transmigration through Scandinavia, 1898-1929*. PhD-thesis, University of Bergen, 2012. Feys focuses on Holland America Line as middleman and active policy agent in the period before the First World War, while Sebak's focus is on DFDS and the Norwegian America Line in the interwar period. See also: Zolberg, Aristide R. *A Nation by Design – Immigration Policy in the Fashioning of America*. 2006.

followed suit in 1896,[381] and some years later the legislations led to calls from physicians in Latin America that similar precautions must be put in place also there.[382]

Despite the participants at the first international leprosy conference in Berlin in 1897 agreeing that "Every leper is a danger to his surroundings",[383] only two physicians advocated quarantine measures: François Henri Hallopeau from Paris and Eduard Arning from Hamburg.[384] Both saw leprosy as an imported disease originating outside the European continent, and just as they believed that the disease had been brought to Europe by the first Christian crusaders, they held the advent of mass migration in the second half of the 19th century responsible for the disease reappearing. According to Arning, most immigrants came from countries where leprosy was endemic (identified as South China and Japan, followed by British India, Russia, Scandinavia and Portugal). While importing foreign labor was in itself justified, the migrants stuck to the way of life they were used to from their home countries, and imprinting that they ought to take proper precautions to avoid spreading their disease was impossible. Hallopeau emphasized that since leprosy was contagious and maritime quarantine had protected France against the other contagious diseases, the lessons learned should be applied also for leprosy.[385]

What Arning and Hallopeau called for at the Berlin conference was inspired by the US Quarantine system: Refusing the landing of lepers, and making the states 'exporting leprosy' responsible for controls at departure. This, they argued, was safer

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[381] Pernet, George. "Leprosy in the British Empire (South Africa and Australia excepted)". *V. Internationaler Dermatologen-Kongress abgehalten in Berlin vom 12-17.September 1904. Verhandlungen und Berichte.* 1904: 46.

[382] The question of preventing leprosy through quarantine was a topic at the Third Pan-American Medical Congress held at Havana, Cuba, in February 1901. The Cuban physician Manuel f. Alfonso, especially, argued that: "Any immigration on a large scale from leprous foci is an important matter as far as the spread of leprosy is concerned." Alfonso, Manuel F. "Leprosy in Cuba". *Lepra Bibliotheca Internationalis*. 1902: 52. See also: Robelin. "La lèpre en Cuba: Algunos datos referentes à la lepra en Cuba*." Lepra Bibliotheca Internationalis*. 1902: Fasc 3; *Revisita de medicina tropical*. March, 1902.

[383] Abraham, Phin. S, Edward Arning, E. von Bergman, Dubois-Havenith, J. J. Kinyoun and G. Thibierge. "The Honorary Secretaries' report from the First International Leprosy Conference" *Mitteilungen 1897*. Bd 2. Abt. IV: 191-192.

[384] Arning, Edmund. "Lepra und Immigration". *Mittheilungen 1897*. Bd 1. 1897: 8-13; Hallopeau. "Die Isolirung der Aussätzigen und die dazu erforderlichen Massregeln". *Mittheilungen 1897*. Bd. 2. 1897: 163.

[385] Quarantine was not a uniform intervention. For a detailed comparison of quarantine measures against cholera, smallpox and syphilis in Britain, France, the German states and Sweden, see: Baldwin, Peter. *Contagion and the State in Europe, 1830-1930.* 1999.

and more practical than controls at arrival, as it was much more difficult to return those who have already arrived. But while none of the delegates explicitly disagreed, none signed up when the question of immigration and emigration was put up for debate under its own heading.[386] Possibly, quarantine regulations were seen as questions for inter-state diplomacy – not a matter for a scientific conference. More likely, quarantine was seen as a derailing from the more important issue, that of segregation. Besides, was quarantine really applicable to a disease such as leprosy?

To stop leprosy at the border was easier on paper than in practice. At the Berlin conference, dermatologist Georges Thibierge from Paris pointed out that health inspection of all subjects from 'contaminated countries' was impractical. Instead, he suggested targeted inspections of those most exposed to the danger of infection: Soldiers returning from military campaigns in leprous countries, sailors, and employees of the colonial- and penitentiary services.[387] His colleague Ernest Henri Besnier agreed: "In most cities it is impossible to stop the leper on arrival, unless they are filthy with evident facial lesions."[388] Instead, he recommended that municipalities should register all foreign residents originating in countries with leprosy, and monitored their health over time.

At the annual meeting of the American Dermatological Association in 1900, physician Prince A. Morrow from New York summarized the three main arguments against quarantine: First, leprosy had an incubation period which could last for years in which not even the person affected was aware of the disease.[389] In comparison, the incubation period of yellow fever and cholera was five days, smallpox two weeks and typhoid fever up to twenty days.[390] Second, the disease was hard to diagnose, especially in its early stages, and the consequences of a mistaken diagnosis were

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[386] *Mittheilungen 1897*. Bd 2. 1897: 121.
[387] Thibierge, G. "Die Isolirung der Aussätzigen und die dazu erforderlichen Massregeln". *Mittheilungen 1897*. Bd. 2. 1897: 180.
[388] "Dans la plupart des villes, il est impossible d'arrèter le lépreux à l'arrivée, à moins qu'il ne soient des lépreux sordides avec les lésions faciales manifestes." Besnier, Ernest Henri. "Die Isolirung der Aussätzigen und die dazu erforderlichen Massregeln". *Mittheilungen 1897*. Bd 2. 1897: 171.
[389] "No system of quarantine has ever been devised that will effectually prevent the importation of a disease so prolonged in its incubation and so little manifest on ordinary inspection as leprosy." (Morrow, Prince A. "The Prophylaxis and Control of Leprosy in this Country." *Lepra Bibliotheca Internationalis* 1901: 171.)

grave. Finally, as by then had been argued at the International Sanitary Conferences for half a century, rigid border control would interfere with commerce. "There can be no commerce or free intercourse between nations or races without an interchange of infectious diseases as well as commercial commodities."[391] In the columns of *Lepra Bibliotheca Internationalis,* Hansen concurred: "I think it is impossible to prevent the emigration and immigration of lepers, because, the disease develops so slowly, that even a physician knowing the disease very well cannot diagnose leprosy for many years, how many, nobody knows, in its beginning."[392] The difficulties of early diagnosis, combined with leprosy having an incubation period lasting years not days, were strong arguments against adapting quarantine measures developed for other contagious diseases: Quarantine might be desirable, but in practice it was impossible to implement.

When the Fifth International Congress of Dermatology convened in Berlin in September 1904 and dedicated the whole first volume of the proceedings to follow-up on what actions had been taken since the leprosy conference in 1897, quarantine measures were hardly mentioned.[393] Nor was it a central topic at the Second International Leprosy Conference in Bergen in 1909.

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[390] Treasury Department. *United States Quarantine Laws and Regulations.* February 24. 1893: 17.

[391] Morrow, Prince A. "The Prophylaxis and Control of Leprosy in this Country." *Lepra Bibliotheca Internationalis* 1901: 170. Morrow did however argue for selective border controls aimed at the Chinese: "Before dismissing this part of the subject it may be said that the Chinese have been the most active disseminators of leprosy in modern times. These pig-tailed Argonauts of the Orient in their world-wide migrations in quest of the Golden Dollar have invaded many lands and almost every land they have touched they have tainted with leprosy." (Op. cit.) Morrow's sentiment against the 'yellow peril' was undoubtedly shaped by the recent US annexation of Hawaii, where Chinese immigrants had been identified as likely culprits already in William Hillebrand's report from 1865, and the locals named the disease "mai pake" ("Chinese disease"). The Chinese Exclusion Act passed in 1882, excluding "skilled and unskilled laborers and Chinese employed in mining" from entering the country shows how Chinese "coolies" as carriers of leprosy was but a part of a more widespread prejudice. (Chinese Exclusion Act, 1882).

[392] Hansen, G. Armauer. "On the prevention of emigration and immigration of lepers." *Lepra Bibliotheca Internationalis.* 1900: 88.

[393] The only exception was French dermatologist Eduardo Jeanselme, who argued that the most important action to be taken by the European colonial powers to stop the spread of leprosy was prohibiting lepers from the colonies travelling to Europe. (Jeanselme, M. E. "Frankreich und Kolonien. Bericht". *V. Internationaler Dermatologen-Kongress*. 1905: 227.). Also Wellesley C. Bailey from *Mission to Lepers* tangented on the topic in his lecture on "How Lepers Travel", which concerned how 'lepers' moved about within India. (Bailey, Wellesley C. "British India." In: Rosenthal, O. *V. Internationaler Dermatologen-Kongress abgehalten in Berlin vom 12.-17. September 1904.* 1. Band. 1904: 98-99.)

Despite several physicians questioning the viability of stopping leprosy at the borders, and no international resolutions to give weight to the arguments, the debate continued. In the spring of 1901, the German government announced that they had reached an agreement with the Persian, Romanian, Russian and Turkish governments aimed at preventing the movement of leprosy patients between the four countries. "To this end it was decided that the authorities of the country interested do not deliver passport to lepers, or even ID-cards."[394]

In France in 1905, the section for medicine and hygiene at The Colonial Congress in Paris unanimously recommended that lepers in the French colonies should be prohibited from crossing state borders.[395] When founding editor of the medical journal *Annales d'Hygiène et de Médecine Colonial* (1898), Alexandre Kermorgant, was made Inspector General of the colonial health service in 1906, he quickly made leprosy one of his top priorities.[396] Pointing at the resolutions passed at the international congress in Berlin in 1897, which concluded that the disease was contagious, he strongly argued that due to increased traveling leprosy in the colonies was a growing threat to the colonizers. France desperately needed to build institutions for the isolation of lepers arriving from abroad so that proper quarantine measures could be put in place.

> We have noted the danger and it is growing every day following the expansion of all colonial peoples, the growing number of relationships with other countries, and facilities provided to passengers to travel the world. In these circumstances would it not be prudent to consider, not the leper colony – that would be medieval – but sanatoriums or nursing homes to isolate lepers who come to us from beyond the sea. (…) Remember, in any case, nothing is more

[394] "Mesures internationales contre la propagation de la lèpre" *Lepra Bibliotheca Internationalis*. 1901: 120, originally published in *La Semaine médicale*, April 10. 1901.
[395] "Voeu du Congrès colonial de Paris de 1905". *Lepra Bibliotheca Internationalis*. 1906. Further, the congress recommended isolating all lepers with open wounds or virulent forms of the disease; banning lepers from trades where they might spread their contagion; and establishing 'maritime leprosaria' – islands where especially vagabond lepers should be deported. Instead of prison-like institutions, they recommended establishing agricultural colonies, where lepers could enjoy relative freedom in collaboration with other sufferers.
[396] Kermorgant had a long career in the navy, first as a surgeon and later as a medical doctor, before being put in charge of the French colonial health services in 1906. For more, see: Weiner, Bernadette and Jean Flahaut.

> dangerous than the failure of any prophylactic measure vis-à-vis a scourge that threatens us. Caveant consules![397]

Kermorgant seems to have had little immediate success in convincing the French government that the precautions were necessary. In 1909, as vice-president of *Société de Pathologie Exotique,* he set down a commission on leprosy in the colonies consisting of himself and three other physicians. After months of discussions they jointly sent their recommendations to the colonial minister. The main message was that leprosy existed in almost all colonies, and that once introduced to a virgin population, the disease proliferated at an alarming rate.[398] Norway was presented as the primary example proving that with proper precautions, the trend could be reversed, but that getting rid of leprosy was both more expensive and more time-consuming than preventing it in the first place. However, to stop leprosy at the borders was but one of eleven recommendations for preventive measures suggested by Kermogrant's commission. Faced with leprosy, quarantine alone was simply not enough.[399]

While the report itself did not dictate action, it shows how physicians increasingly were the driving force in putting prevention on the political agenda.

"Alexandre Kermorgant (1843-1921) témoin de l'état sanitaire des anciennes colonies françaises". *Histoire des Sciences Médicales*. Vol. 33, No. 3, 1999: 267-274.

[397] Kermorgant, M. A. "Des dangers que nous fait courir la lèpre." Inspecteur général du service santé des colonies." *Lepra Bibliotheca Internationalis*. 1902: 43-44.

[398] From the mid 1890s, this line of reasoning was widespread. Physician Isodore Dyer at Hawaii, for instance, argued that: "Leprosy has spread whenever it has been introduced, notably in the West Indies, Sandwich Islands, South America, Mexico and in Louisiana. (…) The Conference of 1897, in Berlin, was held because of the recognition of the appalling proportions of leprous inhabitants on the face of the earth, suddenly realized in all the awfulness of the danger threatening." Dyer, Isodore. "Investigation of Leprosy (from Report of Marine Hospital Service for 1898)." *Lepra Bibliotheca Internationalis*. 1900: 91.

[399] In addition to stopping leprosy through quarantine, the committee recommended spreading propaganda of the dangers of leprosy; preventing lepers from trades where they would be in direct or indirect contact with healthy; surveillance of schools and public places to detect lepers; disinfection of lepers' homes; segregation of leper vagabonds, and denying foreign lepers at the borders. In addition they recommended erecting leprosy camps in the colonies. These should be put under the administration of a physician and surrounded by a 200- meter safety zone. Inside the camps the sexes should be segregated, there should be a hospital for treatment, a separate area for suspected cases, a graveyard, and a system of punishments and rewards (such as permits to visit family members) to ensure proper discipline. Finally, children of leprous parents should be sent to orphanages. (Delrieu, Grall, Jeanselme and Kermogrant. "Rapport de la Commission de la Lèpre de la Société de Pathologie Exotique". *Lepra Bibliotheca Internationalis*. 1909: Fasc 4, originally published in *Bulletin de la Société de Pathologie Exotique*, 1909: 67; "Prophylaxie de la lèpre dans les Colonies Françaises" *Société de*

Among the results were new legislations in the French protectorate of Tonkin, which from December 1909 prohibited lepers from entering the area by law. Lepers were also banned from certain professions, and – explicitly inspired by the Norwegian legislations – those who could not be safely isolated at home were to be forced into an institution.[400]

Quarantine was never a big debate in the columns of *Lepra Bibliotheca Internationalis*, but notifications that similar legislations were put in place in other parts of the world kept accumulating. In September 1909, for instance, the maritime quarantine department in Australia clarified that the 1908 Quarantine Act (Regulation No. 49) also applied to lepers and that lepers arriving in one of the ports should immediately be isolated and returned.[401]

It was not until the third conference in Strasbourg in 1923, where segregation was downplayed in favor of more emphasis on country-specific interventions, that the question of immigration made it to the closing resolution: "1) Laws governing the anti-leprosy campaign should vary according to the country in which they are applied, but in all cases foreign lepers should be barred from entering a country."[402] Calls for international quarantine measures addressing lepers were in turn repeated in the League of Nations' first prophylaxis report published in 1931: "International agreements should be arranged to regulate the movements of lepers from one country to another."[403] Apart from the general encouragement, however, it seems the League's leprosy commission did not see brokering an international agreement as part of their mandate. Nor was leprosy part of the agreements reached at the international sanitary

Pathologique exotique, Meetings February 10, March 10 and April 14, 1909) ; "Discussion du Rapport de la Commission de la Lèpre". *Lepra Bibliotheca Internationalis*. 1910: Fasc 3.)

[400] Audian. "Prophylaxie de la lèpre au Tonkin". *Lepra Bibliotheca Internationalis.* 1910: Fasc. 4, originally published in *Annales D'hygiène et de médecine colonial.* July-September. 1910.

[401] "Commonwealth of Australia – Law as to leprosy". *Lepra Bibliotheca Internationalis.* 1910: Fasc. 4. Prior to the unification of the Commonwealth of Australia in 1901 the different territories had different laws. In New South Wales, for instance, the notification and detention of lepers was made compulsory by law already in 1890: If two medical practitioners agreed the person suffered from leprosy, they were given the choice of confinement for life in the Leper Asylum at Little Bays, or leave the country. (Thompson, Ashburton. "Report on Leprosy in New South Wales for the year 1899." *Lepra Bibliotheca Internationalis.* 1902: 221. See also: Robertson 1997.)

[402] See: Appendix 2c.

conferences. While several countries ended up including leprosy in already existing quarantine regulations, either explicitly or through defining leprosy as a contagious disease, it was never universally adapted.

The rise of segregation: Towards Berlin 1897

Denying lepers from crossing state borders did not stop the disease where it was endemic or had already been introduced, and quarantine was not the only way leprosy became a concern for governments. From the mid 1890s to the early 1920s, the preventive measure that dominated the international medical debates was segregation. As the League of Nations' Leprosy Commission put it their first general report, *The Principles of the Prophylaxis of Leprosy* (1931): "Until recently, the compulsory segregation of lepers in special leprosaria has been the chief - almost the only - measure of prophylaxis".[404] Segregation was not about stopping lepers from entering into an area, but isolating those already present.

As Dorothy Porter and others have shown, in Western countries the health of the population was increagingly seen as a matter for the state.[405] For leprosy, the breakthrough of segregation as an international recommendation can be traced to the first international leprosy conference in Berlin in 1897. The delegates concluded that leprosy was contagious: "Every leper is a danger to his surroundings, the danger varying with the nature and extent of his relations therewith, and also with the sanitary conditions under which he lives."[406] Since leprosy was considered incurable to the individual and detrimental to the society as a whole, adapting legal measures

[403] League of Nations Health Organisation. *The Principles of the Prophylaxis of Leprosy. First General Report of the Leprosy Commission*. (Official No. C. H. 970). Geneva, April 1931: 7.
[404] League of Nations Health Organisation. *The Principles of the Prophylaxis of Leprosy. First General Report of the Leprosy Commission*. (Official No. C. H. 970) Geneva, April 1931: 6.
[405] Porter, Dorothy. *Health, Civilization and the State. A history of public health from ancient to modern times.* [1999] 2005: Part 2.
[406] Abraham, Phin. S, Edward Arning, E. von Bergman, Dubois-Havenith, J. J. Kinyoun and G. Thibierge. "The Honorary Secretaries' report from the First International Leprosy Conference" *Mitteilungen 1897*. Bd. 2: 191-192.

for isolation was seen as a logical consequence: The healthy population needed to be protected. Striking a balance between the rights to liberty of those suffering from the disease and the rights of the healthy majority, the preventive measures put in place in Norway were held up as a golden standard.

Before examining the consequences of these recommendations, I will in the following investigate the medical debates that led up to the Berlin resolutions. My argument is that the resolution was a compromise in the dispute regarding etiology and prevention. On the one hand it was sufficiently severe to satisfy even the most hardline contagionists, on the other it put adequate emphasis on the need for adaptation based on local social conditions to persuade those not convinced the disease was contagious. Most importantly, it established local medical authorities as the ultimate policy experts.

For physicians interested in leprosy in the last decades of the 19th century there were numerous explanations for how leprosy proliferated. The debates illustrate how practical measures towards the disease were entangled with the mechanisms for how the disease spread, but also with what preventive measures were considered practically possible, as well as different interpretations of the outcomes of measures put in place. I will begin with the 'leprosy epidemic' on Hawaii, which in the 1880s and 1890s became a reference point for proponents of several different disease models.

Thirteen years before the Berlin conference, in 1884, president of the Board of Health and Minister of Foreign Affairs on Hawaii, Walter Murray Gibson, sent an enquiry to diplomats in various parts of the world asking for "information as to Leprosy and the social and medical treatment of lepers in other countries."[407] The

[407] Gibson, Walter M. "Introduction to Reports on Leprosy by the Government of British India and Other Foreign Powers". *Leprosy in Foreign Countries.* 1886: 3. Asking for advice in other countries was not the only strategy Gibson pursued. In 1884 he invited the German physician Eduard Arning to investigate the 'leprosy epidemic'. In the fall of 1885 Arning inoculated the convicted murderer Keanu, and two years later he was diagnosed with the disease. When Arning announced his experiment in 1889, this led to huge debates both of the ethics of the experiment and how the outcome should be interpreted. The contagionists withheld that Arning's experiment proved the disease to be contagious, others disagreed. Jonathan Hutchinson, for instance, argued that since the man had been living under the same conditions as others who had developed the disease, and that his son, nephew and brother-in-law were also victims of the disease, the experiment proved nothing.

background for the request was that despite almost twenty years with compulsory segregation, there was little indication that the disease was in decline.

The Kingdom of Hawaii had begun its attempt to "stamp out the scourge by segregation" in 1865.[408] This was a response to physician William Hillebrand's report that leprosy had recently been introduced by Chinese immigrants, and that the disease was proliferating at an alarming rate.[409] The "Act to Prevent the Spread of Leprosy" made it mandatory to report every case of the disease to the sanitary authorities. The police was required to arrest and deliver any person alleged to have leprosy to the Board of Health for a medical inspection, and a remote peninsula on the island of Molokai was set aside for a leprosy settlement. The first seven years, 1.288 persons were examined by the board, and 529 were sent to Molokai. By the early 1880s, approximately ten percent of government revenues were going to the Board of Health, between 50 and 65 percent of which was allocated to leprosy.[410] On average about 140 new cases were admitted to Molokai every year; reaching a peak of 571 new cases in 1888.[411]

Gibson's request resulted in a report with replies from British India, Ceylon, Hong Kong, Siam, the Netherlands and their colonies, the Canary Islands, Norway, Spain, Mexico, Chile, Japan, Guatemala, several states in the United States, and the leprosy institution Tracadie in New Brunswick, Canada. The replies described prevalence, diagnosis, etiology, measures put in place to check the spread of the

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(Hutchinson, Jonathan. "Remarks on some facts illustrating the early stages of leprosy". *The British Medical Journal.* March 8, 1890: 529-531; "The Contagious nature of leprosy". *The British Medical Journal*. April 19, 1890: 909.) For more on this well-documented dispute, see: Edmond 2006: 91; Inglis, Kerri A. "'Cure the dread disease': 19th Century Attempts to Treat Leprosy in the Hawaiian Islands". *The Hawaiian Journal of History,* Vol. 43. 2009: 109-124; Gould 2005: 80-83.

[408] Gibson, Walter M. *Leprosy in Foreign Countries*. Hawaii 1886: 1.

[409] Classifying leprosy as a threat based on race was very much in line with what historian Michelle T. Moran has identified as typical for the US approach to leprosy and public health. (Moran 2007). For similar arguments in Australia, see: Robertson 1999; Bashford, Alison and Maria Nugent. "Leprosy and the management of race, sexuality and nation in tropical Australia". In: Bashford, Alison and Claire Hooker (eds.) *Contagion. Historical and cultural studies.* 2001: 106-128.

[410] Inglis, Kerri A. "'Cure the dread disease': 19th Century Attempts to Treat Leprosy in the Hawaiian Islands. *The Hawaiian Journal of History,* Vol. 43. 2009: 101-124; Gould 2005: 59-109; Edmond 2006: 145-156. See also: *Leprosy in Foreign Countries.* 1886.

[411] Report of the Board of Health of Hawaii, 1909: 186, reproduced in: Hoffman, Fredrick L. *Is Leprosy Increasing?* 1920: 49-50. Between 1866 and 1906, 5.876 'lepers' were sent to Molokai.

disease, as well as direct policy recommendations. The answers varied widely and illustrate the wide range of coexisting etiological explanations for leprosy.

Dr. J. L. Meares, Health officer of the San Francisco Board of Health, saw leprosy as highly contagious and argued that "The isolation of lepers is so important and its necessity so self-evident, that I scarcely think the subject worthy of discussion."[412] Boston physician John C. White agreed: "Lepers belong to the dangerous classes of the community which require perpetual confinement, and the sooner this remedy is applied the less seemingly cruelty will attach to it."[413] The report also included Henry Vandyke Carter's *Prevention of leprosy by segregation* (1884) which argued the necessity of registration and isolation based on the results achieved in Norway,[414] as well as two papers from American medical journals arguing that the leprosy bacillus was proof that leprosy was a specific and contagious disease.[415]

Despite contagion being the ruling paradigm on Hawaii, contagionists arguing for segregation were in a minority in the replies. The largest report came from India, where Alexander Mackenzie, Secretary to the Government, summarized that there was no need for direct action since the disease was not contagious: "In support of this view it may be mentioned that not a single servant of the asylum at Almora in the Kumaun District of the Northwestern Provinces appears to have contracted the disease during the thirty-one years for which there is information."[416] Surgeon General with the Government of Bombay, W. J. Moore, withheld that leprosy was "a latent syphilitic inherited constitutional taint" and that "the means of preventing leprosy is not in reviving the antiquated system of leper asylums, but by measures against the spread of syphilis and by sanitation in the fullest sense of the term. (…) In

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[412] Meares, Dr. J. L. "San Francisco, California". *Leprosy in Foreign Countries.* 1886: 214.
[413] White. "The Question of Contagion in Leprosy". *American Journal of the Medical Sciences.* October, 1882. Reprinted in: *Leprosy in Foreign Countries.* 1886: 222.
[414] Carter, H. V. "Prevention of Leprosy by Segregation" . *Leprosy in Foreign Countries.* 1886: 82-101. More on Carter in the following chapter.
[415] White, Dr. "The Question of Contagion in Leprosy". *American Journal of the Medical Sciences*. October 1882, reprinted in: *Leprosy in Foreign Countries.* 1886: 219-222; Belfield, Wm. T. "The Bacillus of Leprosy." *Journal of Cutaneous and Venereal Diseases*. July, 1883. Reprinted in: *Leprosy in Foreign Countries.* 1886: 223-226.

sanitation I include the prevention as much as possible of whatever entails a state of human system below par."[417] In Moore's view, the disease would decline if the government provided a robust economy, affordable and nutritious food and clothing, improved housing conditions, drainage, "in short, everything tending to improve the condition of the population of a country."[418] T. R. Lewis and D. D. Cunningham, who in 1877 published the conclusions of a sanitary investigation regarding leprosy in India, concluded that the disease was hereditary. Since lepers tended to develop sterility, the disease would gradually disappear without any intervention.[419] While sanitary explanations dominated, the report from India was the one with the largest variety in the explanations of the mechanisms which determined the outbreak of the disease. The vast majority did however have the conclusion in common: There was no need to intervene against leprosy specifically, but improved sanitary conditions would get rid of this and other diseases.

As I will return to in the following chapter, the hands-off approach was the dominant line of reasoning in India from Gavin Milroy's report on leprosy in the British Empire in 1867 to the end of the century. As Dyce Duckworth, the appointed representative of the Royal College of Physicians to the Leprosy Commission to India would argue in 1893: The arguments for contagion were circumstantial at best, and much clearer for other diseases. As long as there were no policies for segregating people with tuberculosis or syphilis, the whole idea of segregating lepers was absurd.

> I know of no trustworthy evidence to prove that a leper in any community is a source of greater danger than is a consumptive patient, and I know that a person suffering from Syphilis is a real and very positive source of danger anywhere. It would therefore be absurd

[416] Mackenzie, A. "Leprosy in India". *Leprosy in Foreign Countries.* 1886: 11.
[417] Moore, W. J. "Extracts from report of August 12, 1885". *Leprosy in Foreign Countries.* 1886: 13
[418] Op. cit.
[419] For a summary of the different anti-contagionist positions (the hereditarian, the dietary, and the sanitarian), see: Pandya, Shubhada S. "Anti-Contationism in Leprosy, 1844-1897". *International Journal of Leprosy and other Mycobacterial Diseases.* Vol 66, No. 3. 1998: 374-384. More on the reports on leprosy in India in the following chapter.

> on the face of it to adopt stringent laws for the leper, and to let the Syphilitic person go free.[420]

While Carter's report argued that the disease in Norway was considered to be contagious, Consul-General for Salvador in Norway, Søren Caspersen, held hereditarism to be the ruling paradigm. When Carter pointed at isolation, Caspersen attributed the reduction of new cases to "The higher civilization, better sanitary habits, and the public asylums".[421] Heredity was also the ruling paradigm in Japan. Surgeon K. Yamamoto reported that the disease was "*highly inheritable* (…) so those who have leprosy, or its tendency, among their families or relatives, are *strongly refused to marry* with other healthy families."[422] The shame the disease brought on the whole family led the lepers to be confined at home and isolated from social relations with others. In Mexico, the disease was regarded as hereditary but only through the maternal line; in Canada and the Canary Islands the physicians withheld that heredity did not cause leprosy in itself, but was a necessary predisposition for contagion to take place. There were also those who argued that leprosy spread through sexual intercourse; while others echoed the arguments from India that climate, poverty and sanitary conditions were either directly or indirectly responsible.

Especially in Britain, where the variety of etiologies was greater than anywhere else, Hawaii was highlighted as proof that segregation was futile. Prominent anti-vaccinationist William Tebb, for instance, argued in 1893 that the law of enforcing segregation of lepers at Molokai was "an admitted failure (...) the most heart-rending and painful experience, in a tolerably long life, I have ever witnessed."[423] Since the only way the disease could spread was through inoculation, the primary means of prevention was to ban vaccination: Segregation was unnecessary, cruel and

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[420] *Leprosy in India, Report of the Leprosy Commission in India, 1890-91*. 1893: 9.
[421] Caspersen, Soren. "Norway." *Leprosy in Foreign Countries*. 1886: 192. According to Caspersen, a rationale for the public asylums was that they segregated the sexes and therefore stopped the new cases from being born.
[422] Yamamoto, K. "Leprosy in Japan". *Leprosy in Foreign Countries*. 1886: 217. Italics in the original.
[423] Tebb, William. *The Recrudescence of Leprosy and its Causation. A Popular Treatise*. 1893: 282.

inhumane.[424] Jonathan Hutchinson agreed, but on radically different grounds: The disease was caused by the consumption of badly cured fish, and claims that the Chinese spread the disease was merely a consequence of them introducing fish to the local diet.[425] In order to solve the problem of leprosy, one simply had to promote an enhanced diet. Compulsory segregation was not just cruel, it was beside the point.[426] Contagionists, on the other hand, held Hawaii as proof in opposition to theories of climate, soil, diet and habits of life, and pointed out that "All these causes acting together for centuries did not produce the disease in the Hawaiian Islands, nor was it known there until some time after the islands were open to foreign trade and commerce with other nations. Hence it is reasonable to suppose that it was imported from other places."[427]

[424] According to Tebb, only way the disease could spread was through inoculation, "both accidental, as in a cut or a sore, and by design as in vaccination. I believe that the instances of communication apart from inoculation of this disease (if they exist at all) are extremely rare." (Tebb 1893: 81). Proper clothing and personal hygiene was in itself enough to prevent accidental inoculation, and thus the theory explained the correlations between prevalence of the disease, culture and climate. Tebb's arguments were well known, but contested. A year before the publication of *Leprosy and Vaccination,* Beaven Rake and George Alfred Buckmaster, physicians and members of the British Leprosy Committee, concluded after comparing the number of people being vaccinated and the number of people with leprosy that "the risk of transmission of leprosy by vaccination is so small, that, for all practical purposes, it may be disregarded." (Rake, Beaven and George Alfred Buckmaster. "An inquiry into the question of communicability of leprosy by vaccination." *Journal of the Leprosy Investigation Committee.* No. 4, 1892: 35.) When the Leprosy Commission in India published their report later in 1893, they too rejected Tebbs theory after a long discussion. (*Leprosy in India. Report of the Leprosy Commission in India. 1890-91.* 1893: 127-135).

[425] "Gardening, poultry-rearing, and fishing are a Chinaman's natural vocations, and he will cook and eat almost everything that has had life. There is no improbability in the supposition that the Chinese may have introduced into the Sandwich Islands novelties in the way of food, and especially in reference to shell-fish and fish. This hypothesis would explain how it is that the Chinese introduce the disease only into certain places and fail entirely in others." (Hutchinson, Jonathan. "Memoranda for Future Investigations as to the Cause of Leprosy". *Journal of the Leprosy Investigation Committee.* No. 1. 1890: 87.) According to Hutchinson, the dietary model also explained how the disease had proliferated in early Medieval Europe: Leprosy was not imported by Crusaders like the contagionists argued, but spread through the advance of Christianity and its promotion of eating fish at least once a week. And it was not segregation, but improved diet that explained why the prevalence later had declined: "I believe that the advance of Christianity, with its salt-fish feasts, and not the Crusades, was mainly conducive to the general prevalence of the disease in Europe during the Middle Ages, that its spreading is always due to food and never to contagion, and its disappearance to an improved diet, and not in the least to enforced isolation." (Hutchinson, Jonathan. "Memoranda for Future Investigations as to the Cause of Leprosy". *Journal of the Leprosy Investigation Committee.* No. 1, 1890: 68.)

[426] "Compulsory segregation would I think involve injustice, and entail much social misery." (Hutchinson in: *Leprosy in India, Report of the Leprosy Commission in India, 1890-91.* 1893: 10.) The British physician was, however, not adverse to voluntary segregation in philanthropic institutions.

[427] Abbott, Samuel W. "Massachusetts: Extracts from the Fourth Annual Report of the State Board of Health". *Leprosy in Foreign Countries.* 1886: 209.

For Gibson, the continued detection and confinement of cases did not indicate that segregation was futile. It merely proved that the disease on the island was especially contagious. "To judge by the number of cases in proportion to the population, the disease appears to be more virulent and malignant in the Hawaiian Archipelago than elsewhere on the face of the globe."[428] Samuel W. Abbott from Massachusetts, in contrast, argued that the problem was not in the legislations, but that the implementation was not sufficiently strict: "If it be urged that isolation and other sanitary measures have not succeeded in controlling the spread of leprosy in the Sandwich Islands, it should also be stated that in the earlier years of sanitary control the execution of the law was opposed very generally, especially by concealment and deceit."[429]

While the Kingdom of Hawaii was one of the first modern countries to legislate compulsory segregation for the prevention of leprosy, the dubious outcomes made it a case in point for the defenders of numerous different etiologies. But Hawaii was not the only country to legislate segregation.

At the conference in Berlin in 1897, Gerhard Armauer Hansen made a more compelling argument for segregation: Statistics which showed that since the practice of sending 'lepers' to state institutions had begun in Norway in 1856, the number of new cases had declined every year. When compulsory registration began in 1856, there were 2870 registered lepers. By 1895, the official statistics showed only 321 known cases. The decline was sharpest in the municipalities where lepers were sent to institutions. According to Hansen, the decline was therefore explained by Norway's increasingly strict practice of isolation, especially the legislations from 1877 and 1885 which he himself had designed.[430] "These results, which we in Norway have achieved

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[428] Gibson, Walter M. *Leprosy in Foreign Countries*. Hawaii 1886: 1. The report had little or no practical consequences. The Bayonet Constitution enforced in 1887 stripped Gibson (who by then held the posts of Prime Minister, Minister of Interior and Secretary of War and Navy) of all his powers.

[429] Abbott, Samuel W. "Massachusetts: Extracts from the Fourth Annual Report of the State Board of Health". *Leprosy in Foreign Countries.* 1886: 212. Likewise, at the first leprosy conference in Berlin in 1897, German consul Henry F. Glade argued that it was first in the 1890s, when segregation was more rigorously enforced, that the 'leprosy epidemic' was finally brought under control. (Glade, H. F. "Die Isolirung der Aussätzigen und die dazu erforderlichen Massregeln". *Mittheilungen 1897*. Bd. 2. 1897: 182-183.

[430] Already at the International Medical Congress in Copenhagen in 1884, Hansen had in the paper "The

through the isolation of lepers, show the benefit of the policies."[431]

In addition to surveillance of those affected by the disease, and voluntary institutions from the mid-1850s, the Norwegian laws had since 1885 demanded compulsory isolation of those affected, either at home or in special institutions. Those who had a private bed (preferably in a private bedroom), their own cutlery (which was not to be washed with the cutlery of others), were cleanly and showed caution in associating with others could remain in home segregation, the rest were to be arrested and sent to one of three state institutions.[432] The power to decide if the proper precautions were taken was granted to the local health committee, headed by the physician responsible for visiting the lepers in the area. As Hansen put it: "This is very democratic, in that it is left to the local community to decide whether they want to protect themselves against the disease or not."[433]

The lessons from Norway were disputed. When the British Leprosy Commission to India published their report in 1893, Hawaii and Norway were both highlighted as examples to prove that segregation had failed.[434] In support of this, they quoted physician W. J. Collins who visited Norway in 1889 and found segregation to be only partial:

Etiology and Pathology of Lepra" presented a similar argument: "Isolation was necessary; for, although the disease was spontaneously curable, they could not cure it; and the practice of isolating the Norway had been effective in reducing the number of lepers in twenty years by nearly one-half." (Editorial. "International Medical Congress: Section of Dermatology and Syphilis". *The British Medical Journal*, August 30, 1884: 426). More on the Norwegian debates on the leprosy legislations in the following chapter.

[431] "Es kann nach den Resultaten, die in Norwegen durch die Isolation der Leprösen gewonnen sind, kaum Zweifel an dem Nutzen derselben erhoben werden." (Hansen, G. Armauer. "Facultative oder obligatorische Isolation der Leprösen." *Mittheilungen 1897*. Bd. 1, Abt. 3. 1897: 4.) In Norway, the legislations against leprosy were considered so successful that they in 1900 were used as a model for a national campaign against tuberculosis. The legislations were designed by Klaus Hanssen, Armauer Hansen's brother. See: Ryymin, Teemu Sakari. *Smitte, språk og kultur. Tuberkulosearbeidet i Finnmark.* 2009: 32-36; Schiøtz, Aina. *Det offentlige helsevesen i Norge 1603-2003*, Bd 2: *Folkets helse – landets styrke, 1850-2003.* 2003: 63-69; Blom, Ida. *Feberens ville rose. Tre omsorgssystemer I tuberkulosearbeidet. 1900-1960.* 1998: Chapter 3.

[432] Hansen, Gerhard Armauer. "Die Isolirung der Aussätzigen und die dazu erforderlichen Massregeln". *Mittheilungen 1897*. Bd. 2. 1897: 163-165.

[433] "Dies ist also ein sehr demokratisches Gesetz, indem es dem Volke selbst freigestellt wird, ob dasselbe sich gegen die Lepra wehren will oder nicht." Hansen, Armauer. "Facultative oder obligatorische Isolation der Leprösen." *Mittheilungen 1897*. Bd. 1, III. Abtheilung. 1897: 4.

[434] "Complete segregation has never been possible. Both in the Sandwich Islands and in Norway it has failed." *Leprosy in India, Report of the Leprosy Commission in India, 1890-91*. 1893: 385.

> To attribute the decline of leprosy in Norway to compulsory isolation is entirely erroneous. In the first place no such powers exist or are likely to be sanctioned by the Norse democracy; if they did exist, it would be impossible without further accommodation to segregate even the reduced number of lepers in Norway at the present time. Indeed, I met many lepers in the streets of Bergen and on the quay, going about their usual vocations.[435]

Collins supported Hutchinsons's dietary theory (that leprosy was caused by eating badly cured fish), and in line with what Milroy had argued in 1867, he believed that "the exaggerated notion of its communicability in the lay man" was due to "the repulsiveness of the disease". It was not segregation that was stamping out leprosy in Norway, "but the increased material prosperity of the people, the growth of foreign trade, the inter-communication with town life, and the opportunities these give for better and more varied subsistence."[436] In other words: Segregation did not address the real cause of the disease, and was therefore unnecessary.

No strong defenders of the sanitary model of leprosy attended the conference in Berlin in 1897, but both the dietary theory and the theory of heredity had their champions. Jonathan Hutchinson pointed out that the theory of contagion did not fit all observations: "The suggestion that it spreads by contagion might well fit some of the facts but it is absolutely incompatible with many others." In some places (such as Hawaii) the disease spread despite precautions, in others it died out entirely "in spite of the utter neglect of precautions against contagion."[437] Further, even direct inoculation experiments had failed to prove that this could cause the disease. The only common feature in places with leprosy around the globe, Hutchinson repeatedly

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[435] Collins, W. J. "Note on the Leprosy Revival". *The Lancet*. May 17, 1890: 1064. Quoted in: *Leprosy in India, Report of the Leprosy Commission in India, 1890-91*. 1893: 386. This interpretation of the Norwegian leprosy system seems widespread among British physicians. But "lepers in the streets of Bergen (…) going about their usual vocations" was representative only of the privately run St. Jørgens Hospital, not the State institutions. (Andresen 2004). The lax segregation at St. Jørgens was a constant concern for contagionists such as Hansen, and in 1891 the lepers were banned from trading their goods at the public market in Bergen. For more on the conflict between the two institutions, see: Vollset, Magnus "Ett år på St. Jørgen – 1885 i dokumenter". In: Irgens, Lorentz M., Yngve Nedrebø, Sigurd Sandmo and Arne Skivenes (eds.) *Lepra*. 2006: 65-70).

[436] Collins, W. J. "Note on the Leprosy Revival". *The Lancet*. May 17, 1890: 1064-1065.

[437] Hutchinson, Jonathan. "Erste Sitzung, Einleitung". *Mittheilungen 1897*. Bd. 2. 1897: 21.

insisted, was the consumption of fish.[438] However, at the conference Hutchinson was indisposed, and when his paper was read by his countryman Phineas S. Abraham it was simply ignored by the rest of the delegates. Zambaco Pacha from Constantinople suffered the same fate when he argued that the disease was hereditary, and that the best prophylaxis was to ban lepers from entering into marriage.[439]

The different etiologies were not mutually exclusive. Physician Julius Goldschmidt from Madeira, for instance, saw leprosy as contagious with a hereditary taint. In 1891 he therefore argued that: "Complete isolation of all lepers and their families is the only reliable measure in order to quickly and totally eradicate the contagium."[440] Furthermore, this should be combined with sanitary measures aimed at alleviating the power of resistance in the rest of the population through abundant food, a moderate quantity of sound alcoholic drinks and cleanliness. At the conference in Berlin he still held predisposition as a possibility, but argued that the policies put in place in Norway had produced the best effects in that the number of lepers had declined.[441] Also the official delegate from the Government of Hawaii, Superintendent Luis F. Alvarez from the newly opened Hospital for the Treatment of Leprosy in Honolulu, concluded that matters would have been worse had segregation not been put in place: "The results have been disappointing to those who had

[438] "...if the food-hypothesis be the true one all measures for the compulsory segregation of lepers are useless and cruel. It is the disuse of all forms of half-cured or uncooked fish is I feel convinced the simple measure which ought to be enforced in districts in which leprosy is prevalent." Hutchinson, Jonathan. "Erste Sitzung, Einleitung". *Mittheilungen 1897*. Bd. 2. 1897: 23.

[439] Pacha, Zambaco. "Des rapports qui existent entre la maladie de Morvan, la Sytingomyélie, la Sclérodermie, la Sclérodactylie, la Maladie de Rayunaud, la Morphée des Contemporains, l'Aïnbum, l'Atrophie musculaire progressive Aran-Duchenne et la Lèpre". *Mittheilungen 1897*. Bd. 1, III Abtheulung. 1897: 21-81. Both would continue to defend their theories in the columns of *Lepra Bibliotheca Internationalis,* but were met with strong criticism. In 1907, for instance, Zambaco Pacha argued that the best way to promote the extinction of leprosy was that a certificate from a physician should be made mandatory in order to enter into marriage, and that lepers should be disqualified. (Pacha, Zambaco. "La contagion de la lèpre en l'etat de la science." *Lepra Bibliotheca Internationalis*, 1907: 129) Pacha was not the only one to connect hereditary with marriage prohibition as a suitable response. In Norway in 1850 when the disease was thought to be hereditary, for instance, a committee suggested that people from 'leprous families' should be banned from marriage. (Vollset 2005: 44-47).

[440] Goldschmidt, Julius. "The Madeira Leprosy". *Journal of the Leprosy Investigation Committee.* No. 4. 1891: 31.

[441] Goldschmidt, Julius. "Vorschläge zur Verhütung und Unterdrückung der Lepra". *Mittheilungen 1897*. Bd. 1, II Abtheilung. 1897: 14-17.

predicted that this law would be the means to stamp out the disease. However the law has been very useful in keeping the disease in check."[442]

The contagionists dominated the Berlin conference. Despite admitting that the precise mechanisms for contagion were unknown, the vast majority of those attending agreed that the disease was caused by a bacillus, that the bacillus was found in every leper and that therefore every leper was a danger to others. As for other pathogenic diseases, the bacillus shifted the focus of disease prevention from a range of environmental and hereditary factors to a focus on humans as carriers of disease. But not everyone agreed, and the bacillus did not entail complete agreement when it came to prevention. What was at stake was finding the right balance between the liberty of those suffering from the disease, and the need to protect those not yet infected. As Hansen argued in the debate the first day, it was a question of rights:

> A sick person has not only rights but also duties, and the primary and most sacred duty must be to not jeopardize the health of his fellow man. The healthy too have human rights, and this obviously includes protection from disease. Fortunately, the healthy are in the majority, and it would be foolish to turn that majority into a minority. (…) I find it more humane to protect people from leprosy than to grant lepers the right and opportunity to spread their disease. I have no doubt that we do mankind a great service if we could free the world from leprosy, and this is indeed possible.[443]

What Hansen argued was necessary, was a system of surveillance and isolation, as well as educating the population that lepers were dangerous. But since the contagiousness of the disease was probably quite limited, Hansen stressed that the requirements for home segregation were not very strict. This position was more

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[442] Alvares. "Die Isolirung der Aussätzigen und die dazu erforderlichen Massregeln". *Mittheilungen 1897*. Bd. 2. 1897: 182.

[443] "Ein kranker Mensch hat neben seinem Rechte auch Pflichten, und die grösste und heiligste Pflicht muss sein, seine Mitmenschen nicht zu gefährden. Ferner haben die Gesunden auch ihre Menschenrechte, und zu diesen gehört offenbar auch, sich gegen die Krankheit zu wehren. Glückweise aber sind die Gesunden in der Majorität, und es wäre unzweifelhaft thöricht, diese Majorität, wenn möglicht, in eine minorität zu verwandeln. (…) Ich finde es dagen viel humaner, die Menschen gegen Lepra zu schützen, als den Leprösen das Recht und die Gelegenheit zu geben, auch andere leprös zu machen, und ich zweifle nicht, dass wir der Menschheit einen

moderate than the legislations in Hawaii, and too moderate in the eyes of some contagionists.

In the months before the conference, the most vocal critic of the Norwegian system was the American physician Albert S. Ashmead, who believed the lepers in Norway were granted too much freedom to spread their disease. He called for "perfect isolation; not isolation as it exists in private houses of Norway to-day (for Norwegian isolation is far from being complete), but real, complete isolation in separated colonies, by decrees enforced by government."[444] His argument was simple: Given that the disease was incurable, those already affected by the disease were beyond hope. The most humane approach was strict isolation, not for the benefit of the sick, but to protect the healthy: "if we take care only of one generation of lepers, and prevent inoculation of children, the disease will be eradicated. Is this not worth fighting for?"[445]

The more highly contagious the disease was perceived to be, the stricter the advice for segregation. While the bacillus was not necessary to the argument, as the case of Hawaii shows, the bacillus was increasingly referred to as decisive proof that contagion was beyond dispute and that this dictated isolation. In 1879, the Swedish physician Fredrik Eklund was the first to use the presence of a bacillus as proof that leprosy must necessarily be highly contagious. Therefore, he argued, lepers should be confined in a separate room, everything they touched should be considered contaminated, and the population should be constantly reminded that those suffering from the disease were dangerous: "There should be a white, oil painted plate above

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grossen Dienst leisten würden, wenn wir dieselbe von Lepra befreien könnten; das aber ist unzweifelhaft möglich." Hansen, Gerhard Armauer. "Erste Sitzung, Einleitung". *Mittheilungen 1897*. Bd. 2. 1897: 17.

[444] Ashmead, Albert S. *Suppression and Prevention of Leprosy*. 1897: 75-76. Also later physicians, such as bacteriologist H. Bayon of South Africa, argued that the leprosy campaign in Norway had been unnecessarily prolonged due to being too lenient: "Norway has also successfully dealt with the disease; but laxity there in methods of segregation, notwithstanding a very complete record of all cases, has made it a very much longer process than was necessary. (…) Leprosy is an unmitigated evil in any country, and if experience or scientific research tells us that the scourge can be stamped out even the most causistic ingenuity cannot find an argument in favour of allowing the cause of so much misery to continue to exist." (Bayon, H. "On the necessity or advisability of segregation in relation to the conditions and spread of Leprosy in South Africa at the present time; the measures to be provided for the prevention and cure of Leprosy; and the suitability of Robben Island as a place of detention for Lepers." *Lepra Bibliotheca Internationalis*. 1914: 134-135.)

[445] Ashmead, Albert S. *Suppression and Prevention of Leprosy*. 1897: 76-77.

the entrance of the lepers home, on which the words 'leper infected' is written with so big large letters that they can be read by a person with normal vision from at least 50 cubits distance [~30 meters]."[446] Finally, the leper should be provided with a Bible promising hope for the afterlife.

Others attending the Berlin conference argued that to the contrary, compulsory segregation went too far. Alvarez, the American physician who pioneered the method for boiling and grinding tissue samples to ensure that no bacilli hid inside them, warned that what the experiences from Hawaii really showed was that treating lepers as criminals destined for exclusion only led people to conceal their disease.[447] The same was reported from South Africa, the British representative Phineas S. Abraham added:

> (…) it appears to me that attempts at the absolute isolation of lepers must in many places be quite futile. Where the lepers are few and public opinion unanimous as to the necessity for their exclusion, such measures might be possible, but where the cases are numerous and not desirous of being seperated from them, harsh measures of isolation and segregation become impossible – their chief result being the concealment of cases.[448]

A third line of reasoning was that the contagiousness of leprosy differed from place to place. This was heralded by Ernest Besnier, medical director of the Hôpital Saint-Louis in Paris, who pointed out that despite 150 to 200 known lepers in Paris alone, the prevalence did not seem to increase. The same had been observed in Britain,

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[446] "Öfver ingången till den spetelskes bostad skall ständigt finnas uppsat en oljemålad, hvit tafla, å hvilken orden «spetelsk smitta» äro anbragta med så store, svarte bokstäfver, att de kunna läsas af en normalseende på minst femtio alnars afstånd." Eklund, Fr. *Om Spetelska (Elefantiasis Græcorum vel Lepra Arabum).* 1879: 87-90, quote om page 89-90.

[447] "I am opposed to the adoption of rigorous or cruel measures against the lepers, because they lead to concealment of cases and thus defect the object for which they were adopted, and, above all, because we ought to adhere to principles of justice and humanity and must not treat the lepers as if they were criminals. The Hawaiians are the most peaceful and law-abiding people in the world, and yeat at times they resist the officers of the law when they attempt to arrest a membef of their family." (Alvarez. "Die Isolirung der Aussätzigen und die dazu erforderlichen Massregeln". *Mittheilungen 1897*. Bd. 2. 1897: 182.) Some six years later, however, after Hawaii had been annexed by the United States, Governor Sanford B. Dole argued to the contrary: The recent decline in new cases being sent to Molokai was due to introducing more rigorous segregation: "From these figures it would appear that with strict segregation the disease has steadily diminished, while, without strict segregation, it shows a tendency to spread." ("Leprosy in Hawaii. Report of Governor of the Territory of Hawaii." *Lepra Bibliotheca Internationalis*. 1903: 98.)

where all but a few of the twenty or so known cases had caught the disease in the colonies where the disease was endemic. With proper hygiene and cleanliness, and an administration set up to supervise this, segregation was not necessary, Besnier withheld – at least not in Europe.[449]

Despite objections, the conference agreed to pass resolutions which highlighted leprosy policies in Norway as the golden standard for others to mimic. The reason was not just that Norway had succeeded in reducing the number of cases, or that it was the discoverer Hansen that had come with the suggestion. Rather, recommending Norwegian-style segregation was sufficiently strict to satisfy those who saw the disease as highly contagious, at the same time the comparatively lax rules for home segregation made the Norwegian ideal acceptable also to those who saw compulsion as counter-productive. Finally the emphasis that 'local conditions' differed, meaning both the capacities of the health system and the relative contagiousness of the disease, and that decisions regarding prevention therefore should be taken by medical experts as close to the problem as possible, made the recommendations universally acceptable. In effect, references to the successful campaign in Norway could be used to justify compulsory segregation one place, and as an argument against compulsion in another.

The final resolution read as follows:

> In view of the virtual incurability of Leprosy and the serious and detrimental effects which its existence in a community causes, and considering the good results which have followed the adoption of legal measures of isolation in Norway, the Leprosy Conference, as a logical issue of the theory that the disease is contagious, has adopted the following resolution proposed by Dr. Hansen and amended by Dr. Besnier.
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> 1) In such countries, where leprosy forms foci or has a great extension, we have in isolating the best means of preventing the spread of the disease.

[448] Abraham. "Sechste Sitzung." *Mittheilungen 1897*. Bd. 2. 1897: 195.
[449] Besnier. "Sechste Sitzung." *Mittheilungen 1897*. Bd. 2. 1897: 171.

2) The system of obligatory notification of observation and isolation as carried out in Norway is recommended to all nations with local self-government and a sufficient number of physicians.
3) It should be left to the legal authorities after consultation with the medical authorities to take the special measures applicable to the special social conditions of the districts.[450]

Push, pull, restrict

The primary goal of all strategies for prevention was to protect the healthy majority from contagion, not to benefit those suffering from disease. Given that leprosy was contagious and incurable, and isolation was the best, if not only, means of prevention, how was this to be achieved? While the local specificities differed, in the first two decades after the Berlin conference three main strategies were pursued: 'Pull', 'push' and 'restrict'.[451]

Hansen and most other physicians who advocated segregation leaned towards push-strategies. This meant employing legislation aimed at forcing lepers into isolation. In practical terms, this required a system for the detection of cases, areas set aside where the lepers were to be isolated from the healthy, either institutions, leper colonies or home segregation, and propaganda aimed at educating the population that lepers were dangerous so that they would be pressured into seeing isolation as a refuge. Push-strategies dominated the medical debates between 1890s and the 1920s.

The pull-strategies relied on making segregation attractive, so that the lepers would seek institutionalization voluntarily. Initially this was advocated by those who

[450] Abraham, Phineas S, Edward Arning, E. von Bergman, Dubois-Havenith, J. J. Kinyoun and G. Thibierge. "The Honorary Secretaries' report from the First International Leprosy Conference" *Mitteilungen 1897*. Bd. 2. 1897: 191-192.
[451] The terms 'pull' and 'push' are adapted from migration research, where the expressions have been used to describe the factors leding people to leave their original homes ('push') and the factors attracting people to a specific destination ('pull'). The origin of the terminology is Everett S. Lee's theory of migration, which presents pull and push as the first of four factors determining migration: "1. Factors associated with the area of origin. 2. Factors associated with the area of destination. 3. Intervening obstacles. 4. Personal factors." Lee, Everett S. "A Theory of Migration" *Demography*. Vol. 3, No. 1. 1966: 47-57; quote on page 50.

saw segregation as unnecessary due to the limited contagiousness of the disease, but increasingly this line of reasoning was adapted also by contagionists who came to the conclusion that the promise of forced and indefinite detention only led those affected by the disease to hide their disease for as long as possible.

The third dominant strategy was aimed at reducing the chances of contagion not through isolation, but through placing restrictions on those suffering from the disease. Especially in India, but also in French colonies and elsewhere, lepers were banned from certain trades, from the use of public transport and from begging in public places. This strategy was more affordable than wholesale segregation, it educated the population that leprosy was contagious, and was an easily administered response to local calls for legislative action.

There were also 'minor' strategies, such as separating the children of lepers from their parents, first proposed in 1879 by William Munro and later propagated by missionaries.[452] This was not part of the first set of recommendations from 1897, but made part of the conclusion in Bergen in 1909 and repeated in Strasbourg in 1923. When BELRA surveyed the prevalence and prophylaxis against leprosy in 1924, they found that early separation of children born to lepers was almost uniformly pursued in the British Empire.[453]

The strategies were not mutually exclusive, and in practice most national

[452] Munro 1879: 95. In a letter to the leprosy conference in Berlin, the founding head of Mission to Lepers, Wellesley C. Bailey, underlined that "if the children of leprous parents can be separated from their leprous relatives and all leprous surroundings, they will in most cases never contract the disease. (…) The Society which I represent has now several Homes for the untainted children of lepers." (*Mittheilungen 1897*. Bd. 2. 1897: 77.) In general, the missionaries appears to have contested conclusions, such as from the Leprosy Commission to India, which claimed the contagiousness of leprosy to be relatively limited. As John Jackson put it in 1900 in one of the first in a series of biographies describing the life and hardships of a missionary to lepers: "it may be fairly inferred that the danger of contagion is by no means, so 'exceedingly small' as the Commissioners suppose. (…) the Committee and their Commissioners were unanimous on one point, viz, that of *incurability.*" (Jackson, John. *Mary Reed. Missionary to the Lepers.* 1900: 49.) In February 1902 the Mission to Lepers arranged a Conference of Superintendents of Leper Asylums in Wardha in central India, which concluded that "The Conference as a body and as individuals is convinced of the contagious character of leprosy. (…) in view of the extremely serious nature of the disease, lepers should be segregated." (Bailey, Wellesley C. "British India". In: Rosenthal, O. *V. Internationaler Dermatologen-Kongress abgehalten in Berlin vom 12.-17. September 1904.* Bd. 1. 1904: 96.)

[453] Rogers, Leonard. "Memorandum on the prevalence of and prophylaxis against leprosy in the British Empire, based on replies to the questionnaire of the British Empire Leprosy Relief Association; with suggestions for dealing with the problem." (LNHO: 12B/42641/29272). 1925: 12.

leprosy campaigns consisted of combinations of these tactics. Emphasis differed and could change over time. Regardless of strategy, more often than not the recommendations from the international conferences were made part of the local argument.

The push-strategies emphasized teaching the population that lepers were dangerous, to add social pressure so that the lepers would seek refuge in the institutions. As Hansen put it at the conference in Berlin; the lepers never acknowledged that they posed a danger to the healthy, and naturally did not want their freedom restricted. Part of the Norwegian success, he explained, was public lectures reserved for the healthy, explaining the virtues of cleanliness, promoting the necessity of isolation, and educating them that lepers were dangerous. "The healthy listen and the main message is that the healthy do not want to have dealings with the lepers. This is my goal. In Norway we have achieved that a leper who wants to hire a servant does not find one."[454]

There was never a universal stigma connected with leprosy. Especially where the disease was endemic, many saw leprosy as an unpleasant but fairly ordinary way of growing old. As Medical Officer to the Hausa Association's Central Sudan Expedition in 1893-95, T. J. Tonkin, presented in a paper in London in 1902:

> The disease is so common that in spite of the repulsive appearance of the sufferers, the general public have, as far as I could make out, no objection to it. They are accustomed to it, and regard it as one of the stable things of the world, and the chance of catching it as one of the ills to which human flesh is inevitably heir. They do nothing so far as I know to limit the chance of contagion.[455]

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[454] "Die Gesunden beachten die Ausführungen, und das ist ja die Hauptsache, dass die Gesunden nicht mit Leprösen Umgang haben wollen. Wenn ich das durchgesetzt habe, ist mein Ziel erreicht. Wir sind jetzt in Norwegen so weit gekommen, dass ein Lepröser, der einen Diener haben will, keinen bekommt." "Die Isolirung der Aussätzigen und die dazu erforderlichen Massregeln". *Mittheilungen 1897*. Bd. 2. 1897: 163. See also: Hansen, Armauer. "Facultative oder obligatorische Isolation der Leprösen." *Mittheilungen 1897*. Bd. 1, III Abtheilung. 1897: 1-6; Pandya 2003: 172.

[455] Tonkin, T. J. "Some general and atiological details concerning Leprosy in the Sudan." *Lepra Bibliotheca Internationalis*. 1902: 135, first read before *The Royal Medical and Chirurgical Society of London*. May 27, 1902.Tonkin himself believed that the disease was contagious, with a diet "wanting in nitrogenous elements" (141) as a contributing cause. See also: Tonkin, T. J. "An Analysis of 220 Cases of Sudanese Leprosy". *The Lancet.* April 16. 1903: 1077-1083.

Hansen, in his autobiography from 1910, noted that similar attitudes were prevalent on the Norwegian countryside. "When I asked them how they had acquired leprosy, the common answer was that 'it's probably destined', or that Our Lord had brought it."[456] Likewise, the Swedish physician Fredrik Eklund pointed out after visiting Norway in 1878: "In my travels I have found that people are not sufficiently frightened by the thought of contagion."[457]

As contagionism gained momentum, the physicians increasingly saw it as their task to teach the population that lepers ought to be shunned. But the chronic nature of the disease made the danger of contagion difficult to convey. Without public fear of the disease, forcing people into the institutions was almost impossible, as Edward C. Long noted in a report from Basutoland (now Lesotho) at the beginning of the 20th century.

> It is in one sense unfortunate that leprosy is such a very chronic and in its early stages a non-disabling, complaint; were it more rapidly fatal the people would soon learn to dread it and easily co-operate in any means decided upon to stamp it out. As things are at present, any attempt to stamp out the disease by forcible segregation, would as I have already stated inevitably lead to concealment.[458]

Where public fear of the disease was strong, push-strategies were easier to implement. Sometimes the precautions taken went further than the physician recommended, such as in New Zealand where the public opinion bordered on lepraphobia. In 1903, Chief Health Officer J. Malcom Mason reported on a Chinese gold digger who was diagnosed with the disease. Despite Mason declaring that the man had no erosion in the surface tissues, and therefore presented little to no danger from a public-health

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[456] "Naar jeg spurte om de hadde nogen greie paa hvorledes de hadde faat spedalskhet, saa var det jevnlige svar at 'de va vel so laga', eller at Vorherre hadde lagt det paa dem." Hansen, Gerhard Armauer. *Livserindringer og Betragtninger*. 1910: Chapter 5.

[457] "Öfverhufvod har jag under min resa funnit, att folk är alldeles för litet uppskråamdt med hänsyn till smitta." Eklund 1879: 42.

