INTERNATIONAL JOURNAL OF LEPROSY
and Other Mycobacterial Diseases

OFFICIAL ORGAN OF THE INTERNATIONAL LEPROSY ASSOCIATION

EDITORIAL OFFICE
Gillis W. Long Hansen's Disease Center
at Louisiana State University
Baton Rouge, Louisiana 70894, U.S.A.

VOLUME 66, NUMBER 3 SEPTEMBER 1998

EDITORIAL

Editorial opinions expressed are those of the writers.

Anti-Contagionism in Leprosy, 1844–1897

"The problem of Leprosy is not for the idle-minded. It is full of intricacy and difficulty. . . . However repulsive the disease itself in some of its phases may be, there is nothing whatever of that nature about its study."[1]

Leprosy, the "great blight" of medieval Europe, disappeared from large parts of the Continent by the seventeenth century. The varied and conflicting reasons advanced for the extinction by commentators in the present,[2] and previous centuries,[3,4] speak for the vast potential for disagreement in matters relating to this disease.

The prime problem of medicine in the nineteenth century was the causation of disease;[5] it was also a time during which the leprosy-free countries of Europe were frequently reminded that the malady continued to thrive in large areas of the world. Regarded simultaneously as a "loathsome" disease of lesser civilizations,[6] and an "aristocrat among diseases,"[7] the problem which leprosy posed to the nineteenth century physician was so intensely challenging, not least in the elucidation of its cause—apparently plain and yet tantalizingly difficult to pin down—that controversy dogged the labors of all who picked up the challenge. So many etiologies were proposed that Hansen in 1895 was provoked to comment "There is hardly anything on earth, or between it and heaven, which has not been regarded as the cause of leprosy."[8]

This essay presents an account of the main anti-contagionist ideologies that participated in the causation debates in the last century; it ends with the First International Leprosy Congress in 1897, an event which marked the retreat of anti-contagionism but, as modern developments show, not its defeat. A notable point brought out in this study is the resilience of the anti-contagionist sentiment in the British medical mind of

[1] J. Hutchinson. "On Leprosy and Fish-Eating: A Statement of Facts and Explanations." London, 1906.
[2] G. Rosen. "A History of Public Health." Baltimore, 1993.
[3] G. White. The Natural History of Selborne. Vol. 1. John Van Voorst, London, 1877.
[4] J. Cantlie. Prize Essays on Leprosy. New Sydenham Society, London, 1890.
[5] W. Bulloch. "The History of Bacteriology." London, 1938.
[6] Br. Med. J. Leprosy in the East Indies. December 6, 1862, 602.
[7] J. V. Klander. Sir Jonathan Hutchinson. Med. Life **41** (1934) 313–326.
[8] G. A. Hansen and C. Looft. Leprosy in its Clinical and Pathological Aspects. Trans. from the German by N. Walker. London, 1895.

the Victorian era. Attention will also be directed to the different perspectives from which contagionists and their adversaries assessed the role and utility of the leprosy asylum. As the medical historian Ackerknecht remarked in reference to anti-contagionism in the context of epidemic disease, "discussion was never a discussion on contagion alone, but ALWAYS ON CONTAGION AND QUARANTINES."[9]

A bird's eye view of the various inherited, acquired and environmental influences bearing on etiology was presented by Rogers and Muir in the first edition of *Leprosy.*[10] The racial and colonial-political tensions resulting partly from the etiologic uncertainties about leprosy in the last century have been well described and critiqued by Gussow[11] and Kakar[12] in relation to the United States and India, respectively.

Opposition to the contagion hypothesis came from three schools of thought: the hereditarian, the dietary, and the sanitarian, with the last proving to be particularly stubborn in resisting the implications of Hansen's discovery.

EIGHTEENTH AND EARLY NINETEENTH CENTURY

To be sure, pre-nineteenth century propositions about the cause of leprosy did not arise *de novo;* the ideological framework of each was bequeathed by over a century of observation in the leprosy-endemic northern and southern fringes of Europe and in leprosy-prone tropical countries with a colonial connection. It was through such studies that leprosy maintained a foothold in mainstream medical consciousness in Europe.

It is possible that the contagion hypothesis historically arose from the outward similarities of the signs of leprosy and syphilis. But, as the shrewd eighteenth century observer Thomas Heberden pointed out, "the leprous infection is of a different nature from that of the pox . . . it is not so easily communicated."[13] He theorized that the venereally acquired leprous contagion lay dormant in the "masses of blood," and produced the full-blown disease in the individual under propitious circumstances. The individual himself was capable of transmitting it to his posterity, "[who] are never secure from it. . . ." Thus, Heberden had already shown an awareness of two possible means of dissemination of leprosy—heredity and contagion. That leprosy was contagious was defended by students of the disease situated as far apart as Scandinavia[14] and Surinam.[15]

Even in a field so geographically vast and varied, observers recognized the archetype (lepromatous leprosy) wherever the disease was met, and a consensus was present on other aspects as well. All recognized the striking tendency to familial occurrence, sometimes spanning several generations. Also the rarity of conjugal leprosy; for example, the offspring of a healthy wife (herself of healthy parentage) could manifest their father's disease in later life.

