THE ROLES OF FAMILIAL SUSCEPTIBILITY AND CONTAGION IN THE EPIDEMIOLOGY OF LEPROSY[1]

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As Frost has said, "Epidemiology is something more than the total of its established facts. It includes their orderly arrangement into chains of inference which extend more or less beyond the bounds of direct observation. But it is not easy when divergent theories are presented to distinguish immediately between those which are sound and those which are merely plausible." This has been especially true when epidemiologic inferences have led into fields not yet well bounded or cultivated. Therefore, it is constructive to turn back to the data from which earlier concepts have been evolved in order to retest them in the light of present knowledge. Thus, erroneously drawn propositions may be amended, or the validities in conflicting hypotheses which have been formed from the same observations may be proved adjustable into a different concept.

There is in the epidemiology of leprosy an illustration in the two divergent theories which have been advanced to explain the transmission of the disease, namely, contagion and heredity. These concepts were derived at different times, not so much from conflicting observations as from resisting inferences evolved from essentially similar observations. They were formed long in advance of developments in the specific fields of science involved, and, with acquisition of knowledge, discrepancies in each and validities in both become apparent.

The approval of either concept necessitates the acceptance of assumptions of doubtful worth in the theory favored and the rejection of valid evidence in the opposition. There is no question

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but that the disease is spread through contagion, but the striking tendency to familial occurrence would make it appear that the result of exposure is determined in large measure by an hereditary influence. Webster has demonstrated that inborn susceptibility is a major factor in the outcome of exposure to certain experimental infections. Studies in poliomyelitis suggest that familial susceptibility plays an important role in the limited and selective occurrence of the paralytic disease (1). Still another example might be mentioned where inherited susceptibility has been thought to play a distinct role in the manifestations of a disease. It is well known that a comparatively small percentage of cases of syphilis develop general paresis. One explanation for this has been the theory that only certain individuals are susceptible to involvement of the central nervous system with this infection, and that this susceptibility is inherited. This idea has been considered entirely plausible by many students of the disease. The present study suggests a similar concept of the epidemiology of leprosy—a concept which would harmonize the acceptable features and adjust the discrepancies in the two opposed earlier theories, namely, that the disease itself is hereditary and that it results solely from prolonged or intimate contact.

The evolution which has taken place in the epidemiology of leprosy extends back to biblical times. Whatever actual evidence may have underlain the ancient theory of contagiousness has been lost in antiquity. It would be difficult to determine to what extent the drastic precautionary measures were based on proof of contagion and how much purely upon fear of the "loathesome" disease. In any case, this concept must be classed as an epidemiologic inference, since it long antedated knowledge of infectious agents. But how close the older notion approached our modern concept of contagion is remarkable and is well illustrated by the meticulous measures outlined for the prevention of the disease, measures which might have been set forth by a trained sanitarian of the present day.

In the Middle Ages, according to the ritual of Paris, the following rules were given lepers by the priest (6):

They were forbidden to enter the church, or the market place, or the mill, or the public fair, or in any company or assembly of people whatsoever (person-to-person contact).

They were forbidden to wash their hands and all necessary things in fountain or in brook or in any water whatsoever, and if they wished to drink were ordered to take water with their own jug or some other vessel (water-borne infection and the drinking cup).

They were not allowed to touch anything that they wished to buy in any place whatsoever, but had to point to it with rod or staff.

While going through the fields, they were not allowed to reply to anyone who might question them except first, for fear they might infect someone, they step off the road to leeward, and also they were not allowed to travel by highway at all for fear of meeting someone (air-borne infection).

If necessity required that they take a path through the field, they were forbidden to touch the hedges, or bush, on either side, except before this they put on their gloves (contamination of objects).

They were forbidden to touch little children or any young people whatsoever and to eat and drink with companions save they were lepers (isolation).

In the middle of the nineteenth century (1848), the age-old belief in contagion was brought into question when the Norwegian scientists Danielssen and Boeck advanced the idea that leprosy was almost exclusively hereditary in origin. This theory prevailed until 1873, when the discovery of the Hansen bacillus afforded such plausibility to the theory of contagion that after a few controversial years the concept of hereditary transmission seems to have been overwhelmed. To quote Rogers and Muir (10):

This threw a flood of light on the etiology of leprosy and revolutionized our entire conception of its epidemiology by displacing the then dominant and paralyzing hereditary theory of its origin by the now generally accepted and more hopeful infective one.

