BIBLIOTECA
INTERNATIONAL
JOURNAL OF LEPROSY

VOL. 8 APRIL-JUNE, 1940 No. 2

# FAMILIAL SUSCEPTIBILITY AS A FACTOR IN THE PROPAGATION OF LEPROSY IN NORTH AMERICA[1]

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Leprosy, not indigenous to North America, has been introduced into all sections of the continent but has not spread except in a few sharply circumscribed areas.[2] One feature of the disease in each of these foci is its tendency to remain restricted through successive generations to certain racial stocks, immigrant from regions of prevalence or in whom the disease occurs elsewhere. Other nationalities within the areas are nearly or entirely exempt. Furthermore, these foci tend to be sharply delimited by the boundaries of the areas populated by the particular groups concerned. The localities involved are widely separated geographically, and differ markedly with respect to environmental influences. Although various nationalities are represented in the several foci, in the two which are the largest and most widely separated, the affected individuals are of a common ancestry.

These observations suggest that the continued occurrence of leprosy in certain localities on this continent is not dependent on the presence of infected individuals alone, or on environmental factors peculiar to these areas, but that, in addition to the presence of cases as sources of infection, it is contingent on some circumstance which is inherent in individuals of the groups involved.

[1]This work was financed by the Harvard Infantile Paralysis Commission and a grant from the Commonwealth Fund.

[2]A consideration of the disease in Mexico is not included in this paper since the necessary data are not available.

LEPROSY IN MINNESOTA

Leprosy was introduced into Wisconsin, Iowa and especially Minnesota, in the middle of the nineteenth century by some 160 cases among the Scandinavian immigrants to the Northwest.

Data on 52 such cases in Minnesota was published in 1900 by Bracken (3); 18 had developed the disease in Europe, 28 after coming to the United States. The longest interval elapsing between immigration and the occurrence of symptoms was twenty years; in 19 cases the period was under ten years, and in 9 between ten and twenty years. These intervals are not inconsistent with the belief that the disease was contracted prior to departure from Europe. Thirty-five of the 52 cases died before 1900 (Table 1). Little is known of the first 17, but according to Bracken "from various reports it is safe to assume that they were all from Norway." Of the remainder, 30 were from Norway and 5 from Sweden.

Hansen visited Minnesota in 1888 for the purpose of studying the propagation of leprosy among the Norwegian immigrants and their descendents in this new environment. He reported (6), in refutation of the theory of hereditary transmission:

> We have demonstrated by our investigations in North America that of the numerous descendents of Norwegian lepers there, not one has developed the disease.

This observation was widely quoted in the literature as convincing evidence against heredity as a factor in the epidemiology of leprosy. Rogers and Muir (9) state:

> The most striking example of this (the diminution or absence of leprosy where it should have continued if essentially or even largely an hereditary disease) is furnished by Hansen's observations on 170 Norwegians, who migrated to the temperate northern portion of the United States of America, especially Minnesota, when either suffering from leprosy or in the incubation period of the disease; yet at the time of Hansen's visit to America not one of their descendents up to the third generation had developed leprosy under the favorable hygienic conditions they lived in, which would not have prevented the occurrence of an hereditary disease.

Thirteen years after Hansen's visit (1901), Burnside Foster (5) reported a case of leprosy in Minnesota in an American-born descendent of one of the affected Norwegian families. Referring to Hansen's dictum Foster said:

> You are all familiar with the statement, so frequently made, that all the cases of leprosy in the Northwest have had their origin in some leprous district or some other country, and that for some unexplained reason the disease was never communicated to others here, although there has been abund-