At the dawn of the nineteenth century, the word "contagion" strictly meant a disease poison ("virus") transferred from the sick to the healthy by touch, while "infection" was used to describe transmission by a medium, usually air. Over the course of the century, disputes arose because the words were used indiscriminately by some physicians, while others required a more precise application. As this essay shows, a great part of the disagreements about leprosy was the result of differing interpretations of the term "contagion."

A hereditary disease was understood to be one which affected more than one generation of a family, either as a structural defect of organs ("solidism") or abnormalities of body fluids ("humoralism"), and was present at or soon after birth.

---

[9] E. H. Ackerknecht. Anti-Contagionism between 1821 and 1867. Bull. Hist. Med. **22** (1948) 562–593.

[10] L. Rogers and E. Muir. Leprosy. Bristol: John Wright and Sons, 1925.

[11] Z. Gussow. "Leprosy, Racism, and Public Health. Social Policy in Chronic Disease Control." Colorado, 1989.

[12] S. Kakar. Leprosy in British India, 1860–1940: colonial politics and missionary medicine. Med. Hist. **40** (1996) 215–230.

[13] T. Heberden. Of the Elephantiasis. Comm. by Dr. W. Heberden. Med. Trans. Publ. College of Physicians in London. **1** (1768) 45–53.

[14] J. Reinsterna. Leprosy in Sweden. Int. J. Lepr. **13** (1945) 101.

[15] G. G. Schilling. "Die Lepra Commentiones" (1778), quoted by B. Scheube in "The Diseases of Warm Countries: A Handbook for Medical Men." 2nd. rev. edn. Transl. P. Falcke, J. Cantlie, J., ed. London, 1903.

Atavism—the transmission of the influence through a generation which itself might be clear of the disease ("skipping a generation")—was held to be a characteristic of hereditariness. Later in the century, the ambit was extended to accommodate "hereditary predisposition," when the disease showed itself in later life; ". . . nature is . . . sparing of the direct transmission of disease, [but] she is not equally so of morbid tendencies. What are called hereditary diseases are so merely by predisposition."—Cyclopaedia of Practical Medicine. 2: (1833).

HEREDITARIANISM

The position of contagion theory became increasingly insecure in the first three decades of the nineteenth century, with influential authors attributing the dominant role to heredity acted on by external factors such as climate and temperature and deleterious living conditions. Important followers of this school of thought were Jean-Louis Alibert in France,[16] Joseph Adams in Madeira,[17] and Whitelaw Ainslie in India.[18]

TRACADIE

The first opportunity to challenge directly the contagionist explanation arose neither in endemic Europe nor in a tropical country, but in British Canada. The occasion illustrates the insuperable difficulty experienced by the early anti-contagionists in anticipating that a chronic disease like leprosy could be contagious in the same manner (if not in the same degree) as an acute exanthem.

In 1844 over two dozen cases of "a frightful and loathsome disease" were detected among the French-speaking inhabitants of Tracadie in New Brunswick, descendants of immigrants from Brittany. A Colonial Commission of Inquiry identified the disease as "the tubercular Leprosy which prevailed throughout Europe during the Middle Ages." Many of the cases were familial, and all were traced to Ursule Landre, a woman who manifested leprosy in 1817 and died of it in 1828. The Commission concluded that the disease was "*not only hereditary, but contagious*" (italics in the original), and unanimously recommended the erection of a lazaretto for compulsory isolation of the affected.

Two vocal critics of the Commission were AIexander Boyle in 1844[19] and Robert Bayard in 1849.[20] Both railed against segregation as an unjustified interference with personal liberty based on ill-grounded inferences. Boyle pointed to the fact that the Commission had not shown from whom and how Ursule Landre herself had acquired the contagion, while Bayard noted that no cases of conjugal leprosy or transmission from diseased child to healthy parent had occurred in affected families in spite of the closest contact with the sufferer. According to him, hereditary transmission was the most likely cause, since the majority of the cases were from consanguineous families and, as such, had some common hereditary endowments.

Using smallpox as the standard, Bayard maintained that "there is no sort of analogy [with leprosy]." For example, the latent period in contagious diseases seldom exceeded a few days or weeks, while in leprosy "several years of continued intercourse intervened between the first communication with the infected person and the appearance of disease in the individuals exposed. . . ." Contagious diseases like smallpox were self-limiting when not fatal, while leprosy was a life-long malady. Last, but not least, in the length of time in which the outbreak had developed and spread in Tracadie, a true contagious disease would have exacted a far larger toll than leprosy (40 cases, including 18 deaths, in a community of 5000 in 28 years). Bayard dismissed the possibility that virulence of a putative contagion

[16] J. L. Alibert "Description des Maladies de la Peau." Paris, 1806.