[458] Long, Edward C. "Report to the Colonial Office on an investigation of the prevalence and distribution of Leprosy in Basutoland." *Lepra Bibliotheca Internationalis.* 1905: 234-241, quote on p. 241. Long explicitly argued that mapping the prevalence of the disease should be combined with spreading knowledge about its contagiousness, and that this would pave the way for more dramatic measures at a later point in time.

point of view, the local county council put his hut under quarantine. A special box was set up, where the man could deposit gold, in return for food. "The food is placed in a box about a quarter of a mile from his hut; the gold is boiled before being handled by the bank, and in this way all possible source of infection is destroyed. The police authorities have been asked to keep an eye upon the man to see he does not break bounds."[459]

The specifics of the push-strategies differed from country to country. In Japan, where leprosy already was connected to family shame, the legislation of 1907 emphasized the responsibilities of the physicians. While a system of state leprosy hospitals was established, it was made obligatory for physicians to notify the government of all cases of leprosy, and disinfect and isolate the lepers and their houses. If the physicians failed to fulfill their duties, they were punished with fines.[460]

From 1906 the Philippines sent more than a thousand lepers to the island of Culion every year. Their campaign, too, was built on convincing the population that leprosy was contagious and that Culion was where the lepers belonged. In 1909, Director of Health Victor G. Heiser explained the strategy: "it was deemed advisable to precede the collection of lepers by a campaign of education and thereby secure the cooperation of the public rather than its opposition."[461] Taking advantage of the archipelagos' geography, a team of educators were to tour an island two or more times, followed by rounding up cases, until the area was considered leprosy-free. Only then would they move on to the next. "By the method persued, the greatest amount of

[459] Mason, J. Malcom. "A case of a Chinese gold digger with leprosy". *Lepra Bibliotheca Internationalis.* 1903: 107. This was not exceptional. One year later Mason told the story of a child developing leprosy being moved 200 metres away from the community. The proscription was "not to allow any one to sleep, eat or live with him. This has been carried out. (…) It will be a blessing when these poor unfortunates are taken by the State and isolated on some island." (Mason, Malcom. "Maru Pomare: Leprosy among the Maories. Public Health statement for New Zealand." *Lepra Bibliotheca Internationalis.* 1904: 129.)

[460] "Leprosy in Japan, report by the Central Sanitary Bureau of the Home Department in Tokyo, 1907." *Lepra Bibliotheca Internationalis.* 1910: Fasc 2. See also: Ohtani 1998: 49-54; Burns 2012:301-327: Sato, Hijame and Minoru Narita. "Politics of leprosy segregation in Japan: the emergence, transformation and abolition of the patient segregation policy". *Social Science & Medicine.* Vol. 56. 2003: 2529-2539.

[461] Heiser, V. "Leprosy in the Philippine Islands." *Lepra Bibliotheca Internationalis.* 1910: 36-39, quote on p. 37. First published in *Public Health Report*. No. 35, 13. August 1909:

territory was freed in the shortest possible time. In military phraseology, the outposts were captured first and the lines gradually moved forward to the strongholds.'"[462]

In contrast to what I call the push-strategies, the common denominator for the various pull-strategies was that they were aimed at attracting lepers through having something to offer. As Jonathan Hutchinson pointed out in the first issue of the *Journal of the Leprosy Investigation Committee* in 1890, preventing contagion was not the only rationale for establishing leprosy institutions. When Norway built its state leprosy institutions in the 1850s, Hutchinson stressed, this was motivated by preventing marriage and thus transmission of disease to future generations. A third and final justification was offering comfort to the sufferers, providing a home with food, amelioration and hope of cure through medical treatment.[463] In other words: You did not have to be a contagionist to argue that lepers belonged in an institution.

When the Leprosy Commission to India published their report in 1893, they concluded that the minimal contagiousness of the disease made compulsory segregation unjustifiable.[464] Instead, the conclusion was that improving the sanitary conditions alone would reduce the prevalence of leprosy as well as a range of other diseases. Besides, the prospect of segregating more than 100,000 individuals made compulsory segregation 'impracticable': "Voluntary isolation is, therefore, the only measure left for consideration."[465] Since leprosy was not considered a major threat to public health, singling out the disease did not justify colonial spending. Local authorities were free to build or enlarge leper asylums through municipal funds or private subscriptions, but in practice philanthropic organizations that justified their activities by offering care in voluntary institutions would dominate the preventive measures in the subcontinent. The largest organization was Mission to Lepers, established in 1873. By 1920, 41 of the 92 leper asylums in India, Burma and Ceylon

[462] Op. cit.
[463] Hutchinson, Jonathan. "Memoranda for Future Investigations as to the Cause of Leprosy". *Journal of the Leprosy Investigation Committee*. No. 1. 1890: 87.
[464] "In no case would the Commissioners suggest an Imperial Act, especially directed against lepers as such, for these are far less dangerous to a community than insane or syphilitic people." *Leprosy in India, Report of the Leprosy Commission in India, 1890-91.* 1893: 388.
[465] *Leprosy in India, Report of the Leprosy Commission in India, 1890-91.* 1893: 387.

were run by Mission to Lepers alone, and ten more by their aid. The Indian government and municipalities ran sixteen institutions, and the native states fourteen.[466]

In addition to sanitary measures and voluntary segregation, the Leprosy Commission to India agreed that if local governments wanted to take further action they could prohibit lepers from occupations where the possibilities for accidental contagion were greatest, such as serving or preparing food, prostitution, working as barbers or washing. They could also prevent vagrant lepers from frequenting public places, to beg, or use public transportation.[467] This was made law in The Lepers Act of 1898. Vagrant lepers that caused public nuisance in urban towns could be forced into the institutions, but as Shubhada Pandya has pointed out, India had in reality no colonial containment policy.[468] The institutions were voluntary sanctuaries for the destitute, places where missionaries could spread the gospel, and not tools for segregation.

In the medical textbooks on tropical diseases and dermatology published after the Berlin conference, the descriptions of leprosy almost consistently included a section on segregation. In *Tropical Diseases: A Manual of the Diseases of Warm Climates* (1898), Patrick Manson highlighted that "the most effectual way of suppressing the disease is the thorough isolation of existing lepers."[469] Since the disease was caused by a bacillus, it was contagious. And since the disease was incurable, isolation was presented as logical consequence. But faced with the real world, one needed to be pragmatic. Isolation was the ideal solution, but the practical difficulties many;

> (…) springing from the rights of the individual, finance difficulties, difficulties arising from concealment or incorrect diagnosis, as well as from the continued introduction of fresh cases

[466] The last eleven institutions were labeled 'unclassified', which likely meant other philanthropic organizations. The numbers were compiled by Frank Oldrieve in: The Mission to Lepers. *Conference on the Leper Problem*. Calcutta, February 1920. 1920: 151.
[467] *Leprosy in India, Report of the Leprosy Commission in India, 1890-91*. 1893: 4-5; 385-390.
[468] Pandya 2003: 151-154.
[469] Manson, Patrick. *Tropical Diseases. A Manual of the Diseases of Warm Climates*. 1898: 417.

> from without. These and other obvious obstacles, incident to any attempt at a wholesale system of thorough isolation are so great that the most that can be hoped for in the present time, and in the present state of public opinion, is some modified system of segregation and isolation, such as has worked so successfully in recent years in Norway.[470]

Reflecting how 'copying the success of Norway' was adapted into widely different strategies, Sir Malcom Morris highlighted in *Diseases of the Skin* (1911) that "Strict isolation is the only trustworthy means of checking the spread of leprosy, as is shown by the experience of Norway."[471] But Morris too stressed that segregation would benefit to the lepers, since institutionalization provided access to medical treatments.[472] Norman Walker's *An Introduction to Dermatology* (1911), on the other hand, argued that "The careful statistics of the leper department of the Norwegian Government clearly shows that the number of new cases is directly proportional to the number of patients at large in a district."[473] According to Walker, the term 'patients at large' did not relate to segregation, but that those suffering from leprosy were put under a strict sanitary regime. "According to Hansen, the most important thing both for the patient and the community is to put the patient in as good circumstances as possible, and to use all measures of personal cleanliness; and the remarkable diminution in the number of lepers in Norway under this able and vigorous régime is the very best proof of the value of these means."[474] In later medical textbooks, segregation was presented as the default, but increasingly contested means of prevention.[475]

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[470] Manson 1898: 418.
[471] Morris, Sir Malcom. *Diseases of the Skin. An Outline of the Principles and Practice of Dermatology*. 5. edition. Printed in London, New York, Toronto and Melbourne. 1911: 535.
[472] "Segregation, if properly carried out, is not only a protection to the community at large, but is greatly to the advantage of the lepers themselves, who thus enjoy far better treatment than they could otherwise, in the majority of cases, command." Morris 1911: 535.
[473] Walker, Norman. *An introduction to Dermatology*. 5. edition. 1911: 290.
[474] Walker 1911: 296.
[475] "The management of leprosy naturally includes a consideration of the means of prevention. There are still great differences of opinion as to the necessity of segregation, and each side of the question has much in its support." (Stelwagon, Henry W. *Treatise on Diseases of the Skin*. 7th edition. 1914: 935.); "While segregation is generally considered the one proven prophylactic measure there are those who question its value." (Stitt, E. R. *The Diagnostics and Treatment of Tropical Diseases*. 3. edition. 1919: 216); "The financial burden of isolating large numbers of lepers is very heavy. Hence the difficulty of carrying out this very necessary method

The main cause for concern, as Alvarez had argued in Berlin in 1897, was that compulsory segregation led to the concealment of cases. At the Fifth Livonian Medical Assembly in Pernau in 1903, physician H. Koppel from Dorpat made the case that the propaganda presenting lepers as dangerous must be coupled with promoting the institutions as shelters offering work and fulfilling lives. The leprosaria must "lose their deterrent character in the eyes of the sick and the healthy."[476] The same argument was repeated in numerous places around the globe, which suggests that this was easier said than done. At the sixth international dermatology congress in New York in 1907, for instance, Walter Remsen Brinckerhoff of Honolulu, recommended that instead of highlighting that the disease was incurable, the physicians should emphasize the occasional spontaneous cures, that they actually could alleviate some of the pain, and that they in some cases managed to keep the disease at bay.

> The popular concept that when a patient once entered a leprosarium, it was to die without treatment; this caused much prejudice against these institutions. In communities where leprosy was likely to occur, the people should be informed that proper treatment would render them more comfortable, even if it will not cure them. In this way only would it be possible to get control of the cases early in the disease.[477]

For diseases such as tuberculosis, which also was met with institutionalization of the sick, you were either cured or dead after a few years. Leprosy, on the other hand, could last for decades. This led physicians to argue that the institutions should differ from other kinds of hospitals. In 1898, Memel began building a set of cottages for 12 persons each in order for life in the leprosy institution to resemble life in family units. The rationale was that "The patients must have room enough to walk about freely and to play, the more so, as they are not allowed to leave the premises. Large sitting

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of protection completely and efficiently." (Castellani, Aldo and Albert J. Chalmers. *Manual of Tropical Medicine*. 3. edition. 1919: 1666.)

[476] Koppel. "Über die Beschäftigung der Leprösen in den Leprosorien". *Lepra Bibliotheca Internationalis.* 1906: 57, originally published in *St. Petersburg Medizinische Wochenschrift*. No. 36. 1903.

rooms, gardens and workshops should never be omitted. For if the patients cannot occupy themselves and so avoid feeling dull, they will never feel at home in the hospital."[478] Of the three main policy approaches, the pull-strategies were the most expensive.

More often than not, the responses to leprosy were a mix of push, pull and restrictions, and emphasis could differ over time. In Estonia the first lepers were accepted at a new leprosarium in Kuda in December 1896, where admittance was voluntary. Under the directorship of Arthur Kupffer from 1901, explicitly inspired by the compulsory elements of the Norwegian model, the policies were reorganized. From 1902 reforms demanded that if those affected by the disease could not document necessary facilities for home isolation, the lepers could forcibly be moved to the institution. The reform culminated in 1905 with regulations deciding whom to be held accountable if a leper broke the rules for home segregation. This soon led to a new patient body in the institutions: Instead of attracting almost only incapacitated cases, a majority of the inmates were relatively healthy and in need of meaningful activities. The solution – weaving, spinning, sewing and gardening for the women, and handicrafts for the men – was however acknowledged to be second rate. "From the lepers' point of view, the institutions should offer treatments or a cure."[479]

As the experiments with various treatments proceeded, increasingly the promise of treatment was held as the main argument for pull-strategies. As H. Bayon in South Africa put it in 1914; "Segregation without treatment appears a harsh, if justifiable measure to take. Once treatment is instituted the leper is not only segregated for the benefit of others, but in his own interests, because any treatment that can be suggested must necessarily last a long time and be regularly administered."[480]

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[477] Brinckerhoff, Walter Remsen. "Proposals for diminishing the diffusion of leprosy". *Sixth International Dermatological Congress*. 1908: 46.
[478] Kirchner, Martin. "Lepra in Memel". *Berliner Klinische Worhenschrift.* Nr. 2. 1900, as quoted by Kuznitsky in *Lepra Bibliotheca Internationalis.* 1901: 52.
[479] Kupffer, A. "Die Lepra in Estland." *Lepra Bibliotheca Internationalis.* 1914: 26-29.
[480] Bayon, H. "On the necessity or advisability of segregation in relation to the conditions and spread of Leprosy in South Africa at the present time; the measures to be provided for the prevention and cure of Leprosy; and the

A matter for the state

The invitations to the leprosy conferences, and later the requests from the League of Nations' Leprosy Commission, asked for the prevalence of leprosy in each country. More often than not, mapping prevalence was both a goal in itself and a means to an end. Producing the numbers meant documenting that action was necessary; this was the necessary first step in asking for measures of prevention to be put in place. Producing numbers was one of four main strategies for making leprosy a matter for the state.

Both high and low prevalence were used as arguments for segregation. In Japan, it was statistics showing that the number of lepers had increased from 23,000 in 1897 to 40,000 in 1905 that led the state to start its anti-leprosy campaign.[481] In Tunisia, director Charles Nicolle at the Pasteur Institute argued that since there were only about 70 lepers in the colony, it was imperative to take action before the situation got out of hand.[482]

Having begun a leprosy-campaign was often used as an argument to expand it. In the Cape Colony in 1907, fifteen years after the introduction of a Leprosy Repression Act, Medical Officer A. John Gregory stressed in his annual report that accommodation at Robben Island (651 inmates) and Emjanya Asylum (473 inmates) was insufficient. The new cases outnumbered the available accommodation. "Of course, every Leper segregated is a source of infection removed, but it is open to question whether it is altogether fair on those Lepers who are compulsory segregated

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suitability of Robben Island as a place of detention for Lepers." *Lepra Bibliotheca Internationalis. 1914: 134.* Although increasingly common, this was not a new argument. Already in 1890, Robson Roose argued that compulsory segregation was to the benefit of the lepers because physicians were the only ones to provide pain relief: "Compulsory isolation in suitable buildings and under proper care is urgently demanded in the interests not only of the general community, but of the sufferers themselves. If leprosy cannot be cured, some of the most distressing symptoms can certainly be alleviated." (Roose, Robson. *Leprosy and its prevention as illustrated by Norwegian experience.* 1890: 96.)

[481] "La lèpre au Japon". *Lepra Bibliotheca Internationalis*. 1908: Fasc 3, originally published in *Bulletin general de therapeutique*. July 15, 1905.

[482] Nicole, C. "La lèpre en Tunisie". *Lepra Bibliotheca Internationalis.* 1907: Fasc. 2. What Nicole asked for was two modern leprosaria for the isolation of the sick, and that lepers should be banned from entering the country.

that their fellows should be allowed at large without restraint."[483] Three years after the unification of South Africa in 1910 bacteriologist Harry Bayon repeated the argument: "The whole matter lies in a nutshell: Either we can go on spending the large sums that are allocated now for the maintenance and segregation of a certain proportion of the leprous population for an indefinite number of years, or the decision can be made manfully to grasp the problem, set aside a sufficient sum for the accommodation of all lepers and get rid of the scourge in ten or fifteen years."[484] The problem, according to Bayon, was not only that only a proportion of the lepers could be accommodated, "and the other half or third remaining is quite sufficient to keep the disease going on for ever", but that the institutions were deeply disliked by the lepers who therefore actively avoided detention.

Presenting leprosy as a danger to the healthy population was increasingly successful. At the first international leprosy conference in Berlin in 1897, 22 governments were represented. In Bergen in 1909, the number had grown to 27. At the third international leprosy conference in Strasbourg in 1923, 34 governments were represented. But even more countries had campaigns to prevent leprosy. At the Fifth International Dermatology Congress in 1904, where following up on actions taken against leprosy in the past seven years was on top of the agenda, there were reports on leprosy in 71 countries.[485] The second international leprosy conference in Bergen in

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[483] Gregory, A. John. "Leprosy in Cape Colony for the year 1906." *Lepra Bibliotheca Internationalis*, 1906: 226.

[484] Bayon. H. "On the necessity or advisability of segregation in relation to the conditions and spread of Leprosy in South Africa at the present time; the measures to be provided for the prevention and cure of Leprosy; and the suitability of Robben Island as a place of detention for Lepers." Report by the Gouvernment Research Bacteriologist to the Department of the Interior, Robben Island. February, 18, 1913. *Lepra Bibliotheca Internationalis.* 1914: 135.

[485] In order of appearance: Belgium, Congo, Bulgaria, China, Chile, Egypt, The British Empire (England, Ireland, Gibraltar, Malta, Cyprus, British India, Bengal, Burma, Mauritius, Seychelles, Ceylon, Federated Malay States, The Gold Coast, Lagos, Sierra Leone, Nigeria, British Central-Africa, East-Africa and Uganda, Canada, Bermuda, Jamaica, The Leeward Islands, Antigua, St. Kitts, Barbados, St. Vincent, Dominica, Bahamas, Grenada, British Guiana, New Zealand), Australia, United States, Cuba, France and colonies (Algeria, Tunisia, Morocco, Senegal, French Guiana, Madagascar, Reunion, French Antilles, French Guyana, New Caledonia, Indo-China, Algeria), Holland and colonies (Dutch West-Indies, Dutch East Indies) Iceland, Danish Antilles, Italy, Montenegro, Norway, Austria, Hungary, Romania, Russia, Sweden, Serbia, Siam, Spain, and Turkey. The reports included a brief history of leprosy in each country focusing on how and when the disease was introduced and what measures had been taken to prevent it. In some cases the reports included legal texts. Further, there were statistics on registered lepers or estimates of concurrent prevalence, and reports on institutions (number of beds, admissions and discharges, drawings of the premises, management and how the

1909 had reports on leprosy in 89 countries and territories.[486] In the opening statement, Edvard Ehlers and Feliz Verdier stressed that the disease was found on all continents, in all climates and among all races. What they all had in common was the leprosy bacillus: Proof that the disease was contagious and that regardless of local conditions, this was the basis for preventive measures.[487]

While producing numbers seems to have been a common first step in all leprosy campaigns, the reports presented at the international conferences reflected great differences in what to do next. Involving the state seems to have been a common goal, but the physicians' did not only disagreed on the necessity of segregation and what strategies to pursue, their relationship within the various legislative frameworks also differed. Physicians were seldom the only actors who had a say.[488] Furthermore, the situation on the ground was not the same everywhere, neither when it came to leprosy prevalence and its relative importance in comparison with other diseases, the capacity of the health systems and the willingness of governments to single out leprosy as a target for an intervention. As George Pernet underlined in his presentation of leprosy in the British Empire in 1904:

> Reviewing the foregoing survey, one feature stands out prominently, viz: the enormous mass of leprosy with which the British have to cope. The magnitude of the task needs no emphasizing when we consider the number and variety of the native races, the hugeness of the population, the vastness of the area under the sway of the English.[489]

Not every country had leprosy campaigns. Following the Berlin conference in 1897,

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lepers were kept occupied). Rosenthal, O. *V. Internationaler Dermatologen-Kongress abgehalten in Berlin vom 12.-17. September 1904*. 1. Band. 1904.

[486] Ehlers, E. and Feliz Verdier. "Géographie de la lepre". *Lepra Bibliotheca Internationalis*. 1909: Fasc. 4.

[487] Op. cit. For a critique arguing that the celebration of Hansen's discovery the leprosy bacillus overshadowed clinical findings and the question of what science could do to help those affected by the disease, see: Deycke, G: "Nachklänge zur zweiten internationalen Leprakonferenz in Bergen". *Medizinische-kritische Blätter in Hamburg*. Bd. 1, Heft 1. 1910: 68-75; reprinted and rebutted by Ehlers in *Lepra Bibliotheca Internationalis*. 1910: 246-252.

[488] For a study highlighting the importance of negotigations between various actors at a local level, see: Buckingham 2002. For an overview of the organization of public health measures in Britain, United States, France, Sweden and Germany, see: Porter 2005.

[489] Pernet, George. "Leprosy in the British Empire". *V. Internationaler Dermatologen-Kongress*. 1905: 62

the Austria-Hungarian minister of Finance and administrator of Bosnia and Herzegovina, Benjamin von Kállay (Béni Kállay de Nagy-Kálló), first commissioned physicians Edvard Ehlers and Leopold Glück to write a report on leprosy in Bosnia. Possibly due to realizing that having the prevalence mapped would trigger demands for a costly state intervention, when the report was finished in 1900 it was banned with an explanation that the findings would be bad for tourism.[490] As I will return to in the following chapter, in India the colonial government saw leprosy mainly as a sanitary problem.

The second line of arguments in involving the state was by pointing at interventions in other countries. While Norway was highlighted as a success to be copied, this was by far the only inspiration. During its fifteen years of existence *Lepra Bibliotheca Internationalis* (1900-1914) published 86 papers dealing primarily with the question of prevention, almost a tenth of the total number of articles. This shows how prevention was an integral part to the medical debate. In addition to numbers from fact-finding missions both domestically and in the colonies, they included policy advice, reports from institutions. 39 of the papers were published under the heading *Prophylaxis, Legislatio etc*, and provided updates on proposed and passed legislations around the world, often including the full legal texts.[491] The medical journals, as well as conference proceedings, created a shared reservoir that the readers to turn to in order to find arguments and examples to use in local debates.

A third prominent line of reasoning was found in *Lepra Bibliotheca Internationalis*' section *Historia*. This consisted mainly of papers dealing with leprosy in Medieval Europe and the reasons for its alleged decline. These papers served two practical purposes: First, they more or less consistently argued that history proved that

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[490] Glück, Leopold. "Die Lepra tubero-anaesthetica, vom klinischen Standpunkte geschildert". *Lepra Bibliotheca Internationalis*. 1908: 2.

[491] Some examples: New South Wales (Thompson, Ashburton. "Report on leprosy in New South Wales, for the year 1897." *Lepra Bibliotheca Internationlis*. 1900: Fasc 1); Denmark (Ehlers, Edvard. "Législation danoise contra la lèpre. Ordonnances Royales du 17. Janvier 1908, visant la lèpre aux Antilles danoises". *Lepra Bibliotheca Internationalis. 1908 : 208.);* Japan (Central Sanitary Bureau of the Home Department in Japan. "Leprosy in Japan. (Tokyo, 1907)". *Lepra Bibliotheca Internationalis.* 1910: Fasc. 2); Indochina ("Arrèté concernant la prophylaxie de la lèpre en Indochine." *Lepra Bibliotheca Internationalis.* 1910: Fasc. 3,

segregation worked. While leprosy never completely disappeared from Europe, consensus was that the prevalence had dropped dramatically. Most authors withheld that this was due to segregation. Second, by emphasizing the worst-case scenarios, concurrent segregation became less cruel in comparison.[492]

History was also used as an argument in several of the papers published in the section for *Geographia,* which not only outlined the concurrent situation in the given geographical area, but often explained how the current situation had developed. The explanations often included legal measures taken, and what practical consequences these had led to (if any). If leprosy was increasing, the standard explanation was in the preventive measures. Either no policies were put in place, the enforcement was not sufficiently strict, or strict policies without sufficient resources had led to concealment.

Forth and finally, the institutional reports published under the heading *Annuaria Leprosarium* explicitly discussed the steps taken in order to found specific institutions, cost per inmate, rules and regulations and what the institution was meant to achieve. While not included in my enumeration above, these reports also provided relevant examples for those arguing that erecting special institutions was a cornerstone in the prevention of leprosy. Institutions needed to be financed, and more often than not the implicit argument was that this was a matter for the state.

Burnet's compromise

The dominant stance until the 1920s was prevention through segregation, and the

originally published in *Bulletin De la société de pathologique Exotique.* 1910)*;* Australia ("Commonwealth of Australia – Law as to leprosy." *Lepra Bibliotheca Internationalis.* 1910: Fasc. 3.)

[492] Medievalist Carole Rawcliffe has convincingly argued that this narrative of medieval 'lepers' being excluded and forced into isolation at the edges of society was based on willful misreading dating from the 19th century. In Britain, at least, leprosaria had more in common with monasteries than tools of segregation. The closing of 'leper houses' had more to do with social and economic factors than the disease disappearing. Rawcliffe, Carole. *Leprosy in Medieval England*. 2006. Rawcliffe's interpretation of Medieval England differs dramatically from what the physicians I have investigated took to be true, but pursing this argument further would go beyond the scope of this thesis.

conference in Strasbourg in 1923 still advocated isolation, banning lepers from certain trades and removing children of lepers from their parents. Still, the era of push-strategies was coming to an end. For the first time the recommendations stressed that "isolation should be humane and allow the leper to be near his family if this is compatible with efficacious treatment."[493] Every country needed an anti-leprosy campaign, but this should be designed according to the circumstances in each country. For those attending the Strasbourg conference, the lessons were clear: Segregation was a necessary evil that should be restricted as far as possibly. Too much emphasis on persecution and the detection of new cases would suffer. Emphasis was shifting from push to pull.

According to Etienne Burnet, secretary of the League of Nations Leprosy Commission, the conference constituted a watershed in that the consensus for the first time was not only that people suffering from the disease had a duty to go into isolation, but that society had obligations in return:

> By that time society had recognized not only its right to defend itself against leprosy but also its duties towards lepers; since it deprived them of a normal existence it realized the obligation to make their life bearable by providing them with human surroundings, hygienic conditions, amusement and occupation, as well as at least a hope of cure.[494]

As I showed in the previous chapter, Burnet saw the hope of a cure in chaulmoogra derivatives, directly inspired by BELRA and the Calcutta-school. This was controversial: As I will show in Chapter 7, most members of the Leagues' Leprosy Commission thought the efficacy of chaulmoogra was overrated and maintained segregation as the best and only prevention. The conclusions reached at the Strasbourg conference was thus more a reflection of the list of attendees more than a dramatic shift in policies everywhere. When Burnet traveled to Europe, Latin America

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[493] *Resolutions and recommendations from the Third International Conference on Leprosy, Strasbourg 1923*. See: Appendix 2.
[494] Burnet, Et. *Provisional Report presented to the Leprosy Commission at its meeting at Tokyo in April 1930.* Part III: General Conditions of Prophylaxis. (League of Nations Health Organisation. C. H./Leprosy/7b) 1930: 2

and the Far East at the end of the 1920s, he found the world divided in two camps:

> (…) the question of segregation still remains a subject of controversy between advocates of freedom on the one hand, and of coercion on the other. The results of the enquiry, which included the consultation of a large number of leprologists, show that any meeting of experts would be divided into two camps; those who may be described – by analogy with another problem of social hygiene – as partisans of abolition and those who are in favour of regulation. The concessions which those two schools are obliged to make in the course of practical work prove that they have some common ground and justify the hope that they would come to an agreement upon a programme of work.[495]

Before arriving at Burnet's compromise between the two schools of thought that in 1931 would herald a new strategy for prevention, I will explain the line of reasoning that made up BELRA's challenge to the segregationist strategy for prevention.

The confidence in the efficacy of early treatments was the first of three distinguishing features of BELRA's approach to leprosy prevention. The second was building on the argument that push-strategies, while ideal in practice, led to the concealment of cases.[496] The vital novelty, however, was the emphasis on the circumstances under which leprosy appeared. The problem was not the individuals suffering from the disease, but the conditions under which leprosy appeared. As Ernest Muir put it in the leaflet *Leprosy: Diagnosis, Treatment and Prevention* (1924):

> It is necessary by propaganda, by teaching the medical profession and through them the rest of the people and by co-operation with educational authorities to familiarize every one with the signs and dangers of the disease till every one learns to abhor not the leper but leprosy and the conditions under which it is likely to spread.[497]

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[495] Burnet, Et. "Report on the Programme of work of the Leprosy Commission." C.H.887 (a); LNHO: 8A/16680/6714. Dated Geneva, August 20, 1930: 1-2.
[496] "The ideal method of prevention would be the hindering of all contact. This was attempted on an extensive and expensive scale in the Philippine Islands and proved a failure as a means of stamping out the disease." Muir, E. *Leprosy. Diagnosis, Treatment and Prevention*. Illustrated. 1924: 28.
[497] Muir 1924: 28-29.

Like with all arguments for prevention, the starting point for the argument was the etiology of the disease. Muir did not contest that leprosy was contagious, but stressed that much was still unknown about the exact means of transmission. For some the uncertainty led to carelessness, for others an irrational and overblown fear of contagion. Both were equally detrimental to a leprosy campaign as they got in the way of early diagnosis and treatment, either due to ignoring the early symptoms of the disease or people hiding their disease to avoid detention.

According to Muir, six factors determined the transmission of the disease: 1) The infectivity of the disease transmitter ("The larger the number of lepra bacilli in the body the more there are to propagate the disease; and the nearer they are to the surface of the body the more likely are they to be carried to other people."); 2) The closeness of contact ("the disease is most commonly communicated where people live under insanitary conditions, crowding together in small dwellings, using the same eating and cooking vessels, and even promiscuously using the same clothes"); 3) The length of contact ("The longer the period of contact the greater the danger of infection); 4) Health of the person exposed to infection. ("This is very important as on it to a large extent depends the resisting power of those who are exposed to infection"); 5) Immunity ("The effect of lack of immunity has been repeatedly seen where leprosy has suddenly been introduced into an island or other isolated area where it had not existed for many generations, with the result that the disease has spread rapidly and in a virulent form"); and 6) general sanitary conditions ("The better these are the less the danger of infection").[498] Undoubtedly shaped by having the main seat in India where the sanitary approach had de facto dominated for more than half a century, the hallmark of BELRA's leprosy approach was that prevention no longer was just a question of leper versus healthy; the conditions of society as a whole must be part of the equation.

Initially, BELRA was not directly opposed to segregation. Around the time of Muir's leaflet, they sent a questionnaire through the British Foreign, Colonial and

[498] Muir 1924: 26-28.

India offices asking for information regarding leprosy. The returns found that segregation had "had a very good effect in reducing the lepers where it has been carried out for some time, while where neglected the known lepers had greatly increased."[499] On the other hand: Not every country was ready for compulsory segregation. Especially in central Africa, "any attempt to enforce such a law would result in hopeless failure due to hiding of cases, quite apart from the greater urgency of devoting most of the available medical grants to combating more important diseases, such as sleeping sickness, syphilis and yaws."[500] There, voluntary isolation was the only possible solution, "as any drastic measures leading to extensive hiding of early cases are likely to do more harm than good."[501] Throughout the 1920s, however, the head of the organization, Leonard Rogers, would be increasingly confident in the efficacy of early treatment with chaulmoogra and in 1930 he argued that segregation should be abandoned altogether.[502]

According to BELRA, prevention should no longer take individual lepers as carriers of a contagious disease as its starting point. Furthermore, improving the sanitary surroundings was important, but alone not sufficient. Instead emphasis should be on identifying early and potential cases in communities where the disease was already present. BELRA's mechanism to achieve this was the dispensary; outpatient clinics. The dispensary had three main goals. First, they should spread information about the disease, especially the message that a cure was possible if treatment started early enough. Second, they should offer voluntary treatment (chaulmoogra). Third, the patients under treatment should be convinced to bring family and friends and have them examined for the presence of the bacillus. According to BELRA the disease was most infectious before clinical signs appeared, but this was also the stage in which the disease was easiest to cure. "Effective

[499] Rogers, Leonard. "Memorandum on the prevalence of and prophylaxis against leprosy in the British Empire, based on replies to the questionnaire of the British Empire Leprosy Relief Association; with suggestions for dealing with the problem." (LNHO: 12B/42641/29272). 1925: 3.
[500] Rogers 1925: 5.
[501] Rogers 1925: 21.

treatment in the earliest stage of the disease is thus the key to present day prophylaxis."[503]

In a Foucault-inspired study of medical knowledge in Britain, David Armstrong has argued that the dispensary represented a new way of observing illness. While the clinic saw disease as situated within individual bodies, the dispensary identified disease in the spaces *between* people, that is, disease in society.[504] Through the dispensary the medical gaze shifted its attention from observing people with disease to looking for the potentially ill, the healthy and the normal. Armstrong's observations fit well with BELRA's vision for the dispensaries in the 1920s: Through early intervention was it possible to stop leprosy through identifying *potential* victims, which could only be achieved in close collaboration with the local population.

Faced with the arguments from BELRA on the one hand, and segregationists both in the Leprosy Commission and in leprosaria around the world on the other hand, Burnet presented a compromise: Instead of seeing the two approaches as competing, they must be understood as complimentary. The problem with the dispensaries was that they did not provide necessary means to remove infection from the community; the problem with the institutions was that they did not reach the communities in the first place. Therefore, while neither leprosaria nor dispensaries were sufficient alone, both institutions were necessary.

> Formerly, the whole prophylaxis of leprosy consisted in segregation in leprosaria; to-day, however, some are opposed to this segregation. It is best to avoid both extremes and recognize that while leprosaria are still necessary they are not sufficient in themselves. (…)

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[502] See i.e. Rogers letter to the Bangkok conference on leprosy in December 1930, referenced in: "Extract from the Report by the Medical Director to the Health Committee. Seventeenth Session, May 4-8, 1931." (LNHO: 8A/30632/29090). 1931: 17.

[503] Rogers 1925: 20.

[504] "The clinical gaze within the hospital had outlined and sustained the passive and discrete body by localizing illness within it. In a similar fashin the Dispensary gaze established the reality of the social by identifying diseases of social spaces, of contacts and of relationship. Thus, as the social basis of the disease justified the Dispensary as the appropriate mechanism to combat it, so too the Dispensary established, by its deployment in the community, the social origins and character of the new health dangers." (Armstrong, David. *Political anatomy of the body. Medical knowledge in Britain in the twentieth century*. 1983: 7-13, quote on p. 11.) Armstrong traces the dispensaries back to a tuberculosis dispensary in Edinburgh, which opened in 1887,

> At the present time, the dispensary (…) is an integral part of the ideal anti-leprosy organization and is no less necessary than the colony; but of it also we can say, as of the colony, that, though necessary, it is not sufficient in itself.[505]

The model Burnet envisioned was institutions for isolation of contagious cases, and dispensaries to act as intermediates between leprosy institutions and the public. The dispensaries were not just to be charged with distributing treatments, but with constant monitoring of the the society to identify early and potential cases.

> "The real object of permanent supervision of social conditions is to keep an eye on the large number of relations and neighbours of lepers who are contacts and suspected cases, and are as important from the point of view of theoretical study as from that of prophylaxis. Cases found among such persons are at the same time the most difficult to diagnose and the easiest to cure."[506]

Burnet's compromise was discussed at the Leprosy Commission's meeting in Bangkok in December 1930 and at the extended conference in Manila a month later. In addition to deciding that the term "segregation" should be replaced by "isolation",[507] the conclusion was that the preventive measures must depend on the particularities of each country with respect to the number of cases, popular beliefs about the disease, and the strength of the health systems. Third, the committee agreed, isolation alone was "not sufficient as a prophylactic measure, but must be supplemented by epidemiological control, treatment and education for patients and public alike."[508] In other words, the goal of eradicating leprosy remained, but the repertoire of prevention had to be expanded.

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followed by a Central Fund for the Promotion of the Dispensaries System which started operating tuberculosis dispensaries in London in 1911.

[505] Burnet, Et. *Provisional Report presented to the Leprosy Commission at its meeting at Tokyo in April 1930.* Part III: General Conditions of Prophylaxis. (League of Nations Health Organisation. C. H./Leprosy/7b) 1930: 1- 4.

[506] Burnet 1930: 5.

[507] The winning argument was that the term "segregation" had historical connotations a final destination, and that this was part of the reason why lepers hid their disease as long as possible.

[508] Extract from the Report by the Medical Director to the Health Committee, 17. Session, May 4-8 1931: 11. (LNHO: 8A/R5930/30632).

The debate resulted in the widely distributed first general report of the League of Nations' Leprosy Commission, *The Principles of the Prophylaxis of Leprosy* (1931), which heralded a the new global consensus on prevention. The report embraced treatment, propaganda, separating children from leprous parents, quarantine, and sanitary measures alike. While the report included new standards for a globally unified terminology of diagnosis, recordkeeping and epidemiological examinations, ironically the premise for the conclusion on prevention was that preventive measures must differ from place to place:

1. Prophylaxis of leprosy is not a problem that admits of solution by the syndication of any one measure, since the means of dealing with it obviously vary with geographical, economic, administrative, financial and social conditions and with the incidence of the disease.
2. There is no reliable system of prophylaxis without treatment, and it is generally accepted that the earlier the treatment is instituted the better will be the results.
3. Leprosy resembles tuberculosis in being, in certain stages, a contagious but curable disease: curable at least in the sense that bacteriological examination becomes negative and other active signs disappear and remain absent permanently or for an undetermined period.
4. The prophylaxis of leprosy may be achieved by a system of medical, educative and legislative measures. It should provide for the isolation and treatment of infectious lepers, and particularly for the treatment of early cases in clinics and dispensaries: also for the periodical examination of suspects. Special measures should be adopted for dealing with the children of lepers and for patients who have recovered either after treatment or spontaneously.
5. It is desirable that each country where leprosy exists to an important degree should have at least one centre for the study of the disease, with research laboratories and special courses for the medical profession and their assistants. Where this is not practicable, men should be sent to some foreign centre for training.
6. Arrangements should be made to include instruction in leprosy in the curriculum of all medical schools and colleges.
7. It is necessary to educate the public in regard to leprosy by modern methods of popular teaching and propaganda.
8. Isolation of infectious lepers is a necessary measure in a comprehensive campaign against leprosy, but it cannot be regarded *per se* as the sole means of prophylaxis. Its drawbacks can

be mitigated by other measures applied concurrently. Isolation should be applied only to cases that are considered infectious.

9. Any form of treatment in order to give satisfactory results requires to be combined with suitable dietic and general hygienic conditions.
10. For special treatment, oils of the chaulmoogra group and their esters and soaps are recommended.
11. The system of prophylaxis must be animated by the spirit of preventive medicine and social hygiene.[509]

Conclusion

There never were any uniform preventive measures against leprosy, but some trends dominated. Before the 1890s, debates on prevention were integral to debates between competing disease models. The attendees at the first international leprosy conference in Berlin in 1897 concluded that the disease was caused by a bacillus, that the disease was therefore contagious, that every leper was a danger to his surroundings and that the policies in Norway should be mimicked. After this, segregation through push-strategies dominated. The goal was to force the lepers into isolation through legislation and educating the population that lepers were dangerous. Less dominant, but locally equally important, were pull-strategies aimed at making the lepers seek voluntary confinement, as well as the practice of restricting the lepers from certain vocations. The rise of contagionism also led to leprosy being included in quarantine regulations constructed to deal with other diseases, but this proved difficult in practice. Quarantine measures along the lines of what Reverend James C. Richmond advocated in 1852 were never uniformly adapted.

The recommendations from the leprosy conferences were never compulsory, but the reports in medical journals and at conferences indicate that having an 'international consensus' to refer to, as well as successes of leprosy campaigns in

[509] The League of Nations Health Organisation. *The Principles of the Prophylaxis of Leprosy. First General Report of the Leprosy Commission.* Geneva, April 1931: 9-10.

other countries, were strong arguments in various settings. On the other hand, the interpretation of the recommendations differed greatly. Even in medical textbooks, Norway was withheld as advocating a 'limited' approach by one author; as an argument for compulsory segregation by another.

In the early 1930s, the League of Nations' Leprosy Commission agreed on a new and universally applicable set of recommendations for prevention. This was a compromise between segregation and voluntarism, and introduced dispensaries as intermediates between institutions and the public. Their role was to provide both treatments and permanent epidemiological control in order to detect new cases before they reached an infective stage. As the discussions on standardization in terminology, classifications and recordkeeping started to bear fruits, the League's prime recommendation when it came to prevention was to expand the repertoire of preventive measures and tailor an approach suited to the local conditions.

The challenge to compulsory segregation originated in colonial India, where segregation was never carried out and sanitary concerns remained part of the etiology of the disease. Researchers organized in BELRA questioned both the premise that the disease was highly contagious, that there were no cure, and that segregation was the best means to remove contagion from society.

In the following chapter I will investigate hvow the theory of contagion was introduced to the medical debates in between the 1860s and the 1890s, focusing on Norway and India. Before this period, leprosy was understood to be due to a combination of hereditary and sanitary conditions in both areas. I will open with a visit to Bergen by the British physician Henry Vandyke Carter. The visit made him convinced that the disease was contagious, and his report was the first to mention the leprosy bacillus in print. Still, convincing others proved difficult: India and Norway would represent two extremes in the debates on etiology, and how reports on leprosy from other parts of the world were appropriated differed greatly.

The next chapter also marks a shift in the perspective. Instead of focusing on the content of the debates and how this changed over time, in the following three chapters my emphasis will be on science as communication; communication that

needed to be organized. Each chapter will also be limited to shorter periods of time, which will allow me to go more into detail.

5. Appropriating contagion

"Is Leprosy the same Disease in Western India and in Norway?"[510] This was the first question to be answered by surgeon-major Henry Vandyke Carter, who visited Norway on behalf of the Secretary of State of India in the fall of 1873 "for the purpose of studying the character, prevalence, and state-treatment of 'Leprosy'."[511] Explicitly arguing that leprosy (Spedalskhed) in Norway was the same disease as True or Black Leprosy (Răktăpĭti) in India was crucial to making Carter's experiences relevant back home.[512]

What Carter found in Norway was a system of leprosy institutions, including three state asylums and a research hospital. He was also told about the network of district physicians who since 1856 had been charged with annual registration of every individual leper living in their area. Carter's use of the term "the Enlightened Kingdom" summarizes his impressions.[513] During his visit, Carter also met with Gerhard Armauer Hansen, at the time an assistant physician working at the Lungegaarden research hospital. Hansen was increasingly convinced that the disease was contagious. In addition to being introduced to Hansen's line of reasoning, Carter was the first foreign visitor to be shown the microscopy that could "point to the parasitic origin of the disease."[514]

In the early 1870s, the dominant understanding of the etiology of leprosy in both India and Norway was that the disease was caused by a combination of hereditary and external factors, such as climate and hygiene. The meeting with Hansen made Carter change from seeing leprosy as an inherited 'taint' to seeing it as a contagious disease. Hansen would remain Carter's main contact in Norway for almost

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[510] Carter, Henry Vandyke. *Report on Leprosy and Leper-Asylums in Norway; with references to India.* 1874: 7
[511] Letter from Carter to the Secretary of State for India, dated London November 24, 1873. In: Carter 1874: A2.
[512] Carter 1874: 6-7.
[513] Carter's report is used as the main contemporary source on Norwegian leprosy control in Zachary Gussow's influential book *Leprosy, Racism and Public Health* (1989), and Gussow borrowed the term "The Enlightened Kingdom" for the title of the chapter on Norway. Later studies in Norway have shown the picture to be a lot more nuanced, see: Andresen 2004, Vollset 2005, Hammerborg 2009.

two decades, and in several publications Carter argued that India should take its cue from the Norwegians. But how was the notion of leprosy being contagious appropriated in these two vastly different contexts? How did the lessons from Bergen influence discussions in Bombay and vice versa? And what was the role of the leprosy bacillus in these discussions?

This chapter is about appropriation. Despite leprosy being put on the agenda in numerous locations around the world, and researchers mainly of Western origin throughout the world communicated and exchanged knowledge, I will show how the same events and reports could be interpreted very differently. Knowledge from other places was read in light of local context and previous experience. This meant that the importance and potential impact of knowledge produced elsewhere could differ dramatically. Competing disease models had different standards for what was considered relevant observations, and both conditions on the ground and political circumstance influenced how knowledge produced elsewhere was adopted. Secondly, I will show that the act of producing knowledge was also active and selective. Not every local event was reported elsewhere.

The chapter will start with Carter's visit to Norway. What did Carter report home, and how did his arguments for contagion play out in the context of the British colony? Then, I will investigate the discussions on etiology that took place in Norway and in the British Empire, and show that while there were clear parallels between them, the political configurations, context and outcomes of the debates differed greatly. Next, I will investigate the event that came to have wide and global consequences: The discovery of the leprosy bacillus. My emphasis will not be the genesis of the bacillus, but rather how it came into play in the medical and political debates. To some, the bacillus was seen as proof that leprosy was the same disease all over the world, but this was but one of a wide range of responses. The importance of the bacillus, I will argue, was that it became a common frame of reference for those arguing for contagion, proof that the disease was the same regardless of location, an

[514] Carter 1874: 27.

impetus for collaboration and circulation of knowledge.

Before leaving India

Henry Vandyke Carter qualified in medicine at St. George's Hospital in London in 1852, and received his doctoral degree from the University of London in 1856. He worked as demonstrator in anatomy, and collaborated with Henry Grey on producing drawings for the influential textbook *Anatomy: Medical and Surgical* (1858).[515] The year Grey's *Anatomy* was first published, in the immediate aftermath of the Indian uprising, Carter moved to India and joined the Indian Medical Service. He would be working in India for the next three decades, the first five years as acting professor of anatomy and physiology at Grant Medical College in Bombay, and later with positions as a surgeon at several hospitals.[516]

In the British Empire leprosy was put on the political agenda in the early 1860s after alarming reports on the spread of the disease from the Governor of the British Windward Islands based in Barbados. The Colonial Secretary responded by asking the Royal College of Physicians to find out whether or not leprosy constituted a threat to the Empire. Based on returns from a questionnaire that the Colonial Office circulated around the Empire, the *Report on Leprosy* (1867) concluded that leprosy was 'essentially a constitutional disorder' best tackled by improving the health, diet and living conditions of the native populations. Similar improvements, the report argued, were what had caused the decline of leprosy in medieval Europe. The report maintained that the disease was mainly hereditary, always non-contagious, and most

[515] The lasting success of the textbook can be illustrated by its numerous editions. In 1883, *Grey's Anatomy* was printed in its tenth edition; by 1901 five more editions had been made. The 40th edition was published as a celebration of the book's 150 year anniversary in 2008. For a study of the context in which the book was created, the people involved and the reception, see: Richardson, Ruth. *The Making of Mr. Gray's Anatomy. Bodies, Books, Fortune, Fame*. 2008.

[516] Roberts, Shirley. "Henry Gray and Henry Vandyke Carter: creators of a famous textbook". *Journal of Medical Biography*. Vol. 8. 2000: 206-212; "Henry Vandyke Carter (1831-1897)". *List of the Carter papers in the Wellcome Institute Institute for the History of Medicine*. Online: http://library.wellcome.ac.uk/assets/wtl040084.pdf . His obituary was printed in *The British Medical Journal,* May 15, 1897: 1256-1257.

importantly – it posed no risk to European colonialism.[517] This led the Colonial Secretary to issue a decree that "any laws affecting the personal liberty of lepers ought to be repealed, [and] any action of the Executive Authority not enjoined by the law, ought to cease."[518]

Behind the decree was an acknowledgement that there were local differences in how leprosy was approached in the Empire. Obviously, the disease must in some places already have been considered a legal matter, but top colonial management desired common standards based on the best scientific knowledge available. Despite reports from the colonies to the colonial administration in London pointing in the direction of contagion being the cause of the disease, the College of Physicians would consistently defend the 1867-report. An often repeated phrase was that other reports lacked "sufficient precision to be regarded as furnishing evidence in proof, or in disproof, of the contagiousness of leprosy".[519]

The worrying reports also reached Bombay Presidency where Carter worked, and in 1867 the presidency Sanitary Commission launched its own leprosy census. But when the Royal College of Physician's report arrived and called off the alarm, writing a report was no longer considered a priority. The returns gathered dust for about three years until Carter, not interested in private practice and with time on his hands, offered to analyze them. The returns contained data on almost 8.000 individuals suffering from the disease, 5.309 of them named, registered in ten (of sixteen) districts in Bombay Presidency. This was "a number equal to 1 affected in 600 of the population".[520] Carter's 170-page publication was structured on the 1867 report from the Royal College of Physicians, and first published in *Transactions of*

[517] Edmond 2006: 51-60.
[518] *Report on Leprosy of the Royal College of Physicians Prepared for Her Majesty's Secretary of State for the Colonies*, London, Her Majesty's Stationary Office, 1867: vi. Quoted in: Pandya 2001: 41-42.
[519] "Recent Official Correspondence relating to Leprosy. Communicated to the Editor". *British and Foreign Medico-Chirurgical Review*. 1874: 204-212, quote on p. 205. The correspondence, it seems, was initiated by the Secretary of State for the Colonies, the Earl of Kimberley, asking the College of Physicians for their comments on reports from the colonies.
[520] Carter, H. V. "The Pathology of Leprosy; with a Note on the Segregation of Lepers in India." *Medico-Chirurgical Transactions*. Vol. 56. 1873: 277. Before this, Carter had been among the physicians who had answered the questioner by the Royal College of Physicians.

the Medical and Physical Society of Bombay in 1871.[521] In it Carter argued that leprosy was caused by a hereditary 'taint', but rejected the competing sanitary notion that leprosy in India was caused by filth and lack of hygiene.[522]

Two years later, Carter published a paper on the pathology leprosy, repeating that "heredity is the common cause of the complaint".[523] More importantly, however, he now argued that leprosy could (and should) be controlled through state intervention: There was no evidence to support the claim that the disease was in a natural decline, and therefore practical measures were needed. These should consist of strict segregation of indigenous lepers, moving them "to a central, cool, and healthy locality" and – should a treatment be found – the erection of special leprosy hospitals. The argument was backed by referring to the decline of the disease in medieval Europe, an ongoing debate where Carter sided with those who believed it was segregation, and not changes in diet and living conditions, that had led to the decline of the disease: "Nowhere in the East does there appear ever to have existed such a machinery; and there the disease is probably as rife as ever, and certainly rendered more inveterate by this tolerance for centuries."[524] Segregation would make the healthy shun the lepers, prevent marriage, and thus keep the disease in check; "relieving burdened poor, and removing from sight a wretched class of mendicants".[525] This was in direct opposition to the official 'hands off'-doctrine spelled out in the 1867 report by the Royal College of Physicians.

These publications were the immediate precursors when Carter in July 1873 received permission from the Secretary of State for India in Council "to proceed, at

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[521] Edmond 2006: 58; Pandya 2001: Chapter 4; Pandya, Shubhada. "Nineteenth Century Indian Leper Censuses and the Doctors". *International Journal of Leprosy*. Vol. 72, No. 3. 2004: 306-316. Leprosy was also part of the nation-wide censuses of 1871/72, 1881 and 1891. See also: Kakar 1996: 215-230; Robertson, Jo. "Leprosy and the elusive M. leprae: colonial and imperial medical exchanges in the nineteenth century." *História, Ciências, Saúde-Manguinhos*. vol.10, suppl.1. 2003: 13-40.
[522] Pandya 2004: 311.
[523] Carter, H. V. "The Pathology of Leprosy; with a Note on the Segregation of Lepers in India." *Medico-Chirurgical Transactions*. Vol. 56. 1873: 276.
[524] Carter 1873: 280.
[525] Op. cit.

the expense of Government, to Norway for the purpose of obtaining all available information on the subject of the disease."[526]

Reporting home; falling from grace

Carter spent two months in Norway in the fall of 1873. There he found five leper asylums with almost 900 beds, two of them in Bergen alongside Lungegaardshospitalet, a specialized research hospital dedicated to clinical studies and finding a cure for the disease. As Carter enthusiastically summarized; "The hospital is thoroughly provided with all the *matériel* necessary for every variety of curative procedure, and for all kinds of scientific research. The supply of drugs is unrestricted, and the library and laboratory are freely maintained by Government in an efficient manner".[527] In an appendix Carter included translations of the decrees to establish the state institutions, the official regulations, including instructions for the various positions, information on diet, and images and floor plans of the Leper-Asylum in Bergen, suggesting that this should be copied in India.

In addition to the institutions, the Norwegian leprosy campaign consisted of a system of compulsory notification of every known leper in the country, dating back to 1856.[528] Fascinated, Carter had the official circulars with instructions on establishing the system translated, including the tables used for the registration. The lists asked for full names, residence, birthplace, sex, profession ("Condition or Station in Life"), age, marital position (including whether the spouse was a leper or non-leprous), family relations with other lepers, number of children and their ages (leprous/not leprous), the duration and form of the disease (tubercular/anesthetic/mixed), and a section for "Remarks, chiefly with reference to possible Causative Influences, such as Mode of

[526] Letter from M. E. Grant Duff to H. Vandyke Carter, dated India Office July 11, 1873. In: Carter 1874: A2.
[527] Carter 1874: 13. Also quoted in: Gussow 1989: 72. The travel to Norway would also result in the publication *Reports on Leprosy, Second Series, Comprising Notices of the Disease as it Now Exists in North Italy, the Greek Archipelago, Palestine and Parts of the Bombay Presidency of India*. (1876).
[528] For details on the different projects that together resulted in what historian Morten Hammerborg has aptly termed 'the Norwegian leprosy apparatus', see: Hammerborg 2009: Chapter 2.

Life, Locality or Residence &c; and as concerns the Married, the Duration of Wedlock". Again, the argument was that the same information should be gathered in India.

Based on these individual registrations, the annually updated statistics documented that in Norway the prevalence of the disease was in decline. With no cure available, this meant only one thing: The reduction of new cases was caused by the institutions. For Carter, the main lesson from Norway was confirming the necessity for prolonged state intervention: Building institutions and educating the population that this was where the 'lepers' belonged.

> It may be sufficient to state, amongst other valid reasons for the interposition of Government, that the malady under notice is an altogether peculiar one in severity and extent, and claims special consideration; that British rule in India would benefit itself and subjects by an intervention to be recommended on grounds of both policy and humanity; that delay is to be deprecated, and that the present time is well suited for enlisting the co-operation of the people, who are tolerant only from an ignorance which it is our duty to dispel by open, deliberate and rationally-founded action.[529]

The appendix also included two translated reports by district physicians on the habits and diet of Norwegian peasantry in leprous and non-leprous districts. This was proof, Carter argued, that cultural or sanitary conditions had no impact on the disease, and a confirmation of what he believed before going.[530] Regarding the etiology of the disease, however, Carter was no longer convinced that heredity alone was an adequate explanation: "the more promising inquiry would be that of its origin from a combination or succession of influences which separately could not produce the affection."[531] While pointing out that the Norwegian institutions originally were erected to segregate the sexes and avoid leprosy being passed on to the offspring, he

[529] Carter 1874: 29-30
[530] Historian Shubana Pandya has argued that also the nation-wide leper censuses in India in 1871/72, 1881 and 1891 were used to support whatever preexisting set of believes the investigators had regarding the disease: "The efforts to clarify the etiology and estimate the virulence of the disease in India by means of statistics were animated by the desire to justify and embellish preconceptions." (Pandya 2004: 315.)
[531] Carter 1874: 27.

also argued "that even a *suspicion* of leprosy being contagious is an item in favour of the segregation of lepers."[532]

Connecting the Norwegian experiences to India, the report included a translation from the Indian weekly newspaper *Loka Mitra* (published in Karanese in south central India in July 1870) on leprosy research in Norway, which indicates that Carter was not the first in India to look to Norway for advice on the disease. Finally, Carter included excerpts from proceedings and official documents in Western India regarding the disease – an implicit argument that state institutions would be a natural continuation of the policies already in place.[533]

While Carter swung both ways on the question of contagion or heredity in his report from Norway, historian Rod Edmond has argued that in Carter's monograph *On Leprosy and Elephantiasis*, which was published only a few months later, the transition was clear:

> The very layout of this work demonstrated the recent conversion to contagion that Carter had undergone. Thus, while the text maintained that 'heredity is the common cause of the complaint', a series of long footnotes add that his recent acquaintance with investigations in Bergen has led him to believe that the contagion case is 'tolerably definite'.[534]

After returning to India, Carter would continue his studies on leprosy, both through statistics and post mortems. Within a few years he would argue that the disease was decisively contagious.[535]

Carter's reports were but some of many studies carried out in order to decide if leprosy was a threat to the British Empire.[536] Among the physicians who held Carter's

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[532] Carter 1874: 26. Italics in the original.
[533] Carter 1874: 31-68.
[534] Edmond 2006: 68-69. See also: Pandya 2001: 64-66.
[535] In 1875 alone Carter conducted fifteen post-mortem dissections of lepers in Bombay. Carter, H. V. "Memorandum on leprous nerve disease". *Transactions of the Pathological Society of London*. 1877: 1-4. For the arguments for contagion, based mostly on aggregated data, see. Carter, H. V. *Modern Indian leprosy*. Bombay. 1876; Carter, H. V. *Reports on Leprosy, Second Series*. London. 1876. As almost all the actors in this thesis Carter also studied other diseases, but this is outside my scope.
[536] For an overview of concurrent leprosy studies in the British Empire, see: Robertson, Jo. "In Search of M. Leprae: Medicine, Public Debate, Politics and the Leprosy Commission to India." In: Dale, Leigh and Helen Gilbert (eds). *Economics of Representation, 1790-2000: Colonialism and Commerce*. 2007: 41-59, esp.: 46-47.

works in high regards, was William Munro, late medical officer to St. Kitts in the West Indies. In his experience, the disease was contagious, and human intercourse was the only means of propagation. By 'intercourse' he meant living together, not that leprosy was a venereal disease. He was also opposed to claims that the prevalence of the disease had increased due to vaccination, which had become a hot topic for debate in Britain after smallpox vaccination through arm-to-arm inoculation was made compulsory in 1871.[537] To explain why not even more people caught the disease, Munro added that the "probable primary cause is a want of salt combined with a deficient vegetable diet".[538] Apart from the emphasis on diet as a necessary component, Munro was fully in line with Carter's arguments, and typically described him as "one of the best living authorities on leprosy".[539] He also expressed "admiration" for Carter's change of position regarding etiology, a not very subtle critique of the Royal College of Physician's unyielding defense of their 1867-report.[540]

In Britain, seeing leprosy as contagious was controversial. The main objection came from the principal author of the 1867-report, Gavin Milroy. One generation older than Carter, Milroy had qualified as a physician in Edinburgh in 1828. According to his obituary, he "never practiced medicine on his own account, but, from his early manhood, made choice of its literary path."[541] Milroy was an active member of the Hunterian Society in Edinburgh (a medical society named in honor of the

[537] In a circular dated Downing Street, September 4, 1873, the Secretary of State for the Colonies, The Earl of Kimberley rejected that vaccination could propagate the disease, but added that "as the success of a system of vaccination must largely depend upon the views prevailing amongst the public on the subject, it will be always as well to use lymph which is beyond suspicion in this respect." ("Recent Official Correspondence relating to Leprosy". *British and Foreign Medico-Chirurgical Review*. 1874: 212.) Leprosy would be a focal point for the anti-vaccination movement in Britain for several decades. See i.e. Tebb, William. *The Recrudescence of Leprosy and its Causation. A Popular Treatise.* 1893, also printed with the title *Leprosy and Vaccination*. For studies on the British anti-vaccination movement, see: Porter, Dorothy. *Health, Civilization and the State. A history of public health from ancient to modern times.* 2005 [1999]: 128-130.

[538] Munro, W. *Leprosy*. Manchester. 1879: Prefactory Note. The work was a reprint from a series of papers printed in the *Edinburg Medical Journal* (September 1876 to November 1879).

[539] Munro 1879: 16.

[540] "I can pay Dr. Carter no higher compliment than to express my admiration for his readiness to give up any received theory which he has given much labour to prove the correctness of, when formerly unknown circumstances show that another theory explains the facts better." (Munro 1879: 77)

[541] "Gavin Milroy". *The British Medical Journal*. February 27. 1886: 425-426.

Scottish surgeon John Hunter), and then the Epidemiological Society of London where he was President from 1864 to 1866. In 1849 he was appointed Superintendent Medical Inspector of the General Board of Health in London, and a year later he was sent to study an outbreak of cholera on Jamaica, followed by assignments as a sanitary commissioner for the War Office in the Crimea in 1855. In 1871 Milroy was sent to the West Indies to investigate an alleged cure for leprosy developed by the French doctor Louis Daniel Beauperthy working at Cumana in Venezuela.[542]

Milroy had marked himself as a public opponent of the quarantine system already in the 1840s, and in 1858 he was appointed Honorary Secretary to a committee set up by the National Association for the Promotion of Social Science to report on the practices and results of quarantine worldwide. His modus operandi was the same then as when later making the report on leprosy: Write a series of queries, have them forwarded to the Foreign and Colonial Secretaries of State for circulation among the Consuls and Colonial authorities, and then analyze the returns. His conclusion was that all suggestions of leprosy being contagious were based on superstition and ignorance – a position he would retain until his death in 1886.

Throughout the 1870s, Milroy would be London's authoritative voice on leprosy in the Empire. In addition to corresponding with the Colonial Secretary, from 1874 to 1880 he published a series of nine papers in the London-based *Medical Times and Gazette* under the title "Is Leprosy Contagious?" insisting that calls from the colonies for compulsory detention were uncalled for. When the Colonial Office informed Milroy that Carter's report from Norway was to be circulated to 'the Colonies in which leprosy prevails', he requested that also his own latest paper arguing that the disease was hereditary and that institutions were unnecessary, was included as an addendum.[543] This would be the pattern also for later research from the colonies: The Colonial Office would circulate the reports, but always accompanied by a corrective from the College of Physicians (meaning Milroy).