TABLE 1. *Leprosy in Minnesota (Bracken, 1900).*

| No. | Nationality | Disease in Europe | Disease in America [a] | Birth | Death | Age | Sex | Social state | Children |
|---|---|---|---|---|---|---|---|---|---|
| 1 | — | Yes | — | 1831 | 1888 | — | M | — | — |
| 2 | — | — | 10 | 1822 | 1878 | — | M | — | — |
| 3 | — | — | 1 | 1843 | 1878 | — | — | — | — |
| 4 | — | — | 10 | 1846 | 1876 | — | M | — | — |
| 5 | — | — | 3 | 1848 | 1878 | — | M | — | — |
| 6 | — | Yes | — | 1815 | 1877 | — | M | — | — |
| 7 | — | Yes? | — | 1848 | 1868 | — | M | — | — |
| 8 | — | — | 9 | 1825 | 1885 | — | M | — | — |
| 9 | — | — | 9 | 1854 | 1885 | — | F | — | — |
| 10 | — | — | 5 | 1839 | 1884 | — | M | — | — |
| 11 | — | — | 7 | 1853 | 1886 | — | M | — | — |
| 12 | — | — | — | 1818–28 | 1888 | — | M | Mar. | — |
| 13 | — | — | — | 1848–58 | 1888 | — | M | — | — |
| 14 | — | — | — | 1829–39 | 1889 | — | M | Mar. | — |
| 15 | — | — | — | 1858–68 | 1889 | — | M | Sin. | — |
| 16 | — | Yes | — | 1849 | 1890 | — | M | Mar. | — |
| 17 | — | Yes | — | 1842 | 1890 | — | M | Mar. | — |
| 18 | Norwegian | — | 7 | 1816 | 1895 | — | M | Mar. | 5 |
| 19 | Norwegian | — | 13 | 1854 | 1892 | — | M | Sin. | — |
| 20 | — | Yes | — | 1830 | — | 68 | M | Mar. | 1 |
| 21 | — | Yes | — | 1840 | 1896 | — | M | Sin. | — |
| 22 | — | Yes | — | 1848 | 1896 | — | M | Mar. | 5 |
| 23 | — | Yes | — | 1820 | — | 78 | M | — | — |
| 24 | — | — | 20 | 1834 | — | 64 | M | Mar. | 6 |
| 25 | Norwegian | — | 9 | 1857 | 1894 | — | M | Mar. | 3 |
| 26 | — | Yes | — | 1838 | 189– | — | M | Mar. | 2 |
| 27 | — | Yes | — | 1840 | — | 58 | F | Mar. | 6 |
| 28 | Norwegian | Yes | — | 1843 | 1897 | — | M | Mar. | 3 |
| 29 | — | — | 3 | 1864 | — | 34 | M | — | — |
| 30 | Norwegian | Yes | — | 1850 | 189– | — | M | Mar. | Some |
| 31 | — | — | 4 | 1850 | 1892 | — | F | Mar. | — |
| 32 | — | — | 19 | 1826 | — | 72 | M | Sin. | — |
| 33 | — | — | 17 | — | 189– | — | F | Mar. | 4 |
| 34 | — | Yes | — | 1871 | 189– | — | F | Sin. | 1 |
| 35 | Norwegian | — | 4 | 1851 | 189– | — | M | Sin. | 0 |
| 36 | Norwegian | — | 16 | 1867 | — | 31 | M | — | 0 |
| 37 | Norwegian | Yes | — | 1852 | — | 46 | M | — | — |
| 38 | Norwegian | — | 8 | 1860 | — | 38 | M | Mar. | 3 |
| 39 | Norwegian | Yes | — | 1853 | 1897 | | M | Sin. | — |
| 40 | — | ? | — | 1845 | 1894 | — | F | Mar. | 6 |
| 41 | Swedish | ? | — | 1845 | 1897 | — | M | Mar. | 2 |
| 42 | — | — | 7 | 1860 | — | 38 | M | — | — |
| 43 | Swedish | — | 4 | 1865 | — | 33 | F | Mar. | 4 |
| 44 | Norwegian | — | 8 | 1867 | 1897 | — | M | — | — |
| 45 | Norwegian | — | 3 | 1843 | — | 55 | M | Sin. | — |
| 46 | Swedish | — | 8 | 1861 | — | 37 | M | Mar. | 2 |
| 47 | Swedish | — | 6 | 1858 | — | 40 | M | Mar. | 5 |
| 48 | Norwegian | Yes | — | 1862 | 1890 | — | F | Sin. | — |
| 49 | Swedish | — | 13 | 1845 | — | 53 | F | Mar. | 4 |
| 50 | Norwegian | Yes | — | 1863 | 1898 | — | M | Mar. | 1 |
| 51 | Norwegian | — | 13 | 1843 | — | 55 | M | Mar. | 8 |
| 52 | Norwegian | — | 7 | 1856 | — | 42 | M | Sin. | — |

[a] Number of years after immigration.

ant opportunity for such communication. This case puts the matter in a new light.