[17] J. Adams. "A Treatise on the Supposed Hereditary Properties of Diseases: with Notes, Illustrative of the Subject, Particularly in Madness and Scrofula." London, 1814.

[18] W. Ainslie. Observations on the lepra arabum, or elephantiasis of the Greeks, as it appears in India. Trans. R. Asiatic Soc. **1** (1826) 22.

[19] A. Boyle. Lepra graecorum contagiosa? Nova pestis adest! Med.-Chir. Trans. **72** (1844) 543–548.

[20] R. Bayard. An essay on Greek elephantiasis occurring in Tracadie and its vicinities. Lancet **2** (1849) 260–262.

differed in leprosy and smallpox; the hypothesis presented "insurmountable difficulties." In the cases not amenable to explanation by hereditary transmission (all hereditarians were forced to acknowledge the existence of such cases), he postulated inoculation of "morbid material," combined with a "ready susceptibility of the person receiving the disease." He concluded his anti-contagionist diatribe with an assertion of the hereditarians' tenet, "some fitness in the constitution, and a corresponding fitness of exciting causes."

NORWAY

Some authors regard the years 1847–1848 as marking the advent of hereditarianism in leprosy. Rogers and Muir, for example, stated that the doctrine was "due very largely to the teaching of the great Norwegian authorities, Danielssen and Boeck, in their book published in 1848." But such an assessment is obviously incorrect. It is more appropriate to say that the theory acquired an enhanced respectability when it was championed in Danielssen and Boeck's publication[21] which Rudolf Virchow said marked the beginning of the "modern biological knowledge of leprosy."[22] In addition to pathological and clinical observations, the authors were the first to state explicitly that leprosy was not a local but a constitutional malady, marked by quantifiable abnormalities in the blood (primarily a disproportion in "albumen" and "fibrine" which led to deposits in the tissues). They concluded that pathogenetically leprosy was a hereditary depraved condition of the blood. This assertion placed the Norwegian authors firmly in the school of neo-humoralism lately inspired by Virchow's rival, the Viennese pathologist Carl Rokitansky.

"How far is it contagious?" they asked, and answered, "Not at all." Direct hereditary transmission was the chief cause, based on a tabulation of 213 patients by blood relationship with another sufferer. Their study showed a rather generous reach of the supposed hereditary influence. Consanguinity in direct or collateral line was present in 88% of all patients. The influence followed the maternal more frequently than the paternal route (1.3:1), was more common in the second and fourth generations than the first and third, and in the collateral than in the direct line (116:69). Their finding that leprosy showed atavism clinched the case for hereditarianism: ". . . [it] frequently skips over one, two, or three generations, to reappear with fearful severity in the fourth."[23]

With the same circular reasoning used by their hereditarian predecessors, Danielssen and Boeck attributed the "spontaneous" cases to residence "under unfavourable circumstances and climates where the disease is endemic."

So strong was the belief as to the hereditary character, that the Norwegian authorities, under Danielssen's influence, were not averse to considering drastic methods to prevent the leprosy patient from producing progeny. Besides sexual segregation in asylums, ligation of the vasa deferentia and prohibition of marriage by patients and their immediate descendants were the possibilities discussed.

Even in 1870, when the germ theory of disease was beginning to rear its head, Danielssen refused to bend. In the Triennial Report of the Lungegaard's Hospital (1868–1870) he showed that he was aware that, according to the infection theory, the blood was not the primary seat of pathology but a medium for dissemination of externally introduced morbific material, and admitted that ". . . many phenomena are undeniably better understood on this theory than on that of a blood crisis." But the old hereditarian refused to be convinced that such a state of affairs applied *in toto* to leprosy: "with regard to leprosy it may yet be that a permanent dyscrasia is its head-spring."[24]

His co-author Carl Boeck was at this time (1869–1870) studying leprosy-affected Norwegian immigrants and their descendants in the United States. When he found a direct or collateral family history in eight of nine cases of the disease develop-

[21] D. C. Danielssen and C. W. Boeck. "Traite de la Spedalskhed ou Elephantiasis des Grecs." Paris, 1848.

[22] O. K. Skinsnes. Notes from the history of leprosy. Int J. Lepr. **41** (1973) 220–237.

[23] Review of Danielssen and Boeck's treatise in Br. For. Med.-Chir. Rev. **25** (1850) 171–182.