Carter's specific recommendations after the journey to Norway were that India

[542] Beauperthy died shortly after Milroy arrived. More on this in Chapter 3.

should start constructing leper asylums for care and containment of possible disease carriers, a leprosy hospital for further research, and learn more about the Indian leper through collecting statistics and conducting special investigations in the 'worst districts'. Carter's report described the Norwegian system as being run by a "Leper Department". Accordingly, the leprosy work in India should be organized independently of the Sanitary Commissions, Agricultural Department, the Cholera Commission, and the military Indian Medical Service where he began his career, "for they concern a special subject, needing special knowledge, close attention, and continuous observation for its elucidation."[544]

Perhaps not surprisingly, this was not well received by India's growing sanitary department, which saw leprosy as clearly within their domain. In India Carter's immediate opponent was not Milroy, but James McNabb Cuningham. Like Milroy, Cuningham had his medical doctorate from Edinburgh. This he received in 1851. Cuningham then entered the Bengal Medical Service, and in 1868 he was made sanitary commissioner to the Government of India, a primary advisor to the government.[545] As historian Jane Buckingham put it:

> Much as the Royal College of Physicians in Britain had used the investigation of leprosy to help develop its professional profile in the 1860s, the sanitary department employed control over leprosy research in its effort to extend, as far as possible, its authority over public health in India.[546]

Cuningham saw the calls for institution building and segregation of leprosy sufferers as an impossible and futile undertaking. The vast number of lepers meant that building institutions would be a huge burden on the government purse. Based on the conclusions of the 1867-report, it was also completely unnecessary. Going through Carter's report from Norway, Cuningham pointed out that segregation in Norway was only very partial. While 764 persons with leprosy were institutionalized in Norway by

[543] Edmond 2006: 68.
[544] Carter 1874: 30.
[545] Buckingham 2002: 149.

the end of 1870, there were 1.286 registered lepers who lived outside the institutions. The partial institutionalization could not explain the decline in prevalence, and it could not be considered evidence to suggest the disease to be contagious. As I will show later in this chapter, similar lines of reasoning were repeated in the *Report of the Leprosy Commission in India* (1893).[547] The only recommendation Cuningham endorsed was the call for more knowledge on leprosy in India – a task he successfully argued belonged to his own sanitary department.

In March 1875, based on a resolution drafted by Cuningham, the Indian Government decided that the next enquiry into leprosy should be a sanitary enquiry, conducted by the sanitary department. Cuningham also persuaded the Governor-General-in-Council that "it would not be advisable for the Government to publish and circulate Dr. Carter's paper, and thereby give it an authoritative character, as though the question of the specific origin of leprosy has been decided, which is far from being the case."[548] The mandate to the sanitary investigators was written by no other than Gavin Milroy himself.[549] This led *The Lancet*, which already in 1867 had argued that Milroy's report had underestimated the importance of contagion,[550] to argue that Carter had been 'elbowed out' of a field on which he had 'thrown light by honest and admirable work'. For Carter, departing from the official dogma would ruin his prospects of promotion.[551]

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[546] Buckingham 2002: 148.
[547] *Leprosy in India. Report of the Leprosy Commission in India, 1890-91*. Calcutta. 1893. See also: Pandya 2001: 67-69.
[548] Maharashtra State Archives, General Department, Vol. 50, 1876: 20, quoted in Pandya 2001: 68.
[549] Pandya 2001: 70.
[550] The review was printed in three parts, where the two latter were dedicated to "the important question of the contagiousness of Leprosy". They especially highlighted N. C. Macnamara from the Indian Medical Service whose independent analysis of the 107 reports from India concluded that the disease was probably contagious. *The Lancet* review argued that "the facts which have been presented to the Committee in the various 'replies' received in answer to the 'interrogatories' are not sufficient upon which to form a just conclusion.". ("The Leprosy Report of The College of Physicians" Pt. I-III, *The Lancet*. 1867: 63-65, 189-190, 253-254, quotes on p. 189.)
[551] "Probably, as Dr. Carter's views in reference to the general treatment of lepers turn out to be not in accord with those of the Government, it may be convenient to deny Dr. Carter any facilities for prosecuting his inquiries any further." (Editorial. "Leprosy in India". *The Lancet*. 1875: 458-459. Quoted in: Pandya 2001: 69; Buckingham 2002: 150). As Buckingham points out, this was not an isolated event. In 1884, Carter was unprecedentedly passed over for the position of provincial sanitary commissioner in Bombay. (Buckingham 2002: 151.)

The sanitary report found 'little or no evidence' to support 'the recent revival of contagion theory'. Instead, its conclusions reaffirmed and strengthened the conclusions from Milroy's 1867-report.[552] There was no reason for further state involvement, other than to continue the sanitary approach that would improve colonial health in general. That many in the general public, including local physicians, withheld that the disease was contagious was seen as a proof that they lacked the necessary unprejudiced view that proper science could offer. Finally, the lepers tended to develop sterility, and the mortality rate of their children was 'abnormally high': "It is therefore evident that unless there be influences other than heredity at work in the locality, tending towards the production of the leprous condition, no serious increment need be apprehended."[553]

On the other hand, Henry Vandyke Carter did have supporters in the Bombay Presidency. In 1875 he was asked to study leprosy in the villages of the Kathiawar peninsula (now Gujarat), a district outside British colonial control.[554] He was made president of the Medical and Physical Society of Bombay and Dean of the Medical Faculty of the University of Bombay.[555] In June 1879, based on Carter's recommendations, the Government of Bombay decided that the Sassoon Infirm Asylum in Poona should accept and maintain leprosy sufferers.[556] Shortly thereafter Carter again visited Europe, but this time due to illness and not primarily as a study tour. After returning, Carter would continue to argue that India should learn from Norway.

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[552] Lewis, T. R. and D. D. Cunningham. *Leprosy in India: A Report*. 1877: 56-58, as quoted in Edmond 2006: 80. See also: Pandya 2001: 71; Buckingham 2002: 147-152. Parts of the sanitary report was also printed in the report *Leprosy in Foreign Countries,* published by the Department of Foreign Affairs on Hawaii in 1886. (Lewis, T. R. and D. D. Cunningham. "Extracts from a report". In: *Leprosy in Foreign Countries.* 1886: 78-81.)
[553] Lewis and Cunningham 1886: 80.
[554] Carter, Henry Vandyke. *Report of a Tour in Káthiáwár*. 1876. Bombay. 1877.
[555] Pandya 2001: 57.
[556] General Department, Bombay. Resolution No. 4900. "Establishment of Leper Asylums". Dated Bombay Castle, December 7, 1882: 1. Addendum to Carter, Henry Vandyke. *Memorandum on the Prevention of Leprosy by Segregation of the affected.* 1884.

Meanwhile, in Norway

The leprosy system Carter met in Norway was the outcome of both a disease model which saw leprosy as hereditary and a model of the disease as caused by filth, lack of hygiene and a low stage of civilization.[557] Before returning to India, I will in the following show how the Norwegian 'leprosy apparatus', as coined by historian Morten Hammerborg, was established. Then, I will investigate how Milroy's report and Carter's visit were appropriated in the Norwegian debates.[558] Finally, what happened when, around the time of Carter's visit, contagion was introduced as a justification for continued state involvement in leprosy?

As I showed in Chapter 2, Boeck and Danielssen had in the 1840s described leprosy as having two distinct clinical forms, the anesthetic and the tubercular.[559] In a literary review tracing the disease back to the ancient Greeks, Boeck distinguished between inner and outer causes which worked in combination. Heredity, an inner cause, could explain almost all cases, while in some the disease developed spontaneously. Boeck saw no shred of evidence for the theory of contagion put forward in the Mosaic laws, a theory both he and Danielssen warned had led to inhumane prosecution of the sufferers in medieval Europe.[560] Other possible inner causes were age (leprosy as part of puberty) and sex (making eunuchs immune to the disease), but neither of these were seen as important. Of the external causes, climate and telluric (soil) influences and foodstuffs (especially fish, but also the combination of malnutrition, poor living conditions and filth) could all have some impact, while mental affections, acts of violence, and divine intervention were presented as unlikely explanations.[561]

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[557] Höegh, Ove Guldberg. *Tabeller over de spedalske i Norge i aaret 1860 samt aarsberetning for samme aar.* 1860: 16, as quoted in Vollset 2006: 49.
[558] Hammerborg, Morten. *Spedalskhet, galeanstalter og laboratoriemedisin – endringsprosesser i medisinen på 1800-tallet i Bergen.* PhD-thesis. University of Bergen. 2009.
[559] Danielssen and Boeck 1847.
[560] The non-contagious nature of the disease was further confirmed through experiments with inoculations. Notably, none of the inoculations were mentioned in Danielssen and Boeck's monograph. For an overview of these early experiments, see: Hansen, G. Armauer. "Uebertragung der Lepra von Mensch zu Mensch". *Mittheilungen 1897*. Bd. 2. 1897: 1-5.
[561] Danielssen and Boeck 1847: 66-80, 90-113.

According to Danielssen, who was responsible for the clinical work, leprosy was caused by an imbalance in the blood, a dyscrasia. This meant that the disease was not particular to the individual leprous lesion: Leprosy was not a skin disease but affected the whole body. The argument was backed by post mortem examinations which showed that also internal organs developed signs of the disease. Furthermore, chemical analysis of the blood, a technique which was much discussed in European medical journals in the 1840s, showed that 'leper blood' differed from healthy blood.[562]

Through examining the pedigrees of those affected, Danielssen concluded that the imbalance was mainly hereditary: "These tables show that of 213 individuals attacked by leprosy, it is inherited in 189, and only in 24 has it developed spontaneously".[563] The leprous dyscrasia could lie dormant in one generation, and return with a vengeance in the next. Of the 213 cases Danielssen investigated, 155 had skipped one generation or more. The disease could also jump 'sideways' through marriage. 120 of the 189 cases of inheritance were in the sidelines.[564]

As historian Michael Worboys has pointed out, to have several explanations for the same disease was not usual in medicine in the mid-19th century.[565] For Danielssen and Boeck, to recognize the disease and to work to find a treatment was more important than reducing it to a single cause. The argument that leprosy was caused by a 'hereditary taint', as in Carter's 1871 report, can be traced via the Royal College of Physician's 1867-report, to a translation of Danielssen and Boeck's monograph made available to the English-speaking medical community through a

[562] The first review of Danielssen and Boecks research based on publications in *Norsk Magazin for Lægevidenskaben* (1842), *Ugeskrift for Medicin for Pharmacie* (1843) and papers presented at the meeting of naturalists in Grätz in Preussia (1844) and at the fourth Scandinavian meeting for naturalists in Christiania (1844), explicitly asked for chemical or microscopical investigations. See: Editorial. "Boeck and Danielsen on the Spedalskhed or Norwegian leprosy". *British and Foreign Medical Review*. 1844: 346-353.
[563] "Af disse Tabeller sees, at af 213 Individer angrebne af Spedalskhed er denne nedarvet hos 189, og at den kun hos 24 har udviklet sig spontant." (Danielssen and Boeck 1847: 263.)
[564] Heredity in the sidelines was contested from the outset. In an otherwise positive first review of the monograph, physician Jens Johan Hjort argued that only direct lineage qualified as heredity, and that the argument that leprosy was a 'family disease' had probably been overstated. See: Hjort, Dr. "Om Spedalskhed ved D. C. Danielssen". *Norsk Magazin for Lægevidenskaben*. 1848: 169.
[565] Worboys 2000.

series of eight articles in *The Lancet* in 1856.[566] Carter's report from Norway contained long excerpts from the 1847-monograph, showing that he too was aware of this legacy.

Unlike in India, there were already institutions for leprosy in Norway in the 1840s. Danielssen's clinical work was based on seven years of observations and post mortems in St. Jørgens Hospital in Bergen, a patient-run independent institution which had operated continuously for more than 400 years.[567] Further north along the coast, Reknes Hospital outside Molde was established in 1716, and Reitgjerdet Hospital in Trondheim had since 1612 had a division for lepers. Based mainly on the argument that the disease was an increasing threat to the nation, that further research was necessary and that science could find a cure, the Norwegian Parliament funded the research hospital 'Lungegaardshospitalet' which opened in Bergen in 1849 with Danielssen as head physician. The research hospital burned to the ground on Christmas Eve 1853, killing six patients and a night nurse, but it was reopened in 1859. Danielssen would keep the position as head of the research hospital until his death in 1894.[568]

By the 1870s, the research hospital was well known outside the national borders. This is evident both from Danielssen's correspondence, publications, visits to Bergen, and numerous honors from medical and scientific societies throughout Europe.[569] When Danielssen in June 1871 was contacted by Dr. Bakewell who wanted to come to Bergen to demonstrate Beauperthy's alleged cure for the disease

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[566] Buckingham 2002: 120; Edmond 2006: 46. The translation was published under the title "On the Nature and Treatment of Leprosy" by Erasmus Wilson. It appeared a year after the French edition (*Traité de al Spedalskhed ou Eléphantiasis des Grecs*, 1848) won the Prix Monthyon from the Academy of Sciences in Paris in 1855. Danielssen visited London in 1853, but going through his private correspondence from this time (which was discovered in his desk drawers in Bergen the summer of 2010 and is currently held by Bergen Museum), I have found no indication that Danielssen corresponded with either Carter or Wilson.

[567] St. Jørgens was first mentioned in a testament from 1411. For a history of the institution, see: Knudsen S. Å., et al. *"De fattige Christi Lemmer"... Stiftelsen St. Jørgens historie*. 1991.

[568] Vollset 2005. In 1895 the patients and research activities were moved to the neighbouring 'Pleiestiftelsen', and two years later the building was bought by the municipality who used it first as an isolation hospital and from 1912 as a sanatorium for tuberculosis. The building was torn down in 1957.

[569] For a list of references to the publications on the activities of the research hospital ("Lungegaardshospitalets Virksomhed") as well as an extensive bibliography of Danielssen's published research, see: http://www.whonamedit.com/person_bibliography/2321/. See also: Brunchorst, J. *Bergen Museums Aarbog 1893. D. C. Danielssen. A biographical sketch.* 1894.

(see Chapter 3), part of the justification for not offering Bakewell any payment was that the results of Milroy's investigations in the Caribbean showed the cure to be ineffective.[570] Danielssen reported this before Milroy had published his report, indicating that he was up to date also on leprosy-related research conducted elsewhere.

As Hammerborg has emphasized, Danielssen and Boeck's framing of leprosy as a hereditary dyscrasia had direct political implications.[571] Between 1850 and 1857, the advisory Medical Committee to Parliament was especially successful in involving the state in leprosy work. All three members of the committee used the theory of heredity as the basis for their arguments, and in 1850 they suggested a law which would make it illegal for people from 'leprous families' to enter into marriage. After some debate, the suggestion was rejected. Inheritance in the sidelines meant the precautions would apply to too many people, and there were worries that a marriage ban would not result in fewer offspring but immoral and 'unnatural sexual behavior'.[572] The committee's other suggestion was to establish state institutions. The argument balanced between compassion and control. That those suffering from the disease needed care was beyond dispute, but those affected were a heavy financial burden on the local communities. The main rationale for the state intervention was to share this economic burden. This was coupled with the threat that without an intervention the disease prevalence would only increase and finally get out of hand.[573]

In 1857 the leprosy asylums Pleiestiftelsen No. 1 opened next to Lungegaards-hospitalet in Bergen with 280 beds.[574] In 1861, both Reknes and Reitgjerdet were expanded to 160 and 180 beds respectively.[575] When Carter visited Bergen in 1873, he was full of praise for the institutions, highlighting the segregation of the sexes "the

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[570] Danielssen, D. "Lungegaardshospitalets Virksomhed i Treaaret 1871-73". *Norsk Magazin for Lægevidenskaben*. 1874: 319-321. See also: Edmond 2006: 61-62.
[571] Hammerborg 2009: Chapter 2.
[572] "Betænkning afgiven til Departementet for det Indre fra dettes raadgivende Medicinal-Comitee, angaaendde Foranstaltninger mod den spedalske Sygdom. Christiania den 30te December 1850." Available online: http://digitalarkivet.uib.no/cgi-win/webmeny.exe?slag=visside&kat=lepra-eng&n1=4&n2=2&dok=rapport.htm. See also: Vollset 2005: 44-47; Hammerborg 2009: 79-86.
[573] Op. cit. See also: Vollset 2005: 44-47; Hammerborg 2009: 74-78.
[574] Andresen 2004: 93-116.

idea being that they [young and active lepers] should not be allowed to propagate their disease to offspring, and this idea was the dominant one leading to the establishment of asylums."[576]

Historian Astri Andresen has in her study of Pleiestiftelsen No. 1 shown that around the time of Carters visit, the initial optimism among the inmates flocking to the institutions hoping to be cured was diminishing. Increasingly, the institution was perceived more like a prison than a hospital. Consequently, those who were sufficiently healthy (and had a place to run to) left the hospital without permission.[577] While Carter in 1873 painted a rosier picture than Andresen, he too noticed that "The helpless and infirm leper must obviously be cared for, and most asylum inmates are of this class."[578]

In addition to the institutions, the Norwegian leprosy apparatus consisted of a network of district physicians who annually registered each individual leprosy sufferer in their district. The genesis of the system was in 1854, when the Medical Committee received permission from the state to appoint two physicians charged with coordinating the national leprosy campaign, who were to report directly to the Ministry of Domestic Affairs ('Departementet for det Indre'). Through popular lectures in the most affected areas they were to ensure that the institutions were used, and that interest in the disease did not diminish.[579] Presenting the apparatus as the outcome of three parallel and competing medico-political projects, Hammerborg has shown how establishing permanent leprosy commissions in all municipalities with lepers was the brainchild of the head physician for leprosy ('Overlege for den spedalske sykdom') in the Northern part of the country, Ove Guldberg Høegh. The commissions, established in 1856, were headed by the district physicians, and included the heads of the local governments. Four years after their inception, in 1860,

[575] Vollset 2005: 58. For Carter's description of these asylums, see: Carter 1874: 13-15.
[576] Carter 1874: 15.
[577] Andresen 2004: 93-116.
[578] Carter 1874: 15.
[579] In 1863, the head physician for leprosy reported that leprosy had been the main topic of 256 public lectures around Norway. (Løberg, T. J. *Tabeller over de spedalske i Norge i aaret 1864 samt aarsberetning for samme aar*. 1864:13, in: Vollset: 2005: 51-52.)

the leprosy commissions were used as a model for local Health Commissions ('Sunnhetskommisjoner') in all municipalities, the backbone of the Norwegian health system for decades to come.[580] Unlike in British India, there were no preexisting sanitary commissions in Norway. Rather, the leprosy campaign was integral in establishing the public health system.

The district physicians were also charged with keeping an updated registry of every sufferer, to be submitted to Høegh annually for compilation and analysis. According to Hammerborg, the questionnaires (which Carter translated and printed in hope they could be used in India[581]) were designed to challenge Danielssen and Boeck's hereditary disease model in favor of a sanitary model.[582] Still, it was more a question of emphasis than a direct challenge. Danielssen and Boeck too had argued that the disease could have multiple causes, and Høegh did not deny a hereditary component. Høegh's principal position, however, was that leprosy was a sanitary concern caused by poor living conditions. In addition to keeping track of every individual leper, the main task of the commissions was to educate the population and work against "bad housing, poor clothing, deficient food and filth".[583]

Høegh died in 1863, and Timandus Jonas Løberg in Bergen remained the sole national Head Physician for Leprosy, alongside a position as physician at Danielssen's research hospital. Løberg saw the disease as caused by 'the low stage of civilization' of the population, connecting it as did Høegh to climate, housing conditions, hygiene and poor clothing.[584] Again, this did not exclude a hereditary component.

There were also other differences between Norway and the British Empire. In Norway leprosy was a domestic disease, not something mainly found in overseas colonies. Hence, declaring the disease not to be a threat to the colonizers, as Milroy

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[580] Schiøtz, Aina. *Folkets helse, landets styrke 1850-2003. Det offentlige helsevesen i Norge 1603-2003*, bind 2. 2003: 26.
[581] Carter 1874: 43 (Appendix 6).
[582] Hammerborg 2009: 86-92.
[583] Höegh, Ove Guldberg. *Tabeller over de spedalske i Norge i aaret 1860 samt aarsberetning for samme aar.* 1860: 14, quoted in Hammerborg 2009: 88; Vollset 2005: 50-51.

did in 1867, was never an option. The Norwegian physicians' warning that the disease would come out of hand without state intervention was also credible. The first census of leprosy sufferers in Norway, conducted by the parish ministers concluded in 1836, concluded that there were 659 'lepers' in the country. Twenty years later, when the national patient registry wae stablished, the number had reached almost three thousand.[585] On the other hand, with less than 3000 lepers registered at any one time, the size of the problem was also more manageable than for instance in the Bombay Presidency with almost 8000 known cases. In colonial India as a whole, 100.000 cases were considered a low estimate. Additionally, in India other diseases (especially cholera) were a larger and more imminent threat to health. While the leprosy census Carter analyzed showed that one of 600 persons in his presidency had leprosy, the proportion was slightly lower than the average death toll to cholera in India every year (1 out of 570). In 1877, which was one of the worst years, 357.430 cholera deaths were recorded in Madras Presidency alone.[586]

A final vital difference was that in Norway the state employed physicians working with leprosy were part of the elite class of public servants ('embetsmenn') which dominated national politics.[587] Unlike in London or colonial India, the Norwegian leprosy physicians were not just advisors, but many were elected representatives to Parliament. Boeck was elected to Parliament in 1845; his younger colleague Danielssen was five times elected to represent Bergen in Parliament (1859-60, 1862-64, 1871-1876); Løberg served three parliamentary terms (1865-67, 1871-76). Danielssen and Løberg were also active in local politics. This means that they had intimate knowledge of how the political system worked and were in a position to influence policies from 'within'.

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[584] Løberg, T. J. *Tabeller over de spedalske i Norge i aaret 1862 samt aarsberetning for samme aar.* 1862: 12, as quoted in Vollset 2005: 49.
[585] Vollset 2005.
[586] Arnold, David. "Cholera and Colonialism in British India". *Past and Present*, No. 113. 1986: 118-151, numbers on p. 122.
[587] In the Norwegian historiography, the term "embetsmannsstaten" ('the civil servant state') is the dominant interpretation of the political system between the country getting its own constitution in 1814 and the introduction of parliamentary rule in 1884. Seip, Jens Arup. *Fra Embedsmansstat til ettpartistat og andre essays.* 1963.

When Carter visited in 1873, Løberg was still administering the leprosy apparatus and Danielssen was still in charge of the research hospital. In Christiania (now Oslo), Carter met both the head of the Ministry of Health ('Medicinaldepartementet') and Carl Wilhelm Boeck. Boeck was at that time the head of the Division of Dermatology at the National Hospital (Rigshospitalet), and had just returned from North America where he had examined leprosy among Norwegian emigrants. His findings, he argued, confirmed his theory that the disease was hereditary.[588]

In the early 1870s, the debates on the nature and causes of the disease had reignited, with contagion as the relative newcomer. The debate was sparked not by domestic concerns, but by reports from abroad. In 1871, the *Report of the Royal College of Physicians* (1867) was introduced to a Norwegian audience by medical professor Ferdinand Lochmann at the University of Christiania.[589] To Lochmann, the report was not a confirmation of heredity or that the disease was a sanitary concern, but a major argument for the contagiousness of the disease. This he supported by quoting the 24 statements (of a total 250 replies) from the report that all supported this view.

Retired physician Johan Jørgen Hjort, who since the 1830s had argued that the disease was connected to hygiene and that the importance of heredity had been overestimated, soon rebutted that Milroy who had compiled and analyzed the replies had reached the exact opposite conclusion:

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[588] Boeck, W. "Spedalskheden i de forenede Stater i Nordamerika". *Nordiskt Medicinskt Arkiv*. Bd. 3, No. 1. 1871. This was the second of three such investigations. In 1863 Jens Andreas Holmboe, argued that his investigation of Norwegian 'lepers' in North America confirmed his theory that the lepers' health would improve with changes in climate and lifestyle. In 1889 Gerhard Armauer Hansen would argue that his investigation confirmed that the disease was contagious and that improved hygiene had stopped its spread. See: Lie, H. P. "Norwegian Lepers in the United States: The investigations of Holmboe, Boeck and Hansen". *International Journal of Leprosy*. No. 3. 1938: 351-356; Gussow 1989: 81-82; Davidsen, Bjørn. ""Forskerstafett" til Amerika 1863-1888." *Bergensposten*. No. 4. 2001: 29-39. Available online: http: //digitalarkivet.uib.no/sab/bp2001/side29.htm.

[589] Lochmann, F. *Om Spedalskheden*. 1871. Incidentally, Lochmann was the model for Dr. Thomas Stockmann in Henrik Ibsen's play *En Folkefiende (An Enemy of the People*) from 1882, especially for Lochmann's use of the popular press to spread his messages.

> The all but unanimous conviction of the most experienced observers in different parts of the world is quite opposed to the belief that leprosy is contagious or communicable by proximity or contact with the diseased. The few instances that have been reported in a contrary sense either rest on imperfect observation, or they are recorded with too little attention to the necessary details as not to affect the above conclusion.[590]

While the report in colonial India gave Gavin Milroy the authority to define the mandate for investigating leprosy as a sanitary concern, in Norway this was but one of several reports from abroad. Gerhard Armauer Hansen, who since 1869 had worked as assistant physician at the Lungegaarden research hospital, also pointed out that Lochmann's interpretation was "odd". A better argument, Hansen argued, was found in the works by the Dutch physician Charles Louis Drognat Landré. In 1867, based on case histories of 12 children of European settlers in Surinam contracting leprosy, Drognat Landré had argued that leprosy was an infectious disease propagated solely by contagion.[591] When Carter visited Norway, the debate on the nature and causes of leprosy was on top of the agenda: "Respecting the *causes* of leprosy – a subject not unconnected with the topic under notice – I have not during my tour in Norway found less debate there than elsewhere."[592]

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[590] "Report on Leprosy by the Royal College of Physicians prepared for Her Majestys Secretary of State for the Colonies", as quoted in Hjort, J. J. "Om Aarsagerne til den spedalske Sygdom." *Norsk Magazin for Lægevidenskaben*. 1872: 125, English in the original.

[591] Hansen presented nine of the twelve case histories, and highlighted Drognat Landré's publication as what 'attracted my attention to contagion not being adequately investigated here.' (Hansen, G. Armauer. "Om vort Kjendskab til Spedalskhedens Aarsager og om vore Forholdsregler mod Sygdommen". *Norsk Magazin for Lægevidenskaben*. 1872: 21). Hansen would later praise Drognat Landré's monograph as "the most valuable of research about this [the contagiousness of leprosy]" (Hansen 1874: 80). The library catalogue from the research hospital in Bergen contains both Landré's Dutch original (*De besmettelijkheid der lepra arabum*. Utrecht. 1867) and the French translation of the treatise (*De la contagion de la lèpre*. Paris. 1867). See: Dethloff, H. G. *Katalog over Lungegaardshospitalets Bibliothek ved udgangen af aaret 1904*. Bergen, 1905: 144. For more on Drognat Landré, see: Menke, Henk E, William R. Faber, and Toine Pieters. "Charles Louis Drognat Landré and Gerhard Henrik Armauer Hansen; contribution from a Dutch colony to the discovery of the leprosy bacterium". *Leprosy Review*. Vol. 81. 2010: 82-86. In comparison, the Royal College of Physicians dismissed Landré's work out of hand, arguing that "from the meagre and unauthenticated details of the nine cases cited in support of the contagion of leprosy, the College feels that the evidence on which these inferences rest is quite insufficient for the solution of any scientific question." (Letter from George Burrows to The Earl of Kimberley, dated December 1872. "Recent Official Correspondence relating to Leprosy", *The British and Foreign Medico-Chirurgical Review*. 1874: 207.)

[592] Carter 1874: 26.

The most sophisticated argument for contagion was presented in Hansen's report to the Medical Society in Christiania in 1874, which he later would refer to as his main scientific contribution. The aim of the report was to find the laws that governed why leprosy appeared.[593] The report was written after he was granted a scholarship from the Medical Society in 1870, which Hansen spent traveling to Germany, Vienna and Venice to learn the latest research techniques.[594] His literary review stressed the numerous positions on etiology that existed among the Norwegian physicians, ranging from inheritance, via degeneration caused by cultural stagnation, to spontaneous appearances, miasma and contagion, as well as combinations of causes. For Hansen, however, the disease was specific, contagious and decidedly not hereditary.

Hansen's conclusions were based on case histories from 69 families, statistics, dissections, animal experiments and microscopy. The main argument hinged on comparing Norwegian districts based both on his own travels, reports from physicians and statistics from the patient registry that Høegh had established eighteen years earlier. Hansen did, however, make a vital alteration to the original statistics: Instead of reflecting the time each individual was diagnosed, he backdated the cases so that the registry instead reflected when the disease first had made its presence known.[595] When comparing different districts using these new figures, it was clear that the disease appeared independent of filth and malnutrition, and regardless of climatic factors such as weather or humidity. Yet, there was a strong correlation between isolation and the number of new cases. In districts where those suffering from the disease had been institutionalized, the number of new cases had declined rapidly, while in districts where no 'evacuation' had taken place, there was a steady supply of new cases. Furthermore, on the whole the decline had taken place faster than could be

[593] Hansen 1874: 1-88. See also: Irgens 1984: 337-343.

[594] In his autobiography, Hansen highlights studying microscopy with Max Schultze in Bonn. Caught in the Franco-Prussian War, Hansen left for Vienna where he picked up Ernest Haeckel's *Natürliche Schöpfungsgeschichte* (1868). Alongside Darwin's *On the Origin of the Species* (1859), this was at the foundation for his worldview. (Hansen 1910: Chapter 4.) Hansen would soon present Darwin to a wider audience through a series of articles in the local newspaper *Bergensposten*, and in the popular science magazine *Naturen* which from 1887 also had its base in Bergen.

explained had the disease been hereditary. The only explanation, Hansen concluded, was contagion. 'Evacuating' lepers to the institutions had had the desired effect.

> Only after leprosy has become endemic do the causes which point towards heritance appear. If the disease was contagious, this could be explained quite naturally in that the first case caught the contagion elsewhere and later it spread to the closes acquaintances – members of the same family or lineage.[596]

Initially the report was intended to be printed as an independent publication, since the columns in the Medical Association's journal *Norsk Magazin for Lægevidenskaben*, the only Norwegian medical journal at the time, were limited. Referring to Carter's interest and intent to produce a monograph based on his Norwegian experience, Hansen insisted that the report instead was to be printed as an appendix to the journal. This would ensure legitimacy, a larger distribution and hasten the publication without disrupting other journal content.[597] The chairman of the Association, Christian Thorvald Kierulf, was also contacted by Carter directly.[598] The 'international' interest was the main argument in the decision to cover the expenses of printing the report as an addendum to the journal, illustrating how knowledge exchanges were not mono-directional. It was also on Carter's suggestion that Hansen the following year published a slightly shortened translation in *The British and Foreign Medico-Chirurgical Review*.[599]

The only immediate response to the publication was a critique by Thorvald Buchholz, district physician from Hadeland and a prolific writer. Buchholz argued that Hansen had gone looking for proof for his theory of contagion, and bent the

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[595] Hansen 1874: 62-63.
[596] "Först efterat spedalskheden er blevet endemisk opträde de tilfälde, der kunde tyde på arv. Var nu sygdommen smitsom, kunde dette have en ganske naturlig forklaring, idet de förste tilfälde, erhvervede ved smitte andetsteds, senere hyppigt smittede sin närmeste omgang, medlemmer af samme familje eller slägt." Hansen 1874: 36.
[597] "Møte den 20de Mai 1874". *Forhandlinger i det Norske medicinske selskab i 1874*. 1875: 140-143.
[598] Op. cit. Kierulf had international contacts of his own: In the early 1850s, he befriended Rudolf Wirchow while studying in Würtzburg, and was instrumental in inviting him to study leprosy in Norway in 1859. (Jæger, Henrik. *Videnskabernes Literatur i Det Nittende Aarhundrede*. 1896: 232-233.)
[599] Hansen 1875: 459-489.

findings to fit this conclusion:

> If the isolated researcher, as Hansen and many with him, start with preconceived ideas and theories in which the entire research should be squeezed and the results bent to fit, it leads to a misleading confusion – and Hansen's report shows exactly such a highly one-sided tendency that it must be an obligation to refute it.[600]

According to Buchholz, the association had made a mistake in printing Hansen's 'speculations'. In his view, the disease was not contagious but the result of an inherited functional deficit brought forward by cultural and intellectual stagnation. Any decline in prevalence was caused by cultural enlightenment. If the state continued to pour money into costly state institutions instead of spending it on educating the population, he predicted the decline in new cases would be only temporary.[601]

Despite disagreements, suggesting that the disease was contagious did not lead to Hansen being 'elbowed out' of further leprosy work, rather the opposite. When Løberg in 1875 was made director of the National Hospital and Birth Clinic in Christiania, Hansen was temporarily appointed the new 'head physician for leprosy' ('overlege for den spedalske sygdom'), a position he would keep until his death in 1912. That Hansen in 1873 married the daughter of his immediate superior Daniel Danielssen was probably of minor significance.[602] More importantly, he was recruited from the same position at the Lungegaarden research hospital that Løberg had had before he was appointed in 1857. Before Hansen inherited the position, this was seen as an administrative chore. With Hansen at the helm, this would quickly change.

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[600] "Gaar saaledes den isolerede Forsker, som *Hansen* og Mange med ham have gjort, du fra forudfattede Ideer og Teoremer, indenfor hvilke altsaa den hele Forskning skal indklemmes og Resultaterne derefter bøies, saa fremkommer herved en misvisende Uklarhed – og netop *Hansens* Indberetning viser en saadan høist ensidig Tendents, som det maa være Pligt at tilbakevise. Kritikk var og er altsaa her paa rette Sted." Buchholz, Thv. "Mer om Spedalskhedens Væsen". *Norsk Magazin for Lægevidenskaben*. 1875: 2.
[601] Buchholz 1875: 42-43.
[602] Stephanie Marie Danielssen died later the same year due to tuberculosis.

Contagion and the law

As head of the Norwegian leprosy apparatus, Hansen reinterpreted the rationale behind the state intervention. The institutions were no longer described as 'good homes' to provide care, but as institutions for the isolation of a contagious disease.[603] This went hand in hand with new legislation. In 1876, Hansen proposed an Act which would make it illegal to accept leprosy sufferers in need of care in the 'legd system' – the traditional system of accommodating poor people at local farms for a period before they were sent to the next farm. For a contagionist like Hansen, this system ensured that the disease would spread. Instead, poor leprosy sufferers should as a rule be admitted to the public leprosy institutions.[604] In addition, the legislation demanded that the clothes and linens of deceased 'lepers' should be disinfected.

Underlying these measures was also a concern that the leprosy institutions had almost 300 unoccupied beds while a majority of Norway's registered 'lepers' remained outside the institutions. This was increasingly pointed out by physicians and members of Parliament who argued that the costly and increasingly unpopular institutions should be shut down completely. In Parliament, however, the supporters of the law argued that it would "bring people's attention to the disease, and this alone will make the Act useful."[605] Voices of opposition were also heard, pointing out that they were not convinced of the premise that leprosy was contagious. Bishop Jacob Liv Rosted Sverdrup, for instance, argued:

> As long as the population suffers from bad housing and malnutrition, a disease like this will remain; as soon as the living conditions are improved, the disease will diminish. This is the main cause for the reduction of the disease; the institutions have never performed miracles, and they never will.[606]

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[603] Odelstingsproposisjon 24/1877; Vollset 2005: 72-74.
[604] "Lov om Forsørgelsen af fattige Spedalske m. V.", passed May 26, 1877: §2.
[605] "(…) denne Lov vil virke til at vække en større Opmerksomhed hos Alle for Sygdommen, og allerede herved vil Loven gjøre Nytte." Ellingsen, Lasse. "Ang. Lov om Forsørgelse af fattige Spedalske." *Stortingstidende 1877, Forhandlinger i Odelsthinget*. 1877: 198; Vollset 2005: 72.
[606] "Saalænge Befolkningen lever slet, har daarlige Huse, slider meget ondt paa daarlig Kost, vil en Sygdom som denne vedblive; saasnart Folkets Kaar bedres, vil Sygdomen mere og mere forsvinde. Det er Hovedsagen

Despite opposition, the law was passed without much debate. The winning argument was statements from the local Health Commissions which showed that the law would confirm an already existing practice. From 1877 those affected by the disease were in Norway sources of contagion in the eyes of the law, and were to be treated as such. Leprosy in Norway was becoming a legal matter.

In 1885 a new and stricter leprosy Act was passed.[607] The new legislation gave the local Health Commissions power to request the police to forcibly move the sufferers into the institutions if they failed to live up to the district physician's demands for isolation at home. At the same time, the district physicians would for the first time be reimbursed for their traveling expenses visiting the lepers living in home isolation. Again, all the preliminary work was done by Hansen, who from 1883 discussed the proposed legislation in all the Municipal Boards ('Herredsstyrer') in Western Norway. He also sent the proposal to the 176 Health Commissions in municipalities with leprosy patients: 148 supported the act, 10 were negative, while 18 sent their own suggestions, ranging from reviving the call for a marriage ban to reorganizing the institutions to make them more attractive for the sufferers.[608]

In parliament, the proposed legislation was interpreted as a technical question which demanded medical expertise. Since the suggestion came directly from 'the men of medical science' several politicians argued that it was their duty to endorse it. As the county governor Carl Lauritz Mechelborg Oppen in Northern Bergenhus county, who supported the Act, put it:

> The question at hand, namely if the danger of spreading the disease through contagion is in reasonable proportion to the restrictions on personal freedoms that the suggestion intends, and if the restrictions are a reasonable measure to eradicate the disease, can hardly be judged by other than the men of medical science.[609]

til at Sygdommen fjernes; man har aldrig gjort Underværker ved Pleiestiftelserne og vil aldrig gjøre det." Smitt, Jacob Sverdrup. "Ang. Lov om Forsørgelse af fattige Spedalske." *Stortingstidende 1877, Forhandlinger i Odelsthinget*. 1877: 198, also quoted in Vollset 2005: 73.

[607] "Lov angaaende Spedalskes Afsondring og Indlæggelse i offentlig Pleie- eller Helbredelsesanstalt m.V", passed June 6, 1885.

[608] Vollset 2005: 80.

[609] "Det, hvorpaa det her kommer an – nemlig, om Faren for den spedalske Sygdoms Udbredelse ved Smitte

Again the argument was that the proposed act would remind the population that the disease was contagious and thus encourage them to take proper precautions. Minister of Justice Aimar Sørensen, however, warned that it was exactly this idea that had led to inhumane prosecution of leprosy sufferers in antiquity and Medieval Europe:

> This lesson was surely known in antiquity, it led to lepers being chased like wild animals into the forests. I believe that the Act can be dangerous if it is used as such a lesson. People are easily educated into seeing these people as outcasts of humanity that no longer possess any human rights.[610]

The Act came into force on June 6, 1885. But Oppen's assertion that the legislation was the collective judgment of 'the men of medical science' soon proved to be wrong. Only Health Commissions in districts with lepers, in other words those who would get increased powers as a result of the Act, were asked for input. After the legislation was enacted, this led to heated debates both in the Medical Society of Christiania, and in two of the national medical journals.

The editor of the *Journal for Practical Medicine* ('Tidsskrift for Praktisk Medicin', established in 1882), Nils Wulfsberg, argued that all health legislations should be discussed in the medical societies and printed in medical journals before being sent to the politicians. The new law would, he argued, result in persecution and conflicts within families, and inevitably be abused. "From now on, leprosy is not a disease that evokes involvement and aid, but a crime to punish."[611] Wulfsberg was not opposed to the theory of contagion, but pointed out that nothing was known about the precise mechanisms involved. For him, Hansen's argument that the institutions

staar i rimeligt Forhold til den Indskrænkning i den personlige Frihed, som Forslaget tilsigter, og hvorvidt saadan Indskrænkning vil blive et væsentligt Middel til den spedalske Sygdoms Udryddelse, – kan vel for Tiden vanskelig bedømmes af andre end lægevidenskabeligt uddannede Mænd." Odelstingsproposisjon 12/1885: 9.

[610] "Den Opdragelse hadde visselig Folk allerede i Oldtiden; thi da jagede man saadanne Spedalske ligesom vilde Dyr til de fjerne Skove osv. Jeg tror, at Bestemmelsen netop kan blive lidt farlig, hvis den skal tjene til at opdrage i den Retning; Folk er meget tilbøielig til at lade sig opdrage paa den Maade, at de betragter disse Mennesker som et Slags Udskud af Menneskeheden, der ingen Menneskerettigheder længere har." *Storthingstidende*. 1885: 193.

[611] "Spedalskhed er fra nu af ikke en Sygdom, der vækker Deltakelse og berettiger til Hjælp, men en Forbrydelse, hvorefter man hjemfalder til Straf." (Wulfsberg, N. "Tvangslov og Stiftelser mod Spedalskhed". *Tidsskrift for Praktisk Medicin*. No, 15. 1885: 295.)

were successful because the number of lepers declined, and the declining number of lepers proved that the institutions were successful, was not enough to justify the new law.[612]

Hansen chose to rebut the charges in his own medical journal, *Medical Review* ('Medicinsk Revue', established in 1884 with Hansen as one of three editors). To him, the law was 'humane'.

> My position in this regard, is that the most humane act is to protect the healthy from catching the disease, and that those who have the disease have not only rights, but also duties – the most important being to not spread the disease to their fellow man.[613]

According to Hansen, when faced with a disease for which there was no cure, a humane approach meant prevention. "We are faced with a disease that everyone acknowledges to be the most horrid and loathsome we know, and the goal is to prevent its dissemination so that as few as possible will suffer from it."[614] Obviously investigations into finding a treatment should continue, but until then "It is simply a question of power and rights, the individual or the society (…) either the healthy must run away, or the sick person must be put outside society, must be isolated."[615]

Wulfsberg also pointed out that almost a third of the beds in the state institutions were empty. Hansen agreed that the number of patients in the institutions

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[612] For Wulfsberg a more likely explanation for the decline of leprosy was that the Norwegian lepers had immigrated to the United States. Hansen rebutted that only 52 known lepers had emigrated from Norway to the US, while at the same time 3.901 have been admitted to the institutions. (Hansen, Gerhard Armauer. "Den nye lov om Spedalskheden." *Medicinsk Revue*. 1885: 362). Wulfsberg, in turn, pointed to the long incubation period of the disease and that that many had probably left at the early onset of the disease to avoid detection. (Wulfsberg, N. "Replik". *Tidsskrift for Praktisk Medicin*. 1885: 407.) This debate was the direct precursor to Hansen's journey to the United States to investigate leprosy among Norwegian emigrants in 1888-1889.

[613] "Min Opfatning i denne Henseende er den, at det er det mest humane at beskytte de friske Mennesker mod at faa Sygdommen, og at de, der har Sygdommen, ikke alene har Rettigheder som Mennesker, men ogsaa Forpligtelser, og blant disse som den vigtigste den at ikke paaføre sine Medmennesker Sydommen." (Hansen, Gerhard Armauer. "Tvangslov og Stiftelser mod Spedalskhed". *Medicinsk Revue*. 1885: 285-286. The reply was also printed in *Tidsskrift for Praktisk Medicin*. No. 19. 1885: 391-397.)

[614] "Vi staar overfor en Sygdom, som af alle erkjendes at være en af de uhyggeligste og tækkeligste, man kjender, og det gjælder om saavidt mulig at forebygge Sygdommens Udbredelse, for at saa faa som muligt skal komme til at lide under den." (Hansen, Gerhard Armauer. "Den nye lov om Spedalskheden." *Medicinsk Revue*. 1885: 352.)

did not fully explain the decline of new cases; rather, the fear of the leper was itself the best deterrent. "I also believe that in certain districts there must be other factors at play. These consist of progress of civilization which leads people no longer to associate with lepers as freely as before."[616]

Numerous physicians joined the debate. District physician Thorvald Buchholz, who ten years earlier had argued it was a mistake to publish Hansen's arguments for contagion, repeated that leprosy was a constitutional disorder brought forward by cultural stagnation and that the legislation was thus based on false premises.[617] His son, district physician Jerome Buchholz pointed out that if the disease was contagious, it would already be covered by the National Health Act of 1860. Referring to Milroy's report from 1867, he concluded that since the disease was not contagious, there was no justification for the legislation.[618]

Head physician Edvard Kaurin at Reknes supported the legislation with reference to isolation or hospitalization being the accepted response to sufferers of other contagious diseases, such as syphilis, typhus and cholera. What made him uncomfortable was not that these were diseases that compared to leprosy usually were resolved in a relatively short time, but the lack of definitive proof for contagion: "Was the contagion in leprosy an undisputed fact, there could be no doubts about the full justification of the law."[619] District physician in Lyster, H. R. Smith, saw the law as nothing more than a natural extension of the relatively uncontroversial 1877 Act:

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[615] "(…) det er simpelthen et Magtspørgsmaal, hvem der har Ret, det enkelte Individ eller Samfundet (…) enten maa de friske Mennesker rømme eller den syge Person må sættes udenfor Samfundet, maa isoleres." (Op.cit: 354.)

[616] "(…) jeg tror ogsaa, at der i visse Distrikter maa være endu andre Faktorer, der gjør sig gjældende, og jeg tror, at disse bestaar i en fremadskridende Civilisation, der lidt efter lidt medfører, at man ikke omgaaes de Spedalske saa ugenert som tidligere." (Hansen, Gerhard Armauer. "Tvangslov og Stiftelser mod Spedalskhed". *Medicinsk Revue*. 1885: 289.)

[617] Buchholz, Thv. "Om Tvangslov og Stiftelser mod Spedalskhed". *Tidsskrift for Praktisk Medicin*. 1885: 383.

[618] Buchholz, Jerome. "Den spedalske Tvangssmittelov". *Tidsskrift for Praktisk Medicin.* 1885: 476-481.

[619] "Var Kontagiositeten et ubestridelig Faktum, kunde der jo ikke reises Tvil om Lovens fulde Berettigelse. Den fordrer jo kun det samme som ved enhver smitsom sygdom: Isolation eller Indlæggelde paa et Sygehus, og jeg har aldrig hørt, at vore Bestemmelser om Syfilis, Kolera, Tyfis etc. er inhumane. De kan være høist ubehagelige og brydsomme for dem, hvem disse Sygdomme rammer, men de er en bestemt Nødvendighed ligeoverfor det hele Samfund." (Kaurin, Edv. "Om den nye Lov angaaende Spedalskhed". *Medicinsk Revue*. 1885: 349.)

> What is then new in the law of June 6, 1885? It gives district physicians' compensation for travels and diet for journeys to control the hygienic circumstances, and gives the Health Commissions a tool to implement their decisions.[620]

Despite taking place after the law was in effect, and failing to achieve a legislative rematch, the debate did have an impact. In a circular to the Health Commissions dated September 15, 1885, Hansen clarified what was meant by the "adequate seclusion" demanded in the legislation: The lepers must have their own bedroom, at least a bed not used by others. They might eat with others but must use their own cutlery. The district physician should "through the Health Commissions attempt to educate people not to see intermingling with lepers as harmless."[621]

While the debate probably made the implementation of the law less severe, it did lead to forced hospitalizations. From 1891-1895, Reitgjerdet alone reported that 13 of their new patients had been admitted against their will.[622]

Outside the national context, however, this controversy was never mentioned. When Henry Vandyke Carter in 1887 referred to the Norwegian legislation in a report to the Bombay Government, again arguing that they were applicable to India, he made no references to the dispute. Instead, Carter would echo Hansen's claim that the reason why the "sagacious and patriotic legislation" was not implemented earlier simply showed "how difficult [it has] proved to overcome prejudice or to interfere with the liberty of the subject."[623] Likewise, when Hansen in Berlin in 1897 proudly

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[620] "Hva Forandring bringer nu Loven af 6te Juni 1885? Den giver Distriktslægen offentlig Skyds- og Diætgodtgjørelse til Reiser for at kontrollere de Spedalskes hygieniske Forhold og giver Sundhedskommissionen et Middel til at faa sine Forskrifter iagttagne." (Smith, H. R. "Loven om spedalskes Afsondring m.v." *Medicinsk Revue*. 1885: 365.) This opinion was echoed by district physician Lund, who saw it as a threat which could be invoked against those who refused to comply with the instructions, but that would never be used in practice. (Lund, Dr. "Den nye Lov om Spedalskes Afsondring og Indlæggelse i offentlig Pleie- eller Helbredelsesanstalt m. v." *Tidsskrift for Praktisk Medicin.* 1885: 397-407.)

[621] "Som De vil se, er dette ikke mer end at forsøge paa gjennem Sundhedskommissionen at opdrage Folket til ikke at betragte Omgangen med Spedalske som en ufarlig Sag." (Hansen, Gerhard Armauer. "Den nye lov om Spedalskheden." *Medicinsk Revue*. 1885: 356.)

[622] *Norges Officielle Statistik, Tredie Række no. 287. Beretning om de spedalske i Norge i Femaaret 1891-1895.* 1896: 89.

[623] Carter, H. V. *Observations on the Prevention of Leprosy by Segregation*. 1887: 2. The following year the City of Bombay did pass a Municipal Health Act aimed at preventing the spread of dangerous diseases such as cholera, but it turned out not to be of much help dealing with a chronic disease such as leprosy as there were no facilities for lifelong segregation. (Pandya 2001: 144-153.)

presented the Norwegian leprosy campaign as a system of segregation to be copied by others, neither the debate nor any of the counter-arguments were mentioned. What made the legislation successful, Hansen argued, was that it gave the local community the tools they needed to get rid of the disease. Then it was up to them to decide if this was something they wanted to achieve.

> I find it more humane to protect people against leprosy than to give the lepers the rights and opportunity to cause other people to catch the disease, and I have no doubt that that we are doing humanity a big favor in being liberated from leprosy, and [through these measures] it is indeed possible.[624]

While Hansen agreed that the disease was not highly contagious, the selective presentation of the legislation probably influenced the appropriations elsewhere. Coupled with Hansen's definition of what a 'humane approach' entailed, namely protection of the healthy majority through removing those suffering from the disease, this is part of the explanation for the huge disparities in the later interpretations of the 'lessons from Bergen'.

There was, however, one more difference between the two legislative debates in Norway: References to the leprosy bacillus. This was not an argument for the 1877-Act in Norway, nor was it part of the debates in either Norway or India in the 1870s. In 1885, however, Wulfsberg emphasized that it was the presence of the bacillus that convinced him that the disease was indeed contagious. The problem, as he saw it, was that the presence of the bacillus did not indicate what kind of contact was necessary for the disease to spread, whether the bacillus could live outside the body for instance as a spore, or whether the disease thus could be spread indirectly. In short, the bacillus did not say anything about how communicable the disease really was, and could

[624] "Ich finde es dagen viel humaner, die Menschen gegen Lepra zu schützen, als den Leprösen das Recht und die Gelegenheit zu geben, auch andere leprös zu machen, und ich zweilfe nicht, dass wir der Menschheit einen grossen Dienst leisten würden, wenn wir dieselbe von Lepra befreien könnten; das aber ist unzweilhaft möglich." (Hansen, Armauer. "Erste Sitzung, Einleitung." *Mittheilungen 1897.* Bd. II. 1897: 17.) The argument was backed up by references to isolation of other contagious diseases, the reduction in new cases, and that isolation gave a socio-economic return on investment. (Hansen, Armauer. "Die Isolirung der Aussätzingen und die dazu erfordelichen Massregeln." *Mittheilungen 1897.* Bd. II. 1897: 162-166.)

therefore not decide what measures were necessary or effective. "The claim that since bacilli are found, the causation is also found is very bold, as no other proof has been presented other than their presence."[625] The bacillus could equally well be an outcome of the disease rather than its cause.

In an independent publication, physician and medical biographer Frantz Casper Kiær supported the legislation with no mention of the bacillus at all. Instead he based his argument on the historical record: Where the lepers had been isolated, the disease has diminished or disappeared – where no isolation had taken place, the disease had been stable or increased.[626] This was a repetition of Munro's argument from 1879.

Hansen, finally, agreed that the presence of the bacillus alone did not prove contagion, but withheld that this was irrelevant: "I have, as mentioned above, not used the contagiousness of the disease as an argument in recommending the legislation, but instead the results of the precautions already taken".[627] Precautions meant isolation. Like in his 1874 report, the bulk of Hansen's argument hinged on statistics comparing new cases, institutionalization and mortality rates in different districts. But this time Hansen also made frequent analogies with bacterial research on tuberculosis, which he stressed had striking similarities with leprosy. Furthermore, the argument that the bacilli were more abundant in the tubercular cases, and that these cases according to the statistics were the most dangerous regarding possible disease transmission, shows how in 1885 the presence of a bacillus was increasingly being activated both as an argument for contagion and isolation, and for deciding whom among the leprosy sufferers constituted the greatest danger to their surroundings.[628]

Before returning to India and the British Empire, it is necessary to examine how the leprosy bacillus was introduced to the debates.

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[625] "Paastanden, at fordi Baciller er fundne, er ogsaa Aarsagen funden, er derfor i høi Grad dristig, fordi intet andet Bevis foreligger end, at de er der." (Wulfsberg, N. "Tvangslov og Stiftelser mod Spedalskhed". *Tidsskrift for Praktisk Medicin*. No, 15. 1885: 382-391, quote on p. 383.)

[626] Kiær, F. C. *Diskussion i det med. Selskab om Loven angaaende Spedalskes Afsondring m. v. af 1885*. 1885: 7.

[627] "Da nu Sagen staar saaledes med Hensyn til det strengt videnskabelige Bevis for Spedalskhedens Smitsomhed, saa har jeg, som ovenfor nævnt, ikke benyttet Sygdommens Smitsomhed som Argument ved min Anbefaling af Lovforslaget, med derimod Resultaterne af vore hidtidige Foranstaltninger mod Sygdommen". Hansen, G. Armauer. "Den nye Lov om Spedalskhed". *Medicinsk Revue*. 1885: 358.

Enter the bacillus

Already in a paper published in Norwegian in 1872, Hansen had suggested that leprosy might be caused by "a virus or perhaps a fungus", which he argued would contradict the dominating theory that the disease was hereditary.[629] At the end of his 1874 report, published in English the following year, he mentioned the results from microscopy which he demonstrated for Carter during his visit:

> "Examining he samples without any additives, one can here and there discover small rods-haped elements either stationary or in a weakly oscillating movement; (…) If one adds a drop of water, the rods move more lively and little by little more and more rods appear; the older the lesion, the more rods. (…) The size is very different, varying from 0,006–0,0015 mm".[630]

While this could be interpreted as a specific cause – strengthening the claim that the leprosy was a specific (and therefore contagious) disease, Hansen underscored that the results were uncertain: "a lot is still missing from the direct demonstration of the specificity of leprosy, but I believe that this report on my research should also inform about the steps I have taken in this direction."[631]

As I have shown, the leprosy bacillus was not an argument for the 1877-Act in Norway, nor was it part of the debate. In fact, when Hansen was made Head Physician for leprosy in 1875 it seems he stopped using the microscope for research for several years. It was not until the fall of 1879 that the bacillus was again presented as a fact, directly relevant to questions of contagion, legislation and treatment. Despite

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[628] Op. Cit. 361.
[629] Hansen, G. Armauer. "Om vort Kjendskab til Spedalskhedens Aarsager og om vore Forholdsregler mod Sygdommen". *Norsk Magazin for Lægevidenskaben*. 1872: 19-20.
[630] "Undersöger man präparaterne uden nogen tilsätning, kan man his tog her opdage stavformige legemer enten I ro eller I svag oscilllerende bevägelse; når cellerne ere bevarede hele, er deres antal ringe. Sätter man nu en dråbe vand til präparatet, kommer stavene i livligere bevägelse og lidt efter lidt träde flere og flere stave frem, jo äldre knuden er, desto flere. (…) störrelsen er meget forsjellig, varierer fra 0,006–0,0015 mm." Hansen 1874: 77.
[631] "det mangler endnu meget til den direkte påvisning af spedalskhedens specificitet, men jeg troede I denne indberetning om mine undersögelser også at berette om, hvad jeg have foretaget i den retning." Hansen 1874: 79.

frequently being backdated to 1873, it was three events in the late 1870s that sparked the lasting interest in the bacillus: Two visits to Norway, new research techniques developed on the Continent, and bacilli being identified and demonstrated as the cause of other diseases.

In the summer of 1878 the Swedish physician Fredrik Eklund traveled to Norway to study leprosy patients, and the same fall he published his findings in a book in Swedish. In the book, based on 36 Norwegian case histories, Eklund casually mentioned having observed numerous bacteria in the ulcers, blood, tears and nose tissues of most of the patients he had examined.[632]

In July 1879 the German physician Albert Neisser visited Norway and met with Hansen who provided him with unstained slide preparations and other tissue samples. Neisser had received his medical degree in Breslau two years prior. Several of his teachers and colleagues were among the pioneers in microbiology, such as the pathologists Julius Friedrich Cohnheim and Karl Weigert who researched staining techniques, and biologist Ferdinand Cohn who had collaborated with Robert Koch in developing smear tests for the identification of bacteria. Only weeks after he had returned to Breslau, Neisser succeeded in staining the samples from Norway with gentian violet and methylene blue, and in October 1879 he demonstrated the bacilli at a meeting of the Silesian Society for National Culture and published the results.[633]

The attention led Hansen to publish a paper in Norwegian, German, French and English titled "Bacillus Leprae" where he claimed precedence for the discovery.[634] The goal of the paper was:

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[632] Eklund 1879. Probably due to language, there were few further references to Eklunds work by other than Scandinavian speaking physicians. For more on Eklund's arguments, see Chapter 4.

[633] Neisser, A. "Über die Aetiologie des Aussatzes". *Jahresbericht der Schlesischen Gesellschaft für vaterländische Kultur*. 1879: 497–500. Neisser obtained his medical degree from the University of Breslau in 1877, and probably learned the histological staining technique from Julius Cohnheim and Karl Weigert before they left Breslau for Leipzig in 1877 and 1878 respectively. From 1880 to 1882 Neisser had a junior faculty position at the University of Leipzig, before returning to the University of Breslau.

[634] Hansen, G. A. "Bacillus Leprae". *Nordiskt medicinsk Arkiv.* 1880: 1-10; Hansen, G. A. "Bacillus Leprae". *Virchow's Archiv*. 1880: 32-42; Hansen, G. A. "Bacillus Leprae, études sur la bactérie de la lèpre". *Archive de Biologie*. 1880: 1-16; Hansen, G. A. "The bacillus of leprosy". *Quarterly Journal of Microscopic Sciences.*

> …to inform about my findings so far in search of the infectious agent, in part to claim priority on the finding, and in part to present the details in the study that I due to the still unsecure results did not present in my report to the Medical Association in Kristiania in 1874 regarding my research into the etiology of the disease.[635]

The main content of the paper was previously unpublished laboratory notes with details on the examination of samples taken from eleven patients at Pleiestiftelsen in Bergen, dating the first observation of the bacillus to February 28, 1873. While Hansen in 1874 had stressed that he was not sure whether what he found were true bacteria or not, now the reason for doubt was presented as practical difficulties in producing convincing samples. The method Hansen had used in 1874, namely adding distilled water and a 1% solution of osmium acid, produced specimens that it took a trained eye and a sympathetic attitude to recognize.[636]

Throughout the 1880s, Hansen and Neisser would continue to publish updates on the bacillus. The later interpretations differ when it comes to how much conflict the question of precedence really created, and to what extent Neisser was really trying to claim the discovery for himself.[637] Although Neisser quickly stressed that "I have never demanded priority for the discovery",[638] the question of precedence was not finally decided until the meeting in Berlin in 1897. There Hansen was declared the discoverer with Neisser as the one confirming the findings. In the intervening years even some researchers visiting Norway referred to Neisser as the authority on the bacillus without mentioning Hansen with a single word, such as the British physician

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New Series. 1880: 92-102. According to his autobiography, it was Daniel Danielssen who convinced Hansen to publish. (Hansen 1910: Chapter 4).

[635] Op. Cit, first page. Quote taken from the English version of Hansen's paper.

[636] Hansen 1874: 77-78.

[637] Czaplewski, E. "Albert Neisser und die Entdeckung des Leprabazillus". *Archiv für Dermatologie und Syphilis*. 1917: 513–530; Flite, G. L. and H. W. Wade. "The Contribution of Neisser to the Establishment of the Hansen Bacillus as the Etiologic Agent of Leprosy and the So-called Hansen-Neisser Controversy". *International Journal of Leprosy*. 1955: 418-427; Vogelsang, T. M. "The Hansen-Neisser Controversy, 1879–1880". *International Journal of Leprosy*. 1963: 74–80 and 1964: 330–331; Irgens 1984: 337-343. For a discussion on priority disputes, see: Merton, Robert K. "Priorities in scientific discovery: A chapter in the sociology of science." *American Sociological Review*. Vol. 22, No. 6. 1957: 635-659.

[638] "(...) erkläre ich hiermit, dass ich nie die Priorität für mich in Angespruch genommen habe, bei Lepra zum ersten Male Bakterien gesehen und auf sie hingedeutet zu haben". Neisser, Albert. "Weitere Beiträge zur Aetiologie der Lepra". *Archiv für pathologische Anatomie und Physiologie und für klinische Medizin*. 1881: 514-542, quote on p. 515.

Robson Roose.[639] The immediate importance of the 'controversy' was, however, that it changed the nature of the debate. While Hansen's cautious statements in 1874 and 1875 provoked little response, the question was now not whether the bacillus was a fact or not, but who should receive the honor for its discovery.

Two more events were central to making the perfect conditions for introducing the leprosy bacillus to the medical community: New staining techniques developed especially in the German-speaking world, and the identification of other bacillus causing contagious diseases. German researchers such as Karl Weigert, Paul Ehrlich and Robert Koch were pioneers in using various dyes for treating and staining samples, making bacteria visible when observed through the microscope. In his 1880 paper Hansen especially highlighted Koch's exemplary study of the etiology of wound infections and his presentation of the anthrax bacillus as the inspiration for resuming his laboratory studies.[640] The plethora of papers and presentations of bacteria in the early 1880s undoubtedly made the leprosy bacillus easier to accept than ten years earlier, when arguing that diseases could have a singular cause was controversial in itself. This illustrates how discussions on leprosy was integrated with and influenced by circulation of medical knowledge in general.