Foster's report, correcting the widely accepted conclusion of Hansen, seems to have passed almost unnoticed.

Thus Hansen's observation was not only premature but was based on insufficient data. Even at the time he was in Minnesota the patient reported by Foster was already suffering from the disease; he developed symptoms about 1885 and died in 1898, but the case was not reported until his brother was diagnosed as leprous. This was about fourteen years after Hansen's visit and forty years after the immigration of the patient's father from Norway. The patient is said to have had a leprous uncle in Norway whom he had never seen, and in infancy to have been nursed by a woman who had two brothers who were leprous. However, since none of his family had had leprosy in America he was not exposed to "prolonged and intimate contact with members of his own family," to which the familial occurrence of the disease is generally attributed.

Leprosy has continued in Minnesota up to the present time, apparently largely in the descendents of affected Norwegian families. Indeed, Bracken (11) has stated, "We have no record of leprosy occurring outside the family of a leper in Minnesota." The cases of leprosy in Minnesota among American-born descendents of lepers are given in Table 2.

A contemporary case is a woman whose grandmother, mother and uncle had the disease. The grandmother was born in Mora, Sweden, where she lived with her foster mother who had leprosy. She came to Minnesota in 1887 and married in 1888. She had four children, two of whom—the mother and the uncle of the present case—developed the disease.

### LEPROSY IN MANITOBA AND SASKATCHEWAN

In addition to the cases of leprosy from the local New Brunswick focus (to be discussed later), 23 patients have been admitted to the Tracadie leprosarium from other parts of Canada. Of this group 13 were sporadic, from various parts of the Dominion, most of them known to have resided previously in foreign leprous areas. The remaining 10 were admitted from Manitoba and Saskatchewan, the only two localities outside of New Brunswick which present any degree of concentration of the disease.

*Manitoba.*—Four patients, all of them Icelanders, were admitted to Tracadie from Manitoba in 1897, three from Winnepeg

and one from Selkirk. Case records are not available, but it may be presumed that they brought the disease from Iceland, where it has existed for centuries and where the reported figures reached the high mark of 226 cases in 1896.

TABLE 2.—*Leprosy in Minnesota; cases in American-born descendents of leprous families.*[a]

| Case | Year of birth | Year of appearance of disease | Year of report of case | Sex | Nativity of parents | Relatives with leprosy | Year of death |
|---|---|---|---|---|---|---|---|
| G. J.[b] | 1870 | 1885 | 1902 | M | Mo.: Minn.<br>Fa.: Norway | Uncle<br>Brother | 1898 |
| J. J.[c] | 1879 | 1892–95 | 1902 | M | Mo.: Minn.<br>Fa.: Norway | Uncle<br>Brother | 1923 |
| L. G. | 1891 | 1897 | 1906 | F | Fa.: Norway | Father | |
| R.E.W.[c] | 1878 | 1904 | 1906 | M | Fa.: Norway | Father<br>Sister | 1911 |
| L.W.H.[c] | 1874 | 1910 | 1911 | F | Fa.: Norway | Father<br>Brother | 1928 |
| E.M.B.[d] | 1891 | 1906 | 1911 | M | Mo.: Sweden | Mother<br>Sister<br>Niece | 1913 |
| A.B.W.[d] | 1894 | 1916 | 1921 | F | Mo.: Sweden | Mother<br>Brother<br>Daughter | |
| P. W.[d] | 1919 | 1921 | 1921 | F | Mo.: Minn. | G' mother<br>Mother<br>Uncle | |

[a] Data from the Minnesota Department of Health.
[b] Brothers
[c] Brother and sister
[d] E.M.B.: uncle of P.W.; A.B.W.: mother of P.W. (see text, contemporary case).

*Saskatchewan.*—Six patients have been admitted from the province of Saskatchewan, five of whom were Russians and one a Syrian.

There are in Saskatchewan small colonies of Dukhobors, a Russian religious sect the members of which are opposed to participation in military service and for this reason were a persecuted people in their own land. In 1840-50 they were banished from the government of Tauris to Transcaucasia, near the Turkish frontier. This region, south of the Caucasus Mountains, between the Black and Caspian Seas and extending into Azerbadjian, Armenia, Turkmenia and the frontier of Iran, is known to be infected with leprosy.