Aside from the bacteriologic evidence, there is an abundance of epidemiologic proof that leprosy is an infectious disease. However, the doctrine of contagion alone is inadequate, since relatively few of those who are exposed develop the disease. The gaps have been filled in by supposed variations in degree or duration of exposure and by "predisposing causes." The extent to which the latter have been invoked is indicated in the following statement by Rogers and Muir:

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. We consider that it is firmly established that in an endemic area less than half of those who are inoculated with the germs of leprosy develop the disease. Man, like the lower animals, when in normal health and living under favorable circumstances is able to resist the onset of leprosy. Anything which lowers the resistance may act as a predisposing cause.... The natural resistance must be distinguished from natural immunity, although the latter forms a part of the former.

Elsewhere Muir states:

Much of the confusion which exists in regard to leprosy is due to want of recognition of the important role played by predisposing causes. There is probably no disease in which predisposing causes play a more important part than in leprosy. Many facts might be quoted to prove that, al-

though the bacilli may be present in the body, some predisposing cause or other is necessary before they can begin to increase in number and produce signs.

He then cites a large number of contributory conditions, such as puberty, pregnancy, malaria, syphilis,[2] chronic bowel disorders, hookworm, unsuitable diet, climate, humidity, lack of exercise, the climacteric, and even laziness, which were regarded as predisposing factors. It is curious that hereditary susceptibility was not included in this sweeping list. It can only be supposed that it did not find a place because the factor of heredity in leprosy was completely set aside with the discovery of the Hansen bacillus, at an epoch when the whole tide of medical thought was turned away from constitution and toward contagion.

Another obstacle to the acceptance of contagion *per se* is the relative infrequency of conjugal leprosy. Gwyther recorded that of 178 wives living with leprous husbands from three to twenty-seven years, only 4 contracted the disease. McCoy and Goodhue give figures of 4.8 percent for females and 5.1 percent for males, and Kitasato found only 3.8 percent of conjugal infections. As a matter of fact, all the figures indicate that husbands and wives do not contract the disease from each other any more often than do unrelated persons (servants, etc.) living in the household. Hayd (4) reports an instance of a healthy husband who buried three leprous wives while he himself remained strong and well. The relative infrequency of conjugal leprosy as compared with familial occurrence usually has been explained on the basis of decreased susceptibility of persons of marriageable age. However, the age distribution of leprosy, even when set back for incubation period and lapse in diagnosis, does not indicate a decrease in susceptibility with age sufficient to account for the low incidence of conjugal leprosy (Table 1).

It is not surprising that the striking occurrence of leprosy in familial lines suggested the hereditary transmission of the disease itself, when it is remembered that the work of Danielssen and Boeck antedated that of Mendel by about twenty years and the recognition of Mendelian heredity by fifty years. Landré, as early as 1869, objected that the alleged hereditary influence showed itself more strongly in the collateral than in the direct line, in his opinion quite contrary to the laws of inheritance, evidently confusing heredity and congenital transmission. Similarly, Muir omitted cases

[2] "Leprosy is the fourth stage of syphilis, a stage that white men are exempt from in the majority of cases. . . by reason of hereditary immunity" (3).

in collateral relatives as affording no evidence of hereditary influence. Holmsen recorded 93 lepers, 12 (13 percent) of whom had parents or grandparents who suffered from the disease. As an argument against heredity, he points out that in no less than 11 of the 12, the parents or grandparents were attacked after the birth of the children.

TABLE 1.—*Probable age of onset (percentages infected at the ages given).*

District affected	Age of patients in years							
	0–5	0–10	0–15	0–20	0–25	0–30	0–35	Over 35
So. Russia (Munch)	6.10	19.40	37.20	54.70	65.50	73.50	80.00	20.00
Indian Commission	8.85	19.59	32.70	47.36	63.11	73.41	83.53	16.47
Vandyke Carter M	——	——	4.70	31.40	65.30	86.10	95.60	——
Vandyke Carter F	——	——	20.00	49.30	76.50	93.70	97.10	——
Molokai (McCoy)	6.10	21.50	40.70	54.70	65.00	72.50	80.90	19.10
Soudan (Tonkin)	25.45	39.08	59.08	73.17	80.89	84.98	89.43	10.57