In many of the papers on bacteriology, the steps taken were explained so that others could reproduce them and ideally achieve similar results. When Robert Koch identified the tuberculosis bacillus in 1882, only weeks passed before Paul Ehrlich demonstrated a new staining method for coloring the bacteria to the Association of Internal Medicine in Berlin. Koch's method for staining the preparations was to use an alkaline solution of methylene blue, the same technique Neisser had used in his

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[639] This was probably more an indication of available literature than a conscious choice in an ongoing debate. Otherwise, Roose concluded that "The leprosy question in India will have to be grappled with some day, and it will become more and more difficult as time goes on. Our knowledge of the disease is doubtless imperfect, but we are fully cognizant of its horrible character, and of the means by which alone its spread can be arrested. Compulsory isolation in suitable buildings and under proper care is urgently demanded in the interests not only of the general community, but of the sufferers themselves." (Roose, Robson. *Leprosy and its Prevention as Illustrated by Norwegian Experience*. 1890: 96).

[640] Koch, Robert. *Untersuchungen über die Ätiologie der Wundinfektionskrankheiten*. 1878. Two years later, the study was translated to English by W. Watson Cheyne and published by the New Sydenham Society: Koch, Robert. *Investigations into the Etiology of Traumatic Infective Diseases*. London. 1880. See also: Gradman 2009.

demonstration of the leprosy bacillus. This rendered the preparations brown with the bacillus remaining intensely blue. By substituting the methylene with aniline oil, the preparation was rendered white with the bacteria colored red. The new method, which was presented with enough detail for reproduction, also cut the time of producing a sample from 24 hours to less than an hour.[641] This probably made it more attractive for others to put tissue samples under the microscope. The technique was printed in other journals and given the name 'Ehrlichs method'. Reports on observations of the leprosy bacillus kept accumulating, and in 1882, the *The British Medical Journal* published an editorial stating that the evidence for the bacillus was becoming stronger every day.[642]

When Carter in 1884 for the first time presented the leprosy bacillus to an Indian audience, he stressed that he had used Ehrlichs method to produce the preparations.[643] Hailing Hansen as the 'discoverer', Carter stressed that "In its fresh state I once saw this organism at Bergen (1873), and soon after at Bombay".[644] A footnote on page 27 of Carter's report from Norway published in 1874 was indeed the first time the bacillus was mentioned in any publication. Then, after referring to the "minute organisms (a species of *Bacterium*) which are present in living leprous matter taken from the interior of a 'tubercle'", Carter had quickly underlined that the inquiries had not terminated in a proper demonstration.[645] Also for Carter, it was only in the 1880s with improved staining methods and bacteria being linked to several diseases, that the observation could safely be backdated to this first time observation.

But how could observations in the microscope be connected to a specific disease? As epidemiologist Lorenz Irgens has pointed out, the gold standard for

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[641] Ehrlich, Paul. "Aus dem Verein für innere Medicin zu Berlin. Sitzung vom 1. Mai 1882." *Deutsche medizinische Wochenschrift*. Vol. 8. 1882: 269-270. Ehrlich was a classmate of Neisser from Breslau and cousin of Karl Weigert. In 1908 received the Nobel Price in Medicine for his work on immunity. For a list of Paul Ehrlich's publications, see: http: //www.pei.de/DE/institut/paul-ehrlich/publikationen/paul-ehrlich-publikationen.html

[642] Editorial. "The Bacillus of Leprosy". *The British Medical Journal.* July 29, 1882: 174-175.

[643] Carter, Henry Vandyke. *Memorandum on the Prevention of Leprosy by Segregation of the affected.* Bombay Castle, March 5, 1884: 7. The paper was also referred to in *The British Medical Journal*, see: Edmond 2006: 82.

[644] Carter 1884: 7.

[645] Carter 1874: 27.

proving a bacillus as the cause for a disease was set by Jacob Henle: 1) The bacillus must be present in all patients with the disease, 2) it must be possible to cultivate outside the organism, and 3) it must induce a similar disease upon inoculation into an animal.[646] Henle's postulates would indicate the direction of further laboratory research. For leprosy, as well as other diseases, the 1880s saw a range of laboratory studies both regarding staining, inoculations and attempts at cultivating bacteria.[647]

Hansen's presentation of his work at the Eight International Medical Congress in Copenhagen in 1884 (where he backdated the discovery to 1871 or 1872) shows the importance of Heinle's postulates. In the presentation, Hansen argued that he had found the bacillus in all tubercular cases he had examined. While he had observed no bacilli in anaesthetic cases, some of these patients developed into mixed cases with bacteria-rich tubercles. Thus, the explanation for the lacking bacilli was reduced to deficits in the staining technique.[648] In the discussion that followed, Neisser added that the presence of the bacillus in the nerve trunks of anaesthetic cases had now conclusively been established.[649] Thus, Heinle's first criterion was met.

Both Hansen and Neisser withheld that they had succeeded in cultivating the bacillus using the same substance that Koch had used for cultivating tuberculosis. Hansen acknowledged that cultivation was extremely difficult, but withheld that his observations that the cultured bacilli had produced spores, probably directly inspired by Koch's similar observations of the anthrax bacillus, proved the cultivation was successful.[650] Neissers' observations differed only slightly: "Regarding the spores, my

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[646] Irgens 1984: 341. The conditions were over time replaced by his student Robert Koch's famous postulates, first presented in 1882, and again at the International Medical Congress in Berlin in 1890.

[647] For an early study linking the inoculation experiments on animals to previous leprosy research, see: Köbner, Heinrich. "Ubertraugungsversuche von Lepra auf Thiere". *Archiv für pathologische Anatomie und Physiologie und für klinische Medicin.* 1882: 282-306. For an extensive review of the later experiments, see: Sugai, T and D. Irisawa. "Gelungene Übertragungsversuche mit Lepra bei Säugetieren". *Lepra Bibliotheca Internationalis.* 1908: 145-160.

[648] Hansen, G. Armauer. "Die Aetiologie und Pathologie der Lepra". *Congrès Périodique International des Sciences Médicales. 8me Session – Copenhague 1884. Comptre-rendu*. Tomb III, Section de Dermatologie et de Syphilidologie. 1886: 27-44.

[649] Hansen 1886: 43.

[650] Robert Koch's demonstration of stained preparations of the anthrax bacillus, and Louis Pasteur's presentation of the famous anthrax immunization experiment at Pouilly-le-Fort were highlights at the previous International Medical Congress held in London in 1881. Both presentations were reprinted in numerous medical journals.

opinions differ from Hansen, but this is irrelevant. The most important is acknowledging the Bacillus Leprae as the cause of the disease."[651]

According to Hansen's presentation in Copenhagen, only the third step, producing the disease upon inoculation in an animal, remained. Despite repeated experiments on cats, dogs, rabbits, fish and apes, all attempts at inoculation were unsuccessful (with a possible exception of a dog inoculated by Neisser that had developed a local lesion but then died before it had developed further). This was probably because these animals were immune to the disease, Hansen argued. Thus, the animal experiments "do not prove that the disease spreads from man to man, but we can unfortunately not experiment on humans, at least not here in Europe."[652]

To contagionists such as Hansen, Neisser and Carter, the bacillus proved that the disease was the same all over the world. In the paper in Copenhagen in 1884 Hansen presented preparations provided by Carter in Bombay, as well as samples from Granada produced by Neisser.[653] When Neisser later that year for the first time presented the bacillus in a general textbook, he added that he had observed the bacillus in samples from Brazil, Romania, Palestine, Hundostan [India], Dutch Guyana and Batavia. In addition he referred to descriptions by Cornil (Spain), Hillaret and John Hillis (British Guyana), B. Hernando (Spain), Köbner (Brazil), Atkinson (North America), Majocchi and Pellizzari (Italy).[654] This indicates both the speedy circulation of the new methods, and that the preparations themselves were easy to transport. Reports that the bacillus was detected in leprosy sufferers also in other parts of the world undoubtedly strengthened their argument for the bacillus.

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[651] "Bezüglich der Sporenbildung differire er mit Hansen, doch seien dies unwesentliche Punkte. Das Wesentlischste sei die Anerkennung des Bacilus leprae als Ursache der Krankheit." Hansen 1886: 44.

[652] "Wenn alle diese Versuche überhapt etwas beweisen, dann ist es jedensfalls nicht mehr als dass wir bisher kein Thier kennen, das leprös werden kann; sie beweisen aber nicht, dass die Uebertragung auf Menschen nich gellingen würde; mit Menschen können wir aber leider nicht experimentiren, jedenfalls nicht hier in Europa." Hansen 1886: 32.

[653] One year after going to Bergen, Albert Neisser visited a leprosarium in Granada, Spain, and continued his bacteriological investigations. In addition, physician E. Wagner sent a specimen from Dutch Guyana. Thus, in 1881 his argument that the bacillus was universal to leprosy was supported by samples from four Norwegian cases, three Spaniards and one patient from Dutch Guiana. The details on attempts at staining, cultivation and animal experiments were all aimed at explaining the process so that the results could be reproduced elsewhere. (Neisser 1881).

At the same time, the discussion of the bacillus made up less than three of the seventeen pages of Hansen's paper in Copenhagen. The bacillus was an addition to the case for contagiousness, not a decisive argument in itself. Like when making the case for stricter legislations in Norway, the bulk of Hansen's evidence consisted of individual and family cases, local hygienic and cultural conditions, analogies with other diseases, and that the build-up of a national leprosy apparatus coincided with a documented reduction in new cases. Based on the assumption that 100 lepers produced 10 new cases every 5 years, he showed mathematically that even partial isolation would lead to a reduction in the number of new cases. The model was then compared with the actual statistics from Norway, concluding that "the numbers show a quite constant reduction in new cases in the following five year period, about 9% to 8%".[655] Since the disease (increasingly meaning the pathogen, the bacillus) was universal, so was the mathematical model.

Not everyone was convinced. In the discussion that followed the presentation physician Démétrius Alexandre Zambaco Pacha, educated in Paris and working in Constantinople, argued that "the Mediterranean leprosy is not in absolute parallel to the one in the North (…) it might have more in common with the leprosy of the Orient".[656] He made no reference to the bacillus, but focused on the clinical diagnosis. Around the Mediterranean the proportion of anesthetic cases was larger and the disease produced changes in skin pigmentation. This fit better with clinical observations from India than the observations from Scandinavia. Seeing leprosy in

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[654] Neisser, A."Chronic Infectious Diseases oft he Skin". In: Ziemssen, Hugo Wilhelm von. (ed.) *Handbook of Diseases of the Skin.* 1885 [1884]: 318.

[655] "Es zeigen diese Zahlen ein ziemlich constantes Procentverhältniss der neuen Fälle in einem Quinquennium zur Zahl der Leprösen im vorigen, ungefähr 9% oder 8%". (Hansen 1886: 39.) When Robert Koch received the Nobel Prize in Medicine for his investigations and discoveries in relation to tuberculosis in 1905, he highlighted "the exceptionally instructive example of the fight against leprosy in Norway" to justify the value of partial isolation, and argued that the same lesson was applicable also to campaigns against tuberculosis. See: http://www.nobelprize.org/nobel_prizes/medicine/laureates/1905/koch-lecture.html.

[656] "La lèpre du midi n'est pas absolument pareille à celle du nord (…) un seul de ces sujets à quelque ressemblance avec la lèpre d'Orient". (Zambaco Pacha in: Hansen 1886: 40.) Interestingly, Zambaco Pacha's objections were not mentioned in the otherwise thorough review in *The British Medical Journal*, possibly because the reviewer did not understand French. See: "International Medical Congress … Proceedings of Sections". *The British Medical Journal*. August 30, 1884: 426.

different parts of the world as slightly different diseases did not initially lead him to question Hansen's findings – he merely declared them locally irrelevant.

Not only did the diagnosis differ, so did the etiology of the Mediterranean leprosy. Zambaco Pacha's argument was, typically, based on his own local experience telling him that leprosy appeared mainly in families. "I have never observed a single case of contagion."[657] When also his attempts at inoculation all failed, experience led him to conclude that in addition to heredity most cases developed spontaneously, possibly influenced by diet, climate and uncleanliness.

In the following decades Zambaco Pacha would continue to argue for this disease model. In his 385 page monograph *La contagion de la lèpre en l'état de la science* (1907), Zambaco Pacha argued that bacteriology, although an attractive theory, could explain neither how contagion happened, nor why numerous examples of people living "even in complete promiscuity" with lepers did not catch the disease. In his opinion, time was overdue for the bacteriologists to apologize for their speculative assertions.[658] While the monograph received a devastating review by the Tunis-based French physician G. Eichmüller in *Lepra Bibliotheca Internationalis*, Zambaco Pacha's publication is one of several examples of how different disease models continued to compete for hegemony well into the 20th century.[659] The review

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[657] "Je n'ai pas vu un seul fait de contagion de la lèpre."(Zambaco Pacha in: Hansen 1886: 43.)

[658] Eichmüller, G. "Zambaco Pacha: La contagion de la lèpre en l'état de la science". *Lepra Bibliotheca Internationalis*. 1907: 128. The book was followed by *L'Hérédité de la Lèpre* (1908), which argued for establishing leprosy colonies to separate the sexes and thus prevent the heredity which he held to be the the single undeniable cause of the disease. This conclusion was reached after decades of research. In the early 1890s, Zambaco Pacha had presented first a study of leprosy in Egypt, Palestine, Greek islands, Cyprus and Crete (Zambaco Pacha, Dr. *Voyages Chez les Lépreux.* 1891), and then the the French Mediterranean in a lecture for the Academy of Medicine in Paris. (Zambaco Pacha, Dr. D. A. *La Lepre dans le Midi de la France en 1893. Communication faite à l'Académie de Medecine le 9 mai 1893.* 1893). Among numerous honors, he received the Prix Monthyon from the Academy for his work. In addition to the International Medical Congresses Copenhagen in 1884 he presented leprosy in international conferences (general and dermatological) in London, Vienna, Rome, Paris, Moscow, Madrid, and Lisbon (Zambaco Pacha, Démétrius Al. *Anthologie. La Lèpre a travers les siècles et les contrées*. 1914: V-VI). At the International Leprosy Conference in Berlin in 1897 he argued that Morvan's disease and several other constitutional disorders were in fact variants of leprosy. (Zambaco Pacha. "Des rapports qui existent entre la maladie de Morvan, la Syringomyélie, la Sclérodermie, la Sclérodatylie, la Maladie de Reynaud, La Morphée des Contemporains, l'Aïnhum, l'Atripie musculaire progressive Aran-Ducheune, et la Léprose." *Mittheilungen 1897.* Bd. 2. 1897: 20-80).

[659] Competing disease models could exist within the same Empire. In the Dutch colonies, for instance, leprosy was in the middle of the 1880s considered non-contagious in the East Indies and a decidedly contagious disease in the West Indies. (Van Eyk, Sprenger. "The Netherland's Colonies". In: *Leprosy in foreign countries*. 1886: 188-190.)

also illustrates how contagion was the hegemonic position in the columns of this international journal. Without exception, all studies not arguing for the contagiousness of leprosy received negative reviews.

Those who recognized the bacillus as the core of the disease probably saw reports from afar as more relevant compared to those who saw the disease as locally or regionally distinct. But concurring that a bacillus was present did not necessarily entail complete agreement. Instead, the demonstration of its presence spawned numerous new disputes. Whether the bacillus was found inside or between the cells was the focal point of a debate between Hansen and physician Paul Gerson Unna from Hamburg that would last for a quarter of a century.[660] In 1886, two years after visiting Bergen, the French dermatologist Henri Leloir suggested that the bacillus could be spread via mosquito-bites or other insects, a theory that also would lead to both controversy and research efforts.[661] The same happened with experiments on animals, where apparently successful inoculations turned out to be difficult, if not impossible, to reproduce.[662]

Around the turn of the century Moscow-based physician W. J. Kedrowski would take Hansen's theory of the bacilli producing spores as the starting point for renewed cultivation attempts, and argue that the acid-fast bacillus observed through

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[660] Unna founded the semi-monthly *Monatshefte für Praktische Dermatologie* in 1882, and in 1884 he visited Bergen. Back in Hamburg he had his own private skin clinic where he also taught international students the latest clinical and laboratory techniques, as well as experiment with therapies. Unna's position was that the bacillus was mainly found in the lymph between the cells. This meant that the bacillus craved oxygen and could be treated by removing the oxygen through drugs or applying pressure. To Hansen and Neisser, who claimed that the bacillus was only found *inside* the cells, this strategy for treatment was meaningless.

[661] Donald H. Currie's dissections of 493 mosquitoes that under controlled conditions had fed on eleven leprosy sufferers at the Public Health and Marine Hospital Service on Hawaii in September 1910 was probably the largest such experiment. For an overview, see: Lebæuf. "Recherches expérimentales sur la valeur du rôle que peuvent jouer certains insectes hématophages days la transmission de la lèpre." *Lepra Bibliotheca Internationalis*. 1914 : Fasc. 1, originally published in *Bulletin de la Société de pathologie exotique*. 1912. See also: Benchimol, Jaime L. and Magali Romero Sá. "Adolpho Lutz and controversies over the transmission of leprosy by mosquitoes". *História, Ciências, Saúde - Manguinhos*, vol. 10 (supplement 1). 2003: 49-93.

[662] For an extensive review of animal experiments on mammals such as rabbits, dogs, apes, guinea pigs, rats, mice, cats and birds, see: Sugai, T. and D. Irisawa. "Gelungene Übertragungsversuche mit Lepra bei Säugetieren". *Lepra Bibliotheca Internationalis*. 1908: Fasc. 3. The largest experiment took place in San Francisco, California, between 1908 and 1911, and consisted of examining 200.000 rats for the naturally occurring 'rat leprosy'', a possible analog to human leprosy. The leprosy-like disease was found in lesions from 186 of the rats, giving a prevalence of one out of 1075. (McCoy, George Walter. "Observations on naturally acquired rat leprosy". *Lepra Bibliotheca Internationalis*. 1914: Fasc. 2, originally published in *Public Health Bulletin*. Washington, July. 1913).

the microscope was but one of several stages in the life cycle of the pathogen.[663] In general, however, the bacillus was not so much a topic in itself, but it was quickly entangled in other debates regarding the nature of the disease, with direct consequence first on legislation and therapies, and later also on diagnosis.

The bacillus was also made part of an already existing narrative based on historical records. In Britain the argument that the global distribution of the disease could be traced throughout history was introduced by William Munro, late Medical Officer to St. Kitts in the West Indies and member of the Pathological Society of London.[664] In his book *Leprosy* (1879), Munro traced the first written sources on the disease to Antiquity (whether it first occurred in Egypt, Africa, India or China remained an open question), and its subsequent introduction to Western Europe from Greece and the Middle East in early Medieval times. Its disappearance from Europe, he argued, was a direct consequence of 'lepers' were being kept strictly apart from the healthy. The disease was then introduced to the New World by the trans-Atlantic slave trade. In the late 1840s, leprosy was transported over the Pacific as a stowaway with the import of Chinese labor. This was also the case for Australia where the disease too, according to Munro, was introduced by the Chinese.[665] A central premise for the argument was that leprosy was the same disease throughout history and throughout the world. Munro frequently referred to reports on prevalence from physicians around the world. Again, relating to the results of researchers in other parts of the world was more relevant for the contagionists, than for those who, like Zambaco Pacha, saw the disease as locally distinct. Munro made no mention of the bacillus, but the bacillus would fit hand-in-glove with the narrative of a disease that throughout history had

[663] See i. e. Kedrowski, W. J. "Über die Kultur des Leprabacillus". *Lepra Bibliotheca Internationalis*. 1901: Fasc. 4, originally published in *Zeitschrift für Hygiene und Infektionskrankheiten*. No. 1. 1901. Kedrowski's bacteriological model had some supporters, see i.e.: Bayon, H. "The Present Position of Leprosy Research (Paper read before the Meeting of the British Medical Association at Cape Town 24. October 1912)". *Lepra Biblioetica Internationalis* 1914: 54. For an extensive (but by no means exhaustive) overview of cultivation experiments, see: Thompson, J. Ashburton. "Experimental Leprosy: A perspective". *Lepra Bibliotheca Internationalis*. 1914, Fasc. 1.

[664] Munro 1879.

[665] For an account of leprosy as a foreign threat (a 'yellow peril') in colonial Queensland, and links between leprosy and xenophobia, see: Robertson 1999. For a similar argument based on comparing the US leprosy policies on Hawaii and the mainland, see: Moran 2007.

spread around the globe. The bacillus did not create the argument, but certainly served to strengthen it.

On the other hand, contagion was not the only disease model that took as its starting point that the disease was the same all over the world. The prominent British physician Jonathan Hutchinson argued that those having extensive local experience "encounter some real disadvantage, in the risk that their attention may be unconsciously drawn too closely to circumstances which are, after all, only local."[666] Since his own experiences with the disease was limited to the few leprosy sufferers that ended up in London hospitals, as well as a visit to Norway in 1867, he argued that he was in a better position to produce an unbiased account of the 'whole picture' compared to those who had worked with the disease their whole life. Given that the disease was the same throughout the world and throughout history; his conclusion was, as I showed in Chapters 3 and 4, that the disease was not caused by contagion but eating badly cured fish.

> "I believe that the advance of Christianity, with its salt-fish feasts, and not the Crusades, was mainly conducive to the general prevalence of the disease in Europe during the Middle Ages; that its spread is always due to food and never to contagion, and its disappearance to an improved dietary, and not in the least to enforced isolation."[667]

According to this theory, the appearance of lepers in the same family or geographical area was explained by them having eaten the same poisonous food. It also explained the global prevalence: "Wherever a community is to be found which subsists chiefly on fish, there leprosy is present."[668]

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[666] Hutchinson, Jonathan. "Memoranda for Future Investigations as to the Cause of Leprosy". *Journal of the Leprosy Investigation Committee*. Vol. 1, 1890: 67-89, quote on p. 67. Throughout his career Hutchinson published about 1200 medical papers, and produced the quarterly *Archives of Surgery* (1890-1900). His list of presidencies in medical societies include the Hunterian Society (1869-1870), the Pathological Society (1879–80), the Ophthalmological Society (1883), the Neurological Society (1887), the Royal College of Surgeons (1889), the Medical Society (1890), and the Royal Medical and Chirurgical Society (1894-1896). He was also the Royal College of Surgeon's representative to the Leprosy Investigation Committee (1889-1896) For a brief biography, see: http: //www.whonamedit.com/doctor.cfm/887.html

[667] Op. Cit. 68.

[668] Op. Cit. 78.

Hutchinson first presented the 'Fish Hypothesis' in 1858, and would support it until his death in 1913.[669] By the Tenth International Medical Congress in Berlin in 1890, he had incorporated the bacillus in the theory as the identification of the poison that was found in the contaminated foodstuffs.[670] Further research, Hutchinson argued, should be to put fish under the microscope to search for the bacillus there. After his theory was blatantly ignored at the First International Leprosy Conference in Berlin in 1897, Hutchinson traveled to South Africa and India to interview lepers in search of proof for the theory. The results of the journey were then presented at the 71st annual meeting of the British Medical Association in Swansea in 1903, where he received a mixed response. Sir Patrick Manson, medical advisor to the Colonial Office was not convinced: "If fish was the medium by which the bacillus entered the body, how did it get into the stinking fish?"[671] George Pernet, who by then had taken over as the London-based editor of *Lepra Bibliotheca Internationalis,* was even less diplomatic: "According to Mr. Hutchinson apparently, lepers who deny ever having eaten fish are not to be believed, but their statement as to never having seen a leper must be taken as an incontrovertible fact."[672] Hutchinson did have some supporters, but in *Lepra Bibliotheca Internationalis*, where Hutchinson was listed as one of the founding editors at the front page of every issue, reports would accumulate from around the world showing no connection between leprosy and the eating of fish. Over time the theory would be increasingly ridiculed to the effect that Hutchinson instead chose to publish his theories elsewhere.[673]

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[669] For a typical defense of the fish-eating hypothesis, see: Hutchinson, Jonathan. "On Leprosy and its connection with the use of uncooked fish". *Mittheilungen 1897*. Bd. 2. 1897: 20-23.
[670] Hutchinson, Jonathan. "The causes and origin of leprosy". *Verhandlungen des Internationalen Medicinischen Congresses Berlin, 4.-9. August 1890*. Bd. 4, part 13. 1892: 43-49.
[671] Hutchinson, Jonathan. "Discussion in leprosy: Its etiology, histology and treatment". *Lepra Bibliotheca Internationalis* 1905: 202, originally published in *The British Medical Journal*, September 26, 1903.
[672] Op. cit.: 203.
[673] After George Pernet's devastating review of Hutchinson's 420 page monograph *On Leprosy and Fish-Eating. A statement of Facts and Explanations* in 1906, Hutchinson's publications were not referenced in *Lepra Bibliotheca Internationalis*. "All modern evidence points to this, that leprosy is the result of the invasion of the human organism by a micro-organism the bacillus of Hansen, from outside, and thus the disease is conveyed from man to man. Medicine has left behind it, for good be it hoped, the dialectical methods of medieval times and the old scholastic disputations of the Sorbonne, so humorously satirized by Rabelais. The publishers are to be congratulated on the get-up of the book." (Pernet, George. "Jonathan Hutchinson, London: On Leprosy and Fish-Eating. A statement of Facts and Explanations (Constable, London, 1906, pp. 420)". *Lepra Bibliotheca*

Proof and human experiments

Even for those convinced that the disease was contagious and caused by the bacillus, the lack of convincing and undispoted proof was problematic. To produce proof for the theory of contagion was the direct motivation for experiments on humans. Some of the experiments, such as the British-born German physician Eduard Arning's inoculation of a man sentenced to death for killing his lover's husband on Hawaii in 1886, were made public and widely discussed.[674] On the one hand the man did develop leprosy after the inoculation, on the other it turned out he had family members with leprosy, ate the same food as other 'lepers' and lived under the same conditions. Hansen's own human experiment in Bergen, though, did not receive attention although it had direct consequences for his ability to do research.

After corresponding with Robert Koch in the fall of 1879 regarding his failed attempts at producing leprosy in rabbits and cats, Koch suggested that these animals were immune to the disease. Hansen then inoculated a female patient at Pleiestiftelsen in Bergen suffering from anaesthetic leprosy. The experiment consisted of injecting her in the eye with a sample from a patient with the tubercular variant. The goal was to produce proof of contagion by using a person that beyond doubt was susceptible to the disease. As Hansen later explained in his police statement; "even though the test subject should suffer some, I chose a patient that had been a leper for several years and therefore would not cause a new disease. I especially considered both the great scientific and national importance of the question at hand."[675]

Internationalis. 1907, Fasc 2.) In 1909, Hutchinson presented his fish-eating theory at the second international leprosy conference in Bergen. From the published proceedings it appears it was simply ignored by the other attendees.

[674] Arning, E, "Eine Lepraimpfung beim Menschen und Demonstration einer Sammlung von Lepraabgüssen." *Verhandlungen der Deutschen Dermatologischen Gesellschaft, I, Congress gehalten zu Prag, 10-12/6, 1889*. Wien, 1889. See also: Tebb 1893: Chapter 3; Edmond 2006: 90-92; Bushnell, O. A. "Dr. Edward Arning. The First Microbiologist in Hawaii." *History of Science in Hawaii*. No. 3. 1967: 1-30.

[675] "Selv om vedkommende forsøgsobjekt skulde lide noget derved, når jeg til sådant valgte en patient, der allerede i flere år havde vært spedalsk og hvem jeg derfor med mit forsøg ikke kunde påføre nogen ny sygdom, og det, især som når jeg betenkte den store både videnskabelige og nationale betydning, afgjørelsen af det forutliggende spørgsmål vilde have." BSA. I.C.c.133. Byfogd og byskriver i Bergen, case number 99/1880. "Forklaring fra G. A. Hansen til Overrettssagfører Mowinckel", dated April 23, 1880. For details and assessments of trial, see: Vogelsang, Th. M. "Gerhard Henrik Armauer Hansen 1831-1912: The Discoverer of the Leprosy Bacillus: His Life and Work." *International Journal of Leprosy*. 1978: 257-332; Robertson, Jo.

After repeated complaints, the hospital minister August Grønvold took Hansen to court, referring to "the fear and bitterness that the medical experiment has produced in all the patients, an exasperation that can lead to unpredictable consequences".[676] In a Royal Decree dated April 17, 1880, Hansen's positions as physician and head of the national leprosy apparatus were separated, and six weeks later Hansen was sentenced in the Bergen Municipal Court to lose his licence to practice at the institution and pay for the costs of the trial. The verdict was in line with the recommendation from the Medical department. Hansen did, however, remain head physician for leprosy.[677]

After the verdict, Hansen's office was moved to Bergen Museum, and his production of new knowledge about leprosy could no longer be based on working directly with patients. In other words: The sentence limited his ways of working, and thereby the way of knowing the disease. The verdict made headlines in Norwegian newspapers.[678] But abroad, the sentence was not brought up in the later public debates regarding the Norwegian leprosy legislations, nor in at any of the conference proceedings or publications until rediscovered by Vogelsang in 1963.[679]

Although legislative texts were part of the knowledge that was circulated, as I mentioned in the previous chapter, the exact details on what went on behind the scenes were seldom mentioned. The physicians were selective in choosing what stories to share with colleagues. Knowledge circulation was thus only the tip of the

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"The Leprosy-Affected Body as a Commodity: Autonomy and Compensation". In: Ferber, Sarah and Sally Wilde. *The Body Divided: Human Beings and Human 'Material' in Modern Medical History*. 2011: 131-165; Vollset 2005: 74-79.

[676] "(…) den Frygt og Forbittrelse som samme medecinske Forsøg har fremkaldte hos dens samtlige Lemmer, en
Forbittrelse, der kan medføre uberegnelige Følger." Bergen State Archives: Kopibok for Pleiestiftelsen. Letter to the supervisory committee, dated November 21, 1879. In: Vollset 2005: 75.

[677] BSA: Byrettssak 99/1880: 5.

[678] The case was referred to on the front pages of *Bergensposten* (June 3, 1880), *Bergen Aftenblad* (June 5, 1880), as well as the Christiania-based newspapers *Dagbladet* and *Aftenposten*. Vollset 2005: 79.

[679] Vogelsang, T. M. "A serious sentence passed against the discoverer of the leprosy bacillus (Gerhard Armauer Hansen), in 1880." *Medical History*. 1963: 182-186. When Hansen in Berlin in 1897 presented an overview of inocculation experiments on humans, he did not include his own experiments. (Hansen, G. Armauer. "Uebertragung der Lepra von Mensch zu Mensch". *Mittheilungen 1897.* Bd. 1, II. Abtheilung. 1897: 1-5.) Hansen did not mention the case in his autobiography. (Hansen 1910). In 1973, Justice Knut Blom of the Norwegian Supreme Court concluded that the sentence was in accord with contemporary law and that the case marked the genesis of the principle of informed consent in Norwegian medical research. (Blom, K. "Armauer Hansen and human leprosy transmission. Medical ethics and legal rights." *International Journal of Leprosy*. 1973: 199-207. See also: Vollset 2005: 74-79).

iceberg, and underlines the need for local studies, as well as studies of transfers of knowledge that can investigate each connection more closely. Although it is safe to assume that not everything was written down, correspondence is a source that might give further details than this study has allowed.

Rematch in the British Empire

Back in Britain, Gavin Milroy ended the public defense of his 1867-report around 1880 due to ill health. In his absence reports from the colonies arguing that the disease was contagious, as well as explicit critiques of the 1867-report, were now printed without immediately being rebutted by the College of Physicians. Both Carter's 1884-paper, where he presented his own bacteriological findings to audiences in India, and his 1887-paper, where he again argued for Norwegian-style segregation, was referred to in the *British Medical Journal* and *The Lancet*.[680] The 1880s also saw several publications arguing for segregation aimed at a general public.[681] Still, it was events in Hawaii, especially reports that the Belgian Catholic priest Jozef de Veuster (better known as Father Damien) had caught the disease, which eventually would force a rematch in the British Empire.[682]

As I mentioned in the previous chapter, the first report a of dramatic increase of leprosy on Hawaii came in the mid 1860s when the German physician William Hillebrand sent a letter to physician Ch. Macnamara in Calcutta where he argued that the disease was in rapid increase on the islands. Macnamara in turn printed the letter

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[680] Edmond 2006: 82; Carter, Henry Vandyke. *Memorandum on the Prevention of Leprosy by Segregation of the affected*. Bombay Castle, March 5, 1884; Carter, H. V. *Observations on the Prevention of Leprosy by Segregation*. 1887. Both are available online from the National Library of Scotland: http://digital.nls.uk/indiapapers/browse/pageturner.cfm?id=74561442; http://digital.nls.uk/indiapapers/browse/pageturner.cfm?id=74559842.

[681] I. e. Lambert, Agnes. *Leprosy: Present and Past*. The Nineteenth Century. 1884; Wright, Henry Press. *Leprosy and Segregation*. 1885; Wright, Henry Press. *Leprosy: An Imperial Danger*. 1889. See: Edmond 2006: 84-85.

[682] Carter was among those who highlighted Damien as a clear example of direct contagion: "[W]ith respect to the seemingly direct communication of diseased to healthy persons living in intimate and prolonged contact with the affected, several such affirmative instances are known; one of the latest, and perhaps clearest, is being that of Father Damien at the Sandwich Islands." Carter 1887: 3.

as an appendix to his report *Leprosy* (1865). In it, Hillebrand claimed that the islands had 230 'lepers' among a native population of 67,000. More importantly, these were all new cases: The disease "cannot be traced farther back than the year 1852, or at most, 1848".[683] It had since spread rapidly, the first six cases in the immediate neighborhood of the first. This showed that the disease could not be hereditary but spread through contact. Again, contagionism did not depend on bacteriology or Hansen's discovery.

On Hawaii, Hillebrand's reports led to the passing of an Act to prevent the spread of leprosy in 1865. As historian Michelle Moran and others have shown, from 1866 more than a hundred leprosy sufferers and sent to the isolated 'leper settlement' of Kalawao on the island of Molokai very year.[684] Between 1881 and 1908, 3,940 'lepers' were apprehended.[685]

When the Royal College of Physicians in 1874 was asked by the Colonial Secretary to comment, they dismissed the observations as "lacking in precision, as well as deficient in details," pointing out that Hillebrand himself had arrived only in 1851.[686] The claim that the disease was introduced by Chinese only three years earlier was therefore considered hearsay evidence:

> The apparent rapid increase of leprosy began to be noticed, too, coincidentally in point of time with the establishment of the Queen's Hospital and Dispensary, the number of lepers who applied for relief at the hospital augmenting year by year. The truth may be that the presence of the leprous amongst the population at large was only more clearly perceived on account of their seeking relief in greater number at the hospital as this became better known.[687]

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[683] Macnamara, Ch. *Leprosy*. Calcutta, February 3. 1865: 53.
[684] Moran 2007.
[685] Brinckerhoff, Walter R. and A. C. Reinecke. "A statistical study of an endemic focus of leprosy". *Public Health Bulletin*. No. 33. 1910: 3.
[686] "Recent Official Correspondence relating to Leprosy". *British and Foreign Medico-Chirurgical Review*. 1874: 206-207.
[687] Op. Cit. In the late 1880s, Eduard Arning, the German physician who inoculated the Hawaiian murderer, examined a large number of skeletons found in burial caves "without ever finding the characteristic lesions of leprosy." ("Debate on Leprosy at the International Medical Congress, Berlin, 1890." *Journal of the Leprosy Investigation Committee*. No 2. 1890: 233-234.; *British Journal of Dermatology*. September 1890;

This did not put a stop to explanations based on contagion. In 1879 the reports from Hawaii was a case in point for William Munro, who withheld that what was unfolding on the islands in the Pacific was a repetition of what had happened in medieval Europe: "The spread of the disease has been like that of all epidemics at their commencement – fearfully rapid – and reminds us of its behaviour in Europe after the Crusades, when everything was in its favour. There were last year no less than 700 lepers in the Leper Settlement which has been established by the Government 20 miles from Honolulu".[688]

Already the year after Milroy died, in 1888, reports from the colonies as well as continued bacteriological findings led the Colonial Secretary to ask the College of Physicians if they still maintained that the disease was hereditary. In the reply the College acknowledged that this opinion had never been accepted by the general public of the colonies where leprosy was endemic, and set down a new Leprosy Committee to again investigate the pathology of the disease.

A month later, on April 15, 1889, Father Damien died of leprosy on Molokai.[689] The news soon reached other parts of the world through the popular press. In Norway, this was seen as a confirmation that Hansen had been right all along, and that the measures taken had avoided a disaster. In Britain it caused a public outcry. For some, it was proof that the disease indeed did pose a danger to Europeans; for some it was an argument for further support to missionaries and their selfless sacrifices. For yet others, it merely showed that the 'Biblical' disease still existed, and that the sufferers deserved better care.[690]

Verhandlungen des Internationalen Medicinischen Congresses Berlin, 4.-9. August 1890. Bd. IV, part 13. 1892: 45.)

[688] Munro 1879: 38.

[689] The Belgian Roman Catholic missionary Damien de Veuster (Father Damien) had worked among the eight hundred lepers in the secluded settlement Kalaupapa since 1873, and in 1884 he was diagnosed with leprosy. To missionaries, his death from the disease later became a powerful symbol of martyrdom, and religious self-sacrifice. See: Edmond 2006: 90-102; Moran 2007: 21ff; Gould 2005: 59-102.

[690] In the Prince of Wales' dinner speech at a fundraising for The National Leprosy Fund in London on January 13, 1890, he took the latter view. The death of Damien was a reminder that it was "imperative that something should be done, not only for the sake of the unhappy lepers themselves, but for t he benefit of the whole community." The pinnacle of the talk was a letter presented by a leper to Lord Lawrence (later Viceroy of India), probably from the mid-1850s, which underscored the desperation on behalf of those affected by the disease: "Hail, cherisher of the afflicted. Be it known to your enlightened mind that your devoted servant has

The most important outcome was the establishment of The Father Damien Memorial Committee with the Prince of Wales as its president.[691] As subscriptions and donations were raised, the Committee was soon renamed The National Leprosy Fund.[692] In addition to erecting a memorial to Father Damien, the formation of a fund for indigent British 'lepers' in the United Kingdom, and sponsoring two 'Leprosy Studentships', reassessing 'the leprosy problem' in India was established as one of the top priorities. Both the College of Physicians and the Royal College of Surgeons were invited to nominate members to a special Leprosy Commission to India, which made the College of Physician's Leprosy Committee obsolete.[693] Sparked to a large extent by events in Hawaii, the National Leprosy Fund would in the early 1890s be the driving force for a rematch over the question of etiology in the British Empire.[694] As I showed in the previous chapter, their conclusions in 1893 would be that although the disease could technically be classified as a contagious disease due to the presence of the leprosy bacillus, claims that the decline of the disease could be attributed to compulsory isolation was "entirely erroneous". Instead, the report repeated that the disease was in practice best addressed through sanitary improvements.

> Though in a scientific classification of diseases leprosy must be regarded as contagious and also inoculable, yet the extent to which it is propagated by these means is exceedingly small.

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been a leper for many years. My limbs have fallen off piece by piece; my whole body has become a mass of corruption; I am weary of life; I wish to die. My life is a plague and disgust to the whole village, and my death is earnestly longed for. It is well-known to all that for a leper to consent to die, to permit himself to be buried alive, is approved of by the gods, who will never afflict another individual of the village with a similar malady. I therefore solicit your permission to be buried alive. The whole village wishes it, and I am happy and content to die. You are the ruler of the land, and without your leave it would be criminal. Hoping that I may obtain my prayer, I pray that the sun of prosperity may ever shine on you." ("Account of the Dinner in aid of 'The National Leprosy Fund.'" *Journal of the Leprosy Investigation Committee.* No. 1, 1890: 14-20, quotes from pages 17-18.)

[691] The Prince of Wales traced his interest in leprosy to 1885, when he visited a leprosy asylum in Norway. (Abraham, Phineas S. "Introduction: Account of the Origin of the National Leprosy Fund". *Journal of the Leprosy Investigation Committee*. No. 1. 1890: 5-20. See also: Edmond 2006: 90-107.)

[692] For details, see: Edmond 2006: 92-99.

[693] The Commission had six members. The National Leprosy Fund nominated George N. Curzon, Under Secretary for India (chairman) and Edward Clifford; the Royal College of Physicians nominated Dyce Duckworth and G. A. Heron; the Royal College of Surgeons nominated Jonathan Hutchinson and N. C. Macnamara. In addition, an Executive Committee had the following members: Ferdinand Rotschild (chairman), F. Londin, Andrew Clark, James Paget, J. Fayrer, W. Guyer Hunter, Jonathan Hutchinson, E. Clifford, Algernon Borthwick, Edward Lawson and J. R. Somers Vine.

[694] Edmond 2006: 107-108.

> (…) Leprosy is indirectly influenced by insanitary surroundings, such as poverty, bad food, or deficient drainage or ventilation, for these by causing a predisposition, increase the susceptibility of the individual to the disease.
>
> Leprosy in the great majority of cases originates *de novo*, that is, from a sequence of concurrence of causes and conditions, dealt with in the report, and which are related to each other in ways at the present imperfectly known.[695]

In Bombay the question of etiology was up for a rematch even before the National Leprosy Fund got involved. After returning to Bombay around 1883, Carter continued to stay in touch with Hansen who forwarded him the latest official statistics from Norway. These provided the basis for reports Carter published in 1884 and 1887, where he again argued that the decline of leprosy in Norway was unexplainable "except upon the hypothesis of contagion, no other explanation of events seems possible".[696] All efforts to suppress the disease, Carter argued, should be based on this premise. According to Pandya, newspaper editorials in Bombay subscribed fully to the model of leprosy as contagious, and argued for legislations aimed at removing especially poor and vagrant leprosy sufferers from the streets.[697] Most of the local physicians, however, referred to Milroy and saw no scientific basis for contagion and segregation.[698] Leprosy in the vast Indian countryside was not an issue.

Towards the end of the 1880s, after pressure from the local elites who feared that the disease was dangerous, the Bombay government felt forced into action. In 1888 the City of Bombay passed a Municipal Act for preventing the spread of "dangerous diseases, such as cholera",[699] but attempts at applying the legislations to

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[695] *Leprosy in India. Report of the Leprosy Commission in India, 1890-91*. Calcutta. 1893: 4. This was a compromise which not everyone agreed to. In a special foreword, five out of ten members of the British Commissioner's Executive Committee categorically denied that leprosy was contagious altogether: "We dissent from the opinion expressed in the body of the Report of the Special Committee on the subject of the Contagion of Leprosy, and from the recommendations founded on that opinion." (op. Cit: 11, Signed: Andrew Clark, James Paget, J. Fayrer, W. Guyer Hunter and Jonathan Hutchinson).
[696] Carter 1884: 6.
[697] Pandya 2001: 181.
[698] Pandya 2001: 189.
[699] Pandya 2001: 146.

leprosy proved of no avail. There were simply no facilities for what given the chronic nature of the disease in fact would mean life-long quarantine.[700]

The following year, in 1889, a 'Draft Leper Bill to make Provision for the Isolation of Lepers and the Amelioration of their condition' was suggested by the central government in Calcutta. The bill was aimed at isolating beggars and vagrant lepers in 'retreats', to be funded by the local authorities. It also opened for voluntary isolation as extensive as local circumstances allowed. Those who escaped were subject to re-arrest, regardless of whether the admission was voluntary or not.[701] After pressure from London the legislation was postponed pending the report of the Commission.[702] The immediate effect of the Leprosy Commission to India was thus to delay action on the ground.

The underlying problem in India was not just opposition from London, but lack of funds. This was made clear in Bombay already when the Sassoon Infirm Asylum in Poona at the beginning of the 1880s asked the government to cover the increased expenses connected to accepting 'lepers'.[703] While on one hand referring extensively to Carter's arguments for segregation, the local government in Bombay declined the request for funds, explaining that this would lead to "a very great cost – so great that however much His Excellency the Governor in Council may wish to alleviate the sufferings of lepers, he cannot undertake to meet the whole of the expenditure with due regard to other claims on the public revenues."[704] Instead, Carter's reports were again circulated "with the hope that they will attract the attention of wealthy philanthropists both in this Presidency and elsewhere."[705]

Despite new legislations drafted in 1895 and enacted in 1898 banning lepers from certain trades, the hands-off approach would be the dominant method dealing

[700] Pandya 2001: 148.
[701] Pandya 2001: 150-1, Appendix 5.2.
[702] Pandya 2001: 144-153, Appendix 5.3
[703] General Department, Bombay. Resolution No. 4900. "Establishment of Leper Asylums." Dated Bombay Castle, December 7, 1882: 1. Addendum to Carter, Henry Vandyke. *Memorandum on the Prevention of Leprosy by Segregation of the affected.* 1884.
[704] Op. Cit.: 5.
[705] Op. Cit.: 5.

with leprosy in India.[706] According to the national leprosy censuses, more than 100.000 individuals were affected, and even on a provincial level the problem was simply too large to handle. The Indian government perceived other diseases (especially cholera), as well as famine, as larger and more imminent threats. As I have shown earlier in this chapter, cholera alone claimed more lives every single year than the total number of individuals affected by leprosy. Sanitary measures and calls for philanthropy became the 'one size fits all'-proscription which also fit the economic capacities of the colonial rule. In Bombay a donation from Sir Dinshaw Petit and support by Municipal Commissioner H. A. Acworth, led to a leper asylum with capacity for 200 patients being opened in 1890 (within a few years expanded to 300 beds), the Homeless Leper Asylum.[707] Similarly, the vast majority of the leprosy institutions established in India were financed mainly through private donations, either local philanthropists or more commonly by foreign missionaries. These measures reached but a fraction of those affected by the disease. By 1920, when the census showed 109.094 known lepers in India, the Mission to Lepers cared for 4.707 'leper inmates'; 2.068 leprosy sufferers were admitted to Government or Municipal Asylums, while 1.021 persons were in Native State Asylums.[708]

Taking the question of etiology up for a rematch, and thereby postponing action on the ground, was not the only impact of the National Leprosy Fund. As part of his duties as secretary of the Fund, Phineas S. Abraham established a medical journal, *Journal of the Leprosy Investigation Committee* (1890-1891). In addition to a thorough presentation of the background for the National Leprosy Fund, Hutchinson's fish theory made up the main paper in the first issue in 1890. Editor Phineas S.

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[706] For the full text of "An Act to Provide for the Segregation and Medical Treatment of Pauper Lepers and the Control of Lepers Following Certain Callings", see: Pandya 2001: Appendix 5.4.

[707] Abraham, Phineas S. "Remarks on Leprosy in the British Empire". *The British Medical Journal.* November 13. 1897: 1411. The name was changed to Acworth Medical Asylum in 1904; in 1955 to Acworth Leprosy Hospital. It is currently a municipal hospital that provides outpatient leprosy diagnostics and therapeutics, in addition to training of physicians. It also has a medical museum. See http://www.theacworthleprosymuseum.org. The genesis and early administration of the asylum is detailed in Pandya 2001: Chapter 9.

[708] *The Mission to Lepers. Report of a Conference of Leper Asylum Superintendents and Others on the Leper Problem in India.* Cuttack, Orissa Mission Press. 1920: Appendix IV and V. The number of patients includes Ceylon and Burma, who were not enumerated by the leprosy census for India. Thanks to Kathleen Vongsathorn for sending me digital copies of this report.

Abraham sent copies of the journal to numerous researchers in the world. The replies, which in turn were printed in later editions, included letters from Norway (G. A. Hansen), Hawaii (A. Lütz), Hong Kong (Patrick Manson), South Russia (Münch), Madeira (Jno Keene; J. Goldschmidt), New Brunswick (J. C. Taché), New Orleans (Ohmann-Dumesnil), Finland (L. W. Fagerlund), Iceland (W. T. McCormick), New Zealand (R. H. Bakewell) and British Guiana (Dr. Castor). In addition to providing local information on prevalence as well as how 'the leprosy problem' was handled in different locations, the columns had discussions on etiology and vaccination, as well as abstracts of publications and debates first published elsewhere. While the journal was a short-lived affair, it can be seen as the first attempt at organizing leprosy as a global concern through the means of a medical journal. Many of those who contributed were invited to attend the Berlin conference in 1897.

As I showed in the previous chapter, the Berlin conference concluded by passing resolutions acknowledging the bacillus as the cause of the disease, and that the measures taken in Norway should be the model for other countries to mimic. On behalf of the British delegation, Phineas S. Abraham presented no less than sixteen Acts passed around the empire, proof that the Empire was not completely passive. On the other hand, in accepting the resolution he emphasized that the measures taken should be "applicable to the special social conditions of the districts" and that "the isolation which be propose[d] is really only partial".

> Accepting as we do that the cause of leprosy in Hansen's bacillus [sic], that leprosy must therefore be regarded as an infective disease, and that the human tissues – so far as we know at present – are the only soil in which these bacilli can develop, – it seems only logical that the isolation of diseased persons is the proper scientific method to prevent the dissemination of leprosy. But from other practical consideration, it appears to me that attempts at the *absolute* isolation of lepers must be in many places quite futile. Where the lepers are few and public opinion unanimous as to the necessity for their exclusion, such measures might be possible, but where the cases are numerous and not desirous of being separated from them, harsh measures of isolation and segregation become impossible – their chief result being the concealment of cases.

> (…) There are still many medical authorities of eminence who strongly disapprove of stringent legislation; and until medical opinion is unanimous and our knowledge of the pathology and etiology more complete, it will be futile to expect all governments to adopt strong measures. The question of legislation, indeed, must be left to the individual governments. This is especially so for the British Empire – where the social and political conditions in her different colonies and dependencies vary so very much.[709]

In Norway, being presented as a model for others to copy was taken a sign of honor. In Britain, it led the Colonial Secretary Joseph Chamberlain to ask the Royal College of Physicians again if they saw reason 'to change their previously expressed opinion' regarding segregating lepers in the Colonies. Despite contagionism still being the challenger to the status quo among the British medical elite, a consensus from an international congress was hard to ignore.

In 1898 The Royal College of Physicians concluded for the first time that Milroy's 1867-Report had been ill-founded:

> That the communicability of Leprosy, by direct or indirect means, from Lepers to the healthy, must now be accepted as an established fact, the evidence in support of this belief being conclusive: and that there is no evidence of the disease arising or spreading in any other way.[710]

As Pandya has pointed out, no physicians from India attended the international leprosy conference in Berlin. Although the medical authorities in London stopped insisting that the disease was not contagious, this had few consequences when it came to actual policies. The prospect of segregating a hundred thousand individuals or more was unrealistic. Leprosy was never very contagious, and compared to other diseases the burden of leprosy was relatively mild. Over decades the health system had been primed towards sanitary improvements, and leprosy was simply not

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[709] *Mittheilungen 1897*. II Abtheilung. Berlin. 1897: 196-197. See also reports on the Leprosy conference in *The British Medical Journal.* 1897: 1196, 1409-1414; Rosenthal, O. *V. Internationaler Dermatologen-Kongress abgehalten in Berlin vom 12.-17. September 1904.* 1. Band. 1904.
[710] *Leprosy Committe Report.* December 6. 1898, in Edmond 2006: 108-109.

considered a threat that needed to be singled out. The funding of leprosy institutions continued to rely on philanthropy, especially missionaries.

Conclusion

The main difference between the debates in Norway and British India revolved around whether leprosy demanded special intervention. In the 1850s, the Norwegian state built institutions and started registering people affected by the disease. The disease was considered to be hereditary, and state involvement was primarily aimed at distributing the economic burden caused by the disease. Hansen's initiative for new leprosy acts was based on the premise that the disease was contagious, but was seen as continuation of existing practices that did not demand new investments.

In the British Empire, however, the Royal College of Physician's report from 1867 established a 'hands off' policy which would remain the official doctrine of the Empire until the end of the century. From the mid-1870s Carter argued that the Norwegian system had led to a reduction in new cases, but his efforts to introduce similar measures in British India were met with a cold shoulder. The colonial government did not have the capacity to invest in leprosy prevention, and compared to the burden caused by other diseases leprosy was a minor problem.

The bacillus was introduced to the debates in the 1880s. New bacteriological methods for staining tissue samples, and bacillus being identified as the cause of other diseases were vital to making the bacillus relevant, but neither in Norway nor in India was the bacillus itself at the center of the argument. Apart from its mere presence, too much was unknown about its role in the pathology of the disease.

The circulation of knowledge did not trump local experience, rather the actual exchanges show that circulation was an active and selective process both in deciding what local knowledge should be disseminated to a wider scientific community and in deciding what experiences from other places was considered locally relevant. When appropriating knowledge from elsewhere, it seems the researchers put most emphasis on their own experiences. They were more welcoming to reports that reinforced the

views they already held than reports which pointed to other conclusions. The more an actor had invested in publicly defending a particular disease model, the less likely he was to change his mind in light of new evidence. This explains why several radically different disease models, all claiming to be universally applicable, continued to exist side by side.

Impacts on local or national practices depended on the relative position of the one presenting the arguments. In Norway, calls for segregation came from the administrative head of the national leprosy campaign. From this position, Hansen managed to bypass the medical elite in the capital of Christiana and convince Parliament to pass the leprosy acts of 1877 and 1885. In India, the argument was presented by a provincial physician as a challenge both to the increasingly powerful sanitary department and the authority of the medical elite in London. There are strong indications that this impeded Carter's chances of promotion. Not all actors shared the same relations to funding bodies like the state, and the social and political circumstances differed from place to place. These mechanisms were not limited to Norway and British India, or the specific period between the 1870s and the 1890s.

The appropriation of knowledge from afar was also shaped by the actual situation on the ground. Despite more than 100.000 people being affected by the disease, in India leprosy was a minuscule threat to public health compared to the staggering death tolls claimed by especially famine and cholera.

Circulating knowledge can be seen as conscious efforts to overcome these local differences. All the actors I have focused on in this chapter actively published, which indicates that they saw their own experiences and conclusions as relevant to others. They also referred to works produced by other actors, and although the interpretations differed, they clearly saw research conducted elsewhere as relevant. Placing local experiences in context with findings from elsewhere was increasingly important in justifying the credibility of a report.

The resolutions from the first international leprosy conference in Berlin in 1897 concluded that leprosy was contagious. The appropriation of these conclusions differed. In Norway, this was but a confirmation that the already established system

was well founded, in London it led to yet another rematch on the contagiousness of the disease. When a number of international experts had reached a consensus, their arguments were hard to ignore. The topic for the following chapter will be the attempts to organize the circulation of knowledge on an international scale.

6. Connecting the world of leprosy

In the fall of 1896, the American physician Albert S. Ashmead announced in several medical journals that he had corresponded with Gerhard Armauer Hansen, and that the government of Norway would soon issue official invitations to an international congress of leprologists to be held in Bergen the following year.[711] Ashmead's goal for the congress was to form a permanent international committee to dictate leprosy policies and channel funds to leprosy institutions in the 'civilized world'. However, his announcement was premature: Hansen was unsure of the merit of the scheme, and when he instead was approached by a competing group of European physicians working for a different vision of international leprosy collaboration, he withdrew his support. What were these visions, how were the conferences planned, and how did they find their form? How were the conferences received?

This chapter will investigate the interlinked roles of meetings, correspondence and medical journals in the circulation of knowledge about leprosy between the mid-1890s and the Great War. How did knowledge move from place to place? What efforts were made to organize the circulation of knowledge in this period? What were the impacts of these efforts?

The first part of the chapter will outline the general background for the international conferences, and then investigate how the leprosy conferences came about. Next, I will investigate the tradition of medical journals, particularly the quarterly *Lepra Bibliotheca Internationalis* (1900-1914). What practices could the founders of this specialized publication draw upon when it came to medical journals? What was the content of the journal, and what was its role in the circulation of knowledge regarding leprosy?

Finally, I will discuss how the various arenas for circulation of knowledge were connected by examining a particular research project that argued for a link

between leprosy and cancer. The case shows how knowledge made available through circulation itself could give rise to new research topics. It also demonstrates how discussions on new knowledge took place not only in the columns of *Lepra Bibliotheca Internationalis*, but how this journal can be seen as the tip of the iceberg for research being published and discussed in a range of medical journals, as well as in meetings in medical societies and official reports. Finally, the case shows how different actors could have fundamentally different understandings of the status not just of reports published in other parts of the world, but of publications published in the past.

The rise of international congresses

In the historiography, the need for international cooperation and coordination in matters of health has been linked both with industrialization, urbanization, increased international travel and commerce, and the successive waves of cholera epidemics that hit Europe from the 1820s and 1830s.[712] From 1851, International Sanitary Conferences aimed at standardizing quarantine regulations to stop the spread of cholera, plague, and yellow fever were arranged more or less every fifth year.[713] As David Fidler has put it: "The nature of the problem – diseases spreading across borders through international trade and travel – demanded international cooperation."[714]

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[711] Ashmead, Albert S. "Congress of Leprologists: Letter to the Editor." Dated New York, August 4, 1896. *The Journal of the American Medical Association.* August 15, 1896: 387. A similar invitation was published in *The British Medical Journal* on October 3, 1896: 974, titled "Proposed International Congress of Leprologists".

[712] See i.e. Bynum, W. F. "The rise of science in medicine, 1860-1913". In: Bynum, W. F., Anne Hardy, Stephen Jacyna, Christopher Lawrence and E. M. Tansey. *The Western Medical Tradition, 1800 to 2000.* 2006: 221-228.

[713] Howard-Jones, Norman. *The scientific background of the International Sanitary Conferences 1851-1938.* World Health Organization. 1975. The following International Sanitary Conferences were held in Paris (1859), Istanbul (1866), Vienna (1874), Washington (1881), Rome (1885), Venice (1892), Dresden (1893), Paris (1894), Venice (1897), Paris (1903, 1912, 1926 and 1938). For a list of diseases discussed at each conference, see: Bynum 2006: 223.

[714] Fidler, David P. "From International Sanitary Conventions to Global Health Security: The New International Health Regulations." *Chinese Journal of International Law.* Vol. 4, No. 2. 2005: 329.

In addition to the sanitary conferences, which aimed at giving policy advice, there were conferences aimed at discussing the latest medical research. The largest were the series of international medical congresses that started in Paris in 1867 and lasted until 1938.[715] The medical conferences had a clear leadership who were responsible both for the practical arrangements and publishing proceedings of the papers and the oral discussions. On the one hand, the international conferences grew out of local or national meetings mainly in Europe; on the other hand they provided exemplary models for further local or national meetings. The number of attendees grew steadily, and had by the 10th meeting in Berlin in 1890 reached almost 6000. The conferences were increasingly criticized for being a victim of their own success; they had grown too large to be of any use.[716] As more people got organizational experience and found others with similar interests, as well as from plain necessity,[717] the conferences were divided into more manageable and specialized sections. From the 1880s different specializations started arranging their own international congresses, such as the First International Congress of Dermatology and Syphiligraphy arranged in Paris in 1889 where leprosy was among the topics discussed.[718]

International conferences were not limited to medicine. In 1907, *Union of International Associations* (UIA) was established with its seat in Belgium by internationalists Henri La Fontaine and Paul Otlet to enhance collaboration between

[715] The following conferences were held in Florence (1869), Vienna (1873), Brussels (1875), Geneva (1877), Amsterdam (1879), London (1881), Copenhagen (1884), Washington (1887), Berlin (1890), Rome (1894), Moscow (1897), Paris (1900), Madrid (1903), Lisbon (1906), Budapest (1909) and London (1913).

[716] 5,737 physicians attended the congress in Berlin. For a typical critique arguing that the number of participants made it hard to facilitate discussions or establishing new contacts, see: Hanssen, Klaus. "X. internationale medicinske kongres i Berlin". *Medicinsk Revue.* Vol. 7, No. 9. 1890: 273-276. "In conclusion one can hardly judge the success of the congresses base don the number of attendees; one is perhaps closer to the truth by describing the benefit as inversely proportional to the number of participants (…). The scientific reunions have undoubtedly had a prominent importance for international scientific work, and the desire to meet with scientists from other nations will undoubtedly continue in the future ensuring there will be more such meetings; it only boils down to finding the arrangement which will make them the most fruitful." (Hanssen 1890: 276).

[717] Weisz, George. *Divide and conquer: a comparative history of medical specialization.* 2006: 229.

[718] The Congresses of Dermatology and Syphiligraphy continued with meetings in Wien (1892) and London (1896). In Paris (1900), the conference was part of the 13th International Medical Congress, before again going 'independent' in Berlin (1904), New York (1907), and Rome (1912). After the disruption caused by The Great War, the Eight International Dermatology Congress was arranged in Copenhagen in 1930.

international associations and to serve as a center for documentation.[719] Although their lists were not complete, UIA document the overwhelming increase in the annual numbers of international congresses in the second half of the 19th century (Figure 2, below).[720]

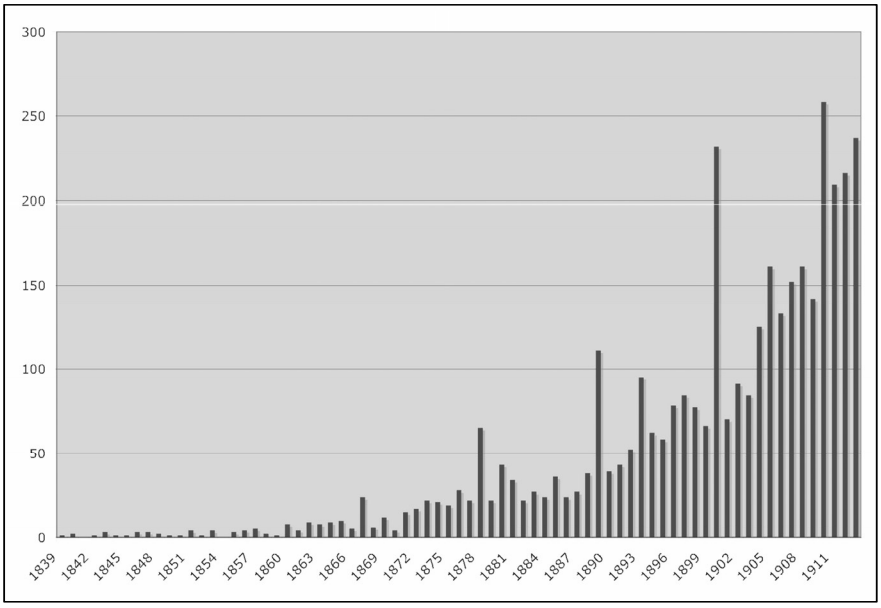

FIGURE 2. Union of International Associations. *Les congrès internationaux de 1681 à 1899. Liste complète*. 1960; Union of International Associations. *Les congrès internationaux de 1900 à 1919. Liste complète*. 1964.