In 1899 about 7,500 Dukhobors emigrated to Canada, where the Canadian government allotted them land in the Province of Saskatchewan, near Yorktown, Thunder Hill and Prince Albert.

They comprise but a minority of the population in Saskatchewan, yet five of the six cases of leprosy from that province have been in Russia, from villages in the region settled by the Dukhobors; one case was from the town of Verigin, named for the Dukhobor leader in Saskatchewan. Professing as they do opposition to form in religion, it is probable that marriage is limited to the members of their own group.

Four of the five patients evidently were born years before the colonization of the Dukhobors on this continent, and presumably were infected in their native land. The interval elapsing before their admission to the leprosarium may indicate a relatively long incubation period, but it is not known how far advanced the disease was when it was detected. The date of birth of the fifth patient would suggest that the individual was born in Canada and developed the disease there; and recently information has been received which corroborates this assumption. The nationality of the sixth patient from Saskatchewan is given as Syrian. Since both the places of origin and of settlement of this individual are adjacent to those of the Dukhobors, the speculation is permissible that he may have been of the same group but had lived across the border from the Dukhobors in Russia.

The immigration of the affected groups in Manitoba and Saskatchewan was recent (the Dukhobors in 1899) and the cases, with one exception, presumably were imported. If we take the Minnesota experience as the criterion it may still be too early to expect to find additional cases in the descendents of the original Dukhobor or Icelandic immigrants. In Minnesota, with a much larger number of immigrant cases, the first occurrence of the disease in an American-born descendent of the affected Norwegian families was reported in 1902, some forty years after the arrival of the family in this country and fourteen years after Hansen's visit and erroneous conclusion. The single known American-born case in the Saskatchewan group was reported in 1930, thirty-one years after the Dukhobor immigration. An imaginary study of the cases in this focus, corresponding to Hansen's study in Minnesota in 1888, would have been made in 1927; and the disease would not have been revealed in any Canadian-born descendent of the Dukhobors, since the first case was not discovered until 1930.

### LEPROSY IN NEW BRUNSWICK AND LOUISIANA

An outstanding feature of established leprosy on the North

American continent is its continued occurrence in the people of a common origin, who live in two widely separated and circumscribed areas.

### NEW BRUNSWICK

Approximately 300 cases have been recorded from 1815 to the present time in northeast New Brunswick, in an area not larger than a county lying between the mouth of the Miramichi River and the Baie de Chaleur. This territory was partially settled by Norman immigrants, many of whom fled from Nova Scotia at the time of the Acadian expulsion of 1755, crossed the isthmus and scattered along the shores, forming settlements at intervals as far north as the St. Lawrence River. The disease was first detected in one of the older settlements, composed almost entirely of French Acadians of Norman descent.

The first recorded case in New Brunswick was that of a French woman whose paternal grandfather came from St. Malo, Normandy, where leprosy existed. She developed the disease between 1815 and 1818 and died in 1828. Her husband subsequently became leprous, as did also two of her sisters. The disease gradually spread thereafter.

The actual origin of this focus is shrouded in mystery. One account is that the first case was contracted by washing the clothes of sailors who came to Caraquet, New Brunswick, from France. It has also been asserted that, in 1815, two Norwegians took passage in "La Florida," a ship which navigated the Baie de Chaleur under Captain Michael Landry. These men, who were in an advanced stage of leprosy, left the vessel at Misonette, opposite Caraquet, and coming to Tracadie spent some days with the family in which the first cases occurred.

Studies in New Brunswick (1) show that the 293 recorded cases of leprosy occurred in individuals bearing only 69 different surnames. Analysis of the data discloses that changes in spelling have taken place in 12 names in the course of years, and that 18 names were later introduced into the family lines by marriage; leaving only 39 family lines in which these cases arose. Forty-one cases with 25 surnames on which no data are available for establishing relationships have been deducted; thus there remain 252 cases in 14 family lines.

All 14 names appear among the earliest admissions to Tracadie. The records show a great deal of intermarriage between these few families, who have lived in this isolated community

since shortly after the expulsion of 1755; and it is reasonable to suppose that they were already interrelated when the first cases were reported in 1815. The restricted and selective character of the disease is well brought out by the fact that its occurrence has been confined to this circumscribed area and to a relatively small number of families for over 150 years. In fact, two patients who went to the leprosarium in 1937 actually bore the same names as the original cases admitted in 1844.