[24] Quoted by H. V. Carter in "On Lepresy and Elephantiasis." London, 1874.

ing after immigration, he felt vindicated: "I want to say that, if I formerly may have doubted the theory about the heredity of the disease, I have now no longer any doubts about it."[25]

RUDOLF VIRCHOW

The Norwegian study tour of Rudolf Virchow in 1859[26] marked the entry of a professional pathologist into the causation debate. Although he collected a large amount of valuable pathological material, the inconclusive epidemiological findings gave him pause on the question of etiology; in 1860, in view of "the great importance of the subject," he issued an appeal for information from other leprosy-endemic countries on the role of inheritance, contagion, geographic (e.g., climate and soil) and dietary (e.g., consumption of spoiled fish) factors. He was disappointed again, as he confessed in the famous lectures on cellular pathology delivered at the University of Berlin in 1862.[27]

The founder of cellular pathology who viewed diseases as specific vulnerabilities of tissue lost no time in criticizing the humoralism embodied in Danielssen and Boeck's "dyscrasia sanguinis." Virchow agreed that a hereditary element was a "long established" fact in leprosy, but preferred to refer to it as a hereditary "predisposition" since "the disease is rarely congenitally present, developing only in later years." Yet he had reservations about full hearted support of the hereditary hypothesis: "the idea of inheritance [is] inadequate," because the spontaneous disappearance of leprosy from large parts of Europe could not be explained "except on the basis of a special cause." Virchow could not bring himself to consider that control of contagion might have been that special cause, even as he acknowledged that "immigrants to leprous countries not too infrequently acquire the disease [spontaneously]." Virchow decided that the contagiousness of leprosy was an "improbable . . . idea more and more [to be] abandoned." He did allocate a special role to locality, which, he pointed out, could be hospitable to the disease (along sea coasts in Scandinavia) as well as hostile to it (in North America). On the whole, his speculations were infructuous: "I can only say that, according to my knowledge, it is not at the present time certain what the determinant causal factor of the disease is." Apologists for Virchow, notably Ackerknecht, maintain that "in disproving the dogma of the hereditary nature of leprosy in 1859, Virchow cleared the way for the discovery of the lepra bacillus by Armauer Hansen."[28] In fact, Virchow not only commented favorably on the inheritance theory but, like other hereditarians before him, chose to be blind to the etiologic import of the "rare" spontaneous case.

THE SANITARIANS AND LEPROSY

The belief that dirt contributed to leprosy was common as early as the the sixteenth century in Scandinavia,[29] but it was not until the mid-nineteenth century that a vocal anti-contagionist sanitarian lobby arose, particularly in Britain. Surprisingly, social factors and poor hygienic conditions had not been alluded to by Rudolf Virchow, an early protagonist of public health reform in Germany, in his speculations on causation.

In 1862, a dispatch from the Governor of Barbados to the Colonial Secretary drew attention to "the increase of this fearful malady in recent years,"[30] and suggested that a full-fledged inquiry into the subject would be widely beneficial. On request, a six-member special Committee of the Royal College of Physicians in London, taking the cue from Virchow, formulated a questionnaire to be sent out to medical officers in the West Indies and other British Colonies. The Committee expected that the more

[25] H. P. Lie. Norwegian lepers in the United States: the investigations of Holmboe, Boeck and Hansen. Int. J. Lepr. **6** (1938) 351–356.

[26] M. Vasold. Rudolf Virchow und die Lepra in Norwegen. Medizinhistorisches J. **24** (1989) 123–137. (I am grateful to Dr. Katie Modi for translating this paper.)

[27] R. Virchow. Virchow's Leprosy. Die Krankhaften Geschwulste (1864–65) Transl. G. L. Fite. Int. J. Lepr. **22** (1953) 71–79.

[28] E. H. Ackerknecht. "Rudolf Virchow: Doctor, Statesman, Anthropologist." University of Wisconsin Press, 1948.

[29] F. Henschen. Tuberculosis, leprosy and fungus diseases. In: "The History of Diseases." Transl. J. Tate. Longmans, 1966.

[30] Report on Leprosy of the Royal College of Physicians Prepared for Her Majesty's Secretary of State for the Colonies. London, 1867.

widely broadcast the interrogatories, the greater would be the amount of "authentic information" on which they could base their conclusions and recommendations.

Within a year of launching the questionnaire, and with only 25% of the eventual number of replies in hand, the Committee informed the government that ". . . a very large majority of the reporters consider the disease to be not contagious or communicable to healthy persons by proximity or contact with the diseased. The replies already received contain no evidence that, in the opinion of the Committee, would justify any measures for the compulsory segregation of lepers." (The Committee was being less than accurate in claiming that their "forcible" interim conclusion was "authoritatively" based; it ran counter to their own earlier comment that none of the replies—whether pro-contagion, anti-contagion or non-committal—had been backed by "satisfactory evidence in favour of the opinion." In other words, they admitted that opinions expressed had not been buttressed by proof.)

The doctrine asserted by the College in their final report published in 1867 formed the heart of the sanitarian ideology, that leprosy was a nonspecific disease and, therefore, did not require a specific strategy for prevention and control. "Leprosy is essentially a constitutional disorder, indicative of a cachexia or a depraved condition of the general system . . . [and that] the hope of extirpating the malady amid a people must rest mainly on the adoption of measures for ameliorating their general health and amending their physical condition, can scarcely admit of doubt . . . ."