J. C. White, in 1882, pointed out that such evidence as had been brought forward in support of the hereditary origin of leprosy was based on inquiries in restricted geographic regions where leprosy has prevailed for centuries among certain classes, and in small districts where affected families have intermarried for generations and where the continued appearance of leprosy in the descendents of such families "may be as good evidence of its communicability as of its hereditary origin," and he contended that the theory of heredity will not hold good in any instance without the absolute demonstration that inoculation has been possible. Rogers and Muir state:

> It may be observed that nearly all the recorded data in favor of the hereditary origin of leprosy belonged to a period when the leprosy bacillus was either unknown or not fully established. It may safely be said that all of the evidence is now in agreement with the statement of Munro in 1879 that no proof can be brought forward that leprosy has the true character of a hereditary disease in being transmitted whether the children live with the parents or not.

Manifestly, such criticisms as these are directed against hereditary transmission of the disease itself and as such are valid.

The data which Danielssen and Boeck presented to substantiate their belief in the hereditary transmission of leprosy reveals the tendency of the contagion to restriction in high degree to family lines (Table 2).

Mauritz reported infection in Hawaii of 9 out of 17 children three to fourteen years of age. Denny's extensive statistics in the Philippines showed 16 percent of infections among children one

to ten years, and as many as 44 percent in those who had lived with leper parents for from seven to ten years. Figures from the Culion leper colony up to February, 1922, showed infection of 308, or 14.2 percent of children born there and not separated from their leper parents, while 18.8 percent more showed suspicious signs of the disease, bringing the total of probable infections to no less than 33 percent. Sand and Lie reported that of 2,010 children of 587 couples, 7 percent showed infection when the father alone was a leper, 14 percent when only the mother was diseased, and 26 percent when both parents were lepers. In Japan, Kitasato found 7 percent of the children of lepers contracted the disease.

TABLE 2.—*Evidence of heredity in leprosy (Danielssen and Boeck).*

Source	Generation				Total cases	Percent	Paternal	Maternal
	1st	2nd	3rd	4th				
Direct descent.....	20	40	1	8	69	32.4	29	40
Collateral descent.....	38	58	7	13	116	54.5	52	64
Spontaneous origin.....	—	—	—	—	28	10.1	—	—

Hansen, in spite of being aligned on the side of contagiousness, recorded 51 out of 210 patients who had leper relatives in the direct line of ascent. Vandyke Carter reported, in the Kattiawar State in the west of India, relationships between lepers who showed some direct or collateral taint in 30 percent. Ehlers of Copenhagen recorded that in 1897 he found in Iceland that 56 of 119 patients had leper relatives: one or both parents in 22, brothers and sisters in 20, and grandparents in 14.

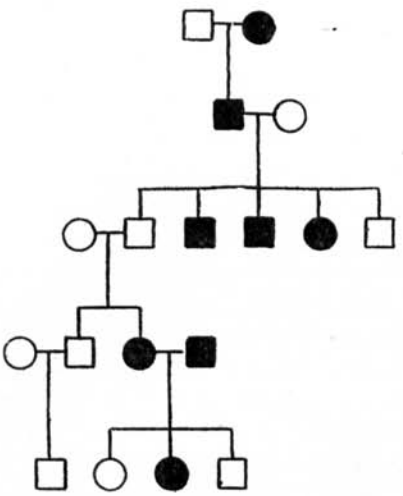
Brinckerhoff (2) reported 84 out of 460 cases which gave some family history of the disease: father or mother, 36 cases; brother or sister, 24 cases; cousin, uncle, aunt, etc., 18 cases; father and mother, 6 cases; son or daughter, 2 cases. He felt that these data only partially portrayed the familial occurrence of the disease,

> .... for it is exceedingly improbable that the patients, whatever their suspicions might be, would reveal the source of their infection. This reticence is broken only when a member or members of the family are already subjects of segregation.

One cause for this secretiveness is the widespread belief among the Hawaiians that leprosy is a disease transmitted by sexual intercourse.

McCoy (8) collected over a period of thirteen years statistics of 461 cases in which there had been known association with a leper; in 316 of these the contact had been with a member of the family.