### LOUISIANA

Leprosy was first recorded in Louisiana in 1766-68, during the administration of Ulloa, when cases occurring among the French were isolated at Balize at the mouth of the Mississippi River. In 1785, Miro established a leper hospital at New Orleans, which had but a brief existence. Subsequent sporadic cases attracted little public interest, and it was not until ninety years later (1872) that Joseph Jones (8) noted the increasing importance of the disease in certain parts of Louisiana. The cases which he observed were principally in persons of French descent, many being offspring of the Acadians who were driven out of Nova Scotia in 1755 and recolonized in Louisiana. The occurrence of leprosy in the Acadians both here and in New Brunswick suggests that it may have been carried to Louisiana by these people, but there is no record of the disease among the Acadians in Nova Scotia, their original home on this continent. The record indicates that the disease was introduced into the two areas long after the Acadians had separated and the groups had gone their different ways.

In 1888, Blanc (2) reported having seen 42 cases in New Orleans in five years. Their histories indicated that they were epidemiologically related to the Acadians, to immigrants from the West Indies, or to more recent immigrants from western Europe.

Denney (4) traced the origin of 471 cases which had developed in Louisiana and found that 391 of the patients were born in the state; 43 came from elsewhere in the United States and 37 were foreign-born. He concluded from this that:

> . . . there is in the state of Louisiana some inherent, although undetermined factor which renders its populace more liable to develop the disease.

That this factor resides in the people rather than in the environment is suggested by the preponderance of the disease in Louisiana among certain racial stocks, the existence of foci in two widely

separated areas each populated by a certain group, and the restriction of the disease to localities occupied by people of the racial stocks involved. The suggestion is that the Louisiana focus, apparently on the wane, took on renewed activity with the introduction of German immigrants, probably including susceptible individuals, since the occurrence of leprosy principally in people of this stock extends into eastern Texas, where there are large German settlements.

Because branches of the same families who went to New Brunswick from Nova Scotia are known to have colonized in Louisiana, studies of the familial occurrence of leprosy begun in New Brunswick have been extended to the Louisiana focus. At one time within recent years there were in the national leprosarium at Carville 106 patients (names not available) who were either born in or admitted from forty-five Louisiana towns. Forty of these towns are in that part of southern Louisiana known as the Teche country.

As a first approximation, telephone directories which were available for twenty-seven of the forty-five towns were consulted. Family names associated with leprosy in New Brunswick were found in twenty-four of the twenty-seven towns. While it is realized that telephone subscribers may not represent a cross-section of the population, a little over 4 percent of 20,000 listings in these twenty-four towns were New Brunswick names. In twenty-two of the towns the names were those of the fourteen family lines to which the majority of cases in New Brunswick can be traced.

Four cases in the leprosarium were connected with the three towns in which no Acadian names were listed. From two of these towns, which are outside the Teche country, there were two cases. One, a Negro, was born in the first town but admitted from outside the state; and the second case, Norwegian-French, was born in and admitted from the second town. The other two patients, French, were born in the third town, in the Teche country, but admitted from elsewhere in Louisiana. Although no Acadians were listed in this small town, it is known that French families reside there.

For the purpose of studying the consanguinity of the families associated with leprosy in New Brunswick and Louisiana, a visit was made in January, 1939, to thirty-six towns in fifteen parishes of the Teche country in southern Louisiana from which cases have been recorded. The names of 114 patients, past and present,

were obtained from various sources. As many of the families as could be located were visited, and through their helpful cooperation genealogic information was obtained concerning 60 of these cases. The data were in many instances extensive, but all histories giving at least the maiden name of the mother of the patient were included. The correctness of the information obtained from one person frequently was corroborated by others. Despite the common opinion that, because of reticence concerning leprosy, such information is difficult to obtain, only two of several hundred individuals interviewed declined to give the desired information.

The 114 patients bear 67 different surnames. Satisfactory family histories were unobtainable in 54 cases, with 41 surnames. The remaining 60 cases bear 26 different surnames. Of this number, 25 have 11 surnames which differ from those of New Brunswick patients, and New Brunswick names do not appear in their genealogies.