The College's bold stand against forcible segregation led to the closure of leprosy asylums, withdrawal of state measures against leprosy in the West Indies and other Caribbean Colonial territories, and the repeal of laws enjoining compulsory isolation in those countries. (Contagionists alleged that these steps were followed by a disastrous increase in leprosy in those countries.)

The pen on the report was that of Gavin Milroy, a leading sanitarian of the Victorian age, whose introduction to leprosy took place in 1853 during a tour of the West Indies to investigate a cholera epidemic. In that report he had come out with a strong anti-contagionist statement on cholera, and denounced quarantine as "useless." None of the other Leprosy Committee members had ever studied leprosy; H. H. Scott surmised that this was to ensure an objective and unbiased conclusion.[31]

In 1873 Milroy got another chance to opine on the question, having used his considerable influence with the College to be appointed a one-man commission to report on "the imputed contagiousness of Leprosy [and Yaws] in the West Indies."[32] But it did not result in any change of his basic views. He re-asserted that leprosy was not a specific disease, but a [possibly hereditary] "distemper" of the whole bodily framework and system, brought on by ". . . [unwholesome] food, unhygienic conditions, climate and malaria . . . ." Quarantine [isolation in asylums] was "not in accord with the teachings of medical experience, and [served] to perpetuate many delusions . . . there is no need, on the ground of public health, for the enforced segregation of leprous patients . . . ." He urged those in public affairs to "expedite and greatly facilitate the introduction of hygienic reforms."

Milroy's bald sanitarianism was forcefully repudiated by N. C. Macnamara, a member of the Bengal Medical Service, who in 1866 independently analyzed the 107 replies sent in from east India to the Royal College.[33] The disease was contagious and inoculable, he said, and "neither climate, kinds of food, nor filthy habits are capable of generating leprosy."

The Royal College faced criticism from another India medical officer, H. V. Carter of the Bombay Presidency. In 1871,[34] with 10 years of personal study to back him, Carter refuted all of Milroy's contentions: ". . . the various subclimates of the Concan and the Deccan have no essential influence on the prevalence . . . malaria [has] no con-

[31] H. H. Scott. "A History of Tropical Medicine." London: Edward Arnold & Co. Ltd., 1939.

[32] G. Milroy. Report on Leprosy and Yaws in the West Indies. H.M.S.O., London, 1873.

[33] N. C. Macnamara. "Leprosy." Calcutta, 1866. Reviewed in Br. For. Med.-Chir. Rev. **40** (1867) 141–142.

[34] H. V. Carter. Report on the prevalence and characters of leprosy in the Bombay Presidency, India, based on the official returns of 1867. Trans. Med. Phys. Soc. Bombay (New Series) **11** (1871) 75–250.

nection with leprosy . . . nor is this disease attributable to . . . diet, or social habits . . . or even to defects of sanitation. . . . [The] existence of a primary dyscrasia or blood change, seems hypothetical and even needless. I still hold that there is no primary cachexia in leprosy."

Carter alleged that the Royal College Report of 1867 had led to a regrettable "discountenancing" of leper asylums, when the need was for more rather than fewer such institutions. Disagreements between Milroy and Carter became more vehement after Milroy's tour of the West Indies and Carter's eye-opening visit to Norway in 1873. The opinions of each on the asylum question had, if anything, hardened. Carter had become a confirmed contagionist after seeing Hansen's demonstration of the bacilli; like that scientist, Carter saw proof positive of the efficacy of asylums in the steep decline of the disease in Norway.

Milroy refuted the allegation that the College had ever "discountenanced" leper asylums—they had merely suggested the "discontinuance" of a policy of compulsory isolation, he maintained, since the disease was not contagious.

The Royal College remained a staunch opponent of the contagion theory. In 1877, even 4 years after Hansen's discovery, they did "not allow that the disease has been shown to be contagious."[35] Their argument was that since only about 30% of Norwegian patients were isolated, the good effects of segregation could just as well be ascribed to "medical and moral treatment" and sanitary measures in asylums.

Anti-contagionist ideology also pervaded the highest echelons of the Indian medical officialdom in the person of J. M. Cuningham, the government's Sanitary Commissioner. Like Milroy, Cuningham had cut his sanitarian teeth on the matter of cholera causation and opposition to "ineffective" quarantines. "Cholera," he wrote, "is favoured by filth, overcrowding and every other condition averse to health. . . . Every sanitary defect must be sought out and, as far as possible, remedied."[36]

Cuningham dismissed Carter's contagionist opinions as "[resting] on a very slender basis of evidence . . . it is not probable that such a partial measure [isolation of a minority of patients in Norway] could have had a very decided effect . . . . segregation commends itself to those who believe that leprosy is in some way or the other contagious, but it would appear that there is very little to support this idea. . . ."[37] Even granting for the sake of argument, he said, that asylums and systematic segregation in Norway were as effective as Carter maintained, he declined on financial and logistic grounds to emulate the measures in India; "it will be impractical to put Dr Carter's theory in practice in this country."