Illustrative of many records in the literature that are indicative of a familial tendency is the report of Thin (12) of the lineal occurrence of leprosy in five generations (Text-fig. 1). No record is given of the disease in collateral lines.



TEXT-FIG. 1. Lineal occurrence of leprosy in five generations. (Thin).

To determine to what extent leprosy was propagating in families in Louisiana, Hopkins and Denney (7) selected for study the first 100 cases at the National Leprosarium in which complete family histories were obtained, and added all subsequent information concerning the appearance of leprosy in other members of these families during the fifteen years that had elapsed since the admission of the last of these patients. These original patients were members of 100 families consisting of 100 fathers, 100 mothers and 474 brothers and sisters, a total of 674 persons in the immediate families which, therefore averaged 6.7 persons per family. Of this group of original cases, 64 represented instances of only one leper in the family without further known propagation of the disease. In the families of the other 36 lepers, however, there developed 83 additional cases, and this group of 119 lepers presents interesting evidence of familial tendency. There were 5 instances in which the disease occurred in a father and one or more of his children, 15 instances in sons of lepers, 21 instances in daughters of lepers, 38 instances among brothers, and 31 instances among sisters. In addition, the following number of cases occurred in less closely related members of the family: 8 uncles, 8 aunts, 18 nephews, 9

nieces, 5 granddaughters. Furthermore, Hopkins and Denney stated that among all the patients admitted to the Louisiana Home (which at that time, 1929, received only exceptionally patients outside the state) an astonishingly large percentage were found to be closely related by blood. As many as 33 percent were parent and child, brother and sister, uncle or aunt, nephew or niece. In one instance reported the woman, who was a conjugal leper, was herself a member of a leprous line; her father, mother, four brothers, one sister and a nephew developed the disease.

It would thus appear that the definite tendency which leprosy shows to restricted occurrence in family lines and its failure to spread to persons who, though closely associated, are not related by blood ties can best be accounted for by familial susceptibility. This conclusion leads to the consideration of a new concept to explain the transmission of the disease: admitting the necessity of exposure and casting hereditary susceptibility as the predisponent necessary in the development of the disease.

FAMILIAL SUSCEPTIBILITY AND CONTAGION

A dual etiology, hereditary susceptibility plus contagion, is by no means a new epidemiologic notion. It was held for centuries in China, and was believed by Virchow, by Liveing and by Solano in Colombia. From the literature on the disease in the 1880's, one gathers that many students of the subject held this view. To quote one observer (11):

> The disease does not appear to me to be hereditary, that is, transmitted *de toutes piéces* from parents to offspring by procreation, or stored in the blood of individuals for generations, in its morbid nature and potential energy, without show of its presence. I doubt not, however, that the greater or lesser susceptibility to contract or acquire the distemper forms part of constitutional inheritance. Families may have received from parents and ancestors innate organic peculiarities, which render their members, or some, or many of them, not necessarily, but eventually, easier preys to the disease, when the exciting cause is brought, with effective force, to act upon them.

In a disease which is determined by both contagion and some selective predisposition, such as hereditary susceptibility, the distribution of cases would tend to resemble that of contagious diseases where the proportion of susceptibles in the population is high; and only where there is little suitable material would restriction to certain categories of individuals become apparent. Thus, the theory of contagion as the sole etiologic factor in leprosy doubtless has received its strongest support in areas where the proportion of susceptibles has been high. On the other hand, in regions in which this factor

has been restricted, as in Norway, the features presented might well have accounted for the hereditary theory.

A dual causation might adjust many of the factors in the existing theories which are at variance with our definitive knowledge of the respective means of spread. It may explain, for example, why leprosy possesses a high degree of communicability for those connected by blood ties, and is well-nigh noncommunicable for unrelated individuals living in close proximity to cases, such as nurses and attendants, husbands and wives. It might reconcile a discrepancy in the theory that the disease itself is hereditary, namely, that sometimes a parent may not develop the disease until after the birth of offspring who later become leprous. The proposed concept might also make clear the reason why the disease spreads extensively when first introduced into a given population, on the grounds that a large number of susceptibles have accumulated over an interval when the group was not exposed to infection, as well as why the disease remains restricted to small foci for long periods of time. In short, a twofold etiology would reconcile the paradox of the low contagiousness in general of a disease which under certain circumstances is highly communicable.