When the schemes for arranging an international leprosy congress were hatched, it was both a continuation of a long tradition of international medical conferences and in line with concurrent trends of increasing specialization.

[719] For more on the Union of International Association, see: http: //www.uia.be/
[720] The table in Figure 2 was compiled by Stijn Van de Perre for the Third International Research Conference of the Voluntary Action History Society arranged at the University of Liverpool in 2008, and is used with permission.

The congress that never was

As historian Shubana Pandya has shown, the first international leprosy conference was the outcome of two competing schemes.[721] Gerhard Armauer Hansen, who after the death of Danielssen in 1894 became the primary reference point for most foreigners interested in leprosy in Norway, was invited to join both schemes. In the following I will investigate the opposing proposals for a leprosy conference. What were the visions for organizing leprosy internationally? How were the conferences planned, and why did only one of them succeed?

The first scheme for an international conference emerged in correspondence between the physicians Albert Sidney Ashmead and Jules Goldschmidt in December 1895.[722] Ashmead was from Philadelphia and since 1882 he had a private practice in New York. Goldschmidt was from Mainz in Germany, and had since 1869 worked as superintendent for the lepers at the Lazaretto Hospital in Funchal at the Portuguese Island of Madeira.

In 1890 Ashmead began to publish on leprosy in Japan, where he from 1873 to 1876 had been Foreign Medical Director and lecturer at the Imperial Japanese Hospital in Tokyo.[723] In the spring of 1895 he received national attention in the United States after reporting in the columns of *The Journal of the American Medical Association* on an anonymous New York resident with leprosy, publicly refusing to

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[721] Pandya 2003: 161-177.

[722] Letter from Goldschmidt to Ashmead, dated Paris 26.12.1895 (CPP: MSS 2/0029, Box 2: Ser. 2.1: Goldschmidt, Jules). Exerpts were printed in: Ashmead, Albert S. "Proposed International Congress for the suppression of leprosy". *Journal of Cutaneous and genito-urinary diseases*. July. 1896: 280-282. See also: Ashmead, Albert S. "What a leprosy congress should be", *The Lancet*, August 14, 1897: 414; Ashmead, Albert S. "Proposed International Congress of Leprologists", *The British Medical Journal,* October 3, 1896; Ashmead, Albert S. "The Leprosy Congresses". *Medical Record.* January 9, 1897: 53; Pandya 2003: 161-177.

[723] Ashmead got the position in Japan after attending to Prince Adzuma, brother of the emperor of Japan, who in 1873 was a student at the United States Naval Academy at Annapolis, Maryland. "Ashmead, Albert S." *National cyclopedia of American Biography*, New York, 1905. (CPP: MSS 2/0029, Box 1: Ser. 1); Ashmead, Albert S. "Tuberculosis and leprosy in Japan. A study in ethological pathology". *The Journal of the American Medical Association*. August 15, 1891: 254-262; Ashmead, Albert S. "Leprosy in Japan - Intermediary Host Function in its propagation". *Journal of Cutaneous and genito-urinary diseases*. June. 1890: 220-227; Ashmead, Albert S. "Syphilis and leprosy in Japan". *Journal of Cutaneous and genito-urinary diseases*. June. 1890: 233.

notify the Board of Health.[724] This was published in a context where the question of what to do with the 'lepers' who had been isolated on North Brother Island outside New York since 1891 was still an open question.[725] Ashmead's case was discussed in the chairman's address at the 46th Annual Meeting of the American Medical Association at Baltimore in May 1895, as part of the argument that the federal government should establish a leprosy colony on the US mainland.[726] In the following years Ashmead published numerous papers on the history of leprosy in the Americas; how the disease was introduced, how it was spread, and what had been done to combat it. His main argument was that left to itself, the disease would inevitably proliferate and get out control.

Goldschmidt's research was mainly based on his own experiences at Madeira, and focused on treatment and prevention.[727] In 1891, Goldschmidt had been among those taking part in the tuberculin trials (see Chapter 3). Three years later he published a survey of leprosy globally, which argued that leprosy was a threat originating in 'uncivilized countries', especially India and China. The disease needed to be stopped through segregation of cases, a stern denial of entry to all diseased subjects, and surveillance of all suspected individuals.[728]

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[724] Ashmead, Albert S. "A puncture with a bone of a living fish in the West Indies, followed by leprosy in a Caucasian subject". *The Journal of the American Medical Association.* March 16. 1895: 396-397; Ashmead, Albert S. "Leprosy Case – Where it should be reported". *The Journal of the American Medical Association.* April 13. 1895: 563.

[725] For more on the Board of Health in New York's controversial decision to isolate suspected lepers on North Brother Island, see i.e.: "The Chinese Lepers Secluded". *The New York Times.* August 16. 1891; "The Lepers at Bellevue". *New York Times*. October 19. 1897.

[726] Montgomery, Liston Homer. "Chairman's Address". *The Journal of the American Medical Association.* August 31. 1895: 346-347. It would be more than two decades before a federal institution was established on the US mainland. See: Moran 2007: Chapters 3 and 4.

[727] Mittelmaier, Carl and Julius Goldschmidt. *Madeira und seine Bedeutung als Heilungsort: Nach* vieljä*hrigen Beobachtungen geschildert***.** 1885; Goldschmidt, Julius. *Die Lepra auf Madeira*, 1891. Goldschmidt sent a copy of the book to Ashmead in 1895.

[728] Goldschmidt, Julius. "Zur Aetiologie und Prophylaxis der Lepra". *Berliner klinische Wochenschrift*. No. 7. 1894. It was in this paper, which Goldschmidt also sent to Ashmead in December 1895, that the idea of an international congress was first voiced. See also, Goldschmidt, Julius. "Vorschläge zur Verhütung und Unterdrückung der Lepra". *Mittheilungen.* Bd. 2. 1897: 16; Pandya, Shubhada. "The first international leprosy conference, Berlin, 1897: the politics of segregation". *História, Ciências, Saúde – Manguinhos.* Vol. 10 (supplement 1). 2003: 164-165. Goldschmidt also took part in the first tuberculin trials on leprosy, see Chapter 5.

The two found each other after Ashmead sent a letter to Goldschmidt containing two studies he wanted feedback on. One argued that leprosy, goitre and dwarfism were signs of degeneration and "evidences of the dying out of a race", the other argued that leprosy was introduced to the Americas after Columbus. The papers were mainly based on information and images gathered through correspondence with physicians and anthropologists around the world. Goldschmidt's reply, with copies of his own publications attached, was printed in *The American Anthropologist* and *The Journal of the American Medical Association.*[729] This was Ashmead's modus operandi, his approach to science. He corresponded with experts and published updates in medical journals, before finally compiling the research into independent publications.[730]

When promoting the idea of an international congress, Ashmead would use the same approach: Write letters to physicians, diplomats and heads of states – and publish the replies as letters to the editor of various medical journals.[731] While he repeatedly stressed that the scheme was Goldschmidt's idea, and the two coordinated

[729] Ashmead's two papers were: "Racial Degeneracy in America: Goitre and Dwarfing", published in the *University Medical Magazine* January, 1896, and "Pre-Columbian leprosy" printed as a 6-part series in *The Journal of the American Medical Association,* April-June 1895, and as special reprint later that year. Goldschmidt's reply (CPP: MSS 2/0029, Box 2: Ser. 2.1: Goldschmidt, Jules) was printed in *The American Anthropologist*, Vol. IX. June 1896: 219 and *The Journal of the American Medical Association.* 1896: 993. As Michel Moran has shown, the connection between leprosy and race would remain central to US discussions on leprosy for decades. Moran 2007.

[730] This way of producing knowledge was not new. In 1860-1862, for instance, after returning from studying leprosy in Norway, the German physician Rudolf Virchow published five papers on the history of leprosy in *Archiv für pathologische Anatomie und Physiologie und für klinische Medizin* ("Virchow's Archive") under the heading "Zur Gesichte des Aussatzes und Spitäler, besonders in Deutschland" (1860: 138-162, 273-329; 1861: 43-93, 1862: 166-198, 459-512). The first paper consisted of an overview of available literature, along with a list of questions on present and past leprosy and leprosaria, with a request for other physicians to contribute. This request was also printed other medical journals, such as *Deutsche Klinik*, April 25, 1860: 159-160, *The Medical Times and Gazette*, July 28, 1860: 93-94, and *The Lancet*, July 23, 1860. The four follow-up papers were based on the replies. See: Rather, I. J. *A commentary on the medical writings of Rudolf Virchow: based on Schwalbe's Virchow-Bibliographie, 1843-1901*. 1990: 116-121.

[731] In addition to *The Journal of the American Medical Association*, Ashmead published 'letters to the editor' in *American Antropologist, Journal of Cutaneous and genito-urinary diseases*, *Medical Record* (New York), *The Magazine of Medicine* (Atlanta), *The British Medical Journal* and *The Lancet.* The practice of publishing the same paper in different journals to ensure a larger audience, contrary to the editors' claims to exclusivity, was controversial. See: Ashmead, Albert S. "The Editor and the Contributor". *The Journal of the American Medical Association.* December 4, 1897: 1177.

extensively behind the scenes, the American soon took over as the driving force in recruiting potential participants.[732]

From the outset, Ashmead and Goldschmidt's brainchild had global and political ambitions. The two physicians shared the view that leprosy was caused by a bacillus, spread by contact from man to man, that there was no efficient cure, and that the only way to stop the threat was through compulsory segregation. The argument was backed up by references to experiences from around the world. In Russia, "during the period in which it was generally believed that the disease was not contagious, the leper hospitals were closed; leprosy increased apace." In the Sandwich Islands (Hawaii) the lack of efficient segregation had caused the disease to "spread in an unheard of manner. In 1865, there were 230 lepers in a population of 45,000. In 1881 and 1882, there were 4,000 lepers. (…) In Colombia, while there was no isolation, leprosy increased in forty years from 400 to 27,000 cases."[733]

The solution, the two agreed, was strict segregation in order to stamp out leprosy already existing, and international regulations to avoid the introduction of new sources of contagion through migration. Probably inspired by the international sanitary conferences, the means to achieve this "philanthropic undertaking" was to arrange a congress for expert delegates representing governments. The task of the congress would be to establish a permanent committee to channel funds, support institutions and dictate policies.

> The delegates of the different governments will form an international committee, to be permanently active. Funds will be raised by that committee in all civilized countries, and applied to the support of leper asylums in those countries where either the willingness or the capacity to help those institutions is wanting or insufficient. All problems concerning leprosy will be submitted to that international committee.

[732] In the two years leading up to the Berlin conference in 1897, Ashmead received 28 letters from Goldschmidt, most of them regarding the planned congress. (CPP: MSS 2/0029, Box 2: Ser. 2.1: Goldschmidt, Jules (i-iii)).
[733] Ashmead, A. S. "Antileprous Serum". *The Journal of the American Medical Association.* January 27, 1897: 181.

> It is hoped that by this common and universal effort against the dreadful scourge it may, in a comparatively short time, be wiped off from the surface of the world.[734]

One of the first to be invited to join the scheme was the Norwegian physician Gerhard Armauer Hansen, whom Ashmead and Goldschmidt both admired as the discoverer of the leprosy bacillus, "the greatest of leprologists".[735] In a letter quoted by Pandya, Ashmead stated that "A Leprosy Congress without Hansen would be a play of *Hamlet* with the Prince of Denmark left out".[736] That Hansen by the mid 1890s had a unique status was reflected also elsewhere, such as in the review of Hansen and Carl Looft's textbook *Leprosy: in its Clinical and Pathological Aspects* (1895) in *The America Journal of the Medical Sciences.* The review opened as follows:

> The name of one of the authors of this book, Armauer Hansen, the discoverer of the bacillus of lepra, is in itself sufficient guarantee that something more than a compilation may be expected, and in this respect we have not been disappointed. Dr. Hansen has for more than twenty years been engaged in studying leprosy in Norway and elsewhere, and the good work that he has accomplished entitles him to be listened to with respect. His words are authoritative.[737]

Hansen had worked as a physician at the Pleiestiftelsen leprosy hospital in Bergen since 1868, and in 1875 he was appointed chief physician for leprosy. As I showed in the previous chapter, Hansen was the architect behind the increasingly stern national leprosy legislations passed in 1877 and 1885, which combined surveillance and segregation either at home or in one of four state institutions.[738] According to Ashmead and Goldschmidt, Norway was the only country which took the threat of leprosy seriously, and they held the segregation policy responsible for the dramatic

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[734] Ashmead, Albert S. "Congress of Leprologists: Letter to the Editor." Dated New York, August 4, 1896. *The American Medical Journal.* 1896: 387. The same argument was also presented in Ashmead, A. S. "What a leprosy congress should be". *The Lancet*. August 14. 1897: 414.
[735] Ashmead, Albert S. "The Object of the Berlin Leprosy Conference". *The Journal of the American Medical Association*. February 27. 1897: 425.
[736] Ashmead to Falcao, 6.9.1896 in: Pandya 2003: 166.
[737] "Review: Leprosy: in its Clinical and Pathological Aspects." *American Journal of Medical Sciences.* November 1895: 586.

reduction in the number of lepers. The Norwegian experience was a success other countries should mimic.

Ashmead had first contacted Hansen in 1894 as part of researching pre-Columbian leprosy.[739] In July 1896 Ashmead invited Hansen to be part of a 'provisional committee' consisting of Hansen, Goldschmidt and himself. After briefly discussing the matter with the director of the national medical service, Michael Holmboe, Hansen replied that it would be a good idea to arrange a meeting in Bergen, "the head-quarters of the story of leprosy".[740] According to Hansen, the purpose of the congress should be for foreign scholars to learn from the Norwegian experience: "we hardly had anything to learn from foreigners while we are capable of giving good information. (…) I beg you kindly to give me your opinion on this point so that the matter as soon as possible can be laid before the Norwegian government."[741] Ashmead interpreted the reply as though only minor practical arrangements were standing in the way before formal invitations could be issued. Immediately after receiving the response, he started spreading the news of the upcoming congress to be held in Bergen in 1897, both in letters to journal editors, physicians, politicians and diplomats.[742]

> It has seemed desirable to publish all over the world the fact that the government of Norway will next year probably convoke a congress of leprologists, and delegates from all civilized

[738] Vollset 2005: Chapter 5.

[739] Did leprosy exist in the Americas before Columbus ('in pre-Colombian times')? Based on the skeletal remains found in burial sites, Ashmead argued that there was no indication that leprosy existed in the 'New World' before being introduced by Europeans and their slave trade. Hansen replied with sending Ashmead photographs from Dr. Kaurin's collection of leprous preparations at Reknes Hospital in Trondheim, Norway, for comparison with bones of the Peruvian mummies. (Hansen to Ashmead, dated Bergen 2.2.1895, CPP: MSS 2/0029, Box 2: Ser. 2.1: Hansen, G. Armauer). One of the photographs, an image showing the disease's effects on the bone structure of a hand, was printed in *The Journal of the American Medical Association*. April 27, 1895: 632.

[740] Hansen to Ashmead, dated Bergen 9.7.1896 (CPP: MSS 2/0029, Box 2: Ser. 2.1: Hansen, G. Armauer). See also Pandya 2003: 167.

[741] Op. cit.

[742] Among the politicians Ashmead contacted were President Grover Cleveland of the United States, Governor General Lord Aberdeen of Canada, Emperor Wilhelm II of Germany and President José Diaz of Mexico. In addition he appealed to the governments of Great Britain, France, Italy, Spain, Mexico, the republics of South America, South Africa, Japan and China. (Ashmead, Albert S. *A Berlin! Open letter to all leprologists*. Dated New York, January 22, 1897 CPP: MSS 2/0029, Box 2: Ser. 5 (iv) "Leprosy")

> countries, especially from those which, by the suffering of their own people, are especially interested in the question of leprosy. Hansen, the discoverer of the bacillus, suggested that this congress should be held at Bergen, Norway (...) Norway and Hawaii have already expressed their willingness to send their delegates.[743]

In the following months, Hansen sent a series of letters expressing worry that Ashmead had misunderstood: No final commitments had been made and he was in no position to make pledges on behalf of his government. Norway was a poor country and Hansen was skeptical of Ashmead's goal of a permanent committee of officials.[744] In September he withdrew from any active part in the planning.[745] Ashmead then contacted the Norwegian government directly and demanded a final decision, and they in turn turned to Hansen. In November 1896 Hansen pulled the plug, stating that "the government did not find it convenient to convoke the said congress."[746]

According to Pandya, Ashmead's presumptuous announcement that the Congress would be funded by the Norwegian government was part of the reason why Hansen in the fall of 1896 opted out of the scheme. In addition, Ashmead's repeated emphasis that Norwegian lepers were responsible for one of the three foci of the disease which together made leprosy a public health problem for the US probably did not make collaboration very inviting.[747]

By Hansen's final withdrawal, Ashmead had received support for the scheme from seventeen leprologists from Berlin (Germany), Bogota (Columbia), Breslau

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[743] Ashmead, Albert S. "Congress of Leprologists: Letter to the Editor." Dated New York, August 4, 1896. *The Journal of the American Medical Association.* August 15, 1896: 387. A similar invitation was published in *The British Medical Journal* on October 3, 1896: 974, titled "Proposed International Congress of Leprologists".
[744] CPP: MSS 2/0029, Box 2: Ser. 2.1: Hansen, G. Armauer; Pandya 2003: 169.
[745] "I should best like if you and Dr. Goldschmidt alone took the task of convoking the Congress on your shoulders; but should you think it very desirable to have my name, you may use it, but on the condition that the Congress is only regarded as arranged by us. If the Congress then will meet in Bergen, I hope still that the Norwegian government will pay the business matters; the best would be if the participating governments each paid their tribute." Hansen to Ashmead, dated Bergen 4.9.1896 (CPP: MSS 2/0029, Box 2: Ser. 2.1: Hansen, G. Armauer). The letter was published in *The Journal of the American Medical Association*. February 27, 1897: 426.
[746] Letter from Hansen to Ashmead, dated Bergen 1.11.1896 (CPP: MSS 2/0029, Box 2: Ser. 2.1: Hansen, G. Armauer).
[747] Pandya 2003: 169.

(Germany), Canton (China), Cape Town (South Africa), Hawaii, Jamaica, Lisbon (Portugal), London (United Kingdom), New York (United States), Paris (France), Rio de Janeiro (Brazil), St. Petersburg (Russia), Tokyo (Japan), Tracadie (Canada), as well as officials from United States, Canada and Mexico. In addition, invitations had been sent to the governments of China, France, Germany, Great Britain, Italy, Japan, Russia, South Africa, the republics of South America, Spain and United States.[748] Hansen's retraction was thus initially not perceived as a fatal blow, but with Bergen off the table, the question of location was brought to the top of the agenda. In a context where a growing number of physicians had experience arranging international medical conferences, two options were seen as especially viable: London or Moscow.

London had numerous successful medical conferences to look back on, including the Third International Congress of Dermatology in early August 1896.[749] There Goldschmidt had promoted the plans for an international meeting for the prevention of leprosy, and several of the expected participants had attended. Ashmead corresponded with the president of the conference, Jonathan Hutchinson, and through the US ambassador in London he secured an endorsement by Queen Victoria.[750]

The other option was Moscow, which was scheduled to host the Twelfth International Medical Congress in August 1897. Many of the leprologists were expected to attend. As the planning proceeded, continuous updates were printed in various medical journals.

> It was at first proposed to hold this congress at Bergen, Norway, out of compliment to Hansen. As the latter has renounced his claim, and left that question to be decided by those who will work for the scheme, and as there is among the present workers a division of opinion as to the comparative merits of London and Moscow, nothing can be said for the

[748] Ashmead, Albert S. *À Berlin! Open letter to all leprologists*. Dated New York, January 22. 1897. (CPP: MSS 2/0029, Box 2: Ser. 5 (iv) "Leprosy".)
[749] The First and Second International Congress of Dermatology were held in Paris (1889) and Vienna (1892). These grew out of the 7th International Medical Congress, hosted in London in 1881, which was the first in the series to have its own section on dermatology. After a successful repetition in Copenhagen in 1884, dermatology became a permanent feature of the international medical conferences.
[750] Letter to John Ridleley Carter, Secretary to the US Ambassador, from Arthur Bigge, Private Secretary to the Queen, dated London Sept. 21, 1896. *The Journal of American Medical Association.* October 24, 1896: 922.

present, as to the place where the congress will meet. Invitations will be issued, we hope, by one government to the other governments, to send each an official delegate. These delegates will form a permanent international committee, whose business it will be to formulate laws suitable for each country.[751]

All talk and no action?

Ashmead contacted more physicians than the seventeen who endorsed the scheme. Especially the goal of involving politicians and establishing a permanent committee was controversial.[752] One of those opposed was the Danish physician Edward Ehlers, who parallel to Ashmead and Goldschmidt worked on a rival plan for an international meeting of leprosy experts.[753] While Ehlers too believed that leprosy needed to be defeated through globally coordinated efforts, he had a different vision of how a world without leprosy should be achieved.

Ehlers had qualified in medicine in 1887, and in the following years he studied dermatology in Berlin, Breslau, Vienna and Paris. In 1891, he received a doctorate degree for a study on syphilis, and started working as a specialist of skin- and venereal diseases in Copenhagen.[754] Four years later his study of leprosy in Iceland won a prize in an international essay competition arranged by the British National

[751] Ashmead, Albert S. "Congress of Leprologists". *The Journal of the American Medical Association.* October 24, 1896: 922. See also: Ashmead, Albert S. "Proposed International Congress of Leprologists". *The British Medical Journal.* October 3, 1896: 973-974.

[752] In his letters, Hansen repeatedly pointed out that "As to your proposal of a permanent international committee, I do not clearly see what good such a committee should do." (Letter from Hansen to Ashmead, dated Bergen September 28, 1896. CPP: MSS 2/0029, Box 2: Ser. 2.1: Hansen, G. Armauer). Likewise, director Wm. Havelburg at the Bacteriological Laboratory at the Hospital Dos Lazaros in Rio de Janeiro, Brazil (who traveled to Europe in the fall of 1896), argued that: "Particular progress in our knowledge of leprosy and its treatment has, in the last years, not been made. Therefore, what are we going to do with a special congress? He who in this matter wants to talk about it, finds ample opportunity in the general congresses (Interior Medical Dermatologic Hygiene, or the International Congresses)." *The Journal of the American Medical Association*. July 25, 1896: 219-220.

[753] Letter from Ehlers to Ashmead, dated Copenhagen, September 18, 1896. *The Journal of the American Medical Association.* October 31, 1896: 968.

[754] Rosekamp, Erik. *Kraks Blaa Bog.* 1929: 238; Limon, C. "Edvard Ehlers". *Annales de Dermatologie et de Syphiligraphie*, Ser. 8, 1937: 358-461; Dansk Biografisk Leksikon: *Edvard Ehlers*. (http://www.denstoredanske.dk/Dansk_Biografisk_Leksikon/Sundhed/L%C3%A6ge/Edvard_Ehlers)

Leprosy Fund.[755] There he argued that the prevalence of leprosy in Iceland had declined due to "popular dread of contagion", but that the disease had increased after the Government in 1848 had decided to close down the four leprosy hospitals as the population falsely believed the disease was no longer a threat:

> Surely the hospitals played but a slight part in the combat against the disease, but it must not be left out of consideration that their existence indicated that the disease was still to be feared. On the contrary, the abolishing of the hospitals and the administration's throwing up the game could not help giving the population the idea, which Iceland's superior physicians persistently maintained, that the disease was on the point of becoming extinct spontaneously, and that it did no longer deserve any interest.[756]

Ehlers agreed with Ashmead and Goldschmidt that the disease was contagious, that isolation of dangerous cases was necessary to protect the healthy, and that Norway provided the gold standard for official leprosy control.[757] He also agreed that the fight against the disease called for international cooperation. But Ehlers' goal was not a congress to produce a permanent supranational committee or international laws. If the politicians were interested they could attend, but for science the chief imperative was to gather knowledge. In line with this, Ehlers' vision was a conference to work out a scientific consensus among experts in the field. As he wrote in a letter to Ashmead: "We do not want to make a congress of leprology, it is a conference which takes place between renowned leprologists. The governments are invited to send delegates, but

[755] Ehlers, Edward. "Conditions under which Leprosy has declined in Iceland". In: Newman, Ehlers, Impey. *Prize Essays on Leprosy*. The New Sydenham Society, London. 1895: 151-189. Ehlers was also involved in organizing the leprosy asylum Laugarnes on Iceland opening on October, 1897, and an asylum at St. Croix at the Danish West Indies in 1903. Both were financed by the Danish Odd Fellows society.
[756] Ehlers 1895: 187.
[757] For Ehlers it was important that the approach was 'humane', echoing an argument first made by Danielssen and Boeck some 50 years prior: "I am for isolation according to the Norwegian system, that is, the isolation of the diseased, who are incapable of taking care of themselves, and whose presence among other persons constitutes a real danger for the well. But I am not an admirer of the barbarians of the Middle Ages, who burned and imprisoned with horrible ceremonies the poor lepers, segregating them from their families and their friends." Ehlers, Edvard. "The Leprosy Questions". *The Journal of the American Medical Association*. December 18, 1897: 1282, translated from French in the journal *Janus*, July/August 1897.

these delegates are not to have in the conference a special position."[758]

Based on taking part in discussions among leprologists in Europe, both through studying, reading publications and regularly attending medical conferences, Ehlers knew that there were competing models among the medical authorities on the nature of the disease. As I showed in the previous chapter, the findings of the British Leprosy Commission in India from 1893 concluded that though the disease was technically contagious, it must be addressed through improvements in the sanitary surroundings. Ehlers' co-winner in the British National Leprosy Fund essay competition in 1895, the British physician George Newman, argued against contagion based on a comparative study of the history of the disease.[759] At the Third International Congress of Dermatology in London in August 1896, where Ehlers also attended, *The British Medical Journal* reported that the discussion which followed the session on leprosy "was very animated, and turned on the contagious and non-contagious character of the disease."[760]

While Ashmead corresponded and saw the letters he received as inherently public and therefore worth publishing, Ehlers recruited support mainly by meeting colleagues face to face. To Ehlers, letters were private. Medical journals should disseminate results of research, they were not part of the decision making process.

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[758] Letter from Ehlers to Goldschmidt dated November 16, 1896, then forwarded to Ashmead. (CPP: MSS 2/0029, Box 1: Ser. 2.1: Ehlers, Edvard). See also: Ashmead, Albert S. "The Object of the Berlin Leprosy Conference". *The Journal of the American Medical Association*. February 27, 1897: 425.

[759] "The whole weight of historic evidence is opposed to the supposition that contagion was the cause of the spread of the disease, and that segregation was therefore the cause of its decline." (Newman, George. "On the History of the Decline and Final Extinction of Leprosy as an Endemic Disease in the British Islands". In: Newman, Ehlers and Impey. *Prize Essays on Leprosy*. The New Sydenham Society, London. 1895: 1-150, quote on p. 91.) Newman's method was to compare the diet and sanitary conditions in medieval Britain and contemporary China, India and Iceland – places where the disease still existed. His conclusion was that "the disease being diffused neither by contagion nor heredity, has *under favourable hygienic circumstances* a tendency to die out. Hence, the decline and final extinction of endemic leprosy was due, not to segregation, but to this general tendency under favouring circumstances, viz. to a general and extensive social improvement in the life of the people, to a complete change in the poor and insufficient diet (...) and to agricultural advancement, improved sanitation and land drainage." (Newman 1895: 109, italics in the original.)

[760] "Third International Congress of Dermatology". *The British Medical Journal*, August 15, 1896: 410-411. Although the 'contagionists' were gaining terrain, prominent physicians such as the president of the congress, Jonathan Hutchinson, withheld that the only model which could explain the global distribution of the disease was consumption of badly cured fish. Hutchinson was also founder and honorary secretary to The New Sydenham Society, which published Ehlers prize essay on leprosy in 1895. (Hutchinson, Sir Jonathan. *The New Sydenham Society: Retrospective Memoranda*. 1911.)

Two months after the congress in London, in the beginning of October 1896, he traveled to Berlin to discuss the idea of arranging an international leprosy conference with colleagues there.

In Berlin, leprosy was already on the agenda after worrying reports of an 'outbreak' of leprosy in Memel. Among politicians, the fear of leprosy as a threat from the east was growing. When Ehlers arrived, Robert Koch, respected bacteriologist and director of the Royal Prussian Institute for Infectious Diseases, had just returned from undertaking a survey of the reported leprosy epidemic on behalf of the Prussian government.[761] He quickly agreed to join Ehler's scheme for an international conference. In addition to his bacteriological research, Koch had extensive experience from attending and organizing conferences, and was well known among European physicians.

Ehlers also recruited Oscar Lassar, who was equally well known among the European leprologists. Lassar had his own private clinic for skin diseases in Berlin and was one of the founders of both *Monatshefte für praktische Dermatologie* (1882) and the *Berlin Dermatological Association* (1886). In 1890 he had been the general secretary at the International Medical Congress in Berlin (where Koch had presented Tuberculin, see Chapter 3). Three years later Lassar left the *Monatshefte* and founded the competing *Dermatologische Zeitschrift*. In 1894, Lassar took the initiative for an international fundraising to set up a memorial in honor of the recently deceased Norwegian physician Daniel Danielssen, resulting in donations from 175 individuals and associations in Europe, Russia and the United States.[762] Like Ehlers and Goldschmidt, Lassar had also taken part in the heated discussions on the contagiousness of leprosy in London in August, 1896.[763]

Agreeing that the disease was contagious, caused by a bacillus and sharing

[761] Koch, Robert. *Die Lepra-Erkrankungen im Kreise Memel*. Jena, 1897. First published in *Klinisher Jahrbuch*. Vol. 6. 1897: 239-253.

[762] The money was used for a commemorative plaque which over the over the entrance to the leprosy hospital Pleiestiftelsen No. 1 in Bergen, which Lassar unveiled. Fourteen of the thirty physicians present at the funeral would later attend the Berlin conference. (Lassar, Oscar. "Zur Erinnerung an Daniel Danielssen". *Dermatologische Zeitschrift.* Vol 2, No. 5. 1894: 534-544.). The plaque is still on display over the entrance of the building in Bergen, now named "Norsk Medisinsk Fødselsregister".

Norway as a central point of reference, they then approached Hansen who agreed to form a second 'preliminary committee'. That the invitation came from three physicians Hansen had met face to face on earlier occasions probably made it more attractive to join this scheme than to cooperate with Ashmead, a physician he had never met, and with a plan he did not fully approve of.

Together, Ehlers, Lassar, Koch and Hansen quickly secured endorsement from the German Emperor for an international congress. Initially the conference was planned to take place in March 1897 under the presidency of Robert Koch, but after Koch was sent to South Africa to investigate cattle plague in December 1896, it was re-scheduled for October 1897.[764] When it became clear that Koch's African engagement would take longer than anticipated (he would stay abroad for 18 months), he was replaced by the renowned German physician Rudolf Virchow. Virchow was widely known for his innovative work on cellular pathology, comparative pathology and social medicine. Like Lassar, Virchow was also a well-known editor. Since co-funding the medical journal *Archiv für pathologische Anatomie und Physiologie und für klinische Medizin* in 1846, he had developed a wide network and was one of the first to review new ideas as they were submitted for publication.[765] In 1859 Virchow had visited Danielssen in Bergen after an invitation by the Norwegian government. The 'father of cell pathology' was also president at the International Medical Congress in Berlin in 1890, and honorary president of the Berlin Medical Association.

The members of the preliminary committee all shared the belief that leprosy was contagious and caused by a bacillus, but agreed that in order to bring the scientific society together, they had to find an angle which did not exclude their European colleagues who strongly opposed contagionism. In February 1897, Ehlers sent the first round of invitations to a conference on "the distribution and geographic extension of leprosy", in German, French and English. The conference was to be held in Berlin in October 1897 with support and assistance from the German

[763] "Third International Congress of Dermatology". *The British Medical Journal.* August 8, 1896: 355.
[764] "The leprosy congress". *The British Medical Journal.* November 21, 1896: 1528.

government.[766] By June 1897, a detailed program was ready for a week-long conference with more than fifty confirmed speakers.[767] In addition to sending the invitations directly to physicians, it was disseminated by Prince Hohenlohe, Chancellor of the German Empire, in official diplomatic dispatches to "all civilized states". The invitations were in turn forwarded to the national medical associations, before finally being printed in full or in abstracts in the medical journals.[768]

The final program was based on the returns to the call. The largest section was on the distribution of leprosy in a total of 35 countries. There were also more than twenty lectures on themes ranging from therapeutics to how best to run a leprosy institution; from pathological anatomy to culturing the leprosy bacillus; from competing etiological explanations to the history of the disease. A few of the papers addressed migration of 'lepers' and to what extent the rise of leprosy in the colonies constituted a danger to the colonizers at home and abroad. Hansen himself raised the question: Should segregation be voluntary or compulsory? Lastly, there were invitations to present objects for an exhibition with anatomical specimens, microscopy, photographs, maps and publications.

Upon learning that Ehlers had recruited Hansen, Koch and Lassar, as well as secured government sponsorship, Ashmead officially renounced his scheme in January 1897.[769] Still, as he learned more of the plans of his European colleagues, he was increasingly critical. First, he saw no reason to extend invitations also to physicians who publicly opposed the doctrine of contagion and isolation, positions

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[765] After the death of the co-founder Benno Reinhard in 1852, the journal was mainly referred to as *Virchow's Archives*.

[766] For the original invitation (in French), dated February 23, 1897, see: CPP: MSS 2/0029, Box 1: Ser. 2.1: "Ehlers, Edvard". Ashmead published the invitation, along with his reply, under the heading "The Leprosy Conference" in *The Journal of the American Medical Association.* March 27, 1897: 613.

[767] Ehlers, E., Hansen, G. Armauer, Koch, R. and Lassar, O. *Einladung und Programm zur Lepra-conferenz. Berlin. October 1897.* 1897. http: //da2.uib.no/cgi-win/WebBok.exe?slag=lesbok&bokid=lepra. From the outset the Europeans dominated. 36 of the 52 speakers were from European countries, four from Latin America, three from North America and the Middle East, and two from Asia, Russia and Africa respectively.

[768] See i.e. "Program of the Berlin Lepra Congress", *The Journal for the American Medical Journal,* July 10, 1897: 82; "A Leprosy Conference in Berlin". *The British Medical Journal.* July 24, 1897: 247. "The Leprosy Congress in Berlin". *The British Medical Journal.* August 21, 1897: 482.

[769] Ashmead, Albert S. *À Berlin! Open letter to all leprologists*, dated New York, January 22, 1897. (CPP: MSS 2/0029, Box 2: Ser. 5 (iv) "Leprosy").

Ashmead considered "un-scientific".[770] Second, the conference lacked plans for tangible outcomes. The problem, as Ashmead saw it, was not a lack of knowledge, but lack of action.[771] To discuss prevalence was a waste of time.

> I do not see that there is any use of further talk about the distribution and geographic extension of leprosy. All these things are on record, and nothing is easier for any person anxious to obtain some light on any subject connected with this question than to consult what all good libraries offer.[772]

That "all these things are on record" was simply false. In addition to occasionally being quite inaccurate when summarizing the views of others and being virtually unknown among the European leprologists, this and similar statements made it easy for the Europeans to dismiss Ashmead as unqualified. In the summer of 1897, Ehlers confronted Ashmead in the columns of the Dutch journal *Janus*, which in turn was translated and published in *The Journal of the American Medical Association*:

> "I see today with a certain displeasure that three of the publications, as numerous as they are little important, of Dr. Albert S. Ashmead have been inserted in the sixth issue of your paper (May and June). One of the publications, that entitled "Leprosy Overcome by Isolation in the Middle Ages," is absolutely useless. The author is pleased in this to attribute to me an opinion which I have never professed, *eide*, that the isolation of the lepers would be useless. *One will never find that opinion expressed in my work.* (…) Will you "kindly" ask Mr. Ashmead to quote in future the passages from my writings to which he refers, in order to avoid writing perfectly useless notes and attributing to me opinions which I do not have."[773]

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[770] "Our idea was, and has always been, that the only way to combat and eventually destroy the dreadful scourge was the isolation of lepers, enforced by law. In one generation, leprosy not being hereditary, the victory would be complete. (…) We declare at once that we are that we are contagionists, and that we do not believe that anything but absolute isolation can destroy a disease which afflicts nearly a million of human beings. (…) In this respect, we are intolerant." (Ashmead, Albert S. "The Object of the Berlin Leprosy Conference". *The Journal of the American Medical Association.* February 27, 1897: 425.

[771] "We want to *do something*; they come together to talk. (…) It is a conference to amuse us fellows, us leprologists, not at all to destroy the most horrible punishment that ever nature has put upon mankind." Ashmead, Albert S. "The Object of the Berlin Leprosy Conference". *The Journal of the American Medical Association,* February 27, 1897: 425-426, italics in the original.

[772] Ashmead, Albert. "Leprosy Conference" (reply), *The Journal of the American Medical Association.* March 27, 1897: 613.

[773] Ehlers, E. "The Leprosy Question". *The Journal of the American Medical Association.* December 18, 1897: 1283 (translated from *Janus* July/August, 1897).

That Ehlers' initial invitation was addressed at "colleagues whose works are well known" only made matters worse. Ehlers intention was probably to include the European colleagues who attended the scientific conferences, while excluding the numerous authors who in the early 1890s, especially in Britain, had published books on leprosy. Unaware of this context, Ashmead interpreted the phrase as an attempt by Ehlers to exclude him as an unqualified amateur. The public conflict that ensued was vicious, and peaked with Ashmead printing a 12-page pamphlet entitled "Ehleriana" where he questioned Ehlers' competence, experience and charged him with "stealing the idea" for the congress after the London meeting in 1896.[774] In the end, Ashmead refused to attend the meeting in Berlin, but the American did send several papers to be read along with proposals for resolutions. They were unanimously voted down.[775]

Interpreting the leprosy conferences

In total 151 persons signed up for the conference in Berlin 11-16. October, 1897. The list of participants included 44 delegates representing 22 governments. According to the published list of participants and staff, 122 attended in person.[776] The members of the 'preliminary committee' constituted the leadership. After a suggestion by Oscar Lassar, Virchow was elected President of the conference, with Hansen and Lassar as Vice-Presidents and Ehlers as Secretary in Chief. Adding prestige, the conference was opened by the Secretary of State representing the Government of the German Empire, Count von Posadowsky. The Imperial Chancellor, Prince von Hohenlohe, later gave a reception in honor of the members of the conference.[777] Within months, the papers

[774] "At that time our work was in full blast, known all over the world, and could not well be unknown to Dr. Ehlers, who knows how to read, and, I suppose, corresponds with some civilized persons. Any unprejudiced person will come to the conclusion that it was our work that stirred him into action." Ashmead, Albert S. *Ehleriana*. 1897: 10. (CPP: MSS 2/0029, Box 1: Ser. 2.1: "Ehlers, Edvard").
[775] *Mittheilungen 1897*. II Abtheilung. Berlin. 1897: 194.
[776] "Liste der Theilnehmer und Mitarbeiter". *Mittheilungen 1897*. I. Abtheilung. Berlin. 1897: V-IX. The list included postal addresses to make it easier for the attendants to keep in touch also after the conference.
[777] Special Correspondence. "The Leprosy Conference". *The British Medical Journal.* October 17, 1897: 1122.

and discussions at the conferences were published in two comprehensive volumes.[778]

For those not attending in person, the immediate outcome depended on where they received their information. *The British Medical Journal* published updates as special correspondence throughout the conference.[779] In an editorial the following week, they congratulated both the organizers and the German government on the achievement and predicted that the official transactions would become "one of the most important contributions yet made in the literature of the subject." For their readers, the main result was the adoption of a 'middle course' regarding leper segregation:

> While acknowledging leprosy to be a bacillary disorder, and in the category of the infective diseases, it recommends only such legal measures as have proved serviceable in Norway – such as the notification of cases, detention of vagrant lepers, and the isolation as far as possible, in their own homes, of such lepers as can be maintained with proper sanitary precautions by their own families.[780]

Ashmead's home journal, *The Journal of the American Medical Association*, concluded that the conference did not "appear to have developed anything specially sensational, either in its discussions or its findings or resolutions, as perhaps might have been expected to be the case." The main impression was that the general tone of the convention "was opposed to the excitement of any general alarm on part of the public as to the possible dangers from this disease, at least as far as the principal civilized countries are concerned". Stressing that many important questions were still

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[778] *Mittheilungen 1897*. Berlin. 1897.
[779] Editorial. "The International leprosy conference, Berlin". *The British Medical Journal*, 1897: 1122; 1197.
[780] Editorial. "The International leprosy conference, Berlin". *The British Medical Journal*. October 23, 1897: 1196-1197. Three weeks later, the recommendations weres reprinted in an issue dominated by the survey of leprosy in sixteen countries in the British Empire that Phinneas S. Abraham had presented at the conference. His argument was that any intervention must depend on local circumstances, that the Empire was not passive, and that the question of contagion was still not settled: "The social and political conditions in the different countries are so varied, that it is manifestly out of the question that any uniform or universal system for the management of lepers will be possible. (…) We must not forget that our knowledge of the etiology of leprosy is still far from complete; and although we may form theories and impressions from the imperfect data which are before us, we are not justified in dogmatically insisting upon conclusions which are practically only based upon such mere theories." (Abraham, Phineas S. "Remarks on Leprosy in the British Empire". *The British Medical Journal*. November 13, 1897: 1409-1414.)

unsettled, the conclusions boiled down to common-sense compromises: "The Congress probably acted judiciously in not endorsing any alarmist opinions, while yet advising reasonable precautions against possible contagion and spread of this loathsome disorder."[781]

To the readers of the Norwegian monthly *Medicinsk Revue,* the main outlet of the medical community in Bergen, Hansen reported that the conference had raised two new and interesting questions: Could leprosy be detected earlier by looking for bacilli in the nasal membranes, and why did some lepers become anesthetic and other tubercular? "It seems fortunate if we in this country could be able to answer these two intricate questions, it would increase our nation's honor."[782] *Monatshefte für praktische Dermatologie* chose to print extensive and non-commented abstracts from the daily discussions.[783]

That the interpretations could differ was well known. On the last day of the conference, the secretaries therefore wrote a condensed report in German, English and French, which they argued "will be especially desirable for those members who are delegated by their respective Governments, and who will have to make reports on the subject". The report concluded that "Every leper is a danger to his surroundings", but that the risk to family and fellow workers varied with the sanitary conditions. The bacillus was "accepted as the virus of the disease", while the theory of heredity "has lost ground in comparison with the now generally-accepted opinion of its contagiousness." As for treatment, it was concluded this had only palliative results. Therefore, emphasis must be put on prevention.[784]

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[781] Editorial. "The late leprosy congress". *The Journal of the American Medical Association.* November 13, 1897: 1021-1022. Two weeks later, Ashmead commented that: "I have no doubt, when the complete report of the transactions is published, you will find that the majority of the conference has not at all so sanguine and non-alarming opinion as you seem to imply. (Ashmead, Albert S. "The Late Leprosy Congress". *The Journal of the American Medical Association.* November 27, 1897: 1125-1126.)

[782] Hansen, G. Armauer. "Nogle leprasprgsmaal". *Medicinsk Revue.* 1897: 324-325. The questions were followed by an official resumé of the general conclusions in German. ("Resultatet af leprakonferensen i Berlin". *Medicinsk Revue.* 1897: 326-327.) In his memoirs, Hansen would later highlight the Berlin conference as the place where he understood that the discovery of the leprosy bacillus had made him famous. (Hansen 1910: Chapter 5).

[783] Jessner, Dr. "Internationale wissenschaftliche Leprakonferenz. Bericht." *Monatshefte für praktische Dermatologie.* 1897: 446-457, 500-508; 550-561.

[784] *Mittheilungen 1897*. II Abtheilung. Berlin. 1897: 190-194. For the full report, see: Appendix 2.

The report was printed in various medical journals, often with editorial notes. Even after reaching an agreement on resolutions and an official report, the interpretations would continue to differ.

Despite not attending, Ashmeads initiative did impact the agenda. Several of the physicians Ashmead had recruited ended up going, and the results of trials with Carrasquilla's serum therapy developed in Columbia and distributed by Ashmead before the conflict with Ehlers arose were discussed (see Chapter 3). And although Ashmead's own suggestions for resolutions were voted down by unanimous decision, they were the direct inspiration for resolutions being passed at all.[785] Ashmead's contributions were, however, not mentioned in any of the reports, and at the conference Ehlers was celebrated as its sole initiator.[786]

While the Fifth International Dermatological Congress in Berlin in 1904 was aimed at presenting the actions taken after the first conference (see Chapter 4), the second international leprosy conference in Bergen in 1909 would take its cues from 1897-conference. Headed by Hansen, a committee was established to make the preparations. The official invitations were sent by the government and spread through diplomatic dispatches, in letters to the attendants at the first conference, and printed in medical journals. At the conference the sessions would be organized in the same manner as in Berlin, with papers read and resolutions passed repeating the recommendations from Berlin (see Apendix 2).[787] Many of the papers were dedicated to establishing the prevalence of the disease in different states; the official opening

[785] Hansen, G. Armauer. "Die Isolierung der Aussätzigen und die dazu erforderlichen Massregeln", *Mittheilungen 1897*. II Abtheilung. Berlin. 1897: 162-166.
[786] *Mittheilungen 1897*. II Abtheilung. Berlin. 1897: 200.
[787] At a special meeting on August 20, 1909, after the conference was officially over, the British and Colonial delegates endorsed the resolutions and passed nine additional points renouncing the anti-contagious sentiments presented by their countrymen in Berlin 1897. The resolutions argued for compulsory notification and to "separate the leprous from the non-leprous by segregation in settlements or asylums" under strong regulations would provide "the most efficient means of mitigating the sufferings of the leper and of assisting his recovery, and at the same time [it] will produce a reduction and ultimate extinction of the disease". The resolutions were signed by delegates from Great Britain, Australia, Cape Colony, Natal, Transvaal, Orange River Colony, Rhodesia, British Guiana, Bahamas and Fiji. Bayon, H. "On the necessity or advisability of segregation in relation to the conditions and spread of Leprosy in South Africa at the present time; the measures to be provided for the prevention and cure of Leprosy: and the suitability of Robben Island as a place of detention for Lepers. Report by the Gouvernment Research Bacteriologist to the Department of the Interior." *Lepra Bibliotheca Internationalis*. 1914: 131-138.

was headed by King Haakon VII who, like the Imperial Chancellor Prince von Hohenlohe, attended a dinner for the conference members. While the first conference in Berlin involved 151 members and 44 delegates representing 22 governments, the second conference in Bergen attracted 168 attendees from 27 countries.[788] After the conference, the transactions were published in two volumes.[789]

Still, some things had changed in the period between Berlin and Bergen. Both an open invitation asking for input in the planning phase, and call for papers and the final program were published in the medical journal *Lepra Bibliotheca Internationalis*.[790] Afterwards, 52 of the papers read were printed in the journal. What was this journal, where did it originate and what was its relation to the conference?

Lepra Bibliotheca Internationalis

While the suggestion of establishing a specialist journal was discussed at the first international leprosy conference, no decision was made. In the following, I will investigate how the journal came about and what the ambitions were. I will then show that similar to international medical meetings, the number of international medical journals also exploded in the second half of the 19th century.

Behind the proposal for "an Archive of Leprosy for scientific and statistical reference" in 1897 were Edvard Ehlers, Gerhard Armauer Hansen, Ernest Besnier and Albert Neisser.[791] All but Ehlers had previous experience in establishing medical journals. Besnier, from 1873 director at the Hôpital Saint-Louis in Paris, was a co-founder of *Annales de dermatologie et de syphiligraphie* (1869). Neisser, from 1882

[788] The conference does merit closer study than what I have found time to. A good starting point for such an investigation would be the digital copy of the archival material from the organization committee available online: http: //da2.uib.no/cgi-win/WebBok.exe?slag=lesbok&bokid=leprakonferanse

[789] Lie, H. P. *II Internationale Wissenschaftliche Lepra-Konferenz: Abgehalten vom 16 bis 19. august 1909 in Bergen (Norwegen). Mitteulungen und Verhandlungen. Band I, Band II. London, Paris, Leipzig. 1909.*

[790] The open invitation was printed in *Lepra Bibliotheca Internationalis* 1908: 75, and was signed Armauer Hansen and H. P. Lie. The program, "Programme provisoire de la Deuxième Conférence Internationale Scientifique contre la Lèpre devant avoir lieu du 16 au 19 Août 1909 à Bergen, Norvège", was published in *Lepra Bibliotheca Internationalis.* 1909: 125ff.

[791] *Mittheilungen 1897*. II Abtheilung. Berlin. 1897: 160-161.

professor of skin- and venereal diseases and director at the University hospital in Breslau, was co-founder of the *German Dermatological Association* (1888), and co-editor of *Archiv für Dermatologie und Syphilis* and *Bibliotheca Medica*. Hansen from Bergen was a co-founder of the Norwegian *Medicinsk Revue* (1884). All regularly attended the international medical congresses. Besnier, Ehlers and Neisser had all attended the discussion at the international congress in London in 1896.

The proposed journal was part a proposal to appoint a 20-man international leprosy commission to be charged with arranging further conferences. The four researchers also suggested that the commission should prepare a general reference work on leprosy. However, after critical remarks by Rudolf Virchow who did not see the need for a specialized journal, only the point about establishing a committee to pave the way for a leprosy society received support from the attendees.[792]

The lack of an official mandate did not stop thirteen of the twenty members of the committee to move forward with the planned journal at their own initiative. Again, the proposal came from Ehlers, who for the lifetime of the journal would remain the main editor and personal owner of the rights to the journal. Co-editors were Ernest Besnier (Paris), Karl Dehio (Dorpat), Gerhard Armauer Hansen (Bergen), James Nevins Hyde (Chicago), Jonathan Hutchinson (London) and Albert Neisser (Breslau). On the front page of all issues were also the names of the 80 physicians who sponsored the publication. With but a few exceptions, all had attended the Berlin conference.

The main objective of *Lepra Bibliotheca Internationalis* was to keep up to date on what was published about leprosy elsewhere. This included papers in medical journals, as well as reports and legislations. As Ehlers put it in the introduction to the first issue:

> We understand that we need more emphasis on how the realization of the studies will be

[792] *Mittheilungen 1897*. II Abtheilung. Berlin. 1897: 160-161. The elected members were: Rudolf Virchow (Berlin, honorary president); Besnier (Paris), Hansen (Bergen), Neisser (Breslau), Ehlers (Copenhagen), v. Düring (Constantinople), v. Petersen (St. Petersburg), Campana (Naples), Kalindero (Bucharest), Lassar (Berlin), Mr. Jonathan Hutchinson (London), Dr. Phineas Abraham (London), Dr. Glück (Sarajevo), Dyer (New Orleans), Engel Bey (Cairo), Falco (Lisbon), Alvarez (Noholulu) and Köhler (Berlin)

> facilitated by considerable centralization of all efforts in a same international body, put together not only scientific papers, but also projects of administrative and legislative order, and inserting in document original work, counts, reports and analysis of all publications related to leprosy.[793]

The papers in the smaller section of original works, *Originalia,* varied greatly both in scope and topics. The larger section, *Relata*, was divided into seven fixed subsections. *Annuaria leproseriarum* featured annual reports from leprosy hospitals around the world, and served a dual purpose: First, it provided insights into the running of institutions. Second, it provided templates on how the annual reports could be written and what kind of information they could contain. *Historia* was a section for discussions on leprosy in the past, with a particular focus on Medieval Europe. How was leprosy introduced to an area, why did it spread, how did it decline? *Geographia* consisted mainly of case-studies from different locations. In addition to discussions of prevalence of the disease (some highlighting culture or diet, others race), this section also featured updates and controversies surrounding erection of new leprosy institutions. *Anatomia pathologica* referred mainly to diagnosis of individuals and discussions following the display of patients at meetings of medical associations. Somewhat overlapping, *Pathologia* focused on disease models, attempts at cultivation and laboratory experiments with animal models. *Therapia* concerned treatment; production, administration and effects of drugs, the effects of exercise, climate and diet, as well as tests on animals. The last category, *Prophylaxis, Legislatio etc*. provided updates and discussions on proposed and passed legislations around the world, often including the legal texts.

The boundaries between the different categories were blurred. Individual cases were often discussed in relation to treatment; country-studies in *Geographia* often included reports both on legislations and history. From 1906, professor Otto Hamann at the Royal Library of Berlin published annual lists of recent literature on leprosy, something which until then had been left to general publications such as Paul

[793] Ehlers, Edvard. "Préface". *Lepra Bibliotheca Internationalis.* 1900: 2.

Clemens von Baumgarten's *Jahresberichte über die Fortschritte in der Lehre von den pathogenen Organismen* (1885-1917). In addition, there were also several book reviews.

The structure of the journal, especially its emphasis on *relata,* was a consequence of what the editors assessed to be most useful for its readers. The decades surrounding the turn of the 20th century saw a host of medical journals which had leprosy as part of their area of interest.[794] The challenge for the leprologists was not to find a journal to publish in, but to keep up to date on colleagues elsewhere.

Over its fifteen years of existence, *Lepra Bibliotheca Internationalis* published papers from more than 220 different sources, including 145 medical journals, 54 different meetings and conferences, and 20 official reports. Some physicians seem to have been responsible for translating from other languages, like A. Grünfeld from Odessa who translated numerous Russian studies into German. Publishing abstracts was the main strategy from the editors to ensure that the journal should be considered the primary resource for leprosy-related research.

From 1900 to 1914, when war made continued publication impossible, *Lepra Bibliotheca Internationalis* was published quarterly in London, Leipzig and Paris (from 1909 also New York). The content was written in German, French and English. After the first few issues where some of the original papers had abstracts in the two other languages, there were no parallel translations. While the abstracts often were in a different language than the original papers, to take full advantage the reader had to master all three languages.

It is not clear what circulation *Lepra Bibliotheca Internationalis* had, or how widely read it was. A consequence of giving references to publications in other journals priority over original content was that the journal itself was seldom referred to. The number of authors indicates the size of the research community: Throughout its lifetime, papers by 523 different authors were printed in full or in abstracts. Only

[794] For instance: *Archiv für Dermatologie und Syphilis* (Wien, 1880), *Journal of Cutaneous and genito-urinary diseases* (New York, 1882), *Monatshefte für praktische Dermatologie* (Hamburg, 1882), *The Journal of the*

three authors published more than ten papers.[795] The ten most published researchers produced a total of 108 papers,[796] the top twenty 166 papers in total. Despite many physicians producing knowledge about leprosy occasionally, the number of actors studying leprosy consistently over time was limited: 328 researchers were only referenced once.

While unpretentious, the abstracts formed a genre in itself and can be seen as incidental steps towards standardization. In addition to usually (but not always) including references to the original reports, emphasis was on outcomes that could be generalized. In some cases this meant focusing on the methods used, in others the results achieved. Increasingly, what could be quantified was underscored at the expense of the particulars. As I discussed in Chapter 3 on treatment, this meant a shift from focusing on individual sufferers to studies conducted on larger groups of patients, usually in institutional settings. Only when the results seemed at odds with the observations of others, local context was highlighted as a potential explanation. More and more the abstracts were void of traces of the local context in which the observations or interventions had taken place. These characteristics of the genre of abstracts were not limited to *Lepra Bibliotheca Internationalis*. Rather, the phenomenon seems to be found in medical journals in general. I have not found a single medical journal from this period which did not publish abstracts of other publications in one form or another.

The ten most quoted journals in *Lepra Bibliotheca Internationalis* were:

American Medical Association (Chicago, 1883); *Medicinsk Revue* (Bergen, 1884), *Dermatologische Studien* (Hamburg/Leipzig, 1886), *Centralblatt für Bakteriologie* (Jena, 1887).

[795] J. Ashburn Thompson, Sidney (20), Edouard Jeanselme, Paris (15) and George Pernet, London (13).

[796] Edvard Ehlers (9), Gerhard Armauer Hansen (9), Isadore Dyer (9), MM. Kermogrant (9), Jonathan Hutchinson (8), Lebæuf (8), H. P. Lie (8).

TABLE 2: **Top ten journals publishing original papers presented as abstracts in *Lepra Bibliotheca Internationalis* (1900-1914).**

Number of papers	Originally printed in	Place of publication (established)
45	Journal of Tropical Medicine (and Hygiene, 1907)	London (1898)
34	Annales d'hygiène et de médicine coloniales	Paris (1898)
26	Tropical diseases bulletin	London (1912)
23	Bulletin de la Société de pathologie exotique	Paris (1908)
17	British Medical Journal	London (1840)
14	Archiv für Schiffs- und Tropenhygiene	Leipzig (1897)
12	Annales de dermatologie et de syphiligraphie	Paris (1869)
10	Deutsche Medizinische Wochenschrift	Berlin (1875)
10	Public Health Bulletin	Washington (1881)
8	Journal of the American Medical Association,	Chicago (1883)

As the list indicates, discussions on leprosy took place in several medical contexts. Leprosy was a tropical disease, a colonial disease, an exotic disease, a skin disease (dermatological), a matter of public health and a part of medicine in general. *Lepra Bibliotheca Internationalis* did not alter the practice of leprosy discussions taking place in other medical journals. What changed was that the subscriber got a way to keep reasonably updated on leprosy as a defined by 'the international scientific community'. It was not education or a specific organization that held the community together, but the journal itself.

The journals that were referenced all had their different histories and local contexts. Often they were backed by local or national societies, often they were established as a reaction to a perceived deficiency in the already existing journals. Hand in hand with the international conferences being more and more specialized, the new journals too specialized. The Norwegian monthly *Medicinsk Revue* ('Medical Review'), founded by the Bergen research community in 1884, is a prime example.

Medicinsk Revue was the third medical journal published in Norwegian. The oldest still in existence was *Norsk Magazin for Lægevidenskaben* ('Norwegian

Journal for Medical Science'), established in 1840.[797] By the late 1870s the journal was increasingly perceived to be elitist, exclusive and more interested in theory than actual medical practice. In 1881, it was supplemented by *Tidsskrift for Praktisk Medicin* ('Journal for Practical Medicine') aimed at bringing practical and useful information to the country's increasing number of medical practitioners.[798] For the research community in Bergen, the two medical journals based in the capital of Christiania were perceived to be provincial.[799] According to the medical community in Bergen, the two other Norwegian journals missed out on the groundbreaking research taking place elsewhere, mainly in Germany. This was the niche the three physicians Eduard Bøckmann, Gerhard Armauer Hansen and his brother Klaus Hanssen wanted to fill by *Medicinsk Revue*, subtitled *Minutes and translations from the Lungegaardshospitalet library*.[800] The editors had the Lungegaarden research hospital as their base, and the catalogue from 1904 shows that library subscribed to 148 different international medical journals.[801]

From the outset, the mission statement for *Medicinsk Revue* was to bring foreign medical knowledge 'home' to colleagues nationally; "through summaries make our medical profession aware of the most significant foreign medical literature".[802] Just as the later *Lepra Bibliotheca Internationalis*, the journal also had a section for original work, as well as summaries of the discussions taking place in their meetings.

The ambition of bringing foreign knowledge to a domestic audience was not a Norwegian invention. Already in 1836, John Conolly and John Forbes had established

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[797] The first Norwegian language medical journal was *Eyr – et medicinsk tidsskrift* (1826-1837). *Eyr* was published by a reading group of physicians in Christiania, which in 1833 evolved into the Medical Association of Christiania ('Lægeforeningen i Christiania') that founded *Norsk Magazin for Lægevidenskaben.* In 1847 the association was renamed *The Norwegian Medical Society* ('Det norske medicinske Selskab') which still exists. See: Larsen, Øivind. "Det norske medicinske Selskab som kunnskapsformidler." *Michael.* 2008: 96-101.
[798] Schiøtz, Aina. "Vårt fag – likeså meget en kunst som en vitenskap – Tidsskriftet 1881-1906". *Tidsskrift for Den norske lægeforening.* 2006: 9-13.
[799] For more on this community, see: Hammerborg 2009: 172-178.
[800] 86 out of the journals in the library catalogue were German, followed by the category "French, Italian and other" (23 titles), "Scandinavian, Danish and Finish" (22), and "English and American" (17).
[801] Dethloff, H. G. *Katalog over Lungegaardshospitalets bibliothek ved udgangen af aaret 1904. Bergen*. 1905.

British and Foreign Medical-Review or Quarterly Journal of Practical Medicine and Surgery dedicated to a similar endeavor. By the 1890s, most medical journals had sections dedicated to publishing abstracts and shortened translations of relevant content published elsewhere. Outside the medical conferences, medical journals were the main communication line for reaching colleagues domestically as well as abroad. First, it reached the immediate subscribers. Second, the results of the studies were printed as abstracts in other journals.

Leprosy and cancer

In addition to continuing the tradition of bringing updates from other journals, providing a common frame of reference, and help streamline the interpretation of the conferences and other events, how did *Lepra Bibliotheca Internationalis* impact the circulation of knowledge regarding leprosy? In the following I will investigate a particular research project linking leprosy and cancer. The case will show how different elements in the circulation of knowledge could work in concert, how knowledge production about leprosy often was motivated by research in other parts of medicine, which role *Lepra Bibliotheca Internationalis* and other medical journals had in circulating knowledge, and how the circulation made it possible to research leprosy without meeting anyone suffering from the disease. It also shows how fundamental debates on epistemology and the status of previous publications could be played out.

In 1911 and 1912, three papers on a possible connection between leprosy and cancer were published in *Lepra Bibliotheca Internationalis*. The researcher was physician Peter Munch Søegaard from Nordheimsund, a rural community by the

[802] "…et Tidsskrift, beregnet paa gjennem Referater at gjøre vor Lægestand bekjendt med det væsentligste af Udlandets medicinske Literatur." Bøckmann, Eduard, Hansen, G. A. and Hanssen, Klaus. "Program". *Medicinsk Revue*. Vol. 1, No. 1, 1884: 1-2.