JONATHAN HUTCHINSON

Jonathan Hutchinson, surgeon and dermatologist, was a doyen of the medical profession in Victorian Britain; he became interested in leprosy in 1859 and remained so until his death in 1906. He had an obsessive belief that a fish diet was the clue to leprosy. During his long career he proved remarkably inventive in adapting his "icthyophagy" theory to accommodate every objection put forward by unbelievers. The fact that leprosy abounded along Scandinavian sea coasts he attributed to the immoderate consumption of fish. When it was pointed out that leprosy was absent in many heavily "icthyophagic" regions of the world, Hutchinson's explanation was: "We may safely hold that the absence of leprosy under conditions otherwise conducive to it may be explained by the abundance of salt . . . ." Leprosy appeared to be spreading along the routes of Chinese immigration to Hawaii and the Far East. Hutchinson explained it thus: "They are skillful cooks, and they can make use of many things which no one else would look at; decomposing fish and potted fish are amongst the delicacies in which they deal. . . ."[38] Leprosy attacked the Brahmins of India who were strict vegetarians. Hutchinson's explanation: "I have been assured by many persons who had enjoyed excellent opportunities in observation that a conscience for truth-

[35] Editorial. The Leper. Lancet, April 21, 1877, p. 584.

[36] M. Harrison. "Public Health in British India: Anglo-Indian Preventive Medicine." Cambridge University Press, 1994.

[37] J. Hutchinson. Remarks on some facts illustrating the early stage of leprosy. Br. Med. J. March 8, 1890, 529–531.

[38] Med. Press and Circular, August 11, 1880.

speaking does not exist in the Asiatic mind . . . ."[1]

Hutchinson continued his anti-contagionist tirade even into 1897: . . . the discovery by G. A. Hansen . . . has had . . . a most unfortunate effect in strengthening the opinions [of the contagionists]. . . . 'Here is a disease which has a bacillus', they say; 'it must be contagious, and absolutely incapable of originating in any other way' . . . and refuse to look . . . at the overwhelming evidence which connects the malady with local and dietetic influences." He was equally outspoken against advocates of compulsory segregation who, he felt, had never "fairly approached the historical problem of the decline of Leprosy in Europe [in spite of inefficient segregation]."[39]

INDIAN LEPROSY COMMISSION

Neisser's success in staining Hansen's bacilli in 1879 and, more dramatically, the death from lepromatous leprosy of Father Damien in 1889, enthused the contagionists. An equally forceful ultra-contagionist lobby also arose, which foretold doom for Britain's empire in India from the "alarming" increase of leprosy in that country.[12]

The Indian Leprosy Commission, composed of three members nominated by the London Royal Colleges and the Damien Memorial National Leprosy Fund, toured the country in 1890-1891 with a mandate to opine on the etiology problem and the segregation question as well. As in the 1860s, only one member, Beaven Rake of the Trinidad Leprosy Asylum (not surprisingly a nominee of the Royal College of Physicians), had any experience of leprosy. He had come to notice earlier by his refusal to find the Damien case at all compelling as proof of the doctrine of contagion since ". . . [Damien] may . . . have absorbed the specific virus (now generally believed to be the bacillus leprae) in many other ways, e.g., in food, water, air, etc."[40]

The Commission's first priority was to demolish the allegation of the ultra-contagionists that leprosy in India posed an "Imperial danger."[41] They pointed out that census enumerations were conducted by untrained village officials, so it was impossible to be accurate about the actual number of leprosy patients in India. Neither were statistics of asylum inmates at all reflective of patient numbers. "Even making all due allowances for errors . . . it is plain that the recent outcry about an alarming increase of leprosy in British India is not based on fact, and that such increase has not taken place. . . ."

The Commissioners personally examined over 2000 patients and made exhaustive analyses of geographic, climatic, telluric, dietetic, economic, racial, hereditary and biological factors that might singly or in combination play a role in causing the disease.

The opening sentence of the chapter on "Contagiousness of Leprosy" in the Commission's Report gave a hint about their own bias: "All modern authorities are agreed that leprosy is an infective disease, that is one caused by a microbe, the bacillus leprae, which obtains access to the body from without . . . [but] the next point to consider is whether leprosy is *contagious"* (italics in original).

The question of contagion was approached by a manipulation of a comment made by the bacteriologist Flugge in 1886 that "the diffusion of leprosy by contagion is *exceedingly rare,* and evidently can only take place *under special and predisposing favourable conditions"* (italics by the Commission). "Now it may be mentioned at once," argued the Commissioners, "that the more weight is attached to 'special favourable conditions'. . . the further contagion disappears into the background. Indeed, for all practical purposes—and these are what the legislator or sanitary reformer has to consider—it vanishes altogether."