The remaining 35 cases either bear the same names as cases in New Brunswick, or have these names in their lines. Twenty-five actually bear nine surnames corresponding to those of New Brunswick patients; six of these are included among the fourteen New Brunswick names which have contributed the largest number of cases in the Canadian focus. Furthermore, seven of these nine families in Louisiana are interrelated.

Text-figs. 1, 2 and 3 show genealogic records of Louisiana families with multiple cases of leprosy. Only so much of the genealogy is given as indicates the occurrence of the disease in blood relatives of the families used as propositi. Individuals with leprosy are shown in solid black, and relationships not exactly traced are indicated by broken lines. Family lines originating in Nova Scotia are indicated by "N.S.," and lines originating in Nova Scotia and in whom leprosy has occurred in New Brunswick by "N.B."

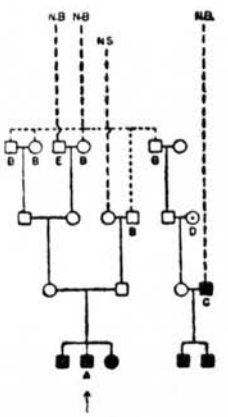
Actual consanguinity has not been directly established between cases in New Brunswick and Louisiana, genealogical records seemingly having been broken off with the expulsion. That these people are related, however, is indicated by the fact that they came from the same place and bear the same names; and there may be added the interesting observation made to me personally by a group of Acadians from the Maritime Provinces who visited in Louisiana that they:

. . . took pleasure in naming people by their family names when meeting them for the first time, the resemblance to people of the same names in New Brunswick being so striking.

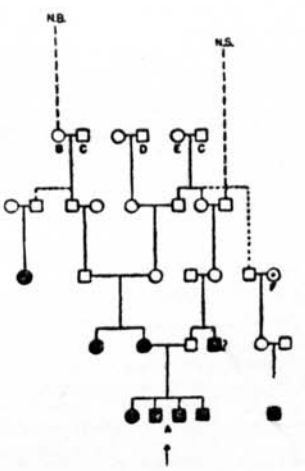
## HEREDITARY SUSCEPTIBILITY

Hereditary predisposition to leprosy was emphasized a century ago by Sir James Simpson (10) in his statement that:

. . . .few facts in the history of tubercular leprosy seem to be more universally admitted by all writers on the disease, both ancient and modern, than the transmission of the predisposition of it from parents to offspring.



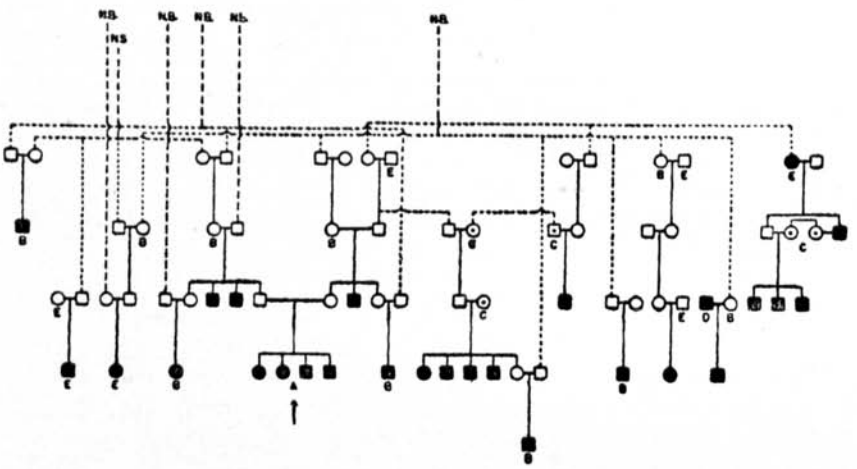
TEXT-FIG. 1. Genealogical record of a Louisiana family with multiple cases of leprosy (see text). A. three cases with three instances of intermarriage in a single family line prominent also in the early cases of leprosy in New Brunswick. B: members of the same family line. C: a case of leprosy in another family line with leprosy in New Brunswick. D: ancestry not known, but said to have relatives with leprosy. E: a second line in which leprosy occurs in New Brunswick.