Hardanger fjord in Norway.[803] Søegaard was not affiliated with any research institution or leprosaria, and his research relied solely on what was already available in medical journals, reports and correspondence. His work, in turn, was discussed in learned societies, reviewed and sparked new research. To relate to previous studies and to publish overviews of current literature was not new, but the case shows how it was possible to open new research topics by combining issues never discussed in the original papers. Søegaard's argument was that leprosy protected against cancer.

Like leprosy, cancer research was organized on an international scale in the first decade of the 20th century. However, the models differed. In 1904 a monthly international journal was established, *Zeitschrift für Krebsforschung*. Two years later an international conference for cancer research was held in in Heidelberg and Frankfurt, followed by the creation of *Die internationale Vereinigung für Krebsforschung* (*The International Association for Cancer Research*) in 1908.[804] Physicians in several countries, including Norway, established national cancer research committees.[805] One widely used research method was 'clinical statistics'; to organize large-scale projects with the goal of gathering as much standardized clinical data as possible, and then analyze it statistically. It was in this context Søegaard in 1909 published his first two papers on cancer, based on statistical analysis of patient records from the small county of Vikør (3.400 inhabitants). The results were

[803] Søegaard, Munch. "Lepra und Carcinom". *Lepra Bibliotheca Internationalis*. 1911: 92-97; "Die relative Krebsimmunität der Leprakranken. (Die Sekundärinfektionen, Kachexie)". *Lepra Bibliotheca Internationalis.* 1911: 172-180; "Weitere Untersuchungen über die Krebsstarblichkeit undet den Leprakranken". *Lepra Bibliotheca Internationalis*. 1912: 128-132. In the 1890s, Søegaard had been a physician in Wisconsin, USA (1892-1894) and Matadi, Kongo (1894-1895) before returning to practice as a physician in different towns in Norway.

[804] The initiative for the journal, conference and association came from the German cancer research committee established in 1900. The association promoted national memberships which committed the member countries to begin practical work against cancer. Britain (1902), France (1906) and the USA (1907) soon established their own national associations. By the outbreak of war, fifteen countries had joined the International Association for Cancer Research: Argentina, Belgium, Chile, Denmark, Greece, Italy, Japan, Netherlands, Portugal, Russia, Switzerland, Hungary and Austria. France left the association in 1913, United States in 1914.

[805] The Norwegian national cancer research committee was established in 1907. Its first initiative was to distribute 'cancer forms' to gather standardized clinical data on individual cancer cases, hoping to get 10,000 replies. By the end of 1911, only 36 percent for the physicians had replied to the call, returning in total 3.200 forms. "This is not in itself negligible, but far from the response we had hoped for." (Unsigned. "Den norske kræftforskning". *Medicinsk Revue.* 1911: 669-670). By the outbreak of war, the question of whether or not the Norwegian committee should join the international association was still up for debate. (Søegaard, Munch. "Samfundet og de kræftsyke". Supplement to *Tidsskrift for den norske Lægeforening*. No. 17. 1914: 80.)

compared to published statistics from cancer research in Britain, Germany, Sweden and Norway.[806]

Similar to leprosy, a major and contested question within cancer research was whether a disposition to cancer could be inherited, if cancer was a sanitary concern, or whether the disease was contagious. The results varied from 1,8 percent of cancer patients coming from families with cancer in a German study, to 46,5 percent in a British study. "But the strangest of it all, is that while dr. Juliusberger with his 1,8 and 3,0 percent, and dr. Williams with his 15 percent argue that their statistics support the assumption of an inherited disposition – Dr. Bashford argues that his 46,5 percent proves the opposite."[807] Søegaard's preferred theory was that in insanitary conditions cancer could be contagious ('cancer à deux'). The experts disagreed, and so did their methods. One strong argument against contagion came from the Imperial Cancer Research laboratories in London, where experiments on mice had failed to produce a single case of contagion through inoculation. According to Søegaard, this only showed that laboratories did not reflect the actual conditions in rural insanitary conditions, which was where 'cancer à deux' appeared.[808]

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[806] Søegaard, Munch. "Omkring kræftspørgsmaalet. Arvelighed. Cancer à deux." *Norsk Magazin for Lægevidenskaben.* 1910: 1045-1075. See also: Søegaard, Munch. "Et bidrag til norsk Kræftforskning". *Medicinsk Revue.* 1909: 305ff.

[807] "Men det merkeligste af alt er, at medens dr. Juliusberger med sine 1.8 og 3.0 pct. og dr. Williams med sine 15 pct. mener at have leveret statistiske tal, der støtter antagelsen af en arvelig disposition, – saa mener dr. Bashford, at hans 46.5 pct. afgiver et bevis for det stikk modsatte." Søegaard 1910: 1050.

[808] In all, Søegaard identified fifteen 'cancer farms' in Vikør; insanitary households where more than one person was affected. The paper received a negative review in *The British Medical Journal,* attacking Søegaard's diagnosis, supporting the value of laboratory experiments: "Dr. Soegaard does not in any instance record a histological confirmation of the diagnosis, and in determining the occurrence of cancer in the families he is apparently quite satisfied with the statement of doctor or patient's relatives that the mother or some other relative died of cancer. The value of his deduction is therefore greatly impaired, because we cannot be quite sure that the reported cases were in reality cases of cancer, and further that the negative cancer did not contain some cases of carcinoma or sarcoma. (…). In attacking Dr. Bashford's deductions that the absence of infection in his mice speaks against any risk of infection, the author regards the conditions obtaining in his Norwegian village as favourable to infection, and those obtaining in the Imperial Cancer Research laboratories as unfavourable. To this proposition exception must be taken. In the former case innumerable factors may be playing a part, and the results obtained are uncertain, since histological confirmation of the diagnosis is wanting; while, in the latter, contact is present and other factors are to a great extent excluded, and scientific investigation of each tumour enables the observer to be certain of every record." (Unsigned. "Inheritance and infection in cancer." *The British Medical Journal*. 1910: 1987). However, it is not clear whether Søegaard ever read this; at least he never rebutted the charges.

Søegaard's interest in leprosy was raised in 1910, by a single paragraph in the American *Annals of Surgery*: "Not a single case of cancer was found among the lepers, notwithstanding the special search instituted in the matter, but this may be due to the fact that the lepers in the United States belong mostly to the non-Caucasian races."[809] Ignoring the question of race, Søegaard hypothesized: "that the lepers in USA should turn out to be immune to cancer has led me to investigate the situation in Norway, where we in this regard have such a rich material that we should be able to reach a final conclusion."[810]

Going through official statistics of recorded deaths in the Norwegian leprosy hospitals, the author found that of 2269 deaths, only 19 were diagnosed with cancer. When ignoring those dying under the age of 40, where it could be objected that the cancer had not had time to develop, the number rose to seventeen out of 1205 deaths. Even these 'least favorable numbers' produced a death rate to cancer of only 1,4 percent. This was well below the general statistics, where the most recent data from 1906 concluded that 8,5 percent of the Norwegian population died from cancer. Could it be that leprosy protected against cancer? If so, why?

> How this should be interpreted, I will leave to the learned. The most reasonable explanation is that the lepers have a strongly reduced susceptibility to cancer. (…) Should it, however, turn out that cancer is an infectious disease it is most certainly significant that the lepers either in institutions or in private isolation do not have freedom to mingle. They have no intercourse with people with cancer, and are therefore not exposed to the same dangers as the rest of us who are free to come and go as we please.[811]

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[809] Levin, Isaac. "The Study of the Etiology of Cancer based on Clinical Statistics". *Annals of Surgery*. Vol. 51, no. 6, June. 1910: 778. The paper was read before the American Surgical Association on May 4, 1910, and presented the first 4000 cases collected by a project instituted by the George Crocker Special Research Fund. The study consisted of sending a 'cancer schedule' with 22 questions regarding individual patients to major US hospitals and then treating the responses statistically – the same method used in cancer research in Norway.

[810] "(…) at de leprøse i U. S. A. skulde vise sig at være immune mot cancer, har foranlediget mig til at anstille en undersøgelse av forholdet her i Norge, hvor vi i denne henseende har et saa rikt undersøkelsesfelt at vi måtte kunne komme til et endegyldig resultat." (Søegaard, Munch. "Lepra og cancer. Meddelelse til Bergens lægeforening." *Medicinsk Revue.* 1910: 635-640, quote on p. 636.) The 'rich material' referred to the national leprosy registry established in 1856. From the 1860s, it also included 'cause of death'.

[811] "Hvorledes dette egentlig skal tydes, det vil jeg overlate til de lærde at avgjøre. Det rimeligste er, at vi virkelig hos de leprøse staar overfor en sterkt nedsatt mottagelighet for cancer. (…) Skulde det virkelig vise sig at kræft maa betragtes som en infektionssykdom, saa tør det ganske visst ha sin betydning at verken stiftelserne

Before his first paper on leprosy and cancer was published in *Berliner klinische Wochenschrift*, the manuscript was sent to the Medical Association in Bergen, and discussed at their meeting on December 1, 1910. In accordance with the long established custom both in Bergen and other medical societies, the paper and following discussion was published in the society's own outlet, *Medicinsk Revue.* In the discussion, questions were raised to why the numbers differed in different leprosy asylums, and whether there was any merit to Søegaard's thesis that through being segregated to protect the population the lepers were themselves protected from cancer in the population. No conclusions were reached apart from encouragement that the research should continue. The author himself had sent the report in a letter, and was not present at the discussion.

Some months later, *Medicinsk Revue* published a short note with statistics from the medical director at the Holdsveikraspitalinn leper asylum in Laugarnesi on Iceland. Having read the presentation in the Norwegian journal, Sæmundur Bjarnhjedinsson confirmed that he too had the impression that leprosy patients were less prone to cancer.[812]

Encouraged, Søegaard wrote a follow-up paper, referring to more published statistics, numbers from hospital reports, as well as papers in sixteen different medical journals.[813] This shows that even for a physician in a rural community it was possible to get access to a wide range of published medical journals. By now, Søegaard had concluded that his thesis that leprosy prevented cancer was proved: "We have in our lepers found a group of people that show strikingly low cancer mortality."[814] Future research, he argued, should focus on explaining this fact.

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eller privatpleiens leprøse har et freies Leben und Treiben. De har ingen omgang med kræftsyke personer, de utsetter sig ikke for de samme noxer som vi andre der kommer og gaar som vi selv lyster." Op. cit: 640.

[812] Bjarnhjedinsson, Sæm. "Lepra og Carcinom". *Medicinsk Revue.* 1911: 336-338.

[813] In Søegaard, Munch. "Den relative kræftimmunitet ved lepra." *Medicinsk Revue*, 1911: 526-536. The journals referred to were: *Annals of Surgery*, *Archive für pathologische Anatomie*, *Medical Chronicle*, *Deutsche medizinsche Wochenschrift*, *Berliner klinishe Wochenshrift*, *Beitrag zur klinischen Chirurgie*, *The Medical Record*, *Wiener medizinsche Blat*, *München medizinsche Wochenschrift*, *Le Scalpel*, *Presse médicine Belge*, *Deutsche Zeitschreift für Chirurgie*, *Archive für Chirurgie*, *Medizinsische Klinikk*, *St. Petersburger medizinische Wochenschrift* and *The Edinburg Medical Journal*.

[814] "Vi har i vore leprøse fundet en gruppe medmennesker, der utviser en paafallende lav kræftdødelighet." Søegaard, Munch. "Den relative kræftimmunitet ved lepra." *Medicinsk Revue*. 1911: 536.

Also this second paper was published in *Berliner Klinische Wochenschrift.* From there the papers were picked up and spread to new audiences through *Lepra Bibliotheca Internationalis,* accompanied by an editorial remark by the main editor Edvard Ehlers asking if others could confirm the findings.[815]

The third and final paper in the series was read and discussed at the meeting of the Bergen Medical Society on April 12, 1912.[816] Søegaard had strengthened the argument that leprosy prevented cancer by comparing the leprosy numbers with registered deaths in mental asylums. These inmates were also isolated under medical surveillance, lived in similar housing, were served similar food and the inmates were recruited from more or less the same strata of society.[817] In the asylums, cancer mortality rates were three times higher than those found in the leprosy asylums. Søegaard also referred to statistics and letters of support from Sweden, Iceland, London, Trinidad, Bombay, as well as from several physicians working with leprosy in China, again showing that distance did not necessarily obstruct circulation. Still, the most interesting was the discussion that followed in Bergen.

H. P. Lie was critical. In addition to being a member of the Bergen Medical Association and co-editor of *Medicinsk Revue,* a member of the Norwegian cancer research committee since 1907 and newly appointed national chief physician for leprosy after the death of Gerhard Armauer Hansen in February 1912, Lie was the director at Pleiestiftelsen No. 1 in Bergen. This institution was by Søegaard presented as 'the most cancer infected leprosy hospital in Norway'. Going through the hospital records Lie found that cancer was first mentioned in 1882. He also found that practices regarding diagnosis had changed over time, including the use of autopsies to provide post mortem confirmations: "It is only after my accession in the 90s that provisions have been made so that dissections cannot be refused when the physician

[815] See: *Berliner Klinische Wochenschrift.* No. 51, 1910; No. 26 and No. 38, 1911. The discussion continued in No. 22, 1912, and in *Lepra Bibliotheca Internationalis.* 1911: 92-97, 172-180 and 1912: 128-132.
[816] Søegaard, Munch. "Videre undersøkelser vedrørende kræftdødeligheten blandt de leprøse." *Medicinsk Revue.* 1912: 276-289.
[817] Søegaard 1912: 287.

deems it necessary."[818] When limiting the statistics to the cases where the cause of death was confirmed post mortem, eight out of 252 deaths were caused by cancer –the same prevalence as those found in the mental asylums. This, Lie argued, meant that Søegaard's thesis was wrong. "A general statistical rule is that statistics which is uncertain should not be used as proof for the rare, unusual or deviant from general rules. Hence, I withhold that the material presented cannot be used to prove that cancer is rare among lepers."[819] Although Lie doubted the validity of Søegaards theory, he suggested that the argument should be presented for the International Leprosy Commission that had been elected at the Second International Leprosy Conference in Bergen in 1909, "requesting that it should be raised as a distinctive case for leprosy research, and then future will tell if, after all, dr. Munch Søegaard's assumptions turns out to be correct."[820]

Søegaard, who again did not attend the meeting in person, probably read the reaction only after it was printed in *Medicinsk Revue*. He immediately wrote a furious response:

> For two generations our leprologists have published their five year reports with extracts, tables and lists. None here at home have studied them, and thus we have not heard a word about their reliability. But then a man arrives, suggesting that there might be golden nuggets also for cancer research among these numbers. He uses the official death lists from the leprosy statistics in the same way as other statisticians have used the lists and tables on contagious disease from state- and district physicians. Only then is the warning call sounded: You must not take our lists and tables seriously![821]

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[818] "Det er først siden min tiltrædelse i 90-aarene at der er kommet bestemmelse om, at sektioner ikke kan negtes, naar lægen anser det paakrevet." Lie, H. P. "Diskussion". *Medicinsk Revue.* 1912: 289-290.
[819] "En almindelig statistisk regel er ogsaa den, at en statistikk, hvis sikkerhet er tvilsom, ikke bør anvendes som bevis for det sjeldne, usedvanlige eller det fra den almindelige regel avvikende. Derfor kan de her omhandlede opgaver efter min opfatning ikke anvendes til bevis for cancerens sjeldenhet hos leprøse." Lie 1912: 290.
[820] "Jeg skulde derfor ville foreslaa for dr. Munch Søegaard, at saken forelægges for den internationale leprakommission, hvori er representanter fra en rekke land med meget lepra, med anmodning om, at dette spørsmål optages som en særskilt sak i lepraforskningen, og da vil fremtiden kunne vise om det dog ikke er som dr. Munch Søegaard antar." (Lie 1912: 290.) Lie did not mention that he himself was a member of the committee, nor does it seem he made any efforts to forward the suggestion himself.
[821] "Her har nu i over to menneskealdrer vore leprologer utgit sine 5-aars beretninger med ekstrakter, tabeller og lister. Ingen her hjemme har studert dem, og saa lenge har vi da heller ikke hørt noget om listernes paalitelighet. Men saa kommer en dag en mand og mener: maaske findes her guldkorn ogsaa for kræftforskningen blant disse tal. Han benytter leprahospitalenes officielt utgivne dødslister paa samme maate som andre statistikere har

According to Søegaard, Lie not only criticized his work, but also all his predecessors for having published 'unusable statistics'. And, if Lie already knew the statistics were unreliable, why was he not warned before making a fool of himself in front of an international audience?

Shortly thereafter, Lie replied, also in the columns of *Medicinsk Revue*. Lie praised the careful statements in the first paper, where Søegaard had suggested that reduced susceptibility might be possible. The problem, as Lie saw it, was that Søegaard too soon moved on to try and explain this connection.[822] As Søegaard was more and more convinced leprosy protected against cancer, Lie reached the opposite conclusions. The statistics were flawed. His main argument was epistemological: The statistics reflected the disease models concurrent to the time of production, and not the currently held models.

> The last half century has changed so much in perceptions of almost all diseases, leprosy in particular, that this also was reflected in medical statistics. (…) If we return to the leading scientists of that time [the 1860s], we will find that affections today not recognized as aspects of leprosy, were perceived to be leprous affections. (…) I therefore withhold that the older notions of what leprosy was must have influenced the physicians' clinical gaze and their perceptions of what other diseases might have been combined with leprosy.[823]

Still keeping the possibility open that there might be a connection, Lie repeated the suggestion of bringing the question to the attention of the International Leprosy Commission so that the question could be properly investigated.

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benyttet stadsfysikatenes og distriktslægernes tabeller over smitsomme sykdommer. Først da gjøres der anskrik: Du maa da ikke ta vore lister og tabeller for det ramme alvor!" Søegaard, Munch. "Kræftdødeligheten paa de norske lepraasyler. Vor medicinalstatistiks paalitelighet. Tilsvar til overlege Lie's kritikk av mine meddelelser til Bergens medicinske selskap." *Medicinsk Revue.* 1912: 453-457, quote on p. 453.

[822] Lie, H. P. "Kreftdødeligheten paa de norske lepraasyler." *Medicinsk Revue*. 1912: 722-726.

[823] "De siste halvt hundreda aar har forandret saa meget i opfatningen av nær sagt alle sykdommer, ikke minst spedalskheten, at de heller ikke for statistikken har kunnet gaa sporløst hen. (…) gaar vi til datidens ledende videnskapsmænd, vil vi finde, at ogsaa disse har regnet til spedalskheden affektioner, som nutiden ikke anerkjender som leprøse. (…) Jeg mener saaledes i det anførte at ha vist, at den eldre tids opfatning av, hva der har været spedalsket, har maattet ha indflydelse paa lægernes kliniske blikk for og opfatning av andre sygdommer, som har kunnet være kombinert med lepraen." Lie, H. P. "Kræftdødeligheten paa de norske lepraasyler. Svar til dr. Munch Søegaard" *Medicinsk Revue.* 1912: 724-725.

Søegaard got the final word: A six point list expressing 'sadness' that the statistics were wrong, that they should never have been published, that Lie undermined his predecessors abilities to make diagnosis (stressing that he himself was not to blame, as he had no hand in diagnosing or producing the statistics), that this was yet another proof that arrogant medical researchers frowned upon practicing physicians trying to contribute with new knowledge; concluding that "I wish Armauer Hansen was still alive."[824] After this he never again published anything on leprosy.[825]

It is unclear if Søegaard ever approached the international leprosy commission. In *Lepra Bibliotheca Internationalis* a short follow-up was published by head physician Dr. Biehler at the State Leprosarium in Riga in 1913. Between 1891 and 1913, 160 of 194 deaths had ended in autopsies, of these ten persons had cancerous tumors. This was a higher proportion of cancer than in the general hospital and close to the general prevalence in the population. He also revisited Søegaard's statistics and concluded that the Norwegian physician had consistently relied on the highest possible numbers regarding prevalence of cancer in the general population. His conclusion was that there was not the slightest indication of any connection between leprosy and cancer immunity.[826]

The hypothesis that leprosy protected against cancer did, to my knowledge, never reemerge. Still, the case shows that a physician in a rural community could have access to a wide range of medical publications, including reports and medical journals seemingly intended for domestic audiences elsewhere. It also shows how publications in themselves could form the basis for the production of new knowledge, but that this was far from unproblematic.

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[824] " Gid Armauer Hansen nu hadde levd." Søegaard, Munch. "Replikk til dr. H. P. Lie". *Medicinsk Revue.* 1912: 727.

[825] Peter Munch Søegaard did, however, continue his research on cancer. The summer of 1914, he traveled to Germany, France, Britain, Denmark and Sweden financed by the Norwegian Roll's scholarship to bring back knowledge of 'the socio-humanitarian aspect' of cancer. This resulted in a booklet, *Samfundet og de Kræftsyke* (1915), which also contained information on cancer work in Austria, Russia, Spain, Switzerland and USA. The report was published with financial support from Norwegian life insurance companies, and distributed as a supplement to *Tidsskrift for den norske Lægeforening*. No. 17. 1915. All surpluses from sales were channeled to the Norwegian Cancer Committee.

[826] Biehler, R. "Die Krebssterblichkeit unter den Leprakranken des Rigaschen städtischen Leprosariums". *Lepra Blibliotheca Internationalis.* 1913: 148.

The debate between Søegaard and Lie clearly shows how views on medical statistics and scientific truths could differ. For Lie, medical statistics were a reflection of the best knowledge available at any given moment. Medical science was thus not merely a matter of accumulation: New standards for diagnosis did not make previous observations completely obsolete, but they could not be taken at face value. Looking back in the records required special expertise. For Søegaard, however, the observations were either true – or should not have been published in the first place.

Finally, the case shows circulation in practice. First, Søegaards study was based both on previously published research and correspondence with experts elsewhere. Second, the papers he wrote were discussed in local medical meetings and consequently published in the local medical association's medical journal, comments included. The publication in turn sparked responses from readers elsewhere, in this case Bjarnhjedinsson on Iceland, and later Biehler in Latvia. This was common for the medical journals and an integral part in the dynamics of circulation. Third, papers published one place could be reviewed elsewhere, sometimes without the authors' knowledge. Although Søegaard's first paper was published in a Norwegian and German journal, the only direct response I have found was a negative review in *The British Medical Journal* which it seems unlikely that Søegaard ever read. Circulation of knowledge did not mean everyone had access to everything that was published, not even everything that was directly relevant for their ongoing research projects. Fourth and finally, although it is not clear if this was Søegaard's intention, his publications in *Berliner klinische Wochenschrift* were put under scrutiny in *Lepra Bibliotheca Internationalis*. This was in line with the editors' strategy to ensure the journal's status as the primary resource for leprosy-related research around the world. Without the journal, it is unlikely that Søegaard's hypothesis would have received as much attention, nor that it relatively quickly could be put to rest after having been tested elsewhere.

Conclusion

Prior to the First International Leprosy Conference in Berlin (1897), circulation of new medical knowledge regarding leprosy was integrated in circulation of medical knowledge in general. Observations, and conclusions based on these, were disseminated through travels, personal meetings, books, journals, reports and correspondence. The widespread practice of publishing abstracts from other medical journals formed a genre in itself, and can be seen as a step towards international standardization. Over time, local particularities were downplayed also in the original research papers.

Not every study circulated. For the leprologists, the lack of a unifying 'international body' made it hard, if not impossible, for researchers to keep up to date on relevant studies produced elsewhere. There was not one center and one periphery, but different centers and different peripheries. Some centered on institutions, others around medical journals. Different disease models (accompanied by different ways of producing knowledge) were living side by side.

The initiative to organize leprosy workers were inspired by shared local circumstances, namely the perception by Western physicians and governments that leprosy was on the rise and that it had to be stopped before overwhelming the world. Norway became a focal point, and was frequently highlighted as the only civilized country that through consolidated efforts successfully had turned the trend and was about to get the leprosy problem under control. But when the American physician Ashmead argued that time had come for action, his European counterparts argued that gathering more knowledge and reaching a consensus among the experts had priority. Both schemes had their supporters, but the latter argument won out. The Europeans were the first to secure government support. It seems that having met face to face at prior medical conferences made collaboration easier than corresponding with unknown colleagues. The result was the first international leprosy conference held in Berlin in 1897, and soon thereafter the journal *Lepra Bibliotheca Internationalis*.

The journal and international meetings passing resolutions and recommendations did not mean that appropriation, as discussed in the previous

chapter, suddenly stopped being important. This is reflected for instance in the various reviews of the Berlin conference. In *The British Medical Journal* (London), it was hailed as an important success – stressing that the recommendations were already implemented in most of the British Empire. In *Medicinsk Revue* (Bergen), it was seen as an opportunity for new research to bring glory to the nation. In the *Journal of the American Medical Association* (Chicago), the conference did not live up to the hype.

Over time the leprosy conferences and the journal created a shared space which facilitated dissemination, collaboration, standardization, and also new ways of producing knowledge. The ambition of the journal was to present updated overviews of ongoing leprosy related activities worldwide, especially (but not limited to) medical research. Søegaard's hypothesis that leprosy prevented cancer shows that *Lepra Bibliotheca Internationalis* was not only an expert forum. A physician with no institutional affiliation could have his results published and tested by leprosy researchers around the world.

That leprosy was the same disease all over the world was an implicit premise for the relevance of knowledge produced elsewhere, and only in exceptional cases a topic for debate. Possibly this reflected the editors' position: The initiatives for both journal and international conferences came from researchers who were convinced that the disease was caused by the leprosy bacillus, and that its presence could be demonstrated everywhere. This was not the only disease model, but its hegemonic position was illustrated for instance in reviews of research based on alternative views.

The outbreak of World War I brought *Lepra Bibliotheca Internationalis* to an abrupt stop after two issues in 1914. In the following chapter I will discuss the competing and complimentary attempts at reestablishing the global leprosy research community in the interwar period, focusing on the travels and meetings of the relatively small group of individuals involved.

7. Interwar globalization

On June 29, 1926, the Brazilian physician and leprologist Heráclides César de Souza-Araújo arrived in Bergen. He had left his position as head of the rural sanitary service ('Serviço de Saneamento Rural') of Pará district in northern Brazil two years earlier, and a month before embarking on what was to become a three year journey, he had opened Brazil's first agricultural colony for lepers, Lazaropolis do Prata. Boarding a ship in Rio de Janeiro in July 1924, the journey had taken him through North America, across the Pacific to Hawaii, Japan, Hong Kong, the Philippines, Singapore and India, through the Middle East and finally Europe.

The voyage was undertaken under the auspices of the International Health Board of the Rockefeller Foundation and the Oswaldo Cruz Institute in Brazil. It was made "in the hope of stimulating the beginning of a general campaign against this terrible scourge. (…) exclusively to study the serious problem of leprosy, hoping to use the knowledge thus achieved in the control of this disease in my own country."[827] What did de Souza-Araújo find around the world? And how did his mission to import knowledge on leprosy from abroad to Brazil end up as an initiative to organize leprosy work on a global scale?

As I showed in the first part of the thesis, in the interwar period the belief in strict segregation that had dominated before the war was challenged by arguments for voluntary treatment. In this chapter I will investigate the initiatives for consolidating leprologists globally in the 1920s and early 1930s, focusing on the actors and organizations involved behind the scenes, such as de Souza-Araújo, the Rockefeller Foundation, the League of Nations, the Leonard Wood Memorial, Société de Pathologie Exotique and BELRA. As I showed in the introduction to the thesis, the outcome of these efforts was a global apparatus for leprosy that would last for 70 years. What did it take to achieve this? Who were involved and what were their backgrounds? What happened behind the scenes?

The outbreak of war in 1914 brought the journal *Lepra Bibliotheca Internationalis* to a complete halt. Traveling was restricted and consequently no conferences were arranged. Even correspondence was increasingly difficult. In addition, more pressing issues surpassed leprosy both among physicians and on the public agenda; leprosy would never get the same attention as before the war. While *The British Medical Journal* published an annual average of 50.2 papers on leprosy from the turn of the century to the outbreak of war, that average dropped to 14.6 during the war.[828] In the 1920s and 1930s the attention given to leprosy, as measured in number of published research reports in the journal, stabilized at around 35 publications per year.

In some ways, the events in the 1920s and 1930s were a repetition of the endeavors discussed in the previous chapter, but with new actors and new geographical centers of gravity. Most of the people involved in the interwar period had not been involved in the events described in the last two chapters, but this past provided inspiration of a past golden age of collaboration. This was a lesson that through collaboration and sharing experiences they could build on each other's results and avoid repeating the mistakes made by others. The goal of ultimately eradicating the disease remained, and establishing new infrastructure for circulating knowledge was seen as a key step in fulfilling this aspirations.

Finally, the chapter will investigate the legacy of Bergen. The reason why de Souza-Araújo went to Bergen was primarily because of its famous past, and not the research going on at the time of the visit. How did the role of Bergen change? Was Bergen still relevant?

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[827] De Souza-Araújo 1929: 3.

[828] Before the war, the annual number of publications in *The British Medical Journal* peaked at 74 in 1903; the lowest was 27 in 1908. During the war, there were 19 publications on in 1915 and 1916, while in 1918 only 8 stories on leprosy were published. Source: Free text-search on "leprosy" at http: //www.bmj.com/search.

The world is not the same

During the journey, de Souza-Araújo experienced huge differences when it came to how "the problem of leprosy" was coped with around the world. In India, where the number of leprosy sufferers now was estimated to be between 200.000 and 700.000 individuals, less than nine thousand persons were institutionalized. The majority of the establishments were run by missionary organizations, and more often than not the inmates were free to come and go as they pleased. In Iraq with an estimated 300 lepers, they were treated as outcasts and de Souza-Araújo was told that many fled to larger towns to hide in the crowds. Establishing a leper colony had been considered, "but its cost is a drawback and the compulsory segregation of individuals of different tribes, races and religions presents almost insurmountable difficulties."[829]

At the other end of the spectrum was the Perpetual Asylum ('Zensei Byo-in') 37 kilometers west of Tokyo. This was one of five state run institutions; the backbone in Japan's expanding anti-leprosy campaign. De Souza-Araùjo was present when three groups of sanitary experts from the Far East received parts of their 45 day training course organized by the League of Nations:

> We were accompanied on this visit by all the technical staff of the asylum, who wore aprons and leather boots (top boots). To each visitor they furnished apron and cap. Two nurses wearing gauze masks, carried trays with pieces of gauze and balls of cotton wool drenched in disinfectants, and the usual objects for clinical and dermatological examination. Every time a doctor touched a patient the nurse offered him the means to disinfect his fingers or hand. The visit finished, which was long and very interesting, we retired to the medical post where our aprons and caps were taken off. There, while we washed and disinfected our hands, we kept our feet on mats drenched in a strong disinfectant solution, as is done in the leprosaria of DUTCH GUIANA. Passing from this disinfectory to the building of the administration, we were invited to gargle with a disinfectant solution and to wash our faces with gauze soaked in

[829] De Souza-Araújo 1929: 264. De Souza-Araújo later served on the World Health Organization (WHO) Expert Panel on Leprosy and on the council of the International Leprosy Association.

> alcohol. Later I observed that these hygienic precautions are customary in the establishment.[830]

At the remote island of Culion in the Philippines, where de Souza-Araújo had spent Christmas in 1925, he found the leper colony organized to mimic society in general, with its own police force, fire brigade, post office, shoemaker, and court system. Seven hundred healthy persons were hired to provide care for more than 5.300 lepers, making this the largest leprosy colony in the world:

> The leprosarium is almost a town: it has 47 concrete buildings, 19 of strong heavy material and 30 of light material. The lepers own 616 houses of their own, in the village and in the farming district, most of them being built of light material. They have still 25 business houses, of which there four societies [sic], being two funerary and two athletic, a band and an orchestra.[831]

At the Molokai leper settlement in Hawaii, de Souza-Araújo found a similar parallel society. On his first day there he observed the juridical system in action: "On September 26th I was present, at the trial of a Corean leper, (A case of nodular advanced leprosy) accused of having made and sold pineapple wine. The barrel, containing half the liquid made, was brought to the Court by the Police Officer, who accused the prisoner of having sold the drink, because it was impossible that he had drunk in 2 days, nearly 20 quarts."[832] The judge was a 45 year old 'neural-leper' who was interned in 1906. Unlike Zensei Byo-in and Culion, however, the number of inmates at Molokai was in decline following new legislations encouraging voluntary isolation – making forced segregation the exception. In 1925 Molokai had 533 lepers, down from 1.213 in 1890.

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[830] De Souza-Araújo 1929: 109.
[831] De Souza-Araújo 1929: 146. See also: Wade, H. W. and Jose Avellana Basa. 'The Culion Leper Colony'. *American Journal of Tropical Medicine*, Vol. 8, No. 5, 1923: 405-6; Robertson, Jo. "Culion, the 'Island of the Living Dead': or Another Look at Leprosy and Citizenship". In: Andresen, Astri, Kari Tove Elvakken and Tore Gronlie (eds.) *Politics of Prevention, Health Propaganda, and the Organisation of Hospitals 1800-2000*. Bergen. Rokkansenteret, Report 10. 2005: 81-92.
[832] De Souza-Araújo 1929: 84.

When de Souza-Araújo arrived in Bergen in the end of June, 1926, he expected Bergen to be one of the highlights of the trip. In the research literature, Bergen was presented as the birthplace for scientific investigations into the disease: This was the place where Danielssen and Boeck in the 1840s had distinguished leprosy from other affections of the skin, and thus established what the German pathologist Rudolf Virchow after a visit in 1859 had termed "the foundation for modern leprology".[833] De Souza-Araúja was proud to own a copy of Johan Ludvig Losting's colored atlas that accompanied the work.[834] However, with the passage of time this was no longer considered a go-to textbook but a treasured relic of the past.

Bergen was also the site where the leprosy bacillus was first observed by Hansen.[835] By the end of the 1920s the bacillus was accepted by the medical research collective as the cause of the disease, and it was at the center of a whole range of practices around the world; from bacteriological detection in the laboratory being the final proof in contested cases, to the rationale for various social policies aimed at controlling those individuals who were infected. The bacillus was seen as proof that leprosy was the same disease all over the world, and the bacillus was a cornerstone in the argument that leprosy was a global disease, a disease which required global coordination to control and ultimately eradicate.

In addition to Bergen being presented as the first global capital of leprosy research, Norway was repeatedly highlighted as the first country in the world which had raised a successful and modern campaign against the spread of leprosy. De Souza-Araújo highlighted Norway as *the* example for other countries to follow. According to the "highly expressive statistics", the number of new leprosy cases in

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[833] Skinsnes, O. K. "Notes from the History of Leprosy". *International Journal of Leprosy*. No. 41. 1973: 220-237. De Souza-Araújo paraphrased Virchow with the following expressions: "The city of Bergen, Norway, where Danielssen, Boeck and Hansen established the foundations of the modern leprology." (De Souza-Araújo 1929: Fig 4, caption) and "BERGEN, the cradle of modern leprology" (de Souza-Araújo 1929: 15). The secretary of the League of Nations Leprosy Commission used the following phrase: "The moderen era opened with the clinical research work of Danielsen and de Boeck, the bacteriological phase with Hensen's discovery." (Burnet, E. *Provisional report presented to the Leprosy Commission at its meeting at Tokyo in April 1930*"*. Geneva, January 1930: 1. LNHO: CH/Leprosy/7.) *The meeting in Tokyo never took place, instead the report was presented in Bangkok, December 8-12,1930. More on this later.

[834] De Souza-Araújo 1929: 319.

Norway had been reduced from around thousand new cases per five years in the 1850s and 1860s, to less than ten in the 1920s.[836] Upon returning to Brazil, de Souza-Araújo argued that the Norwegian experience showed that a successful campaign against the disease went through three 'logical' stages: First, private initiative characterized by material assistance and collaboration of doctors and benefactors; second, official medical attention and establishment of a state leprosy apparatus; and third, forced segregation: "finally, after there was a considerable number of beds for the sick and the process of transmission was known, the government decreed and executed its severe measure."[837] Brazil was still at the first stage, de Souza-Araújo argued. His desire was that the Brazilian government should imitate the Norwegian experience: "The result will be identical."

In 1926, de Souza-Araújo stayed in Bergen only for a week. Unlike most other places he visited he felt it was more to learn from the past than the present. He visited Pleiestiftelsen and St. Jørgens hospital, the two remaining leprosy institutions, but found them largely deserted. Pleiestiftelsen had 280 beds, but only 57 patients, "most of whom very aged people".[838] At St. Jørgens hospital, which had almost 150 patients when Danielssen did his first clinical, anatomical and chemical investigations in the 1840s, only seven remained.[839]

Accompanied by district physician Dr. Høygaard, his son and daughter, de Souza-Araújo traveled to the nearby island of Herdla to experience the Norwegian home seclusion in practice. This was one of the three main components of Norway's "mixed segregation system", the others being state institutions and surveillance of each individual leper. The first patient they visited was a 19 year old man, whose

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[835] The first images in de Souza-Araújo's monograph were photographs of these three Norwegian physicians, "In memorandum".
[836] De Souza-Araújo. 1929: 321.
[837] De Souza-Araújo, H. C. "Desigualdade dos problemas da lepra nos países europeus ou A História da lepra na Europa". *Boletim da Sociedade de Assistência aos Lázaros e Defensa Contra a Lepra.* Vol. 2, no. 12, 1930: 11-14, referred to by Poorman, Elisabeth. "The legacy of Brazil's leper colonies. O legado das colônias de lepra no Brasil". *Cadernos saúde coletiva,* Rio de Janeiro, Vol 16, no. 2. 2008: 313. As Poorman argues, this is a typical example of how the Norwegian leprosy campaign was reframed and used in national debates: A specific national model was given legitimacy by being presented as an imported success.
[838] De Souza-Araújo. 1929: 324.

father, uncle, sister, grandfather and grandmother had all had died from leprosy. The man was, however, impossible to find.

> He had fled away from home, and we had no chance to meet him.
>
> He does the same, Dr. HOYGAARD told me, every time he foresees a visit, least they may transfer him to the asylum, and conceals himself. We entered his house and room. We asked his neighbours for information about his way of living. The reply was that he frequented their houses and used to play and have intercourse with other young people in the village.
>
> This patient having once more infringed the law of prophylaxis, the district physician decided to have him arrested and transferred to BERGEN.[840]

The next patient was a fisherman's wife and mother of six.

> "I examined her and her children. I did not notice any sign of active leprosy in her, nor any symptom of the disease in her offspring. Be it as it is, they all live in a candid promiscuity. Her husband appears at home only on Saturdays."[841]

A second disappointment was that he did not get to meet the head of the Norwegian leprosy campaign, H. P. Lie, who after the death of Hansen in 1912 had taken over the position as chief physician for leprosy. When de Souza-Araújo arrived, Lie was off on an inspection journey in Finmark in the far north of the country. Instead the Brazilian met with another foreign visitor: The Soviet professor Alexis Wladimiroff, director of the Institute of Experimental Medicine in Leningrad and president of the Commission of Studies on Leprosy in Russia. After a meeting among Russian leprologists in Leningrad, Wladimiroff had been commissioned on behalf of the Soviet government to create an updated report on prophylaxis of leprosy and policy advice on how to protect the wider society against the disease. He was therefore sent

[839] From 1896, St. Jørgens was not allowed to accept new patients. For a discussion of the increasingly tense relations between the private and the state institutions, see: Vollset 2006: 65-71.
[840] De Souza-Araújo. 1929: 325.
[841] De Souza-Araújo. 1929: 325.

to Bergen.[842] In Lie's absence, de Souza-Araújo ended up giving Wladimiroff the information he was asking for.[843] Chance meetings and chance events were common in the transnational history of leprosy.

The interwar period saw a soar of international journeys, although traveling as such was not a novel activity among physicians working with leprosy. As mentioned in Chapter 2, Danielssen and Boeck going on long travels abroad was a central part in establishing Bergen as the world capital for leprosy research eight decades earlier. A study by Hanne Winge Kvarenes shows that about fifty percent of the Norwegian physicians working during the period 1840-1880 had been on study trips abroad.[844] I do not know how representative this was for physicians in other countries, but among those involved in organizing medicine internationally, traveling was an activity they shared. What changed in the interwar period was that the journeys were faster, longer and less expensive than earlier. Both in terms of expenses and practicalities, the world was shrinking.

The most common reason for physicians traveling was to receive training, and de Souza-Araújo began his journey in North America with a stay at the newly inaugurated School of leprology at the National Leprosarium at Carville, Louisiana.[845] He also took special courses in Hygiene and Public Health at John Hopkins and Columbia University in the USA.[846] A second reason was to bring home experiences

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[842] Letter from H. C. de Souza-Araújo to Prof. Alexis Wladimiroff, dated July 3, 1926, written in Bergen. (LNHO: 12B-R898/52570). See also De Souza-Araújo. 1929: 15.

[843] De Souza-Araújo 1929: 15; Letter from de Souza-Araújo to Dr. L. Rajchman, medical director of the League of Nations Health Committee, dated October 6, 1926, titled "Leprosy as a world's sanitary problem', written in Siracousa, Sicily. (LNHO: 12B-R898/52570, p. 24)

[844] Kvarenes, H. W. "Travel accounts in the Norsk Magazin for Lægevidenskaben, 1840-1880." In: Ø. Larsen (ed.). *The Shaping of a Profession. Physicians in Norway, Past and Present.* Science History Publications/USA. 1996: 276. During the same four decades, 78 travel reports were published in *Norsk Magazin for Lægevidenskaben* (*Norwegian Magazine on Medical Science*), including information on travel grants and transcriptions of international meetings.

[845] The year de Souza-Araújo visited Bergen, in 1926, 31 physicians, 56 nurses, 60 students of medicine and 75 dentists visited the hospital with the purpose of familiarizing themselves with leprosy. De Souza-Araújo 1929: 34.

[846] De Souza-Araújo 1929: 8. For a study of how Japanese physicians traveling to Germany between 1868 and 1914, see: Chen, Hsiu-Jane. *"Eine strenge Prüfung deutscher Art". Der Alltag der japanischen Medizinausbildung im Zeitalder der Reform von 1868 bis 1914.* Abhandlungen zur Geschichte der Medizin und der Naturwissenschaften, heft 109. 2010. For a study of the impact of the educational schemes of the League of Nations and the International Health Board of the Rockefeller Foundation, with focus on a Spanish setting, see

in order to inform new policies and practices, which was the explicit goal of de Souza-Araújo's travel. A third reason for traveling was to attend meetings and conferences and meet colleagues there, which de Souza-Araújo also took part in. A fourth, which de Souza-Araújo was not involved with, was to charter new territory: To look for specific diseases such as leprosy. This was mainly done by Europeans in colonial possessions.

The visits only provided information on the actual situation on the ground at the given moment of the visit. Most of the information de Souza-Araújo collected was based on a combination of interviews, reports, papers and other researcher's monographs. Each of the 40 chapters in the volume he published after the trip, one for each country visited, included photographs of the premises, a presentation of the local history focusing on how the disease (meaning the leprosy bacillus) originally was introduced to the country, and details on the efforts taken to control the disease.[847] Names and places were in capital letters both to credit those who had contributed with information and to show the readers who they could get in touch with for more details. In addition, he portrayed the local practices and approaches to epidemiology, pathology, serology and therapeutics, legislation, and statistics on number of known lepers in- and outside the institutions. Each chapter ended with a list of relevant literature.

De Souza-Araújo's main interest was in the institutions erected to combat the disease, including architecture and experiences on organizing the daily lives of the inmates down to details on diet and cost per day per inmate. The expressed intention was to present both successes and failures, so that the readers could avoid making the same mistakes as had been made elsewhere. However, in practice failure to de Souza-

Barona, Joseph L. "Public health experts and scientific authority". In: Andresen, A, W. Hubbard and T. Ryymin (eds). *International and Local Approaches to Health and Health Care*. 2010: 31-49.

[847] The 'fourty countries' investigated by de Souza-Araújo were, in chronological order: United States of America, Canada, Hawaii, Japan, Korea, China, Philippine Islands, Federated Malay States, Malacca Straits Settlements, Dutch East Indies, India, Kingdom of Iraq, Syria, Palestine, Kingdom of Aegypt, Tripoli, Malta, Tunisia, Algeria, Marocco, Greece, Turkey, Roumania, Bulgaria, Yougoslavia, Hungary, Austrian Republic, Tscheco-Slovakia, Poland, Germany, Denmark, Iceland, Sweden, Norway, Great Britain, Holland, Belgium, France, Switzerland, Italy, Spain and Portugal. The 400 page report *Leprosy Survey made in Fourty Countries* was published as a book in both English and Portuguese (with the title *Lepra em 40 Países)* in 1929.

Araújo meant not taking the problem of leprosy seriously, success meant detection, erection of institutions and increasingly strict segregation of those affected. In the 1920s this was an increasingly disputed position.

A contested legacy

As I showed in Chapter 4, the question of forced segregation versus voluntary treatment was the main scientific controversy regarding leprosy in the 1920s. Both camps praised the Norwegian leprosy apparatus, but with different emphasis. While de Souza-Araújo saw Norway as proving his theory that a successful campaign went through three necessary steps leading to strict segregation, Sir Leonard Rogers and Ernest Muir had firmly established an opposite camp. For Rogers and Muir, Norway's campaign was successful because of its persistence and humanitarianism, and the measures were not universally applicable:

> The success of the great Norwegian test of segregation has thus completely demonstrated once for all the efficacy of that measure under conditions permitting of its thorough and persistent application, and has furnished a model and humanitarian example for the imitation by other countries, which can be relied on to be effective in exact proportion to the efficiency with which it is put into operation.[848]

Based on Hansen's presentation of the history of leprosy in Norway given at the first International Leprosy Conference held in Berlin in 1897, Rogers and Muir too highlighted the rapidly declining number of leprosy sufferers in Norway. In other words, both camps agreed that Norway had led a successful campaign – but disagreed on what had caused it. While de Souza-Araújo argued that the Norwegian experience showed that 'severe measures' were necessary, Muir and Rogers saw it as proof that eradicating leprosy could be achieved using a 'humanitarian approach'.

[848] Rogers and Muir. 1925: 110. Both had worked at the Calcutta School of Tropical Medicine, and were in 1924 founders of the British Empire Leprosy Relief Association, BELRA.

Unlike de Souza-Araújo, Rogers and Muir repeatedly stressed that the Norwegian success was due to ideal conditions which could not be expected in colonial contexts. There the approach would have to be modified "to suit the conditions pertaining to various countries presenting different degrees of advance in hygiene, education, financial and political evolution, and with public opinion, which all greatly influence the problem to be dealt with".[849] Thus, Muir and Rogers argued, the Norwegian approach was applicable only to 'civilized' countries. This is consistent with what Helen Joy Power argued in her PhD thesis on Sir Leonard Rogers: As a committed imperialist, he saw the best way to manage imperial possessions would be to westernize their inhabitants.[850] According to Rogers, a general and compulsory segregation would be impossible in "poor and backwards" countries: Policies that were passed were seldom implemented, and strict segregation only forced lepers to go into hiding – an argument that had been a minority view among leprologists since before the turn of the century. Finally, even if these obstacles could be overcome, it was not realistic that countries such as India could build enough institutions, whether it was self-sustaining agricultural colonies, hospitals, asylums or leprosaria, to accommodate the great number of people affected by the disease.[851] The latest decennial census from India (1921) showed that India had 102,513 lepers, "but there is no doubt that the returns are much below the truth, especially when we take into account the large number of early cases not recognizable except after careful examination by an experienced medical man".[852] A survey among

[849] Rogers and Muir. 1925: 126.
[850] Power, H. J. *Sir Leonard Rogers Frs (1868-1962): Tropical medicine in the Indian medical service.* Thesis submitted to the University of London for the Degree of Doctor of Philosophy. 1993: 276. (Wellcome Trust Library). See also: Boyd, John S. K. "Leonard Rogers. 1886-1962". *Biographical Memoirs of Fellows of the Royal Society.* Vol. 9, 1963: 261-285.
[851] Rogers and Muir 1925: 105-106. For an in-depth classification of the different missionary leprosy asylums in India between 1886 and 1947, see: Robertson, Jo. "The Leprosy Asylum in India: 1886-1947". *Journal of the History of Medicine and Allied Sciences*. Vol. 64, no. 4. 2009: 474-517. For an exemplary study of practices and negotiations at a local level, see: Buckingham 2002.
[852] Rogers and Muir 1925: 36-37. According to the secretary of the League of Nations Leprosy Commission, Etienne Burnet, Muir estimated that the numbers were at least seven times as high. (Burnet, E. "League of Nations Health Organisation. Report on the Study Tour of the Secretary of the Leprosy Commission in Europe, South America and the Far East. January 1929 - June 1930." (C. H. 887). Geneva, 1930: 15-16.)

thirty of the patients under Muir's care showed that only two had been entered in the census as 'lepers'.[853]

Rogers and Muir strongly argued that leprosy was more contagious in certain stages than others, and that the chance of catching the disease declined with age. This had decisive impact on the question of segregation: Children of lepers should be segregated as early as possible (preferably at birth) to avoid contagion. At the same time, only those with active ulcers were considered highly contagious and therefore a possible threat to public health.

Compared to de Sousa-Araújo, Rogers and Muir presented a different way of knowing leprosy. The ultimate goal of getting rid of leprosy was the same, but they had a different stance regarding what observations were considered relevant knowledge. While the Brazilian focused on leprosy as prevalence in populations, and saw segregation as a means to bring down the number of new cases, Rogers and Muir put emphasis on treatment of individual sufferers and alleviating their surroundings. In their view, leprosy was curable as long as the treatment started early. The two were pioneers in developing and testing treatment based on injections of chaulmoogra oil (see Chapter 3). In their monograph *Leprosy* (1925), the Norwegian experience was used to underline that segregation was futile: Even in Norway, five of six cases were overlooked for three years or more after the onset of the disease, "clearly demonstrating the impossibility of discovering the great majority of leprosy cases in the early stages as long as their only prospect was isolation from their relatives with no appreciable hope of recovery."[854]

Compared to de Souza-Araújo's report, however, Rogers and Muir's monograph would have the greatest impact, both immediately and over time. In the summer of 1925, only months after *Leprosy* was first published, Evald Tomanek from the League of Nations Health Section was sent to London to meet with Rogers. Tomanek concluded that "the publication 'Leprosy' by Sir Leonard Rogers represents

[853] Quoted in de Souza-Araújo 1929: 231.

a monograph written by authority in our days on the subject", and that "there was no need for the League to publish another monograph."[855] New editions of *Leprosy*, updated with the latest research into therapeutics, were published in 1940 and 1946.

Writing a book can be seen both as an outcome of the effort it took to produce it, and a vessel to get the knowledge to those who could use it. The monographs by de Souza-Araújo and Rogers and Muir represent two radically different perspectives, both on how to go about gathering knowledge, what observations were considered relevant, how to present the findings, and in advice on what practical measures should be taken based on this. Both relied on the writings of others, but supported their conclusions through conscious choices of what they considered relevant in the vast body of medical literature and empirical observations. The authors were all considered prestigious medical scientists, they met and discussed, and when de Souza-Araújo published four years after Rogers and Muir, he appropriated the content by referring mainly to the sections in *Leprosy* which he agreed.

Rogers and Muir's take on leprosy was also spread through courses held at the Calcutta School of Tropical Medicine and Hygiene, and several key actors involved in making leprosy a global issue received their training in Calcutta. The school officially opened in 1920 after Rogers as professor of pathology at the Calcutta Medical College had promoted it for a decade. The same year Rogers returned to London and Muir, a former medical missionary in Bengal, was recruited as head researcher of the new school. The Calcutta School soon became a renowned center for training of physicians for out-patient clinics, as well as a center for further research into treatment. The school was not a closed leprosy asylum, but relied on people choosing to be diagnosed and receive treatment for three hours every day voluntarily. When de Souza-Araújo spent the whole of February 1926 at the School of Tropical Medicine in

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[854] Rogers and Muir, 1925: 110. The monograph was organized in six sections: History and distribution, epidemiology and communicability, prophylaxis, etiology, clinical aspects and treatment. The first half was based mainly on available literature, the second on observations from India.

[855] Tomanek, Evald. *Memorandum concerning the statistics of leprosy to the Medical Director*, dated July 21, 1925. Medical Director of the League, Ludwig Rajchman, agreed. (LNHO: 12B-R898/42641/29272. The reply from Rajchman is found in the same folder.)

Calcutta, Muir was the director. The 'Calcutta approach' to leprosy was written on the wall, quite literally:

> In the waiting room of the dispensary, there exists exposed a graphic with lots of advices to lepers. Among these, there are the "Golden Rules" reading as follows:
> 1. – Leprosy is acquired by contact with lepers. Children are especially liable to infection. Infectious lepers should occupy a separate room, and should use separate clothes, bedding, eating and drinking utensils and should not touch food, clothes, furniture, or other things which are used by healthy people.
> 2. – Take plenty of exercise: walking, dumbbell, games, etc.
> 3. – Take nourishing food in abundance, but not excess, well-cooked, but not over-cooked. Take plenty fresh fruits and vegetables. Avoid the following: a) stale food; b) partly decomposed food; c) food preserved by salting; d) highly spiced food; e) highly milled grains; f) narcotics as alcohol, opium, ganja, tobacco; g) an excess of meat or fish.
> 4. – Keep the bowels open, by diet if possible, otherwise by means of laxatives.
> 5. – Keep the skin clean. Bathe at least once daily and apply friction to the skin, rubbing on chaulmoogra oil.
> 6. – Keep the mind active and cheerful.
> 7. – Keep the body strong and try to avoid other diseases.[856]

To de Souza-Araújo, this was humbug. In a lecture at the Faculty of Medicine in Madrid on November 29, 1926, de Souza-Araújo expressed his misgivings for the "exaggerated optimism in the modern treatments and the influence of the so-called 'Indian System' of prophylaxis". In his view, the treatments did not work, and propagating false hope would eventually backfire. Referring to the more liberal policies recently implemented at Hawaii, he argued that the public administration "must re-start the systematic seclusion of all its lepers, otherwise incurs the risk of a disaster, which would have effect in discrediting the modern prophylaxis, producing a bad influence, of pessimism, in the public opinion."[857]

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[856] De Souza-Araújo 1929: 244ff. The referred to poster is also found in E. Muir. *Leprosy. Diagnosis, Treatment and Prevention.* Cuttack: Orissa Mission Press. 1924: 23.
[857] De Souza-Araújo 1929: 97-98.

Publishing monographs were but some of several strategies the two parties had in common. They also created organizations organized teaching.

Organizing and educating

In 1924, four years after returning from India and being appointed lecturer at the London School of Tropical Medicine, Rogers established the organization British Empire Leprosy Relief Association (BELRA). January 27 the following year, an Indian Council of BELRA with headquarters at the Calcutta School of Tropical Medicine and Hygiene's Department of Leprosy was established by Muir. The same year, a second Indian headquarters opened at the Red Cross Society Office in New Delhi. The organization had prominent leadership, with the Prince of Wales as patron, and Secretary of State for Foreign Affairs, The Secretary of State for the colonies, Secretary of State for India, The Viceroy of India and the Governor-Generals of Canada, South Africa and New Zealand as vice-presidents. This shows both Sir Leonard Rogers status among the political elite in the British Empire, but also that leprosy research still was considered an important issue among its top officials.

BELRA had three main objectives: To collect and disseminate knowledge related to diagnosis and treatment of leprosy, including training courses in treatment; to encourage and carry out research; and finally to carry out propaganda with the overriding principle that the stigma attached to the disease should disappear.

Between establishing BELRA and de Souza-Araújo's visit in July 1926, Rogers had spent time raising funds for the association, formulating its policy and establishing contact with people working with leprosy within the British Empire (and occasionally outside its borders). In 1924, Rogers sent a questionnaire to all British possessions through the British Foreign, Colonial and India offices asking for information regarding leprosy. This provided the basis for a report that was sent to both policy makers and the League of Nations, stressing that leprosy was not a highly

contagious disease and that segregation in most cases was futile.[858] In addition to giving courses in Calcutta, Muir prepared a booklet ("Diagnosis, treatment and prevention") and two pamphlets ("Truths about leprosy" and "What the people should know about leprosy") which were distributed to all who contacted the organization. He also gave public lectures.[859] The main message in these early BELRA-publications was that the solution to the problem of leprosy was to educate the public so that they could recognize the signs of the disease and avoid the carriers:

> The ideal method of prevention would be the hindering of all contact. This was attempted on an extensive and expensive scale in the Philippine Islands and proved a failure as means of stamping out the disease. What stamped leprosy out of England was to a large extent the effort of individuals and bodies of people to keep lepers from mixing with healthy people. It was through an enlightened public opinion which was aware of the contagiousness of the disease. Other conditions, such as improved diet and improved sanitation, were also helpful, but these are useless apart from enlightened public opinion and public spirit.[860]

Through the publications and training, BELRA was put in touch with active leprosy workers who in turn could supply information, case-histories, statistics and reports, as well as do clinical trials.[861] From 1928, BELRA published a quarterly journal which distilled both findings and literature, *Leprosy Notes.* In 1930, under the editorship of Dr. Robert Cochrane, it was renamed *Leprosy Review* and shaped into a medical journal.[862] The ambition was that the journal should serve as a common ground, an

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[858] In the report Rogers stressed that "the guiding principle should be that in the long run *any advantages gained by rigid segregation of advanced cases may be dearly bought at the expense of driving the early ones to hide themselves until no longer amenable to the now available improved methods of treatment.*" Rogers, Sir Leonard. "Memorandum on the prevalence of and prophylaxis against leprosy in the British Empire, based on replies to the questionnaire of the British Empire Leprosy Relief Association; with suggestions for dealing with the problem." 1925: 29, italics in the original. Addendum to letter from Rogers to Ludvig Rajchman, head of the League of Nations Health Organization, dated London October 27, 1925. (LNHO: 12B- R898/42641/29272).
[859] See i.e.: Muir, Ernest. *Popular lecture on leprosy, illustrated with lantern slides.* Lahore. 1925.
[860] Muir, E. *Leprosy. Diagnosis, Treatment and Prevention.* Cuttack: Orissa Mission Press. 1924: 28.
[861] Power 1993: 222. Among the leprosy workers who contacted BELRA were physicians working for American Near East Relief at the leper colony Spinaloga near Crete, made famous in Victoria Hislop's novel *The Island* (2005).
[862] Editorial. "Leprosy Review: Origin, policy, content, circulation, finances and the future." *Leprosy Review*. Vol. 70, No. 2. 1999: 126. Available online: http: //www.leprahealthinaction.org/lr/June99/editorial.html. When

arena for leprosy workers to update and keep updated on research that went on elsewhere.[863]

During his journey, de Souza-Araújo too established an international organization for leprosy researchers, *La Société Internationale de Leprologie.* This was officially founded in Professor Edouard Jeanselme's home in Paris on the last day of 1926, and the total (but meager) income from de Souza-Araújo's monograph was set aside for the organization.[864] Unlike BELRA, which saw its geographical area primarily as the British Empire, and secondarily English speaking leprosy workers elsewhere, de Souza-Araújo had global aspirations. In addition to facilitate cooperation between leprologists all over the world through an international medical journal on leprosy, de Souza-Araújo wanted the collaboration to result in an updated prophylaxis for leprosy control, international centers for research and training, and that the organization should work out technical advice to stimulate government involvement in leprosy work.

In his monograph, de Souza-Araújo argued that the idea for the organization came during his travels, and was a topic for discussions from his first stop at the Carville in 1924. After experiencing the efficient collaboration between leprologists

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the quarterly *International Journal of Leprosy* began publishing in 1933, BELRA's abstracts from *Tropical Diseases Bulletin* were highlighted as "most valuable, but they have hardly lessened the need of a regular full-scope journal of leprosy". (H. W. Wade. "The International Journal of Leprosy. An editorial Statement." *International Journal of Leprosy*. Vol 1. no. 1. 1933: 2.)

[863] While the scope was similar to that of *Lepra Bibliotheca Internationalis,* BELRA's main reference was Mission to Lepers in India and the Far East. Mission to Lepers had been established in 1874, and from 1897 they published the quarterly journal *Without the Camp.* Rogers, himself a devote Christian, believed the missionaries put too much emphasis on the gospel and not enough on research. Several of the active members of BELRA in the 1920s were recruited from Mission to Lepers, such as the organization's first secretary, Reverend Frank Oldrieve who had been the Secretary for India for the Mission to Lepers, and his successor Dr. Robert Cochrane and Muir, who both had backgrounds as medical missionaries in Bengal. For discussions on the activities of Mission to Lepers in India, see i.e. Joseph, D. George. "'Essentially Christian, eminently philantropic': The Mission to Lepers in British India." *História, Ciências, Saúde-Manguinhos,* vol. 10, suppl. 1, 2003: 247-275; Robertson, Jo. "The Leprosy Asylum in India: 1886-1947." *Journal of the History of Medicine and Allied Sciences.* Vol. 64, no. 4. 2009: 474-517. See also: Bailey, W. *Fifty Years' Work for Lepers 1874-1924*. London. 1924; Miller, A. Donald. *An Inn Called Welcome.* 1965; Davey, Cyril J. *Caring Comes First: Story of the Leprosy Mission.* 1987.

[864] Edmundo Jeanselme was the president of The Third International Leprosy Conference in Strasbourg in 1923, and had been attending the conferences in Berlin in 1897 and Bergen in 1909. The last section of de Souza-Araújo's monograph includes summaries and abstracts of more than forty letters and statements made by researchers all over the world, summarizing the efforts to organize the society. De Souza-Araújo 1929: 18-19, 366-388.

from Zensei-Byoin in Tokyo and the world's largest leper colony at Culion in the Philippines in December 1925, the main obstacle was identified by chief pathologist Herbert Windsor Wade: "But how could we think of organizing a Society of Leprology since we ignore who are the Leprologists? We have first of all to discover them."[865] Establishing contact with possible collaborators thus became another rationale for the journey around the world.