They insisted that "the abstract and scientific" meaning of "contagion" be separated from the "practical" one. Scientifically leprosy might be a bacillary disease, but its contagiousness appeared to be no greater than that of tuberculosis, and certainly was not of the order of the "par excellence" contagious diseases diphtheria and erysipelas. (Like their ideological forebears of the 1840s, late nineteenth century anti-contagionists of a literal bent of mind saw no analogy between leprosy and exemplary contagious diseases.)

The Commission considered it regrettable that bacteriology had generated a con-

[39] J. Hutchinson. On the present position of the leprosy problem. Edin. Med. J. NS. **1** (1897) 121–123.

[40] B. Rake. Preliminary remarks on the spread of leprosy. J. Lepr. Inv. Comm. **1** (1890) 47–52.

[41] Leprosy in India. Report of the Leprosy Commission in India 1890–91. Calcutta 1892.

tagionist bias, disregarding the fact that contagion might not be "the natural mode of infection," as evidenced in the patients' histories. "The native leper, in an overwhelming majority of instances, . . . denies that he has ever had any contact or intercourse with lepers. . . . In other cases a man will relate that many years ago he came into contact with a leper. . . ." Such instances were regarded by some authors as proof of a long incubation period, but the Commission averred that ". . . the assumption of an irrationally and disproportionately long incubation period is certainly a weak point in any theory of contagion."

The Commission also fell back on the time-honored argument of the rarity of conjugal leprosy, and refuted Hansen's assertion that familial leprosy was proof of contagion. They said the disease never spread "sufficiently" within a family to "warrant the conclusion that it is contagious to any extent."

The label "minimalist" if not "nihilist" might well be applied to the Commission's philosophy, which proved that even in the 1890s it was possible to arrive at an etiologic conclusion without so much as a mention of the bacillus leprae.

Firstly, they made it clear that they did not claim worldwide applicability for their opinions which "only apply to this [India] empire."

Next they excluded hereditary transmission, contagious dissemination, and environmental, dietary and sanitary factors *per se* as causes of leprosy.

They then stated their conclusion that, in the vast maiority of cases, a combination of deleterious natural and sanitary factors produced a predisposition which increased the susceptibility of the individual to the disease, i.e., leprosy was the result of predisposition acquired partly from sanitary factors.

Lastly, the Commission delivered its judgment on the matter of great concern to the government of India: "Since . . . under the ordinary human surroundings the amount of contagion . . . is so small that it may be disregarded, *no legislation is called for on the lines either of segregation, or of interdiction of marriages with lepers"* (italics in original). They considered "lepers to be far less dangerous to a community than insane or syphilitic persons."

The high-level Evaluation Committee in England found themselves divided on the Commission's report.[42] One group (which included a pro-segregationist missionary, a bureaucrat, and the contagionist N. C. Macnamara) strongly disagreed that the contagion factor was "exceedingly small," and that segregation was therefore impracticable and undesirable.

The other group (which included two anti-contagionist India veterans and the anti-segregationist Jonathan Hutchinson) endorsed the conclusions. Given the anti-contagionist leanings of the Indian medical establishment, it is not difficult to guess which of the views the government of India chose to accept.

THE BERLIN CONFERENCE

The largely attended Berlin Conference held in 1897 produced a Final Statement which was a triumph for Hansen's long-held positions on contagion and the absolute necessity for national state policies for segregation and isolation, based on the Norwegian model:

"(1) The Leprosy Bacillus is the Real Cause of the Disease.

"(2) Leprosy is infectious but not hereditary; man is the only animal affected.

"(3) The worse the social conditions, the greater the danger of infection.

"(4) The success of the segregation method in Norway is a strong argument in its favour . . . the segregation of lepers ought to be compulsory. If the measures in vogue in Norway could only be put into universal practice, the disease would be quickly eradicated."

A significant section of the leprosy world was not represented at the Conference. There being no official delegate from India, it was left to the British moderate Phineas Abraham to point out the unenforceability of "harsh measures of isolation where lepers are numerous, and their friends still more numerous."[43] In India the serious dif-

[42] Memorandum on the Report of the Leprosy Commissioners, as Prepared by Special Committee, Appointed for the Purpose, and Endorsed or Annotated by Members of the Executive Committee of the National Leprosy Fund, London, 1890.

[43] The Times of India. November 10, 1897, p. 6, col. 4.

ficulty faced by the Government in enforcing compulsory segregation and sanitary measures had been demonstrated in the then raging plague epidemic.[44] Abraham's may be regarded as a last ditch attempt of colonial pragmatists and sceptics of contagionism to downplay the much-touted effectiveness of isolation asylums.