TEXT-FIG. 2. Genealogical record of a Louisiana family with multiple cases of leprosy (see text). A: four cases in the same family; cases on both father's side and mother's side; father and mother are second cousins. B: family in which leprosy has occurred in New Brunswick. C: family line from France. D: Spanish. E: German. F: ancestry unknown, but said to have relatives with leprosy.

But largely on the strength of the studies of Danielssen and Boeck in Norway, in 1848, hereditary transmission of the disease itself came to be generally accepted. Then, in 1874, the discovery of the Hansen bacillus deflected the emphasis from heredity to the infectious agent, and there arose the school of contagionists, who attributed the high incidence of the disease in the offspring of lepers to intense or prolonged exposure to the infectious agent. For some years there was controversy between this group and those who held to hereditary transmission of the disease. But

both the evident discrepancies in the theory of heredity and the hopelessness seen in it, together with the more hopeful measure of segregation, gained for the contagionists the ascendency which has persisted to the present day.



TEXT-FIG. 3. Genealogical record of a Louisiana family with multiple cases of leprosy (see text). A: twenty-two cases of leprosy in blood relatives of four cases in a family. B: family lines which originated in Nova Scotia and appear in New Brunswick leprosy. C: families are French; ancestry not traced, but cases of leprosy are known in relatives. D: ancestry not known. E: family line is French, but it is not established whether they came from Nova Scotia or directly from France.

Epidemiology has been built, to a large extent, upon the demonstration of the occurrence of disease in those associated in one way or another with the sick. This method of study is more easily applied to acute diseases in which the whole epidemiologic process may take place under the eye of a single observer. Furthermore, epidemics press for study to a greater extent than do endemic diseases, though the latter may exceed in numbers. This attitude in epidemiology probably has been an influence in the retention of the contagionist view of leprosy, with attention focused on the more immediate factors in the causation of the cases of the moment. But where the infectious and incubation periods are long, or the accretion of susceptible individuals is gradual, the epidemiologic process may be long drawn out or widely dispersed and hence the determining factors far more difficult to discern. The development of such diseases, both in the individual and in the community, is slow and likely to extend beyond the range covered by customary epidemiologic

procedures. An understanding of their behavior may require combined studies in widely separated places, over long periods of time.

Leprosy is such a disease. Hirsch (7), a proponent of hereditary predisposition, said in 1885 that the:

> ....best ground on which to try this question is obviously afforded by the small, closely circumscribed, and therefore easily surveyed leprosy spots, with a fixed population subject to no changes, where the state of health in the several families may be learned with the least possible trouble and followed through a long series of generations. Areas of observation of that kind existed at the beginning of the century at various points on the coast of Provence, France .... in several of the coastal districts of Sweden, .... and we will meet with them in southern Russia and the Caucasus, .... and in New Brunswick .... For all of these places do we in fact find, in the authorities quoted, classical proofs that the disease clings to particular families as a consequence of continuous inheritance from generation to generation.

There has been immigration to this continent from such regions of prevalence, with importation of cases. Some of these immigrant groups have tended to remain intact where they colonized, and foci of leprosy have developed which still persist. The disease has now been recorded for a sufficiently long period of time in several of the affected areas to give a comprehensive picture of the long drawn out epidemiologic process.

Where leprosy has taken root on this continent, it has been propagated largely in certain racial stocks immigrant from regions of prevalence, or in whom the disease occurs elsewhere, failing to spread to people of other nationalities living in the same districts. The affected areas, furthermore, are delimited by the boundaries of the localities occupied by the particular racial groups involved. In two large foci, widely separated, the individuals are of the same racial origin.

The studies in these two foci, which are recorded in this paper, indicate that leprosy tends to recurrence in successive generations in certain family lines in which intermarriage is common. Moreover, though the two localities are widely separated, the same family lines have been involved in both places. In another smaller focus the records of cases show a high frequency of relationship to previous cases in the same and in distant localities. In still other groups, where family studies have not been carried out, the restriction of the disease to certain minority groups, originally from areas of prevalence, are indicative of the operation of the same familial factor in the occurrence of the disease.

## CONCLUSION

Collected studies covering several generations of family lines

in which leprosy continues to occur in several localized areas indicate that hereditary susceptibility is a major factor in the propagation of leprosy on the North American continent.

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