De Souza-Araújo's vision was to restart *Lepra Bibliotheca Internationalis*, and let this be the spearhead for a global leprosy organization. A week before arriving in Bergen, he visited Edvard Ehlers in Copenhagen and was formally given the ownership of the journal. The accompanying organization should have 120 expert members elected by leprologists in countries with the disease, 120 representatives of the sanitary services, research institutions and leprosy associations (such as BELRA and missionary organizations), as well as ten honorary presidents. It is clear that the Brazilian expected that many of the people he had discussed the scheme with during his travels would fill key positions. The organization was to be split in an Eastern and a Western branch. After a suggestion by Ehlers, he pointed out H. P. Lie in Bergen to be the general secretary of the European, African and Latin-American branch of the association, as well as the regional editor for the planned journal. After being contacted by mail, Lie prepared a front page for *Lepra Bibliotheca Internationalis,* starting with volume XVI.[866] But moving the organization and the journal from the drawing board into the real world proved difficult.

First, not all leprologists were interested in the society. When the scheme was presented at the Third Congress of Dermatology for French speaking doctors in Brussels in 1926, "the matter proved to be of little interest to French dermatologists."[867] In France, leprosy was already one of several diseases covered by *Société de Pathologie Exotique*. Next, BELRA had already plans for expanding their

[865] De Souza-Araújo 1929: 366. The researchers at Culion also collaborated with colleagues at Hawaii and several of the physicians had received training from Muir in Calcutta. It seems Wade also kept in touch with his predecessor, Dr. Oswald E. Denney, who in 1921 was recruited from Carville to be the director at newly nationalized US National Leprosaria at Carville, Louisiana.
[866] De Souza-Araújo 1929: 373.

activities, or alternatively establish an English-speaking organization in collaboration with Wade and others.[868] Third, the League of Nations was already in the process of establishing its own leprosy commission. Finally, unlike BELRA, de Souza-Araújo's organization lacked friends in high places to give legitimacy and the necessary financial support to get it off the ground. But the initiative was not in vain. When leading leprologists met in Manila in 1931 and established the International Leprosy Association and the *International Journal of Leprosy* they took de Souza-Araújo's framework as the starting point for the discussions. With the exception of Jeanselme, the leadership of the new organization was identical to the one de Souza-Araújo had proposed.[869] The Brazilian was to hold the position as vice-president for the Western Branch of the International Leprosy Association from 1932 to 1956.

After spending the rest of the year in Western Europe and North Africa visiting laboratories, hospitals, and attending several conferences (including meeting Sir Leonard Rogers in London and Ludwik Rajchman of the League of Nations in Geneva), de Souza-Araújo embarked on S. S. Cap Polanio in Lisbon January 15, 1927. After eleven days he was back in Rio, and was appointed head of leprosy research at the Oswaldo Cruz Institute, a position he would keep until 1956.[870] The task of spreading the lessons he had learned on his three year journey started immediately. In February 1927, he gave a 40 hour course on leprology to 23 pupils; 21 physicians and two pharmacists who were students at the Oswaldo Cruz Institute.[871] When de Souza-Araújo in October 1931 enrolled 31 students it was the third such course. By then, the program had expanded to twenty days with five hours

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[867] De Souza-Araújo 1929: 366.

[868] Immediately after de Souza-Araújo's visit to Culion, Wade sent questionnaires to his contacts around the world and invited them to help 'find out who the leprologists are'. Rogers and Muir initially refused to support de Souza-Araújo's organization with reference to their own initiative, but after discussing it with Wade, they both agreed to give it a chance – provided the terms of membership and policies was still open for debate: "Failing that we shall probably probably proceed with our original scheme, I trust in friendly co-operation with your own association." (Letter from Rogers to de Souza-Araújo, dated July 21st, 1929, in Souza-Araújo 1929: 375).

[869] For de Souza-Araújo's full list of members of *Societe Internationalis de Leprologie*, dated Paris December 15, 1926, see: LNHO: 12B-R898/29272/52570.

[870] Poorman 2008: 312.

[871] Letter to Dr. L. Rajchman from H. C. de Souza-Araújo, Rio de Janeiro, April 26, 1928. (LNHO: 8A-R5872/2634)

of lectures each day. Topics dealt with were history, geographic distribution, epidemiology, etiology, pathology, clinical features, treatment and prophylaxis. The course also included visits to various anti-leprosy organizations in Rio and Sao Paulo.[872]

The knowledge was spread not just to other Brazilians. Before leaving Europe, in January 1927, de Souza-Araújo was involved in organizing a Regional Technical Committee for leprosy in Portugal. Later the same year he did the same in Brazil, Argentina and Uruguay, as well as setting up a provisory committee for Paraguay.[873] In 1939 he was invited by the Colombian government to assess the country's control program against leprosy, and gave a course on the disease which was attended by 32 physicians, 24 of whom were members of the leprosy staff of the Colombian Ministry of Hygiene.[874] In Colombia he promoted the establishment of a leprosy society with forty members, ten of whom were later elected members of the International Leprosy Association set up in Manila in 1931.

De Souza-Araújo also kept his interest in spreading knowledge through medical journals. Upon returning to Brazil, he took over as editor in the journal *Memórias do Institudo Oswaldo Cruz.* In Colombia, he promoted the establishment of a quarterly leprosy journal, *Revisita Colombiana de Leprología.*

Setting up scientific societies and giving specialist training were both a goal in itself and means to spread knowledge. In these areas de Souza-Arújo succeeded. In the short run, however, his plans for an international organization failed due to a mismatch between funding and ambitions. Several of the people he met on his journey were interested in collaboration but de Souza-Araújo failed to keep them updated about his work.[875] Yet, in the long run de Souza-Araújo's organization set the

[872] *Rev Med.-Cir. Brazil*. Vol. 40 (1932), referred to in *International Journal of Leprosy*, No. 1, vol. 1. 1933: 110.
[873] De Souza-Araújo 1929: 386-387. The committees were tasked with studying the problem of leprosy in the country; focusing on statistics and epidemiology, etiology and prophylaxis, clinical studies and therapeutics, and to work to create 'centers for studies'.
[874] Obregón, D. "The anti-leprosy campaign in Colombia: the rhetoric of hygiene and science, 1920-1940." *História, Ciências, Saúde - Manguinhos*. Vol. 10 (supplement 1). 2003: 200-201.
[875] One of those who raised this concern was H. P. Lie of Norway. See: Letter from Lie to Madsen, dated Bergen August 25, 1930. (LNHO: 8A-R5894/27029/6714)

premises for how the later *International Leprosy Association* and *International Journal of Leprosy* should be organized. In comparison, BELRA was a success due to influential members and secure funding within the British Empire. Although the two organizations shared the goal of eradicating the disease, they promoted opposite and competing approaches on how to reach the goal.

However, these were only two of several interrelated initiatives aimed at eradicating leprosy on a global scale. The League of Nations was also involved with leprosy. The credit for leprosy ending up on League's agenda was much due to another successful medical association, *Société de Pathologie Exotique*.

The League gets involved

Since the early 1920s leprologists had expressed the hope that an alliance with the League of Nations would put pressure on member states to raise campaigns to eradicate leprosy. As de Souza-Araújo wrote in a letter to Ludwik Rajchman, the director of the League's Health Organization after being asked to summarize the results of his travels: "The *coordination* of efforts of all nations and the international *cooperation* (…) are *imperative* for a general campaign against leprosy, and the Health Section [of the League of Nations] has prestige and power to obtain it."[876] De Souza-Araújo met Rajchman in the Far Eastern Bureau of the League in Singapore in 1925, and when the two met again in Geneva in September 1926 the Brazilian urged the League to get involved. By then the League was already pursuing several strategies for engaging in leprosy work on a global scale.

The initiative for an alliance between the leprologists and the League had come from the Third International Leprosy Conference held in Strasbourg, July 28-31, 1923. In one sense the conference was a continuation of the leprosy researchers'

[876] Handwritten addendum to letter from Souza-Araújo to Dr. L. Rajchman, medical director of the League of Nations Health Committee, dated October 6, 1926, written in Siracousa, Sicily. (LNHO: 12B-R898/52570). Italics underlined in the original.

previous initiatives to achieve global consensus on the disease, and the preceding conferences held in Berlin 1897 and Bergen in 1909 were frequently referred to. Like its predecessors, it was a conference for physicians working with leprosy in various places. In total, 34 different countries were represented. Several of the participants had taken part in all the previous conferences, such as professor Jeanselme from the Faculty of Medicine in Paris, who was the President at the Strasbourg conference. On the agenda were sessions on statistical data, etiology, pathology, treatment, prophylaxis and legislation. It resulted in a set of resolutions and recommendations as well as the publication of the proceedings (See appendix 2). Just as Prussia had sent the invitation to the first conference, and the Norwegian government invited to the second, the official invitations in 1923 were sent from the French Foreign Ministry.[877]

The initiative behind the Strasbourg conference came from a group of mainly French physicians who was interested in leprosy as one of several 'exotic diseases'. The leading organizers of the Strasbourg-conference were deeply involved in *Société de Pathologie Exotique*, established in 1907. Emile Marchoux, General Secretary at the conference, was one of the *Société*'s founding members. Both Marchoux and Jeanselme had attended the Second International Conference in Bergen in 1909 as the society's official representatives.[878] Ten more of the thirty members of the organizing committee were active members of the association.[879]

Although the organizers presented the conference as a continuity of the Berlin- and Bergen conferences, the conference marked a break with pre-war leprosy work with regards to participation. Of the fifteen persons who at the second international leprosy conference in Bergen in 1909 had been elected and charged with organizing a third conference, only three attended the Strasbourg meeting.[880]

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[877] Copy of the official invitations, as well as program and the list of members of the organizing committee, are found in LNHO: 12B-R898/29272.
[878] *Anmeldte deltagere i Leprakonferencen i Bergen, 15.-19. aug. 1909.* Regional State Archives of Bergen. (http: //da2.uib.no/cgi-win/WebBok.exe?slag=lesside&bokid=leprakonferanse&sideid=4)
[879] *Membres de la Société de Pathologie Exotique*. Archives de l'Institut Pasteur. (Available online: http: //www.pasteur.fr/infosci/archives/f_spe2.html);
[880] In Bergen in 1909, the initial 20-man Leprosy Commission from Berlin was reduced to fifteen members. "Wahl neuer Mitglieder der Internationalen Leprakommission." *Lepra Bibliotheca Internationalis*. 1910: 375.

It could be argued that the *Société de Pathologie Exotique* provided an inspirational model for both BELRA and de Souza-Araújo's aspirations to create an international leprosy organization. The mainly French society did not merely provide legitimacy when dealing with policy makers, but also proved that a national organization could provide the necessary resources to organize journals and international meetings. In 1908 it began publishing the periodical *Bulletin de la société de pathologie exotique*, which soon became one of the most frequently referred journals in *Lepra Biblioetica Internationalis*, with 22 abstracts of original papers on leprosy in the six years the two journals coexisted.

The leaders of the organization made important policy decisions, such as setting the agenda for international meetings, choosing the venue, and deciding whom to invite. The Third International Leprosy Conference was organized in the French city of Strasbourg mainly because it coincided with the celebration of the Pasteur centenary. Many of the founding members of *Société de Pathologie Exotique* considered themselves 'Pasteurians', and to be part of anniversary appears to have been a goal in itself.[881]

In addition to inviting physicians who had published on leprosy, a personal invitation was sent to Ludwik Rajchman, from 1921 to 1939 director of the League of Nations Health Organization. Rajchman did not find time to attend, but after consulting with the president of the Health Committee, the Dane Thorvald Madsen, he decided to send staff-member Dr. Hector Rulot.[882] The official invitation which arrived in early June was signed by the Foreign Office of a member state, the Health

That the Strasbourg conference could be interpreted as a 'coup' was pointed out in a letter from H. P. Lie to Dr. Wade, January 31st, 1931: 4. (LNHO: 8A-R5894/27029)

[881] The centenary was celebrated with international meetings as well as an international scientific exhibition (L'Exposition Internationale du Centenaire de Pasteur) which was opened on June 1, 1923, in the presence of the President of the French Republic, members of the French government, and scientific delegates from all over the world. However, when the Third International Leprosy Conference took place on July 28 – 31, most of the prominent guests had already returned home.

[882] Letter from Dr. Rajchman to Prof. Madsen, dated 28. juni 1923; letter from Professor Madsen to Dr. Rajchman, dated 3rd July, 1923. (LNHO: 12B/26213X/11346)

Committee was still expanding its scope of operations, and Rajchman was definitively open for suggestions.[883]

According to Rulot's report to Rajchman after the conference, it was "not very interesting or fruitful, and that it appeared from a practical point of view, that the 3rd Conference will not produce much effect on the various Governments concerned."[884] In his opinion, the only glimmers of hope were Dr. Lie's presentation of the anti-leprosy campaign in Norway, and Dr. Rabello's presentation of the laws and sanitary regulations in Brazil. Those were the only papers that gave insights other states could learn from.

As Rulot saw it, the main problem was that the conference had not been able to establish the geographic distribution and prevalence of leprosy throughout the world. This would be "essential" in order to:

> a) discover if leprosy is a real danger to the world, and how contagious it is;
>
> b) establish the necessity or otherwise of taking specific steps, either national or international.
>
> The appeal to the League of Nations shows that the Conference felt its helplessness, and gives the League an opportunity of stepping onto the breach and demonstrating the value of its existence.[885]

That the conference did not live up to the expectations seems to have been the consensus also among those who attended. After a discussion where two of the delegates promoted an alliance with the *Office International d'Hygiène Publique* (founded in 1907 as a continuation of the International Sanitary Conferences with

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[883] For a thorough study of the League of Nations Health Organisation, see: Borowy, Iris. *Coming to Terms with World Health. The League of Nations Health Organisation, 1921-1946.* 2009. See also: Borowy, Iris. "International Social Medicine between the Wars: Positioning a Volatile Concept". *Hygiea Internationalis.* Vol. 6, No. 2. 2007: 13-35.

[884] "Note by Dr. Rulot on the 3rd International Leprosy Conference." (LNHO: 12B-R898/29272)

[885] Op. cit. Shortly after this, Dr. Rulot moved on to a scheme on interchange of specialist medical officers working with tuberculosis, which began early in 1924. Delegates from thirteen countries, accompanied by Dr. Rulot of the headquarters staff of the Health Section of the League, visited eight countries, and studied the organization of the anti-tuberculosis campaign in each, illustrating how circulation of knowledge and international aspirations were not specific to leprosy. ("Medical News". *The British Medical Journal*. August 22. 1925: 364)

headquarters in Paris), the majority of delegates voted to seek an alliance with the League of Nations. The last three of the six recommendations were a plea for help addressed directly to the League:

> 4) The League of Nations should resume publication of the periodical "Lepra-Archives Internationales de la Lèpre".
> 5) The League of Nations should establish an international information office on Leprosy.
> 6) The League of Nations should make endeavors to secure statistics on cases of leprosy throughout the world.[886]

According to Rulot, such collaboration would be mutually beneficial. On the one hand, the leprologists were in desperate need of help and legitimacy from the League for its international aspirations to succeed. On the other hand, this would give the League's Health Committee a chance to prove its raison d'être. In June 1919 the Treaty of Versailles had directed the League to take steps in matters of international concern for the prevention and control of disease,[887] but until 1924 the Health Organization existed only as a provisional committee.

Rulot's suggested strategy from October 1923 was to start with the countries that already counted leprosy among notifiable diseases, and adopt their standards of diagnosis and surveillance. The League should contact these countries and prepare a preliminary report based on their experiences.[888] However, no initiatives could be made before the issue had been formally discussed in the Health Committee. The question of leprosy was consequently added as issue number 67 for the second session of the Health Committee, held May 7 the following year.

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[886] Resolutions and recommendations from the Third International Conference on Leprosy, Strasbourg 1923. (LNHO: 12B-R898/29272). For the full report, see Appendix 2.
[887] Article 23 (f) in Covenant of the League of Nations, adopted in Paris on April 29, 1919, states that the Member of the League "will endeavour to take steps in matters of international concern for the prevention and control of disease." (http: //cil.nus.edu.sg/rp/il/pdf/1919 Covenant o 20the League of Nations-pdf.pdf) See also: Weindling, Paul. "Introduction: constructing internationa health between wars". In: Wendling, Paul (ed). *International health organisations and movements, 1918-1939.* 1995: 7-8.
[888] Unsigned. "'Statistics on Cases of Leprosy'. Scheme for a preliminary enquiry on the notification of Leprosy in the various countries and on the available statistics of cases in response to the 6th recommendation of the International Conference on Leprosy, 28th-31st July, at Strasbourg." Dated 13.10.23. (LNHO: 12B-R898/29272/29272)

To present "the problem of leprosy" for the Health Committee, Rajchman invited Emile Marchoux, general secretary at the meeting in Strasbourg. Marchoux painted a picture of leprosy as a scourge which affected millions all over the world, that devoted scientists since 1897 had done all in their limited powers to stop the disease, and that the League needed to step in to foster global cooperation. He presented the recommendations passed at the Strasbourg meeting and urged the League to take action.

After a short discussion the League agreed to follow up on the last of the Strasbourg resolutions, namely establishing the global prevalence of the disease. "On the proposal of Sir George Buchanan, the Committee decided that the Medical Director should publish statistical information concerning leprosy and that the publication of a monograph written by a duly qualified person should be taken into consideration later."[889] As historian Iris Borowy has shown, this was in line with the League's involvement in other diseases.[890] The plea that the League would take upon itself to publish a scientific journal, which was what the leprologists gathered in Strasbourg had considered the most important undertaking, was declined. Again, the British representative Sir George Buchanan voiced the decisive argument: "It would be impossible to undertake the publication of the paper referred to above because it would create a precedent."[891] Buchannan, skeptical of the League infringing on the sovereignty of independent states, saw no need for the League to expand their activities and act as editors for a niche medical journal.

In order to publish statistical information concerning leprosy, the League needed to gather the necessary expertise. Despite the limited mandate, Ludwig Rajchman started several parallel investigations. In June 1925, he sent staff member Evald Tomanek to London to meet with BELRA. One year earlier, its secretary Reverend Frank Oldrieve had published a paper in the journal *International Review of*

[889] Extract from P. V. of the First Meeting of the 2nd Session of the Health Committee. 7. May, 1924: 2. "67. The question of Leprosy." (LNHO: 12B-R898/29272)
[890] Borowy 2009.
[891] Extract from P. V. of the First Meeting of the 2nd Session of the Health Committee. 7. May, 1924: 2. "67. The question of Leprosy." (LNHO: 12B-R898/29272)

Mission where he had estimated the number of lepers in the world to be about 3.6 million. Oldrieve argued that due to improvements in treatment, the goal of ridding the world of leprosy was within reach:

> Ten years ago it would not have been possible to speak of ridding the world of leprosy as being a practical proposition. Now, however, the conditions have so greatly altered that I believe we ought to look forward to getting rid absolutely of this terrible disease which has afflicted mankind for so many thousand years; we ought not to be willing merely to contemplate this as a desirable aim, but to set to work immediately to accomplish it.[892]

Rajchman saw BELRA as a potential partner. BELRA's initiative of sending questionnaires on leprosy to all British possessions through the Foreign-, Colonial- and India- offices in 1924 went hand in glove with fulfilling the League's mandate of providing statistical information on the disease. Oldrieve himself spent the last six months of 1924 traveling in India and the East on behalf of BELRA to get first-hand knowledge about the situation on the ground.

Rajchman did not rely on BELRAs report alone. A second initiative involved Emile Marchoux. Inspired by BELRAs initiative, Marchoux was commissioned to prepare a questionnaire on leprosy which the League then sent to the ministers of health of all countries in the world, both members and non-members of the League. The questions asked were almost a direct copy of the program at the Strasbourg conference where Marchoux had been the General Secretary: Prevalence, diagnosis, sex and age upon discovery, localization of lesions, treatment, prevention, institutions and legislation. The questionnaire underscores the commission's continuation of the leprosy conferences.[893]

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[892] Oldrieve, Frank. "Ridding the World of Leprosy". *International Review of Mission*. Volume 13, Issue 4. 1924: 595. Exactly what these 'altered conditions' entailed was not spelled out, but it seems likely that Oldrieve referred both to improved means of communication and the promise of chaulmoogra based treatments.

[893] Marchoux. *Proposed questionaire on leprosy*. Dated November 10, 1926. (LNHO: C.H./Leprosy/1). The initiative was also a recognition that the Health Section's request for "a brief statement about the number of leprosy observed the last ten years, as well as any observations on this disease in your country", sent out in November 1925 failed to produce useful feedback. The 1925-request was an addendum to a request from the Italian delegation concerning the study of prevalence and prevention of Trachoma. (LNHO: 12B-R898: various

The League in Latin America

The League of Nation's third and most consequential initiative involved the Brazilian Carlos Chagas. Since 1917 he had been the director of the medical research institution "Instituto Oswaldo Cruz" (formerly "The Federal Serum Therapy Institute") in Rio de Janeiro, and from 1920 director of Brazil's Department of Public Health. His views on leprosy were radically opposite to Rogers' insistence on voluntary treatment. Chagas was the architect behind Brazil's newly implemented anti-leprosy campaign, which consisted of compulsory notification and obligatory isolation in agricultural colonies for those who were able to work, asylums for invalid lepers, and sanatoria for rich lepers. The legislation opened for police assistance in examination and isolation of suspected lepers.[894]

In 1922 Chagas was appointed as a member of the League's Health Commission. On what seems to have been his first visit to Geneva for the Fifth Session of the Health Committee in October 1925, he gave a presentation that stressed the importance of raising a coordinated international campaign against leprosy. After convincing the committee that "the problem of leprosy" needed more attention, he was charged with preparing a report "on the prevalence of leprosy and the measures applied to prevent its spread in Latin America."[895]

At this time political relations between the League and Latin America, especially Brazil, were strained. When it was clear that Germany in 1926 would be offered a permanent seat on the Council of the League of Nations, Brazil (followed by Spain) felt bypassed and decided to withdraw from the League. Some weeks before Brazil pulled out, *Time Magazine* reflected a common view when it an editorial

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folders). The limited replies showed that more specific instruction and increased standardization was needed in order to produce comparable responses.

[894] De Souza-Araújo, H. C. "The Leprosy Problem in Brazil". *The American Journal of Tropical Medicine.* Vol. 5, No. 3. 1925: 222ff. The first agricultural colony was opened by de Souza-Araújo in Para a month before he departed on his travel around the world. To finance the anti-leprosy campaign, 'Law No. 4440, December 31, 1921', established a special fund for the prevention of leprosy in Brazil. The fund constituted 30 percent of Federal taxes from the sale of alcoholic beverages. However, the annual income of about $3.000.000 was soon used for other purposes.

[895] Report on the Work of the Fifth Session of the Health Committee, Geneva, October 8-14, 1925. (LNHO: 12B/47209/31035).

argued that "It suggests that League states may avoid unwelcome responsibilities by dropping out of the League, and yet expect to continue to reap many of the advantages of League membership by 'collaborating' in the manner of the US."[896]

In the shadow of this political conflict, Carlos Chagas was the architect behind a scheme to establish an international center for leprosy research in a partnership between Brazil and the League. In 1928 he invited Rajchman and Madsen to visit public health administrations and research institutes in Argentina, Brazil and Uruguay. As Iris Borowy has argued, the tour was made to promote the little-known League of Nations and avoid more South American countries from withdrawing. The leprosy research center was its most successful outcome.[897]

The ambition was that the leprosy center should deal with prophylactic and therapeutic aspects of leprosy, and be placed at Chagas' Oswaldo Cruz Institute in Rio de Janeiro.[898] The expenses were to be split between the Brazilian government, the League of Nations and the Brazilian philanthropist Guilherme Guinle.[899] The League's contribution would be reserved for covering the costs for international specialists visiting the research center.

Political instability, uncertainty regarding Brazil's relations with the League, along with the League wanting a second opinion on Chagas' scheme, meant that the plans were postponed until after Getúlio Vargas military coup in 1930. When the situation had stabilized, the Brazilian government offered to set up the center and put

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[896] Editorial. "The League of Nations: Brazil Out". *Time Magazine.* Monday, May 21, 1928. The United States was never a member of the league, but collaborated closely and had members on several of its committees.
[897] Borowy 2009: 214ff.
[898] In promoting the plans, it seems Chagas worked both sides of the table. On the one hand he used his position in the League of Nations to advocate the scheme towards Brazilian politicians. On the other hand, he used the politicians support to promote the center in the League. See for instance the letter from Premier and Foreign Minister of Brazil, Octavio Mangabeira from 1929, which welcomed Chagas' scheme with open arms: "…the Brazilian Government attaches the greatest importance to this scheme of international co-operation, which would include the conversion of the present classes in public health at the School of Medicine (Facultade de Medicina) at Rio de Janeiro into an international school of public health, for the technical education of medical officers in Brazil and other countries of Latin America, and the creation in Brazil of an international centre for the study of leprosy." (Letter from Mangabeira to the Secretary General Eric Drummond of the League of Nations, dated March 17, 1928. (LNHO: 8A-R5872/2634/2634; C. H. 710.))
[899] Guinle was the owner of the port of Santos, and had previously sponsored a central hospital, a research hospital and a network of dispensaries for Rio de Janeiro. (Borowy 2009: 217, note 132).

it at the League's disposal. Its main objective was outlined in a letter dated April 15, 1931:

> To undertake any work which may contribute towards the prevention of leprosy by means of epidemiological, clinical and biological research, with special reference to treatment; and by means of a specialized course of instruction open to scientists, doctors and hygienists of the countries which may desire to take advantage of this course, to promote universal co-operation in the campaign against leprosy.[900]

The center opened in April 1934 and was active until the philanthropist Guinle (appointed first Chairman of the Committee of Management) in 1939 announced that he would not renew his grant, reducing its funding by almost 50 percent. Facing this, the Brazilian government decided to pull the plug. The numerous reports in the League following up on the center contain several complaints from foreign visitors that the working language was Portuguese, that the skills in other languages were poor, and that this made it hard to fulfill the ambitions as an international reference point for leprosy research. The death of Chagas in November 1934, only six months after the center opened, also came as a blow. The activities of the center, however, merits further investigations.

An unforeseen consequence of the international research center in Rio was increased international attention to the library service of the Leprosy Prevention Department (Departemento de Profilaxia de la Lepre) in the Brazilian capital of São Paulo. Opened at around the same time as the center in Rio de Janeiro, the department worked to compile a complete catalogue of references to leprosy in research papers. By 1944 their library contained more than 100.000 references, and answered an annual average of 185.000 consultations by correspondence from all over the world. To lift the burden of replying to the individual requests, a 1.935 page bibliographic

[900] International Centre for Research on Leprosy at Rio de Janeiro. (LNHO: CH/Leprosy/9). The center was built on the same model as the International Institute of Intellectual Cooperation in Paris, inaugurated January 16, 1926, and the International Institute for the Unification of Private Law in Rome, inaugurated May 30, 1928.

index of leprosy was published in three volumes in English and Portuguese, containing about 30.000 references to literature available at their library.[901]

The League's Leprosy Commission

Although the research center in Rio was his main interest, Carlos Chagas' *Preliminary Report on the Problem of Leprosy* (1926) was also the genesis of the League of Nations' Leprosy Commission. Instead of merely focusing on Latin America, Chagas saw leprosy as an issue for all countries in the world. Based mainly on reports written by the same physicians who at the leprosy conference in Strasbourg in 1923 had argued for the League to involve itself with leprosy in the first place, as well as selected government statistics, Chagas argued that "Leprosy is a medico-social subject which demands the attention of the Health committee of the League of Nations in order that the co-operation of all civilized countries may be obtained in combating this terrible scourge."[902]

Leprosy was presented as a disease which made special appeal to both human pity and fear, and was a problem found in every part of the world. While some countries made great efforts to combat the problem, others lacked technical resources for effective prophylaxis. Especially the 'highly civilized countries' needed to step in, regardless of whether they had the disease or not. They had the necessary knowledge when it came to organizing a civil defense, and it was in their interest to stop the disease before it was introduced from abroad. The underlying threat was that if no action was taken it was just a matter of time before the problem of leprosy got out of hand. This bears striking similarities with the arguments used on a national level, for instance in Norway in the 1840s and on Hawaii two decades later.

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[901] Keffer, Luiza. *Índice Bibliográfico de lepra. 1500-1943*. Departemento de Profilaxia de la Lepre de São Paolo. Brasil. Vol 1-3. 1944-1947.
[902] Chagas, Carlos. *Preliminary Report on the Problem of Leprosy.* 1926: 1. (LNHO: 12B-R898/50806/29272)

The conclusion of Chagas' preliminary report was that the League should set up a special commission to study the problem of leprosy in all its aspect:

> I. To spread in the main centers of leprosy modern knowledge regarding the disease, in order that measures may be taken which will effectively restrict infection.
> II. To promote and assist investigation into the treatment of leprosy.
> III. To endeavor, with the aid of specialists, to elucidate the mechanism of infection, an exact knowledge of which is indispensible as the rational basis for prophylaxis.
> IV. To arrange for the exchange of specialists between countries in which leprosy exists, and organize special leprosy research centers.
> V. To promote and assist the publication of an international review, the special purpose of which would be to spread knowledge concerning leprosy.
> VI. To promote international legislation with the object of preventing the transmission of leprosy from one country to others.[903]

The report was discussed at the sixth session of the Health Committee held at Geneva from April 26th to May 1st, 1926, and in a situation where the League felt it was about to lose its foothold in Latin America, Health Committee gave full support to Chagas' plans. This meant a wide expansion of the League's ambitions related to leprosy. Even the question of establishing an international medical journal, which two years prior had been flatly declined, was now back on the agenda.

The first step in fulfilling the new mandate was to find collaborators. Chagas was charged with finding international experts to a special Leprosy Commission to tackle leprosy on a global scale. He went to medical experts who were already collaborating with the League. Five of the six members of the Leprosy Commission were already on the permanent Health Committee: Carlos Chagas (Brazil, Chairman of the Leprosy Commission), Surgeon General Hugh Smith Cumming (USA), Colonel J. D. Graham (India), Doctor Thorvald Madsen (Denmark, also President of the Health Commission) and Doctor Ludwig Rajchman (Poland, also Medical director of the League). The last member of the Leprosy Commission, Professor Maturo

[903] Op. cit.: 9.

Nagayo (Japan), did not have a prior position with the Health Organization but was already involved in the League's exchange program for foreign medical researchers through courses at his Government Institute for Infectious Diseases at Tokyo.[904] All the members were medically trained, all had high-ranking positions in their national health systems, and all had prior experience with leprosy work.

The first official meeting of the Leprosy Commission was held in Paris, on May 14, 1928. Until then the League had been operating more or less as an extension of the International Leprosy Conference. Now, the League itself would be the driving force behind international collaboration regarding leprosy.

However, already the first preliminary exchange of views showed that collaboration would prove difficult. Chagas, Cumming, Nagayo and Madsen argued that the League should promote state segregation. Rajchman was undecided, while the Public Health Commissioner with the Government of India, Colonel J. D. Graham, insisted that the situation on the ground was too differentiated for a 'one size fits all'-approach. Instead, Graham argued, leprosy work must be country-specific and any expenditure on public health measures should correspond to the relative importance of the disease. Based on his experience in India, leprosy could not be considered a major threat to public health, and segregation was both impractical and counter-productive.

To sidestep the potential conflict, the commission initially decided that the main focus should be on collecting knowledge, not on how it should be applied. This was based on the same line of arguments that resulted in the international leprosy conferences. Still, the means differed: The League's joint program focused on facilitating research and training in early diagnosis, methods of communication and treatment.[905] This should be done through international research centers, the already mentioned center in Latin America and one more to be situated in the Far East.

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[904] League of Nations Health Committee: Minutes of the Ninth Session. LNHO: C. 107. M. 38. 127. III (C. H./9th Session/P.V), 1927: 49.

[905] "Communication from the Commission for the Study of Leprosy to the League of Nations Health Organization". Dated Geneva, May 24, 1928. (LNHO: C.H./Leprosy/3, found in LNHO: 8A-R5887/4621/4621).

Most importantly, the committee agreed to hire a secretary. They chose the French physician and bacteriologist Dr. Etienne Burnet, who earlier had collaborated with the League on scientific cooperation regarding health in post-war Poland and Russia. He had also worked as deputy director at the Pasteur Institute in Tunis but left the position after a conflict with its main director Charles Nicolle. Since 1926, Burnet had been a member of the *Société de Pathologie Exotique.* From October 1, 1928, Burnet was officially employed by the League of Nations staff.[906] He was to be the only one working full-time for the Leprosy Commission.

Burnet did not start from scratch. Since 1925, scattered statistics on leprosy was reported from the Oriental Bureau of Epidemiology of the League of Nations (more often referred to as the Far Eastern Bureau).[907] De Souza-Araújo, who in the beginning of September 1926 met Ludwig Raichman in Geneva, had prepared a 31-page document detailing his findings from traveling around the world.[908] There were also piles of responses to Marchoux' questionnaire, giving details both from states, institutions and missionary organizations.[909] Before Burnet arrived, it appears that these reports had more or less been collecting dust. Burnet also soon realized that not all states had bothered to answer the requests for information. Finally, although

[906] Burnet would keep the position as secretary for the Leprosy Commission until August 31, 1936, when he was appointed director at the Pasteur institute in Tunis following the death of Charles Nicolle.
[907] The office was opened in 1925 to facilitate the exchange of epidemiological intelligence from 35 ports in twelve countries. During the first years its network of epidemiological intelligence had expanded to 76 ports in 27 countries. It was soon authorized to act as a center to coordinate scientific research and instruction for medical statistic, and to distribute information on international health work. (Borowy 2010: 15.)
[908] De Souza-Araújo, H. C. *Leprosy as a world's sanitary problem*. October 5, 1926. (LNHO: 12B-R898/52570).
[909] There were also letters to the League from individuals wanting to contribute. On December 26, 1923, Cathrine Blomefield from Johannesburg, South Africa, contacted the League with a special print of her *Thesis on the Prevention of Leprosy* (1923), an 11-page pamphlet which argued that leprosy "is produced by the lack during a lifetime of a diet containing animal fats and fresh meat" (1923: 5). Blomefield, the daughter of the English Major W. B. Arnison of Penrith, requested the Health Director of the League to promote her as a candidate for the Nobel Prize in medicine for 1924. Rajchman acknowledged receiving the document, but took no action. Over the following month, Blomefield wrote more letters, including a three-page "Part II" of the thesis, and asking to be nominated as a member of the Health Commission. Frustrated by the lack of support, the correspondence ended with Blomefield wanting the documents returned: "If the information in typed manuscript is not to be used may I ask for its return, (properly stamped please) as the labour of copying is a consideration, and the Leagues lack of interest in the prevention of leprosy is not so great as to cause me to spend another £23 in printing." (LNHO: 12B-R898//33565x/29272)

Marchoux had given the recipients detailed instructions, the replies were not comparable.

In order to learn about the leprosy situation around the world, it was not enough to read documents. Almost the whole of 1929 he did as de Souza-Araújo had done some years earlier: He went on the road. First, Burnet and Chagas visited leprosy centers in Europe. Then Burnet left on a five-month journey to South America to follow up on Rajchman and Madsen's journey the year before. The goal of the journey was both to promote the League and to gather knowledge about leprosy: Incidence, legislations, provision of leprosaria and dispensaries, methods of treatment and the latest research efforts. Burnet found that while the public health administrators were positive and welcoming, they lacked trained personnel and a system which could produce basic health statistics.[910] In lectures on leprosy, Burnet recommended isolation of contagious cases, rigorous separation of children from leprous parents and the creation of leprosy societies to study the etiology, pathology and bacteriology of the disease.[911] He was also looking for potential sites and collaborators for the planned international research center, and ended up agreeing with Chagas that his set-up in Rio de Janeiro would be best suited for a center of scientific, epidemiological, therapeutic and experimental studies into leprosy.

A matter for technical experts

With Burnet traveling, nothing much happened in the Leprosy Commission. This was a source of frustration. At the first meeting in Paris, each member had been charged with preparing reports on the research taking place in their home countries. Graham commissioned Muir from BELRA and the School of Tropical Medicine and Hygiene

[910] Borowy 2009: 218. For Burnet's report of the travels in Europe, Latin America and the Far East, see LNHO: C. H. 887.
[911] Obrégon 2003: 185.

in Calcutta to write a bibliography on leprosy research in India from 1900 to 1928, but initially refused to share it with the League:

> We also here have prepared a bibliography of leprosy research in India and I think it is a very complete one. In view, however, of the fact that I have received no information of any kind from Dr. Burnet, Secretary of the Leprosy Sub-commission, and I consider that some information at least is due from the Secretary of the Commission to its members – I do not propose to send anything meanwhile to the League until I hear more. The leprosy discussions reported in the proceedings of the Health Committee are not very illuminating and it is extremely difficult for me to form any idea of what is happening.[912]

Only after Ludwig Rajchman stepped in and pointed out that Graham himself had been present at the thirteenth session of the Health Committee when Burnet's journey had been approved (in the letter he even attached the minutes of the meetings), did Graham apologetically agree to forward the report containing 192 references to leprosy research in India.[913] While Chagas officially was the leader of the commission, and Burnet its secretary, it appears that Rajchman was involved in most, if not all, decisions. Burnet consistently asked for his consent before taking any action. Apart from initial recruitment and promoting the research center in Brazil, Chagas does not seem to have been deeply involved with the work of the commission.

In addition to lack of communication and disagreements on the merits of segregation, the question of missionaries was to become another contested issue. The replies to Marcheux' questionnaire showed much of the leprosy work around the

[912] Letter from Colonel J. D. Graham to Dr. Frank Boudreau, Chief of the League of Nations Epidemiological Intelligence and Public Health Statistical Service, dated Simla, July 12, 1929. (LNHO: 8A-R5887/14019). The report from Muir was dominated by the 'Calcutta school': About a quarter of the references, 52 papers, were written by Muir; 21 by Rogers. (Muir, Ernest. *Researches on Leprosy in India (1900-1928*). (LNHO: C.H./Lèpre/6)).

[913] Letter to Graham from Rajchman, dated Geneva August 27, 1929. (LNHO: 8A-R5887/14019). Five days earlier, acting director of the League's Health Section Frank Boudreau had written a letter to Graham which stressed that there had been no other meetings in the commission, and that since there was no news, there had been no need for further communication. That Rajchman himself stepped in might suggest that he feared the British would withdraw from the commission altogether. In his letter, Rajchman explained that Burnet would use the information gathered to write a general report to the Leprosy Commission and the Health Committee, that this would be an important report, and that he personally hoped that the Indian bibliography would be included.

world was not considered a state issue, but was instead left in the hands of religious orders and missionary organizations. In 1924 the Mission to Lepers, in collaboration with 33 missionary agencies, provided for the maintenance of 96 institutions for lepers in twelve different countries.[914] Many of the medical missionaries had decades of experience in the field, and missionary organizations had their own networks for circulating knowledge, including journals, newsletters and conferences for asylum directors. According to BELRA's first secretary Frank Oldrieve, himself a former missionary:

> Missionaries have always been in the forefront in carrying on leper work, and they will probably still lead. (…) it is generally recognized that the best leper work in any country is that which is done under missionary superintendence. It needs a man or woman who has been touched by the hand of Christ to a deep sympathy with those who are in the greatest need successfully to carry on work amongst these poor outcasts.[915]

Should missionaries be invited to the League of Nations Leprosy Committee?

The most vocal opposition came from the American Hugh Smith Cumming. Since March 3, 1920, he had been Surgeon-General of the United States Public Health Service, as well as director of the Pan-American Sanitary Bureau. In 1921 he had been in charge of nationalizing the National Leprosy hospital in Carville, USA, which until then had been administered by the missionary organization *Daughters of Charity* for almost three decades under the name Louisiana Leper Home.[916] In Cumming's experience, most missionaries lacked medical training and were therefore unqualified to give advice: "My attitude has been that we should have on the Leprosy Commission men whose views on the etiological and medical phases of the subject

[914] Oldrieve 1924: 597. The remark was made addressing a missionary audience. In total the Mission to Lepers ministered to some 18,000 lepers and un-tainted children. Most of their institutions were small: 31 of the 46 asylums were in India had fewer than 100 inmates, and the total of 5,428 lepers institutionalized in India was about the same as Culion alone. (de Souza-Araújo 1929: 241)
[915] Oldrieve 1924: 600-601.
[916] Moran 2007: 123ff.

are to be regarded as quite sound."[917] This meant ignoring both the European Mission to Lepers and the American Mission to Lepers (whose general secretary Dr. W. Danner was a "Doctor of Divinity"), despite their active involvement in various leprosy institutions around the world. Leprosy was a public health issue, and missionaries were with their religious perspectives considered unreliable.

Cumming also explicitly opposed involving Sir Leonard Rogers and the British Empire Leprosy Relief Association. While BELRA presented itself as a voluntary scientific organization, it collaborated closely with Mission to Lepers and several of its leaders were former missionaries. In both meetings, letters and telegrams, Cumming consistently referred to Rogers' views concerning therapeutics as "unsound". The sources do not indicate why, but it could be argued that Rogers' insistence that leprosy was curable and not highly contagious, and that the best way forward was voluntary treatment in out-patient clinics, was a direct challenge to the policies of segregation that Cumming had implemented in the United States and that he also promoted through the Pan-American Sanitary Bureau. That Rogers from time to time jazzed up his documents with peculiar proverbs made it easy to label him as 'unscientific'. His Memorandum to Rajchman from 1925, for instance, concluded like this:

> In South Africa part of the money being saved by release of uninfective cases is to be devoted to research, and when we remember how much advance has already been made within a single decade, we should press on with this work in the assured faith that if you cast your bread upon the waters you shall surely find it after many days.[918]

[917] Letter to Dr. C. L. Park, Assistant Director, League of Nations Health Section, from Surgeon-General H. S. Cumming, dated Washington, November 15, 1929. (LNHO: 8A/16440/4621) Cumming was also involved in organizing de Souza-Araújo's journey, and supplied him with a letter of introduction. (De Souza-Araújo 1929: 7)

[918] Rogers, Sir Leonard. "Memorandum on the prevalence and prophylaxis against leprosy in the British Empire, based on replies to the questionaire of the British Empire Leprosy Relief Association; with suggestions for dealing with the problem." 1925: 29. For more on BELRA and its collaboration with Christian missionaries, see: Worboys 2000: 207-218.

The rest of the commission agreed that leprosy was a 'technical' issue, and that this excluded missionaries. Despite Cumming's repeated protests, Rajchman did indeed invite Rogers to be part of the Leprosy Commission. After all, he was considered Britain's leading expert on leprosy, Britain was an important member of the League and a large proportion of the world's lepers lived in the vast British Empire. Tuesday October 15, 1929, Rajchman sent a telegram to all members proposing Rogers as a new affiliate. When Cumming's telegraphic reply ("Rogers views concerning therapeutics leprosy considered unsound here suggest marchoux unna or mccoy = cumming ="[919]) arrived in Geneva at 19.00 in the evening two days later, a personal invitation to Rogers had been sent by telegram only a few hours earlier. Rogers, however, declined the invitation due to lack of time.[920] Unknown to Rogers, this way the commission dodged a potential conflict.

In total the committee was expanded with four new members: Dr. G. Alexander Mitchell (Chief Health Officer for the Union of South Africa, Pretoria), Professor Ernest Muir (School of Tropical Medicine and Hygiene, Calcutta, India), Dr. H. Windsor Wade (Chief Pathologist of the Public Health Service of the Philippine Islands and Medical advisor to the Leonard Wood Memorial for the Eradication of Leprosy, Manila), and Dr. N. E. Wayson (surgeon, Public Health Service of the United States of America, Leprosy Investigation Station, Honolulu, Hawaii).[921] The new members had similar background as the existing six: All of them were trained physicians, all of them had experience from leprosy work, all of them were in leading positions in their home countries, and none of them were missionaries or persons with other academic backgrounds.

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[919] Telegram from Cumming to Rajchman dated Washington, October 17, 1929. (LNHO:8A-R5887/15292/4621)

[920] This was not the first time Rajchman tried to recruit Rogers to collaborate with the League. Two years prior to the official meeting in Paris, Rajchman had invited Rogers on behalf of BELRA to attend a preliminary meeting with Marchoux and Chagas. Then, too, Rogers declined: "I should like to be able to comply with the request, but I very much fear I should be of no use to the Committee, as unfortunately owning to a bad ear for sounds I can neither speak or understand French or any other language than English, and when I attended the Strasbourg Leprosy Conference I was quite unable to follow either the papers or the discussions, so unless Professor Chagas has a good knowledge of English (which I learned at the leprosy conference Professor Marchoux has not) it would be quite useless for to go over Paris confer with them." (Letter from Rogers to Rajchman, dated London, May 22, 1926. (LNHO: 12B-R898/51481).)

Leprosy in the Far East

Burnet stayed in Geneva less than four months before he set off on another journey, this time to the Far East. Leaving Geneva on December 20, 1929, Burnet's itinerary shows that on the way he visited Calcutta, Singapore, Saigon (now Ho Chi Minh City, Vietnam), and the Philippines before arriving in Japan. From there he proceeded to Hawaii, went on to San Francisco, crossed the United States by train and finally returned to Europe departing from New York. The main goal was Osaka, where the next meeting of the Leprosy Commission was planned to be held in conjunction with the quadrennial Congress of the Association of Medical Science of Japan in April 1930. On top of his agenda was to assess the possibility of establishing a second international research center in Japan. Burnet's requirements were that it must be placed in a country where leprosy was endemic and clinical material "as abundant as possible". Furthermore, a center had to build on an existing institution near a large town with a university and scientific institutions; it should have access to laboratories for therapy, bacteriological tests and pathological anatomy (including autopsies), and be ready to accept an international staff on temporary basis and provide practical training.[922] Japan qualified on all accounts.

By the time Burnet set sail for the planned meeting in Osaka, leprosy had long been on the public agenda in Japan. Three decades after Japan's opening to the West in 1854, Christian missionaries had started to erect 'leper houses'.[923] A national survey in 1904 indicated that the number of lepers was increasing dramatically, and that there were more than 30.000 lepers in the country. A Leprosy Prevention Law was enacted in 1907, which divided the country into five health regions. In 1909 a public sanatorium was established in each region to accommodate vagrant lepers.[924]

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[921] LNHO: C. H./Lèpre/8.
[922] Burnet, Etienne. *Report on the Programme of work of the Leprosy Commission.* 1930: 9-10. (LNHO: 8A-R5893/16680/6714; LNHO: C.H.887(a).)
[923] Gould 2005: 133-141.
[924] Ohtani, Fujio. *The Walls Crumble. The Emancipation of Persons Affected by Hansen's Disease in Japan.* 1998: Chapter 2. The book is a translation from Japanese of "The History of the Repeal of the Leprosy Prevention Law: Love Conquers, the Walls Crumble", published in 1996 to celebrate the repeal of the "Leprosy Prevention Law".

Like Brazil, Japan was on a path towards forced segregation. In 1931, the laws were expanded to encompass all lepers: "Any and all patients who suffered from leprosy would be institutionalized in a state or prefectural government administered sanatorium, and the expenses involved in such institutionalization would be covered by the central or prefectural governments."[925] The establishiment and expansion of institutions went hand-in-hand with medical research. Before Burnet left from Geneva, Maturo Nagayo provided his fellow members of the Leprosy Commission a two-volume index of 348 commented research papers on leprosy produced in Japan between 1890 and 1928.[926]

Burnet was thrilled by what he found in Japan. "The organisation of the leprosaria (in Japan) is one of the best in existence, and each establishment has a remarkably well-equipped laboratory attached to it. All the dermatological clinics of the universities have consultation clinics for lepers."[927] However, unlike in Brazil, discussions with the Japanese Government were still at a preliminary stage. The plan was that the strategy should be discussed in conjuncture with the meeting in Osaka, where the Leprosy Commission would be present in full. But despite the Commission at this point only consisted of six members, coordination across the globe proved complicated. Chagas, Cumming and Rajchman found it impossible to fit the meeting in their schedules, and on February 24, the decision was made to postpone the meeting since only Madsen, Graham and Nagayo would be able to attend.[928] By then, Burnet was well underway. Surprisingly, the highlight of the journey turned out not to be Japan, but rather the Culion leper colony on Philippines.

Inaugurated in 1906 by Victor G. Heiser, then the Director of Health of the Philippines, Culion had initially been a remote island where leprosy sufferers had

[925] Ohtani 1998: 61.
[926] Nagayo, Maturo. *Recherches sur la lepre au Japon (1890-1928)*. (LNHO: C.H./Lépre/5).
[927] Burnet, Etienne. *Report on the Programme of work of the Leprosy Commission*. 1930: 13f. (LNHO: 8A-R5893/16680/6714; LNHO: C.H.887(a).)
[928] Letter from the Deputy Secretary-General of the League of Nations to the Director of the Japanese Bureau to the League of Nations, dated Geneva February 24, 1930. (LNHO: 8A-R5887/13191/4621)

been dumped and left to die, out of sight – out of mind.[929] During the first four years of its existence, between 1906 and 1910, 5.403 lepers were sent to Culion: 3.154 died and 114 ran away. The worst year was 1908 with more than three funerals per day. 1.603 new patients were admitted and 1.221 deaths were registered.[930] By the mid-1920s, it was also the largest leprosy research center in the world with almost six thousand patients.

The architect behind the research facilities was Herbert Windsor Wade. In 1921 Wade had been recruited from the Philippine General Hospital and appointed chief pathologist and acting chief physician at Culion by the newly appointed Governor-General to the Philippines, Leonard Wood. Both Wood and Heiser, now director of the International Health Division of the Rockefeller Foundation, strongly promoted the use of Culion as a center for isolation and treatment of leprosy patients. Wade was given liberal budgets.[931] In the first year Wade hired eighteen physicians and twenty-one trained nurses – in addition to the nuns who were already working there. New buildings were erected, and experimental treatment tested on a grand scale. By 1925, about one third of the Philippine health budgets were spent on leprosy and it had spawned public criticism that expenses were out of proportion to the

[929] Victor G. Heiser would later argue: "There were twelve hundred new cases developing every year and practically nothing was done about them. (…) Segregation is always cruel. We did not want to separate husband and wife or children and parents. But segregation is cruel to relatively few whereas non-segregation threatens an entire people. I believed that isolation not only protected others from contracting leprosy but, furthermore, was the most humane solution for the leper himself. Instead of being shunned and rebuffed by the world, he could have an opportunity to associate with others of his kind in pleasant relationship." (Heiser 1936: 227.)

[930] De Souza-Araújo 1929: 154, Table No. 26.

[931] Long, Esmond R. "Fourty Years of Leprosy Research. History of the Leonard Wood Memorial (Americal Leprosy Foundation) 1928 to 1967." *International Journal of Leprosy*. Vol. 35, No. 2. 1967: 239-253. Victor George Heiser was himself a physician specializing in tropical medicine with a special interest for leprosy. As I showed in Chapter 3, his encouraging results of treating some of the patients with the 'Heiser-Mercado'-mixture, a blend of chaulmoogra and camphor oils and resorcinol, was important in redefining leprosy from a herbal palliative to a curative drug. (*US Public Health Report*, supplement no. 20, 1914.) Acting as Associate Director of the International Health Division of the Rockefeller Foundation, Heiser was also the main sponsor of de Souza-Araújo's journey in 1924-1927. Heiser was also an avid traveler, who reportedly circled the world 17 times on his medical missions. His travels are recounted in his autobiography *An American Doctor's Odyssey: Adventures in Forty-Five Countries* (1936). This became the sixth best-selling non-fiction book in the United States in 1936, with more that half a million copies sold. (Wall, Rosemary. "The International Health Division's Views on Nursing Practice, Policy and Education in British Malaya." *Rockefeller Archive Center Research Reports Online*. 2010: 2. (http: //www.rockarch.org/publications/resrep/wall.pdf)

number of those affected.[932] It was also increasingly clear that the military-inspired strategy of eradicating leprosy from one island at the time through segregation (see Chapter 4) had failed.

Inspired by Muir's Calcutta school, a national "Dual Plan" was suggested by the Culion Medical Board in September 1925, which was led by Wade. The plan consisted of new cases being assigned to out-patient treatment stations in the principal leprous regions, and only those whose treatment failed were to be transferred to the leper colony of Culion. This would, over time, cut costs. However, lack of funds made it impossible to establish the necessary number of out-patient treatment stations. Besides, although Wade and Chief Physician Casimiro B. Lara at Culion were able to present cures from 15% to 20% of advanced cases, the majority remained beyond treatment.[933]

Instead of cutting the expenses, Wade's solution was to look for alternative funding. Dorothy Paul Wade (Wade's wife) moved back to the United States in 1925 in order to organize a fundraising-campaign. The goal was to rise two million dollars for research and treatment. She recruited Pierre Burgess from the New York fund raising firm "Ward, Wills, Dreshan and Gates", and together they set up the "American Committee for the Eradication of Leprosy". Like BELRA, it had a high ranking executive committee.[934] Until then, the model of private fundraising was used primarily by missionary organizations. To avoid stepping on their toes the committee agreed to confine their attention to medical research, leaving the welfare field open

[932] Chapman, Ronald Fetts. *Leonard Wood and Leprosy in the Philippines. The Culion Leper Colony, 1921-1927.* University Press of America. 1982: 91ff. In comparison, approximately 30,000 Filipinos died from tuberculosis annually. (Chapman 1982: 105).

[933] Obregón 2003: 188. They did however register 'improvement' in a majority of cases. More on treatments in Chapter 3.

[934] The executive committee included James G. Harbord (former Chief of Staff of the United States Army and Chairman of the Board of the Radio Corporation of America), Hemry L. Stimson (Governor General of the Philippines after Wood, and later US Secretary of State and Secretary of War), Charles Evans Hughes (former Secretary of State and later Chief Justice of the Supreme Court), Owen D. Young (Chairman of the Board of the General Electric Company and former Chairman of the Board the Radio Corporation). Also involved were William Howard Taft (Chief Justice of the Supreme Court and former President of the United States), and George Horace Lorimer (editor of the Saturday Evening Post).

for voluntary organizations such as the American Mission to Lepers who by then had expanded from Latin America to also engage with leprosy work in Asia.[935]

In the midst of the five year fundraising campaign, in 1927, General Wood died during surgery in the United States.[936] The complete organization was set up in New York in his name as a nonprofit organization: "The Leonard Wood Memorial for the Eradication of Leprosy". Their mission statement was "to carry on, maintain and support laboratory investigation, clinical observation and all manner of research with respect to the disease leprosy; to disseminate information concerning the source, diagnosis, treatment and prevention of leprosy". Burgess was made its director in 1928 and became the first President in 1930; Mrs. Wade served as Permanent Secretary of the Board, while Mr. Wade was officially hired as Medical Director in January 1931.

When Burnet arrived in the Philippines and was welcomed by H. W. Wade, the construction of laboratories at Culion and Cebu was well underway. More than four thousand patients took part in clinical trials for new treatments. In 1930, the journal of the Philippine Islands Medical Association reported that 1.600 patients were rendered bacteriologically negative over a period of seven years, and were consequently allowed to leave the leprosaria.[937]

In addition to research and the national leprosy campaign, Wade believed that international collaboration and coordination of efforts were necessary to fulfill the goal of eradicating the disease. With two million dollars pledged from over fifty thousand subscribers in the United States, the Leonard Wood Memorial had the

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[935] Long 1967: 241-2. Several missions were also actively engaged at Culion, illustrating how religion and science was in no way mutually exclusive when it came to practices towards leprosy sufferers.

[936] According to de Souza-Araújo, the death of Wood was the main reason why his *Société internationale de Leprologie* remained inactive: "this sad event having happened in the UNITED STATES in July 1927, caused some trouble amongst the official elements of that remote country [The Philippines], and the collaboration works between them, Europe and ourself to strengthening the basis of the International Society of Leprology suffered an awfully long interruption, of now two years, without any improvement of the situation." (Souza-Araújo 1929: 388, dated September 1929.)

[937] Between 1922 and 1930, 1.845 individuals were released on parole or discharged after consistently testing negative for the presence of the leprosy bacillus for at least two years. During the fifteen years prior, mere 47 persons were discharged for similar reasons. (Report from Jacobo Fajardo, Director of Health of the Philippine Health Service, to Burnet, dated March 4, 1932, p. 28. (LNHO: 8A-R5931/36851/29090))

finances to move the scheme forward.[938] When Burnet explained that the next meeting of the League's Leprosy Commission was intended to be held in the Far East, Wade saw the opportunity of including the League's members in his scheme. The goal was the same as de Souza-Araújo's: To facilitate international collaboration through an international leprosy organization that could publish an international medical journal. The way forward would be to invite the League's Leprosy Commission along with key researchers to a prolonged meeting in Manila, with all expenses covered by the Leonard Wood Memorial.[939]

After Burnet's visit, Wade started contacting people in his network, asking them to suggest persons to invite to the planned Manila meeting. As he stated in the official invitation: "The attendance will be limited to those having had a considerable personal experience with leprosy and its problems."[940] Although officially the initiative came from the Leonard Wood Memorial alone, the Manila meeting was scheduled for early January 1931 so that the members of the League of Nations Leprosy Commission in personal capacity could go there directly from their meeting, which by then had been rescheduled for Bangkok in December 1930.

Each name on the list of attendees was discussed and approved by Wade on behalf of the Leonard Wood Memorial, Victor Heiser on behalf of the Rockefeller Foundation and Etienne Burnet (in collaboration with Ludwig Rajchman) on behalf of the League of Nations. Again, the question of missionaries and the position of Sir Leonard Rogers were controversial. After receiving a list of potential participants from Ludwig Rajchman containing both the names of Muir and Rogers, Wade wrote in reply to Burnet: "from his [Rogers] writings one can but gather that his mentality

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[938] F. P. G. "Special Correspondence: The Leonard Wood Memorial for the Eradication of Leprosy". *Science.* June 9, 1933:562-562.

[939] This was not a new suggestion. Already in April 17, 1925, the secretary of BELRA, Frank Olderve had in a letter to Dr. Tomanek of the League of Nations suggested pursuing a similar strategy: "I have for some time had in mind that before very long an attempt ought to be made to get together half a dozen men, or perhaps a few more than that, who are conversant with leper work in various countries where a good deal is being undertaken, so that we might discuss the question of dealing with the whole problem on a world basis. As a matter of fact we have had a little talk about it here, and we rather strongly feel that in the near future it would be a very good thing if a small conference could be convened, perhaps here or in Geneva, to deal with this matter, and as far as we can be of service we should be delighted to do everything that we can." (LNHO: 12B-R898/42389/2942).

has not improved of recent years (…) he seems to be little more than a propagandist for Muir's work. These two may be expected to argue and vote as one." Wade also blocked the attendance of missionaries: "they would, I fear, burden our conference with non-contributors to technical discussions."[941]

An unprecedented compromize

After the cancellation of the Osaka-meeting, the second meeting of the Leprosy Commission was held in Bangkok during the Eight Congress of the Far-Eastern Association of Tropical Medicine on December 8 to 12, 1930. To hold the meeting in concert with a large medical conference was mainly a question of having experts close at hand and being able to quickly disseminate the outcomes, but was also a way to cut the costs. As Chagas himself was unable to attend, professor Bernhard Nocht, Director of the Institute of Tropical Diseases in Hamburg, was invited to be the president of the meeting.[942] There, finally, Burnet reported on what he had learned during his travels around the world.

> …a whole complex of circumstances was hampering progress in the study and prevention of leprosy, viz., insufficient organisation, unsystematic use of resources, uncertainty on points of paramount importance for the establishment of the bases for prophylactic work, and a certain lack of contact and agreement between medical men and research workers in different countries.
>
> (…)
>
> Thus the question of segregation and prophylaxis still remains a subject of controversy between advocates of freedom on the one hand, and of coercion on the other. The results of the enquiry which included the consultation of a large number of leprologists, show that any

[940] LNHO: 8A-R5914/20600/20600. Discussions on practicalities and whom to invite are also found in this folder.
[941] Letter from Wade to Burnet, dated Manilla September 2, 1930. (LNHO: 8A-R5914/20600/20600)
[942] Nocht was for a time considered the post of director of the planned International Centre in Rio de Janeiro, and six months after the Bangkok meeting he was sent there to inspect the organization of the center. (LNHO: 8A-R5872/19117/2634). The Brazilian government, however, insisted the post was to be given to the philanthropist Guinle as a reward for donating half of its budgets.

> meeting of experts would be divided into two camps; those who may be described – by analogy with another problem of social hygiene – as partisans of abolition and those who are in favour of regulation. The concessions which those two schools are obliged to make in the course of practical work prove that they have some common ground and justify the hope that they would come to an agreement upon a programme of work.
>
> We have given our reasons for thinking the present time is ripe for international co-operation for the prophylaxis of leprosy. Leprologists of all countries have welcomed the Health Committee's action in regard to this matter and it is no exaggeration to say that they hope and expect that the Leprosy Commission will promote the organization of concerted action, even becoming to some extent a centre for this work.[943]

The first point on the agenda was establishing international research centers in Rio de Janeiro and Japan. The plan was that the two centers should provide training for leprologists from all over the world. The workers at the centers were in turn required to spend time in the research centers in Calcutta, Culion and on Hawaii to build on their experiences. In addition, the centers should address the contested question of treatment. Access to a large number of patients would make it possible to follow up on their work with comparative experiments with drugs such as the chaulmoogra derivates. While the center in Brazil was in operation between 1934 and 1939, the research center in Japan was never put under the auspices of the League. Planning had not come to an end when Japan on March 27, 1933, in response to the international condemnation of the occupation of Manchuria in September 1931, announced its withdrawal from the League of Nations.

The next point on the agenda was Burnet's draft report on prophylaxis. All agreed to the need for closer collaboration around the world and more standardization on methods and terminology. In an attempt to avoid historical connotations of persecution, both camps agreed to instead of "segregation" use the term *isolation*, and that further work was necessary in order to reach a global classification of especially diagnosis and treatment. Beyond that, the commission was split. One group focused

[943] Burnet, Etienne. *Report on the Study Tour of the Secretary of the Leprosy Commission in Europe, South America and the Far East.* January 1929-June 1930. Geneva. 1930: 1-2. (LNHO: 8A/R-5893/16680/6714)

on special treatment, the other on isolation, mirroring the debate between de Souza-Araújo, Rogers and Muir discussed at the beginning of this chapter, as well as in the end of Chapter 4.