COMMENT

It has to be admitted that the Berlin Congress Statement imposed a somewhat blinkered perspective on the causation problem. By designating the bacillus as the "real" cause, the Congress implied that it was a necessary and sufficient one. In awarding such autonomy to the infectious principle, the participation of hereditary or acquired predisposition, Virchowian specific cellular vulnerability, inter-current disease and racial factors were heavily downgraded. Deleterious living conditions were viewed as merely enabling transmission of the infectious principle.

Such a unicausal etiology contrasts with the multidimensional approach of some other late nineteenth century commentators, e.g., Robson Roose[45] and George Thin,[46] who did not deny infection, but insisted that its effect was inextricably linked with biological and external circumstances.

The polar opposite of strident contagionism was the implacable sanitarian ideology entrenched at the Royal College of Physicians, which viewed leprosy as a nonspecific debility caused by substandard living conditions. That this view was not an isolated aberration of British medical authority is shown by Howard-Jones with regard to that country's stand in the nineteenth century causation debates on cholera and plague.[47] It is not surprising that British sanitarians put up stiff resistance to the contagion hypothesis, considering that Britain was the mother-country of the nineteenth-century public health movement. The central dogma of the ideology was the nonspecificity of disease, so succinctly expressed by Florence Nightingale, a leading light of the movement. "For diseases, as all experience shows, are adjectives, not noun substantives." Diseases were children of "conditions, as a dirty and clean condition . . . [which are] under our own control . . . ."[48]

Not only ideology but etymology also fanned the causation controversy, because sanitarians tended to interpret the term "contagion" literally, while contagionist opponents unreservedly used it in the general sense of communicability.

Surprisingly, all schools of thought, except the libertarians (who were either critical of any interference with the personal freedom of sufferers or who, like Hutchinson, thought the money could be better spent on research) were agreed that asylums were desirable institutions. Of course, their reasons differed.

Hereditarians such as Danielssen hoped that enforcement of sexual segregation in asylums would prevent hereditary transmission of the disease. Contagionists such as Hansen and Carter regarded systematic segregation as the only feasible method of eradication of the disease from a country, and proudly pointed at Norway's record in justification. Sanitarians saw asylums as providing shelter, food, "moral and sanitary improvement," and therapeutic occupation to demoralized victims of bad living conditions. Missionaries quickly saw the opportunity for evangelization among captive asylum inmates. Alarmists in England regarded asylums as strategic defenses against infection of the mother country, while their more cautious and prudent counterparts in India supported the erection of asylums to incarcerate pauper lepers who were a blot on the fair face of the colonial cities of Calcutta, Bombay and Madras.

The credo of all groups was "Be good to the leper but lock him up."

From today's multidimensional perspective, the difference between conditions and

[44] D. Arnold. "Touching the Body: Perspectives on the Indian Plague 1896–1900." Subaltern Studies V. Guha, R., ed. Delhi: Oxford University Press, 1987.

[45] R. Roose. "Leprosy and Its Prevention as Illustrated by Norwegian Experience." London: H. K. Lewis, 1890.

[46] G. Thin. Leprosy. London: Percival, 1891.

[47] N. Howard-Jones. "The Scientific Background of the International Sanitary Conferences 1851–1938." Geneva, 1975.

[48] C. E. Rosenberg. Florence Nightingale on Contagion: The Hospital as Moral Universe from "Explaining Epidemics and Other Studies in the History of Medicine." London, 1992.

causes is not as clear cut as the Conference made out. Today, inherited, acquired, and racial vulnerability to leprosy are considered subjects of scientific study. Considering that aerial dissemination of leprosy is (and was, as acknowledged by the Berlin Congress) a probability, and the absence of any effective treatment until recently, it is highly unlikely that isolation alone eliminated leprosy from either medieval Europe or Hansen's Norway.

Dr. C. Heineken of Madeira wrote in 1826: "The exciting causes appear to be confined to such as poverty and its attendant evils engender . . . insufficient shelter and clothing, spare, unwholesome diet, great and sudden alterations of temperature, and filth, in their various combinations, appear to be necessary for its production."[49]

Dr. L. M. Bechelli of the World Health Organization wrote in 1973: "additional factors such as [improvement] in the socio-economic situation, education, hygiene and housing, play a role in the control of the disease."[50]

Plus a change, plus de la meme!

—Shubhada S. Pandya, M.B.B.S.

11 Shanti Kutir
Netaji Subhash Road
Bombay 400 020, India

Acknowledgment Reference work for this study was conducted at The Wellcome Institute for the History of Medicine under a travel grant from The Wellcome Trust, London, in May–June 1997. I am thankful to the Trustees.

[49] C. Heineken. Observations on the leprosy of Madeira. Edin. Med. Surg. J. **26** (1826) 15–25.

[50] L. M. Bechelli. Advances in leprosy control in the last 100 years. Int. J. Lepr. **41** (1973) 287–288.