The final report of the commission was a compromise. It recommended simple and elastic laws focusing on notification and isolation of bacteriologically positive cases in state facilities in accordance with the conditions in the particular country. Based on the premise that most of those affected caught the disease as children, they also recommended that children of lepers should be removed from their parents and periodical examination at schools. These measures were to be supplemented by education and propaganda aimed at the general public, both to recognize the disease in its earlier stages and to prevent new cases.[944] The widely distributed report on prophylaxis was arguably the most important document the League's Leprosy Commission produced (see Appendix 4).

After presenting their conclusions in a plenary meeting of the congress of the Far-Eastern Association of Tropical Medicine, the members of the commission (apart from Mitchell who had to return to South Africa) traveled together to Manila to attend the Wood Memorial conference from January 8 to 23, 1931. By attending in personal capacity the Memorial could cover all expenses and the question of acquiring an official invitation from the Philippines was bypassed.

The Manila conference took off where Bangkok ended, but now with 23 members.[945] It was reported back to the League of Nations Health Commission in full

[944] The outcome and summary of the discussion is found in LNHO: 8A-R5930/30632/29090.

[945] The complete list of attendees was as follows: Dr. Etienne Burnet, Secretary of the Leprosy Commission of the League of Nations Health Organisation (Geneva); Dr. Robert G. Cochrane, Secretary of the British Empire Leprosy Relief Association (LNHO: don), Mr. H. I. Cole, Chief Chemist at Culion Leper Colony (Philippine Islands); Dr. J. Fajardo, Director of Public Health at the Philippine Islands; Major-General J. D. Graham, Commissioner for Public Health with the Government of India; Dr. G. Gushue-Taylor, Superintendent of the Mackay Memorial Hospital (Formosa , later Taiwan); Dr. V. G. Heiser, Director for the Far East of the Rockefeller Foundation (New York).; Dr. Lee S. Huyzenga, Superintendent of the Mission Hispital in Jukao, Kwangsi (China); Dr. H. Joyeux, Director of the Public Health Bureau in Hanoi (Tonkin, later part of Vietnam); Dr. A. N. Kingsbury, Director of the Research Institute in Kuala Lumpur (Federate Maly States, later Malaysia); Dr. P. H. J. Lampe, Director of Public Health (Surinam); Dr. C. B. Lara, Principal Medical Officer at Culion Leper Colony (Philippine Islands); Dr. A. C. Leroy des Barres, Inspector-General of the Public Health Service (French Indo-China, later Vietnam); Dr. J. Lowe, Medical Inspector at Dichpali Leprosy Hospital (India); Dr. J. L. Mazwell, Lister Institute of Medical Research in Shanghai (China); Professor Ernest Muir, Leprosy Research Laboratory at Calcutta School of Tropical Medicine and Hygiene (India); Dr. E. E. Neff;

as an extension of the Bangkok meeting. Instead of formal papers, the whole time was devoted to discussions. The conference was split in eleven sub-commissions dealing with definition and description of lesions, clinical questions, prophylaxis, classification of leprosy, special treatment, program of research, and the foundation of an International Leprosy Association and an international medical journal. The committees prepared memoranda which in turn were discussed by the whole session before being incorporated into the findings of the committee.[946]

Again, the two schools of thought were present, and again the conference made unprecedented efforts at reaching compromises. Over two weeks, which included a lengthy visit to the Philippine leprosy institutions of Cebu, Zamboanga, Iloili and Culion, the members had time to get to know each other's views and find a common ground. There was unanimous agreement on the need for international interchanges and study tours, standardization of methods and terms used in recording and reporting leprosy work. The conference gave its full support to the Leprosy Commissions Prophylaxis report and that the League's Leprosy Commission should encourage and coordinate research.[947]

Relating directly to *La Société Internationale de Leprologie*, the organization that was set up by de Souza-Araújo in 1926 but never had started any practical work, the conference decided to establish a new organization using *La Société* as a model.

Medical Inspector at Mogokai Central Leper Hospital (Fiji Islands); Dr. Bernhard Nocht, Geheimer Medizinalrat, Director of the Institute of Tropical Diseases, Hamburg (Germany); Dr. M. Ota, Tohoku Imperial University, Sendai (Japan); Dr. J. N. Rodriguez, Inspector of Treatment Stations for the Public Health Service (Philippine Islands); Dr. J. C. Tull, Government Pathologist at the Singapore Straits Settlement (now Malaysia); Dr. H. Windsor Wade, Chief Pathologist of the Public Health Service of the Philippine Islands and Medical Director of the Leonard Wood Memorial for the Eradication of Leprosy (Philippine Islands/New York); Dr. N. E. Wayson, Surgeon at the Leprosy Investigation Station on Honolulu for the Public Health Service (United States).

[946] Cochrane, Robert G. "The Campaign against Leprosy: International Conference in Manila." *The British Medical Journal.* April 18. 1931: 680-681. In June 1929, Robert Greenhil Cochrane replaced Frank Oldrieve as Medical Secretary of BELRA. Prior to this he had been Medical Advisor to Mission to Lepers, practicing in the leprosy Asylum at Purulia, Bengal. Cochrane's extensive travels are reflected in his list of early publications: *Leprosy in India: A Survey* (1927), *Leprosy in Europe, The Middle and Near East and Africa* (1928) and *Leprosy in the Far East* (1929). All books were published on World Dominion Press that mainly specialized in missionary literature from all over the world. In 1926 he published the booklet *How to rid a country of leprosy.* His obituary was printed in *The British Medical Journal.* Vol. 291, August 31, 1985: 608.

[947] Report from the Leprosy Commission to the Health Committee, 1931: 15-22. (LNHO: 8A-R5930/30632/29090)

The temporary officers charged with finalizing the rules and regulations of the organization were all men with previous experience from international anti-leprosy work: President Victor G. Heiser from the Rockefeller Foundation; Vice-President for the Western Section, Professor Carlos Chagas of Rio de Janeiro; Vice-President for the Eastern Section, Dr. Ernest Muir of Calcutta; General Secretary Dr. Robert G. Cochrane of London; General Treasurer, Mr. William H. Brown, Ph.D., of Manila; Sir Leonard Rogers of London, Dr. Etienne Burnet of the League of Nations Secretariat and Professor de Langen of Batavia were elected general Councilors.

The most important task for the association was to establish an international journal of leprosy, "to serve as the principal, and a truly international medium of publication of worthwhile articles on leprosy, and as a collecting medium for all other material of similar nature that is not published therein originally."[948] By the first issue of the journal, the association had almost 340 members from all over the world.

The symbol of Bergen

By the early 1930s, to what extent was Bergen and the heritage of a successful campaign that had brought the problem of leprosy under control still considered relevant? When the history of leprosy in Norway was presented at the Third International Leprosy Conference in Strasbourg in 1923 by head of the national leprosy apparatus, H. P. Lie, the case was perceived to be one of the highlights. But, as I have argued, the legacy of Bergen was contested. Researchers like de Souza-Araújo argued the Norwegian experience showed that strict segregation was necessary in a campaign to eradicate the disease, others, such as Muir and Rogers,

[948] Wade, H. W. "The International Journal of Leprosy. An Editorial Statement." *International Journal of Leprosy*. Vol. 1, No. 1. 1933: 2. Regular membership with rights to vote was, however, "limited to persons with recognized medical degree, and those with other scientific qualifications who are, or have been, actively connected with leprosy work." (Cochrane, Robert G. "Association News". *International Journal of Leprosy*. Vol. 1, No. 1. 1933: 94).

argued that Norway was proof that the goal could be achieved using a humanitarian approach.

In Etienne Burnet's report on leprosy in Europe, South America and the Far East (1930), Norway was the first country that was presented. This was the birthplace of scientific investigations into leprosy, and according to Burnet, Norway was still relevant: "A visit to the special hospital at Bergen and to Dr. Lie's laboratory is essential for all students of leprosy."[949] But the interpretation of the historical success had changed. "Norway is the classic example of the progressive eradication of leprosy through the systematic isolation of lepers – not, however, by forced internment in leprosaria."[950] This was in stark contrast to Hansen's explanation at the first leprosy conference in Berlin some three decades earlier, where the success was explained by succeeding in keeping the lepers away from the healthy. By the early 1930s, emphasis was shifted in favor of the progress of hygiene and general welfare:

> Segregation on liberal lines has therefore been sufficient to bring about a decline in leprosy which promises to lead to the total eradication of the disease. Norway is a sparsely populated country, where the standard of living is improving, education is widespread and social hygiene is very well advanced.[951]

This interpretation was in line with Lie's explanation for the cause of the decline of the disease in Norway: Isolation did play a considerable role, but the decline of the disease could not be explained without connecting it to sanitary and economic improvements: "The great decrease in the prevalence of the disease since 1856 must therefore be regarded in the light of the great progress the country has made during that time in all respects, and not least in hygiene and sanitation."[952] As I discussed in Chapter 2, leprosy was no longer simply a matter of the presence of a bacillus alone,

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[949] Burnet E. *League of Nations Health Organisation. Report on the Study Tour of the Secretary of the Leprosy Commission in Europe, South America and the Far East. January 1929 - June 1930*. (C. H. 887). Geneva. 1930: 6.
[950] Burnet 1930: 6.
[951] Burnet 1930: 7.
[952] Lie, H. P. "Why is leprosy decreasing in Norway?" *Transactions of The Royal Society of Tropical Medicine and Hygiene*. No. 4, 1929: 357-366, quote on page 366.

predisposing causes and sanitary conditions were also part of the explanation. It was increasingly accepted that leprosy could be addressed by sanitary improvements, and the explanation of the past changed accordingly.

Throughout the 1920s, Lie was the only Norwegian who was in touch with the group of actors actively engaged in making leprosy an international concern. He still believed there was much to be learned, and was a strong supporter of global centralization of leprosy work.[953] Lie was among those invited to the Manila meeting, but declined the invitation. Instead he wrote a series of heated letters to Thorvald Madsen, the newly elected head of the Health Commission, where he predicted that the Manilla meeting would be a failure because it lacked sufficient international representation. None of his contacts in South America had heard of the plans. Besides, an international society already existed (*La Société Internationale de Leprologie*) and Lie had already prepared a frontpage for its planned journal.

> In my opinion, this Conference in the Philippines cannot adopt any resolution of an obligatious character for a great number of States interested in the Leprosy question. Its dealing with the question of the establishment of an International Leprosy Society and the creation of an International Leprosy Periodical can only be of a quite temporary character.[954]

Lie's predictions proved wrong. The International Leprosy Association (ILA) exists to this day, and in September 2013 it arranges the 18th International Leprosy Congress, this time in Belgium.

The meeting at Manila in 1931 established a division of labor between the ILA and the League of Nations which was continued by the World Health Organization. The *International Journal of Leprosy* was published for 73 years until 2005 when due

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[953] H. P. Lie's detailed his call for centralization in this way: "a place, an institution or a journal where one could find information from all areas and all countries regarding leprosy and its suppression – and where one could seek advice and guidance in this question. To create such a center must therefore in my opinion be on top of the agenda when now working to suppress leprosy, that all countries and all people belong together and need each others help to a greater or lesser degree." Letter from Lie to Madsen, dated Bergen August 8, 1930. (LNHO: 8A-R5894/27029)

[954] Letter from Lie to Madsen, dated Bergen August 25, 1930. (LNHO: 8A-R5894/27029)

to raising costs it became financially unviable. The Manila-meeting thus established a lasting framework for leprosy as a global concern.

Despite not attending, Lie was at the meeting in Manila appointed secretary-treasurer for the Western section, associate editor of the International Journal of Leprosy and ex-officio member of the General Council. This was the same position that de Souza-Araújo had envisioned for him in his planned organization. De Souza-Araújo himself was appointed vice-chairman of the Western branch. Marchoux, who after being general-secretary at Strasbourg was the first to present leprosy as an issue for the League, was appointed chairman of the Western branch. In the Eastern section, Ernest Muir from Calcutta was appointed chairman, Kensuke Mitsuda from Nagashima vice-chairman, and James L. Mazwell associate editor with the same status as Lie.

Lie was not the only high-ranking official appointed to the organization not present in Manila. In February, 1932, a general council was held in London and officers and councilors were officially nominated for five-year terms in office. H. P. Lie accepted his position. He also exhibited the insignia that had been used for the second International Leprosy Conference held in Bergen in 1909, which to this day is the official seal of the International Leprosy Association:

> This bears, in formal representation, Saint George, the ancient patron of European lepers, overcoming a dragon; it has neither sectarian nor nationalistic significance. The suggestion of struggle and idealism in this medallion appealed to the members of the General Council present, and after discussion it was voted to adopt it, with certain modifications, as the insignium of the Association.[955]

Despite being highlighted as an 'essential' place to visit by Burnet, by the early 1930s Bergen had been reduced from a leading world capital of leprosy research to a unifying symbol of a past success, a symbol open for interpretation. Everyone agreed the Norwegian campaign had been a success, but the interpretations of why the

[955] Cochrane, Robert G. "International Leprosy Association. Present status of the Organization of the Association." *International Journal of Leprosy,* Vol. 1, No. 1. 1933: 98.

campaign had succeeded depended on what policies the one making the comparison wanted to promote. The Norwegian physicians themselves ensured that Bergen continued to stay relevant through reinterpreting the past through the lens of new epidemiological models.

Conclusion

This chapter has shown that in the 1920s, several concurrent and parallel initiatives were involved in making leprosy an international concern under global coordination. Several actors were involved. They came from different locations and used different strategies to reach their goal: Traveling around the world, establishing international organizations, forming alliances with the League of Nations and Rockefeller Foundation, setting up international research centers and publishing monographs. At the same time, the number of actors involved was relatively limited. Several knew each other personally and collaborated on various schemes. They all had medical training, experience with leprosy work, and most came from high-ranking positions within national health systems. Although the actors and their organizations were new, travelling, establishing organizations and forming alliances were not particular to the period between 1920 and 1933 but a central mechanism in organizing the circulation of knowledge.

Despite a deep divide between the advocates of compulsory segregation and advocates of voluntary treatment, the initiatives came together in an International Leprosy Association with its own international journal. This was achieved by building on the previous initiatives and gathering 23 individuals with direct experience in leprosy work around the world for a two week meeting in Manila. The attendees were hand-picked and approved by H. W. Wade, representing the private foundation Leonard Wood Memorial which was the main funding body of the association, Etienne Burnet, representing the League of Nations Leprosy Commission, and Victor G. Heiser, representing the Rockefeller Foundation. Despite the fact that much leprosy work on the ground was left in the hands of philanthropic organizations

(mainly various Christian missions), missionaries were explicitly excluded. Regular membership in the International Leprosy Association was "limited to persons with recognized medical degrees, and those with other scientific qualifications who are, or have been, actively connected with leprosy work."[956]

The objective of the association was to encourage global collaboration and coordination with the shared goal of eradicating leprosy.

> The purposes of the International Leprosy Association are to encourage and facilitate mutual acquaintance between persons of all nationalities who are concerned in leprosy work and the co-ordination of their efforts; to facilitate the dissemination of knowledge of leprosy and its control; to aid in any other practical matter the anti-leprosy campaign throughout the world; and to this end to publish a scientific journal of leprosy.[957]

The division of labor between the physician's International Leprosy Association, the *International Journal of Leprosy* and the League of Nations Leprosy Commission would be the main structure for coordinating leprosy work globally for the next seven decades. After the Second World War, the activities of the League were taken over by the World Health Organization. Missionaries and representatives from other philanthropic organizations were excluded from positions of formal power, but were allowed to be non-voting members and encouraged to make donations. Publishing in the columns of the journal was, at least in theory, open for all with a special interest in leprosy. This setup did not entail agreement, nor did it force a unification of practices. What it did provide, was a shared context which brought almost all leprosy workers together in a global collective, directly- or indirectly.

Leprosy had become a global disease. Leprosy was globalized.

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[956] Cochrane 1933: 95.
[957] Cochrane 1933: 94.

8. Final remarks

How did the knowledge of the leprosy bacillus move from being an entity observed by one person in a laboratory in Bergen to being accepted as the defining character of the disease all over the world? In this thesis I have argued that this simple question has a complex answer.

As I have written conclusions to each chapter of the thesis, the purpose of this concluding chapter is to take a step back and give an overview of the thesis as a whole. I will begin with arguing why using the moment of discovery as a starting point would have been problematic. I will then recapitulate my strategy and findings and point out what I believe to be the strengths and drawbacks of my approach. Finally, I will return to the three debates I presented in the introduction and summarize my contributions.

Rethinking a genesis

It would have been possible to create a linear narrative which started in the office of Gerhard Armauer Hansen on February 28, 1873, and ended with the unveiling of the bust in August 1901 celebrating Hansen as a discoverer. This story would have shown how the bacillus was first mentioned as a footnote to Henry Vandyke Carter's report from his visit to Bergen in 1873, and then in two reports by Hansen in 1874 and 1875. In this narrative, it is clear that the importance of the bacillus initially was overlooked. In these publications from the 1870s the bacillus was not presented as a great discovery but preliminary and uncertain results of ongoing research into the causes of the disease. The reception was poor, and began to change only after Hansen's claim to priority in 1880, which is when the date of the discovery first was published.

Hansen's claim was prompted by Albert Neisser's successful staining of the bacillus in preparations he had received during a visit to Bergen in the fall of 1879. Hansen's publications in French, German, English and Norwegian the following year

were the first time the bacillus received any real attention. Increasingly, the question was not whether the bacillus existed or not, but who should receive the honor of the discovery. This was first settled at the first international leprosy conference in Berlin in 1897, where Hansen was hailed as the discoverer and Neisser as the one who had confirmed the findings. In the intervening period, some pointed to Neisser as the authority on this new entity, but from the early 1890s Hansen was increasingly highlighted as the discoverer.

The linear story would highlight how events outside the narrow field of leprosy made the bacillus easier to accept in the early 1880s than seven years earlier. In 1873, the claim that a disease had a singular cause was controversial in itself. Demonstrations of germs as the cause of other contagious diseases made the bacillus easier to accept. Improved staining techniques developed especially in the German-speaking world, as well as demonstrations of the presence of the leprosy bacillus in samples from other parts of the world using these techniques, helped make the existence of the bacillus more secure. In 1884 the bacillus was for the first time mentioned in a medical textbook. Still, it was first after the conference in Berlin that the bacillus was generally included as part of the presentations of leprosy in medical textbooks.

Apart from its mere presence, demonstrating a direct causal relationship between leprosy and the bacillus proved difficult. Instead the argument for the entity as a cause rather than an effect rested on two analogies. The first was the resemblance with the bacillus causing tuberculosis. This entity was of the same size, behaved in similar ways and could be stained using the same methods. The second rested on epidemiological and historical evidence which showed that when the disease was introduced into a new area it spread rapidly, and that this could be reversed when people affected by the disease were isolated.

At the conference in 1897, Hansen was able to document that the number of new cases had gone down. This success, Hansen argued, was explained by the Norwegian leprosy campaign. While Hansen had relied on epidemiological findings when architecting and arguing for the increasingly strict leprosy Norwegian

legislations in 1877 and 1885, in 1897 the bacillus was used as an argument also in debates on the prevention of the disease. From the first decade of the 20th century a widespread line of reasoning began with the bacillus as proof that the disease was contagious, and ended in segregating the sick from the healthy as the logical conclusion. In 1901 a bust of Hansen was unveiled in Bergen, funded by 'colleagues and friends from all countries'.

As a postscript to this linear story, I could have argued that the final recognition came only after the second international leprosy conference in Bergen 1909. Here the bacillus was presented as the common denominator: Proof that leprosy was the same disease all over the world. The bacillus explicitly made knowledge produced elsewhere locally relevant. After the conference was officially over, also the delegates from the British Empire endorsed the bacillus as cause of the disease, that the disease was contagious and that it should be met with segregation.

All these elements are found in this thesis. However, early in the research I realized that the linear narrative of a bacillus conquering the world was problematic. First, its importance was not to be found at the moment of discovery. Rather, recognizing the discovery as relevant and significant was a process that took several years. Exactly what impact the bacillus had differed from place to place, and so did the time and way it was introduced to the debates. It was not until others were convinced of the existence and relevance of the bacillus that agreeing on its origins (the discovery) became important. Agreement on a story of the genesis of the bacillus was reached only in the first decade of the 20th century. The genesis must therefore be seen as *outcome* of a process – not its start. Using the genesis as the starting point would mean accepting this backdating and turning a blind eye to the different meanings the bacillus had in different places and at different points in time.

Second, a narrow focus on the bacillus as a genesis would have meant giving it a privileged position that is not supported by the sources. Not everyone knew about the bacillus, not everyone saw it as relevant, and not everyone agreed that the bacillus could explain every significant aspect of the disease. The bacillus was but one of several coexisting and competing ways of knowing leprosy.

Third, even when accepting the existence of the bacillus, its implications differed from place to place and changed over time. As the bacillus increasingly was introduced to already existing debates, the outcomes differed. The most obvious example is in prevention: Systems of segregation were not implemented everywhere, or in the same way. But also when it came to diagnosis, even in the interwar period when the leprosy bacillus was generally accepted as the cause of the disease, its role differed from place to place.

In order to answer the main research question I therefore had to develop a different narrative. The main novelty in this thesis has been to separate the content of the debates from how the circulation of knowledge was organized.

Diagnosis, treatments and prevention

In the first three content chapters I focused on the arguments that were presented in debates on diagnosis, treatments and prevention between the 1850s and 1930s. All these themes existed prior to the bacillus. I have shown how the bacillus was made relevant in different locations, in different debates and at different points in time. De-centering the bacillus made it possible to show both the breadth of the debate, how the dominant positions changed over time, as well as give symmetrical attention also to the arguments that in hindsight turned out to be wrong. The approach let me investigate the impacts of the bacillus without giving it an a priori privileged position.

Before the advent of bacteriology, deciding if a person was ‘a leper’ depended on clinical diagnosis: If you looked like a ‘leper’, you had leprosy. At the Berlin conference in 1897 Hansen insisted that detecting the leprosy bacillus was required to establish a diagnosis, but this was never unanimously accepted. Observing the bacillus in the laboratory was a welcome confirmation in uncertain cases, but not finding it was seldom enough to reverse a verdict based on clinical signs and symptoms. The new way of knowing (bacteriology) was an addition, not a replacement for the old. Still, it drew attention to the drawbacks of clinical diagnosis, especially the need for specialist training and the fact that different physicians could

reach different conclusions. Attempts at making a mechanically objective test which could bypass the need for clinical judgment failed. In the 1920s, there were still local differences, and in borderline cases the verdict could depend on where the diagnosis was made. Yet, from around 1910 the bacillus did have an important impact: It made leprosy a disease you could get well from. If a bacillus had been detected, its disappearance meant that the disease had disappeared. Terms like 'arrested', 'bacteria-free' or later 'burned out case' would have been meaningless without accepting that the disease was caused by the leprosy bacillus. This practice was first adapted in South Africa and then spread. Still, that people affected by leprosy could be paroled was never universally accepted. In Japan, for instance, the official doctrine remained 'once a leper, always a leper'.

When it came to treatments, the leprosy bacillus became part of the rationale for testing new drugs from the early 1890s. After the failure of Carrasquilla's serum therapy at the end of the decade, explaining how a drug would interact with the bacillus became a requirement for new drugs. Testing drugs was an impetus for collaboration between physicians working in different locations: Each individual case was unique, and therefore the efficacy of a drug could only be determined in larger trials. However, the observation in the microscope did not disentangle the disease from the person affected by the disease, and testing new drugs had to be conducted on those suffering from the disease directly. From the first decade of the 20th century, collaboration with professional manufacturers made the new substances more accessible, but this did not solve the main problem: While some of the numerous drugs tested against leprosy reportedly did some good for some of those affected by the disease, at least initially, the goal of producing consistent cures failed.

Measures for prevention were closely intertwined with etiology. Contagion was linked to isolation or segregation, heredity to marriage prohibition, the fish eating hypothesis to diet, and where insanitary surroundings were seen as a cause, sanitary measures were presented as the best means of prevention. There were also hybrid etiologies with hybrid solutions. The argument for state institutions in Norway, erected in the middle of the 19th century, was primarily to alleviate the economic

burden of the disease. This seems to have been an exception: As long as the leprosy was believed to be hereditary, the disease was generally perceived to be a problem for the individual and their families. From the 1880s and 1890s, the bacillus became entangled with the argument that leprosy was contagious and that segregation was the best means to prevent others from falling victim to the disease. Contagion made leprosy a problem for the healthy. Contagion existed before the bacillus based on epidemiological data and historical evidence, such as in the case of Hawaii and in Munro's narrative of leprosy as a disease that throughout time had reached a global distribution, but the bacillus served to strengthen the argument.

The belief in contagion and segregation was at its strongest between the 1890s and the 1920s. The recommendations from the international leprosy conferences concluded that since leprosy was caused by a bacillus every leper was a danger to his or her surroundings, and the best means of prevention was segregation. The Norwegian system was highlighted as a golden standard in finding a balance between the freedoms of the individual and society's need for protection. The exact nature of the lessons from Norway was contested: For some, this was an argument for strict segregation, for others it was proof that a relatively mild approach was sufficient. In any case, the recommendations gave supporters of contagion an international consensus to refer to. Proponents of other explanations had no such agreement to put forward in local debates.

The bacillus alone did not decide what policies should be pursued, but three strategies stood out in the debates that followed: Segregation Acts and educating the healthy that 'lepers' were dangerous so that they would be forced into institutions (push), presenting the institutions as attractive refuges providing proper medical care (pull) and placing restrictions on the activities of those suffering from the disease, such as banning them from certain trades. The researchers seldom saw proscribing policies as conflicting with the goal of producing knowledge. Rather, more often than not, the initiatives for doing something about 'the leper problem' came from the physicians themselves.

Discussing diagnosis, treatments and prevention in separate chapters has had some drawbacks. Often these topics were interlinked, such as in the controversy surrounding chaulmoogra in the interwar period. Chaulmoogra was a treatment that by the time it was introduced to Western medicine already had been in use as a natural remedy in parts of Asia for several centuries. After being regarded mainly as palliative, from the turn of the 20th century collaboration with chemists searching for its active components led to derivatives that some claimed to be a potential cure. From the 1920s, BELRA was the primary proponents of chaulmoogra as a cure, provided the treatment started early enough and was combined with diet, exercise and cleanliness. Proponents of chaulmoogra argued that since a cure now existed, identifying the ill and potentially ill in local communities through dispensaries and offering voluntary treatment should replace segregationist institutions. In other words: Treatments meant that strategies for prevention should change. Opponents pointed at the bacillus: Since the derivatives did not interact with Bacillus tuberculosis, there was no reason why chaulmoogra should have any effect against Bacillus leprae. Another line of criticism was that the efficacy of chaulmoogra had never been demonstrated in comparative trials. Indirectly this linked chaulmoogra to diagnosis, as it brought attention to the fact that diagnosis differed from place to place, and that there were no international standards for classifying cases.

While the bacillus did become an argument in already ongoing debates, it also gave rise to new and lasting controversies, such as whether it was found inside or between the cells, whether it could spread via insects, or whether or not the entity had a life-cycle of its own with the acid-fast bacillus as but a stage in a complex morphology. In my narrative, this aspect has been pushed into the background. Furthermore, the bacillus was enlisted as an argument in more disputes than those I have investigated. Although the narrative of a disease that throughout history had spread around the globe already existed, accepting the existence of the bacillus had a huge impact on the question of the natural history of the disease. In the thesis I have tried to reflect the debates also when they went beyond my predefined categories, but

in hindsight I am not sure whether this aspect of my argument has been sufficiently clear.

Making knowledge move

In the last three content chapters I investigated how medical knowledge about leprosy was made to move, how the circulation of knowledge was organized and the mechanisms involved. Each chapter was limited to shorter periods of time, focused specific aspects, and the relatively few actors involved. My emphasis was on appropriation (Chapter 5), journals and conferences (Chapter 6) and travels, meetings and organizations (Chapter 7), but I also touched upon textbooks, correspondence and education. Again I tried to give attention also to the initiatives that ended up as blind alleys.

I found *appropriation* to be a key concept for explaining how the same observations could be given different meanings in different places. Circulation of knowledge was an active and selective process both in what knowledge should be shared, how it was made available to others, to what extent actors in other locations considered it relevant, how the knowledge was interpreted, and to what extent the knowledge was accepted by others. For instance, I have shown how the same report could be given radically different meanings when introduced as arguments to debates in other contexts. A report that in the British Empire would establish that the official doctrine should be a hands-off approach to leprosy was in Norway introduced as an argument for contagion. A thesis from Surinam that in Norway inspired new research into contagion was in Britain dismissed as 'lacking in necessary precision'. The outcomes of conferences were reported differently from journal to journal, and it would take more than eight decades before the human experiment that led Hansen to lose his right to practice as a physician at the leprosy institutions in Bergen was made known to the rest of the world. I have also shown that the relative position within local hierarchies of the one introducing new knowledge could be decisive. Besides,

the relative importance of leprosy compared to other diseases differed from place to place. Local context did matter. Not everything circulated.

In general, the actors I have investigated seem to have put more emphasis on their own experiences than reports produced elsewhere. The more an actor had invested in defending a particular disease model, the less likely it was that he would change his mind in light of new observations or arguments. In effect, several radically different disease models existed simultaneously. While there still were local differences in the 1930s, however, the scope of explanations was much narrower than eighty years earlier. The explanation, I have argued, is to be found in how the circulation of knowledge was organized.

In the 1850s, discussions on leprosy were part of medicine in general. As Western medicine became increasingly specialized in the latter part of the century, leprosy was discussed as part of dermatology, bacteriology, and as a tropical or colonial disease. Leprosy became a specialty of its own right around the turn of the 20th century through the birth of a transnational research community with international conferences and a shared medical journal as their main unifying features. The leprosy conferences set out to establish the prevalence of the disease in various parts of the world and were an important arena for debate and exchanging experiences on how to treat and prevent the disease. The scheme aimed at creating a global organization of experts that could dictate measures for leprosy prevention failed. But also the international conferences were aimed at political changes, as evident by the topic for the follow-up conference in Berlin in 1904, which asked what actions had been taken in light of the conclusions reached at the first conference.

I have shown that relatively few actors were involved in organizing the conferences, and that for them, the bacillus was an impetus for collaboration. Securing the support of Hansen was seen as vital. Hansen was not only hailed as the discoverer of a bacillus, he was also head of the Norwegian leprosy system. By 1897, Norway was the only country in the world which could document a success in reducing the number of new cases. In a context where leprosy was seen as a possible

threat, and the health of the population increasingly important for Western states, this was something to be mimicked.

In addition to establishing the prevalence of the disease in 22 countries, the bacillus was the main topic for debate. Based on its presence and Hansen's argument that the successful campaign in Norway was due to segregation, most of the attendees agreed that every leper was a danger to his or her surroundings and had to be isolated for the benefit of the healthy. At the next international leprosy conference in Bergen in 1909, where experts from 27 countries took part, the bacillus was presented as the common feature of the disease. Regardless of local conditions, the bacillus was the same. The bacillus proved that knowledge produced elsewhere was locally relevant.

In addition to the international leprosy conferences, the medical journal *Lepra Bibliotheca Internationalis* (1900-1914) was instrumental in making leprosy a specialty. The journal did not stop leprosy for being relevant for medicine in general or a range of other specialties, but was aimed at presenting an overview of studies and reports published elsewhere. This increased the circulation of knowledge, and contributed to an increased streamlining of how to produce knowledge about leprosy. Based on the reviews of publications arguing for disease-models which did not center on the bacillus, it is clear that accepting the bacillus as a scientific fact was necessary to avoid ridicule.

While disagreements continued to exist, and local practices remained unique, the publications gave easy access to observations, arguments, legislations, and experiences from other places. This, in turn, was used as arguments in local debates and added to the prestige and expertise of the actors involved.

In the interwar period, the French association *Société de Pathologie Exotique* took the initiative to a third international conference in Strasbourg in 1923. The main outcome was that the organizers succeeded in making leprosy a concern for the League of Nations. In addition to establishing an international leprosy center in Brazil, issuing policy advice and encouraging states to organize production of knowledge about the disease, The League of Nations Leprosy Commission was one of several actors involved in organizing leprosy workers globally. The main fruit of this

labor was the International Leprosy Association (ILA), established in Manila 1931, with its own medical journal, *International Journal of Leprosy* (1931-2005). Other key actors in the interwar period were The Leonard Wood Memorial, Victor G. Heisser from the Rockefeller Foundation, and Heráclides César de Souza-Araújo who, after a three year worldwide journey financed by the Rockefeller Foundation, established the organization *La Société Internationale de Leprologie.* While the society never had a single meeting, it provided the organizational model for the ILA.

Despite trying to emphasize also the 'failed' attempts at organizing the circulation of knowledge, one unintended consequence of my approach of investigating medical debates has been that the medical actors have ended up as the ones with the most agency. This was partly true, in that most of the actors I have investgated had authoritative positions towards those affected by the disease and often were actively engaged both in producing knowledge and in advocating leprosy policies. But although I have tried to look for the voices of those affected by the disease, for the researchers I have investigated the subjective experiences of being affected by the disease were increasingly seen as less important than their own expert observations. The leprosy bacillus only increased this tendency. Furthermore, as previous localized studies have shown, the physicians were not the only actors involved in the decision making processes, nor was Western medicine the only system for producing medical knowledge. Additionally, in parallel to the physicians, missionaries also developed their own transnational networks for the circulation of knowledge which should be investigated further.

Wider contributions

In the introduction I presented three debates which this thesis relates to and seeks to add to: To what extent bacteriology constituted a radical break with previous understandings of disease; debates within the history of science regarding transnationalism, localism and the places of knowledge; and the growing body of research on the history of leprosy. In the following I will summarize my contributions.

In regards to the debate on whether bacteriology constituted a revolution in medical thinking about disease, I put Michael Worboys and Andrew Cunningham forward as representative of opposing positions. Worboys has argued that 'there was no bacteriological revolution', while Cunningham has withheld that the introduction of single causes of disease constituted a shift so radical that the identities of disease before and after bacteriology became 'incommensurable'. Worboys is right in that most physicians were not involved in bacteriological research and that to them, bacteriology had no immediate practical implications. Cunningham is equally correct in that bacteriology over time led to a radical epistemic shift in the understanding of the causes of disease. The difference between the two conclusions is not just choice of period (1870-1910 or 1895-1930) or actors (general practitioners or vanguard actors), but how they relate to the practice of 'backdating', which none of them reflect upon. The physicians who accepted bacilli as the cause of disease only later dated the knowledge back to when it was first observed. Despite the bust of Hansen being erected in 1901, for instance, the justification was the discovery that happened three decades earlier. While further research is necessary to establish whether my findings on leprosy are generalizable to other fields of knowledge, it does seem evident that agreeing on a moment of a significant discovery could only be established in hindsight. This agreement had several implications. First, establishing a universal genesis of new knowledge hid the fact that it took time before this knowledge was made relevant elsewhere and that this played out at different times and at distinct ways. It also backdated arguments and clarifications that were made during this period of negotiation. In addition to bacteria being observed using the same methods in different locations, shared narratives of their origins and their discoveries added to the universal character of bacteriological knowledge. While bacteriology must be considered an addition and not a replacement for other ways of knowing disease, and the immediate impacts of bacteriology were probably too small to justify the term 'revolution', the term is meaningful to describe the way actors in the 1920s and 1930s gave meaning to the epistemological changes that had happened over the past half century.

Regarding the debates on transnationalism, localism and the places of knowledge, I have tried to follow James Secord's suggestion of seeing science as communication. For more than three decades historians have successfully brought science down to earth by showing how the production of knowledge is a practice that takes place in specific locations. It seems increasingly clear that something falls between the 'gaps' when investigating science in single locations at a time. Local production of knowledge does not happen in a vacuum. In this thesis I have argued that studying the circulation of knowledge as a practice in its own right might be a way to bridge these gaps. An important motivation for scientific publications was that the knowledge should benefit and be recognized by colleagues in other locations, and relating to knowledge produced by others has been at the core of any scientific community. My contribution to this debate has been to provide an in-depth example of how such a study can be conducted, namely through investigating both the content of the transnational debates and how the circulation of knowledge was organized.

In the period from the 1850s to the 1930s, leprosy moved from being primarily a local/national concern to an issue which facilitated global cooperation and coordination. I believe this mirrors changes that also occurred in other parts of society, and not necessarily limited to the sciences. Although the history of leprosy is unique, with internationalization and tension between unification, standardization and claims of universality on the one hand, and local specificity on the other, this seems relevant beyond the history of leprosy. I have tried to read the sources not in search of timeless truths but as arguments in ongoing debates. This has made it possible to show how different and competing truths about leprosy coexisted. There were different opinions regarding what constituted the cutting edge of research, which actors could provide the best expertise, and what places should be considered centers and peripheries. This approach has allowed me to show how interpretations changed over time, including interpretations of the past, and might be a useful insight also for other transnational studies.

My main contribution to the growing body of literature of the history of leprosy has been to provide a detailed overview of how the transnational circulation of

knowledge was organized, the ongoing debates regarding diagnosis, treatments and prevention, and how dominant positions changed over time. Although I have not investigated every transnational relation, I believe this study provides a summary of the international 'outside' that will benefit future localized studies. In addition to focusing on what went on in a specific place, these studies might also investigate how the local actors related to knowledge produced elsewhere and how this knowledge was appropriated. However, this is not to say that historians should all become transnationalists. Although the circulation of knowledge was important in providing access to arguments and experiences elswehere, localized studies are necessary in order to further nuance how these played out in local debates. As this study has built extensively on previous and mainly localized studies, I will be the first to argue that a transnational perspective must be seen as an addition and not a replacement. The actors I have investigated came from different local backgrounds, and it would have been unobtainable to investigate all of these contexts without building on already existing literature.

I began the thesis with the unveiling of the bust of Hansen in the park of Bergen Museum in 1901. My final argument is that this was a more important event than the observations made by Hansen on February 28, 1873, in that the bust representeded an international acceptance (although not complete) that Hansen's discovery was important and relevant. It was not until the 1880s that the bacillus was made part of the medical debates on leprosy, and another two decades went by before its existence was more or less universally accepted. Instead of understanding discoveries as fixed points in time, perhaps a more appropriate perspective would be to see them as the outcomes of negotigation that take years or even decades.

APPENDIX 1. "The Discoverer of the Leprosy Bacillus", 1901

1897-1900 have been prepared by the Imperial Insurance Department, and are placed at the disposal of Congress. They relate to 21,176 consumptive persons. The insurance societies intend to ascertain in future the results as to cure for five successive years, and only cases in which incapacity for self-support has not recurred will be regarded as "successful."

The two published tables express the results of treatment in the years 1897, 1898, 1899, and 1900. According to these, the immediate results of the treatment in the four years varied from 68 to 74 per cent. in all the men and from 68 to 73 per cent. in all the women under treatment. The three last of the years first referred to show better results than those of 1897, a success which is partly due to the more careful selection of cases, and partly to the experience gained in that year. The beneficial effects of the treatment of 1897 lasted only two years in 27 per cent. of the men and 36 per cent. of the women; whereas of those treated in 1898, 38 per cent. of men and 44 per cent. of women were able to support themselves after the same lapse of time. It is worthy of note, however, that of the men treated in 1897, 26 per cent. were still able to work in 1900. Positive conclusions as to the duration of cure cannot yet be drawn, but the greater success of the> morl' recent years is striking. The effect of the treatment is more lasting in women than in men.

Such are the general results of the efforts to combat consumption of the German compulsory workmen's insurance, aided as it has been by local bodies and institutions for the public welfare, as well as by private charity. The experience of several years shows thab this warfare could not have been successfully waged in Germany without the workmen's insurance, but by means of it we feel confident of victory in the end.

THE DISCOVERER OF THE LEPROSY BACILLUS.

ON August 10th a bust of Dr. G. Armauer Hansen, the discoverer of the leprosy bacillus, was unveiled by Professor Visdal in the garden of the Museum at Bergen, in the presence of many Norwegian and foreign medical men. An address was delivered by Professor O. Lassar of Berlin, and Drs. Sandberg and Lie of Bergen also spoke. Congratulatory messages were sent from all parts of the world, and a letter from Professor Virehow was read, in which the veteran pathologist, after expressing his regret at his inability to be present, went on to say that Dr. Hansen's work had definitively cleared up a large and difficult field of pathology, and that his name was known and celebrate<l. throughout the whole world as a benefactor of mankind. The Kinof Norway conferred on Dr. Armauer Hansen the distinctiOn of Commander of the Order of Ola.

Dr. H. P. Lie, the present Director of the Leprosy Hospitals of Bergen, contributes an interesting biographical sketch of Dr. Gerhard Henrik Armauer Hansen to the current fasciculus of *Lepra* (vol. ii, Fase. 3, 1901, p. 121, with portrait). Dr. Hansen was born in Bergen in 1841, and received his early education in the Cathedral College of that town. After passing his medical examinations, he became a resident in the Rigshospital of Christiania, and later spent some time as medical officer to the great Lofoten fisheries. In 1868 he became Assistant Medical Officer to the Bergen Leper Hospital (Lungegaards-Hospital), of which Dr. Daniel C. Danielssen was director. It was under the influence of this enthusiastic teacher and acute observer that Armauer Hansen began his life-long study of leprosy. His first investigation was to work out the significance of the so-called globi, or leprous cells of Virchow. These bodies had been already referred to in 1840 by Danielssen. who thought they were characteristic of the disease. This idea he afterwards gave up, and came to the conclusion they were the results of an involutiOn process, that is, fatty degeneration. Hansen was not satisfied with the latter explanation; he thought that Danielssen's first idea was more likely the right one. This led him to investigate the matter, and in 1869 he published his results in the Norwegian Archives of Medicine *(Nordisk Mediciniskt Arkiv,* vol. i,·1869, No. IJ).

At this time Hansen spent a year at various universities, but on his return to Norway he again applied himself to the difficulties which surrounded the etiology of leprosy. Various views were held at that time on this point. As a result of his observations and investigations, he published an important paper in the *Norsk Magasin for Lcegevidenskaben* (1872), which gave rise to much discussion in Norwegian medical circles. .tlis researches pointed to the contagious and specific nature of the malady. The Medical Society of Christiania voted a sum of money for him to continue his research. In the course of his journeyings through the country he came across instances of the diseasE, which were more readily explained by contagion than by any other theory. An account of his additional investigation was published in the *Norsk Magasin for Lcegevidenskaben* (Third Series, vol. iv, 1874), and an abstract of his paper appeared in *The British and Foreign Medico-Chirurgical Re'Biew* (vol. lv, 1875). Unfortunately this important contribution to the subject was but little known outside of Norway. His views confirmed those of Drognat-Landre, who had worked at leprosy in Surinam, and published a book entitled *La Contagion:seule cause de la Lepre* (Paris, 186g).

This led Hansen to reinvestigate the peculiar bodies (globi-brown corpuscles);previouslyreferred to, for he held that if the disease is contagious there must be some specific virus at work. His labours were rewarded by the discovery, in unstained preparations, of bacilli. These were ultimately stained, and are what we know them to be, the bacilli of leprosy. His discovery, be it noted, was made in 1873, that is about ten years before the bacillus tuberculosis was made known to the world by Koch.

For years Hansen has repeatedly tried to cultivate and inoculate the bacillus leprre, which is also deservedly known as Hansen's bacillus, but up to the present fruitlessly. One great point, however, has been gained, namely, that it is now practically admitted by all those engaged in the study and observation of leprosy, that the disease is contagious. In Norway, practical legislation on this basis has given the best results, and leprosy there is gradually and surely diminishing.

Dr. Armauer Hansen has recently (July 29th) celebrated his 6oth birthday, and:the tribute to his lifelong work and devotion above recorded will be gratifying to all lovers of science. Norway is a small nation, but while she has such sons she may well hold her head high among the proudest.

THE PREVENTION OF TUBERCULOSIS.

WESTMORLAND.

THE accompanying circular may be of use to any sanitary authorities throughout the Kingdom who propose to follow the example of the Camberwell Borough Council, the Kendal Town Council, and the Bowness-on-Windermere and Ambleside Urban District Councils, in examining milk for tubercle bacilli, and prosecuting the purveyors who have it for sale.

BOROUGH OF KENDAL.
TO COWKEEPERS, DAIRYMEN, AND PURVEYORS OF MILK.

D rl ted , b ifl!: mtt i; '¥i : k Ys ;;i>lis'of t u l a P; el 0.i Pf ;ld! 1 rf e ':h% ?I1J'1 fuh rfu'b , r; i: rr be found.

By Section 6 of the Sale of Food and Drugs Act, 1875 :–

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By Article 15 of the Dairies, g:r, h de1Jfl k"!:_ops Order, 1885, as amended

If at any time disease, including in the case of a cow such disease of fst:st tfeei' i eddaPi;ao ! 't 'l, r i ,.t u fd\ e : the milk of a diseased cow therein *(a)* shall not be mixed with other milk; and *(b)* shall not be sold or used for human food.

Any person guilty of an offence against the above order is liable for every such offence to a penalty of £5, and in the case of a continuing offence, to a further penalty of £2 for each day after written notice of the offence from the local authority.

By order of the Kendal Urban District Council.
August, 1901. JOHN BOLTON, Town Clerk.

AUSTRIA.

Dr. von Kusy, an Under-Secretary in the Austrian Home Office and Chief of the Sanitary Department of that Ministry, is now engaged in drawing up a series of Bills and administrative regulations to protect the population of Austria against tuberculosis. According to the *Fremdenblatt* the duty of notification is impressed upon the doctors, while speciP.l

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[958] Editorial. "The Discoverer of the Leprosy Bacillus." *The British Medical Journal.* August 14, 1901: 494.

APPENDIX 2. International Leprosy Conferences, resolutions

a) Berlin, 1897

"At the close of the debates of the International Leprosy conference, Berlin 1897, the Secretaries have the honour to present the following short report of the general conclusions of the Conference.

They believe that such a resumé will be especially desirable for those members who have been delegated by their respective Governments, and who have to make reports on the results of the Conference.

As might be expected, a considerable portion of the discussion has related to the bacillus Leprae, which the Conference accepts as the Virus of Leprosy, and which for upwards of 25 years has been known to the scientific world through the important discovery of Hansen and the able investigations of Neisser.

The conditions under which the bacillus grows and develops are still unknown, as well as the way of its invasion into the human system; but from the discussions of the Conference, it seems probable that an unanimity of opinion will soon prevail in reference to its modes of subsequent dissemination within the human body.

Very interesting observations have been brought forward in connection with the elimination of the bacilli in large quantities by means of the skin and the nasal and buccal mucous membranes of lepers; it is desired that such observations be confirmed where opportunities occur.

The question is of very great importance to those who are entrusted with the care of the public Health, as leprosy is now acknowledged to be a contagious disease.

Every leper is a danger to his surroundings, the danger varying with the nature and extent of his relations therewith, and also with the sanitary conditions under which he lives.

Although among the lower classes, every leper is especially dangerous to his family and fellow workers, cases of leprosy frequently appear in the higher social circles.

The theory of heredity of Leprosy is now further shown to have lost ground, in comparison with the at present generally accepted theory of its contagiousness.

The treatment of Leprosy as only had palliative results up to the present time.

Serum therapy has so far been unsuccessful.

In view of the virtual incurability of Leprosy and the serious and detrimental effects which its existence in a community causes, and considering the good results which have followed the adoption of legal measures of isolation in Norway, the Leprosy Conference, as a logical issue of the theory that the disease is contagious, has adopted the following resolution proposed by Dr. Hansen and amended by Dr. Besnier.

1) In such countries, where leprosy forms foci or has a great extension, we have in isolating the best means of preventing the spread of the disease.
2) The system of obligatory notification of observation and isolation as carried out in Norway is recommended to all nations with local self-government and a sufficient number of physicians.
3) It should be left to the legal authorities after consultation with the medical authorities to take the special measures applicable to the special social conditions of the districts."[959]

[959] Abraham, Phin. S; Edward Arning, E. von Bergman, Dubois-Havenith, J. J. Kinyoun and G. Thibierge. "The Honorary Secretaries' report from the First International Leprosy Conference" *Mitteilungen und Verhandlungen der internationalen wissenschaftlichen Lepra-Conferenz zu Berlin im October 1897*. Bd. 2: 191-192.

b) Bergen, 1909

"1. The Second International Scientific Conference on Leprosy confirms in every respect the resolutions adopted by the First International Conference of Berlin, 1897.

Leprosy is a disease which is contagious from person to person, whatever may be the method by which this contagion is effected. Every country, in whatever latitude it is situated, is within the range of possible infection by Leprosy, and may, therefore, usefully, undertake measures to protect itself.

2. In view of the success obtained in Germany, Iceland, Norway and Sweden, it is desirable that other countries with leprosy should proceed to isolate their lepers.

3. It is desirable that lepers should not be permitted to follow certain occupations which are particularly dangerous in respect to the contagion of leprosy.

In every country and in all cases the strict isolation of leprous beggars and vagrants is necessary.

4. It is desirable that the healthy children of lepers should be separated from their leprous parents as soon as possible, and that these children should remain under observation.

5. An examination should be made from time to time of those who have lived with lepers by a competent physician.

6. All theories on the etiology and the mode of propagation of leprosy should be carefully examined to ascertain if they accord with our knowledge of the nature and the biology of the bacillus of leprosy.

It is desirable that the question of transmissibility of leprosy by insects should be elucidated, and that the possibility of the existence of leproid diseases among animals (rats etc.) should receive early study.

7. The clinical study of Leprosy induces the belief that it is not incurable. We do not at present possess a certain remedy. It is desirable, therefore, to continue the search for a specific remedy."[960]

c) Strasbourg, 1923

"1) Laws governing the anti-leprosy campaign should vary according to the country in which they are applied, but in all cases foreign lepers should be barred from entering a country.

2) In countries with little leprosy, the isolation of cases in hospital or in the leper's house, is recommended, as practiced in Norway.

3) In endemic centers of leprosy, isolation is necessary; -

a) This isolation should be humane and allow the leper to be near his family, if this is compatible with efficacious treatment.

b) If cases exist among paupers, nomads or vagabonds and other persons who cannot be isolated in their homes, isolation should be enforced in a hospital, sanatorium or agricultural colony, according to circumstances, with suitable medical treatment.

c) It is advisable to take the children of lepers from their parents and keep them under constant observation from birth.

4) All members of a leper's family should be subjected to periodic examinations.

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[960] *Internationale Wissenschaftlige Lepra-Konferenz. Abgehalten vom 16. bis 19. August 1909 in Bergen (Norwegen). Mitteilung und Verhandlungen.* Bd. 3: 418-419. (Distributed in *Lepra Bibliotheca Internationalis*. 1910: Fasc. 1)

5) The general public should be informed that leprosy is a contagious disease.

6) Professions liable to spread infection should be barred to lepers, but in that case the duty arises of helping the sick person and those dependent on him.

Recommendations.

1) Clinical, histological and bacteriological researches should be undertaken to elucidate the real nature of tuberculous leprosy.
2) Continuous study should be made to establish the connection between human and nicerine [=rat] leprosy.
3) Research concerning a specific remedy against leprosy should be continued.
4) The League of Nations should resume publication of the periodical "Lepra-Archives Internationales de la Lèpre".
5) The League of Nations should establish an international information office on Leprosy.
6) The League of Nations should make endeavors to secure statistics on cases of leprosy throughout the world."[961]

[961] LNHO: 12B-R898\29272\29272.

APPENDIX 3. "Norwegian Leprosy", 1852

'f"KIUlLt:proay.
I"rmn the 1-:cc ugtl•rul r:a!hulic.
BEao, Norway. Sept.'10. 1552.

llfy D£AR Dn . WAt!<WJ<lUIIT : **It** w1ll probably **surprse you to learn that the Oriental leprosy, as** dE'acnhed hy Moss:s and hnalerl by our Sllvinur, **e.xistt!l at tllts morncrrt in Nonvay. It is not** *:ll,* **huur eincc J httvc t>r:11over a hundrt-!d cn.,ttif of** thiM fughtful and loath•ome dt6eaoo, whtch i• here exactly the JSamP. that you fnund llt N AhlouR Rncl **cbt:9.·here iu Palt-stiuo, aud det crtlJcd. in your u P_atb\,·aJs and Abtding Places of our Lord."** Th•• <li•e""e formerly exi ;ted in Englanrl Rnrl France, ,,.hence it was finally extirpated by tho most severe and tyrannical regulationR, uch "" **c?n uc,-er Uo Cl forc4:d iu o•tr· dtt.y Ktlcl C•'•ntry. f. ·ery person, wtthout exception, that \Vas allhcted with lcpros}·, was brought into a puhlic** •• lr.:prtlAy **hou tic." audutht: di** =-l-uBc **its nut cuu!.ngiouts l.Jut hf»red1tary. 1t** Vt":\8 **thus ahnoat. if not entirety, rooh·cl out of th""o couutries. It** wntt **originally** brou ;ht 11110 Fraue by pil!lrims, wb J had ·Inter. **m:urtcd** **l n1eRt!ne; and ll) the ninth Ci!ntury it watJ carrted luto N or"'·ny, aHer thu oxcur :tiOu'! of** the !'\orthmen imo Norm.,nrt)· ; for these countries oecm to have bcet mortJ closely connected in ,.,.,. **c1ent un,cttbau 1n our own.**

That tho diseaoe has been importer!iY evitlent from tlte foct that it chiclly exidrs upo>n the V.'cllt: **ern coast, bordering upon tbe .Sorth tic•, anti** ex tends along the whole lin e of it, frum thu Nllzc to he !liorth Uaf.le. After more than eilht cen. **tUrhowevrr, Jl has begun tn pc tefmflfend,** hy tlu' det"p furcle and gulfd which oumetion"'d itt· **tlt:nt the \\'t!Hltn: cuRst of Norwtty, for a dbt:uu e** of loctwceu ouo llld two hlltHlrcd miles. **It** id **1:O\V sometimes fnurut far in the interior, l.lut never** in thE' Ea bt l'tn part of thP. country, nnrin S werlen. lct·1 lld """' t·••Lirt·ly rlopopulat!•d *by* tho pl r,ue, and recolor.tzed !rom Norway, and there the dis. **eat-t':tlt;ts.**

Thu Government ha,·o a t ""Sth t:>kt>u tho mat- **ter in ha nd, a_nd in addition to a·-** ***leprosy hou.•c,"*** which haH c-xt>te<l for fh·e hunrlrArl years.and *nmv* CUIltalll" o1le hullllrcd at,<:! thirty-five t>atiuur.s, they h•"e recenlly built a hospital, *tht: fir: t that tht< ?Curiri C!Jcr knew* for the *cure* of lt!prnsy. It Ha. been only three years in 0fJ£tatiou, under tbem!In· a ;cnol'nt. of Dr . DANU .I.M< KN; anrl tllift g•m tle. man loeheves that he ha" succeeded iu curing eight lel'crs, tho fir,;t tht<t have 0'er been cured hy **nwdu:al n•cantt. lio lals auah ji,ll•l til e hlruul** •)f **U!c deC&tied pcrHOflB aud Jjudtt it to conraiu tuv much albun1en, and { hrile nJatter, and uf course directti hi-fTorts to** (**ho Yplnnvnl of thn** ext nsa:.. Jle h3s pnhlishcd a scientific work, w1th IJiatus. 111 the Frcr.clt ar.d DaniHh la nguages. 1.'hu tuotitu. lion is well pruYidcd with fresh and sea wa:er, and VftpOJ t.,thH.

I h vu to-dRy Hcen a uuuolocr of hi patient **\Vhnn• he CO!t tdcrs nearly cured, and th•le;e wbu** h:ne " "" sont home are going on well. He estt. **tuatctJ \hu wholo tJUtnbcr in Norway at thrno thousant1· nltd HS}"B it 11:1 iulrcntting. null wouhl hRve hf·en ntuch Luger but fi)r the cht\racter of** tho c.uuntry. **I** have just t avclecl across Norw,.y, nnd It lo OlJt'gr"" mouutam or rock, with fruir.fnl crevicerather thun valle)><, not *o11e hr.,.drt,th* t»srt *c·f* the whole surface being under cult1va•iuu · Jt iK by fer thl! mc>bt thinly inhalHtP.d conntry I' ever vi•itt!d. The peopldwell eo tar apart. that thu rliSf'DH\ has &prf'arl ttself but slowly. Yet the in- **crctH.O hos 310\l::'Cfl the Storthing, or Nor\'egian** Congresstn grant 35,000 s,rect R ($37,00U). t•>· :wards lmtldm:; a thJtd hqpltal 1n liorgen. 'I'llis **ls a very 1:-nge r\\JJn for a country likA Nor- 'vay, anti the rceulttof Dr.** I>A fELL E="'· **ex..** perimPuts arc louked fur with the groatet anx- **ter.y , ns the disease is consiUered a great JJ.ational cahunirv.**

JJut the puint to which **I** wi:h to call a·tmltiou **is this: In un1e }Jincr·1 on tho Wc tcrn C•la t of Or\vay tho D';Jnthcr of lopertt nrnoug tht! pue)pln JS.11enrly oue 1n tifty. and it is pr cihely fr•J;u tho W Fstern COR!t.** ***that lhc inr.rt a Jng ctnlg raiivttu*** *Amcnca. taf.:iiJ plnce.* Dr. D.\SII<t-L•a•<r< s1lys "" **18 ccri:RJH tha[the dJFease ""Ill ehow itself amoni! tbc t:tuigrauts ; fnr ht' know6 Jcprt)UIl individualS ,,:ho havc gone t(l thP lJuitcd Stn.trfl. aud nne In** particular, quite rocr.ntly, wnom he cnrl.,,wo rorl in **"aiu to ditcsuadc, and in wborn the dt:t::Jiee wa-1** ouly tJcginuiug to> nppcar. !'lnw, thrmgn it i• never contagious, ;t is olwavs. •ntl withnnt fail, hererlnnry, nr.d if there ie the least 9pot in eithar of the p:ucuta, it ia absolutely certain lO appea<

in the descendants, though, like other heredit!lry **eflU tions, it often paeses over c:-to gonertti'ln.** Owmg to our larger nod increasing population, it may become a prcvaleut disease 111 our Western .States. But as yet it i& comparatively in our o•vn **power; Jor** IIHlt!-t **of the Norwegians are settled** to•g..thcr, nt;d nhnost every case might now be found.

For the future, **I** would propose some remcrly of tho full owlllg Illltllre : Many veOAelY with Ami- gla!•ts now sail annually frr.m Norway to tile Urrltctl Stt>tes. They lanri chietlv In N&w-Ynrk. **et** the City or the State enact a law, anrl malta It .know_n 111 Norwa.v, appointiug a ph)'oician to u,qmre tf the dieel1se extsts amoug the emigrant" who enive, onrl if any llUch be found, let them have their choice between being tranaferred to " hn"T•llal or returned to thetr own couutry. The l..n.P.rly ma y act harshly in ""rne inrlividual ens"'• hut it is by no means morn tyrannical than the Quarantine laws that already oxiH. Jt wilt **h'tld tt' til'curu futuro gt-tmutiurut ngalnst one of** the IIIOHt fcl1rful cnlamitiet1 tb:'lt cnn become por- ll•HIItllot Rmong a pcopte.

Could you h1lve seen, aa indfled you did in the Et< t. tho po.>r creatures, some a:licterl witb that L)I"' of tho di••u•• which covers thu face and ov"u rltc <:yo:lid, with rerl tubcrd""• and, by the **g,ow lh Pf the ttJtme witbin the throat, destrovs** the •pe ch, reduces **it** to a husky and hoarse ef. fort, while the poor leper in a few y ears rt.,scenrls to the tomb; or could vou behold tho limba by degrees dropping from tho bod y, and while they n·Iuain tiO dt· tllufc of fl'uling that Ute poor sulfur- llrl<. lreq llNttly lourn them•clvcs with deep scars ht'lurc tbt•y aro cvrm awa re of tho beut, yon would uot wondtr that [should wish to arouse such at- tcntiou, bcloro **it** be too lnte, as m'ly sec1lro pos- tt:rlty 3gutu xt thtR lontbHOtno llffltclinn.

Et'er most atfc ctionatelv yonre,
JUIES C. ftCIIMOND.

tet,Kt\11 fiork 0imts
Published: December 27, 1852

APPENDIX 4. The Principles of the Prophylaxis of Leprosy, 1931

"SUMMARY AND CONCLUSIONS.

1. Prophylaxis of leprosy is not a problem that admits of solution by the application of any one measure, since the means of dealing with it obviously vary with geographical, economic, administrative, financial and social conditions and with the incidence of the disease.
2. There is no reliable system of prophylaxis without treatment, and it is generally accepted that the earlier the treatment is instituted the better will be the results.
3. Leprosy resembles tuberculosis in being, in certain stages, a contagious but curable disease : curable at least in the sense that bacteriological examination becomes negative and other active signs disappear and remain absent permanently or for an undetermined period.
4. The prophylaxis of leprosy may be achieved by a system of medical, educative and legislative measures. It should provide for the isolation and treatment of infectious lepers and particularly for the treatment of early cases in clinics and dispensaries : also for the periodical examination of suspects. Special measures should be adopted for dealing with the children of lepers and for patients who have recovered either after treatment or spontaneously.
5. It is desirable that each country where leprosy exists to an important degree should have at least one centre for the study of the disease, with research laboratories and special courses for the medical profession and their assistants. Where this is not practicable, men should be sent to some foreign centre for training.
6. Arrangements should be made to include instruction in leprosy in the curriculum of all medical schools and colleges.

7. It is necessary to educate the public in regard to leprosy by modern methods of popular teaching and propaganda.
8. Isolation of infectious lepers is a necessary measure in a comprehensive campaign against leprosy, but it cannot be regarded *per se* as the sole means of prophylaxis. Its drawbacks can be mitigated by other measures applied concurrently. Isolation should be applied only to cases that are considered infectious.
9. Any form of treatment in order to give satisfactory results required to be combined with suitable dietetic and general hygienic conditions.
10. For special treatment, oils of the chaulmoogra group and their esters and soaps are recommended.
11. The system of prophylaxis must be animated by the spirit of preventive medicine and social hygiene."[962]

[962] "League of Nations Health Organisation: The Principles of the Prophylaxis of Leprosy. First General Report of the Leprosy Commission", Geneva, April 1931: 9-10.

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