It would hardly be expected that members of certain family lines would be confined to very sharply localized areas, or that the infectious agent itself could by any conceivable chance be so restricted. Sharply localized foci of a disease might well exist, though each of the two factors is less limited since there is a diminished chance of the two spreading in the same direction. The outer boundary of a disease resulting from either contagion or heredity would be far more extensive than that of a disease resulting from the coincidence of the two in the same individual.

The results from human inoculations are in keeping with the concept of dual causation. Danielssen, Profeta, Cagnina and Bargilli failed to infect themselves, but in Arning's well-known case of the convict Keanu, the disease did develop after inoculation. Although this case is somewhat spoiled as an experiment by the fact that the man had lepers among his relatives with whom he was in contact before and after inoculation, there would seem to be little question from the fact that he experienced pain in the inoculated arm in one month and a nodule appeared four and one-half months later, that the disease in this instance might well have been due to the fact that the inoculated individual possessed an inherited susceptibility to the infection.

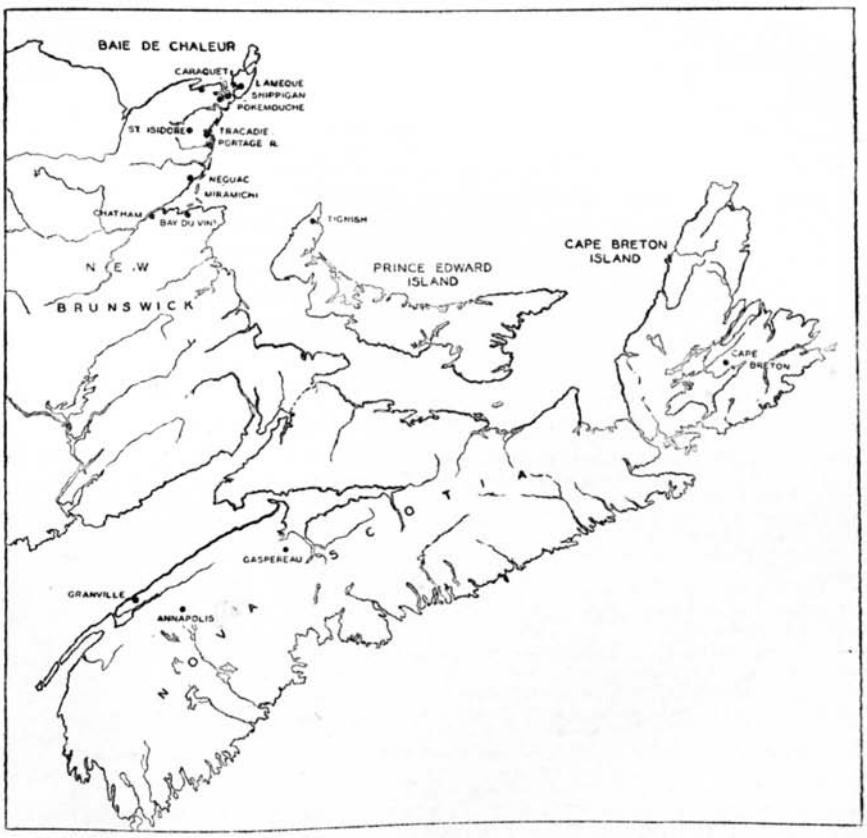
Belief in a dual etiology in leprosy has been expressed many times by students of the disease, but no specific studies seem to have been made to verify such a conception. Since the establishment of the infectious nature of the disease, much of the data available in regard to familial occurrence has been incidental to efforts to trace the sources of contagion, which in a high percentage of cases have been blood relatives. In order to test the concept of hereditary predisposition, an exhaustive study should be made of the disease in family lines. Specifically, it is proposed: (1) to evaluate the significance of the frequency with which the infection is communicated to relatives in comparison with others who are in similar close proximity to cases; (2) to ascertain whether conjugal infections, when they occur, are not largely restricted to individuals who are themselves members of leprous lines; (3) to investigate epidemiologically a third group, those cases who are members of leprous families, but who contracted their infection from sources other than members of their own families.

The obvious and permanent manifestations of leprosy, as well as the fact that a large proportion of cases have been recorded and put under institutional observation, remove the large error resulting from missed, forgotten or unknown cases which would be encountered in diseases which run a rather temporary course. The disease on this continent, because of its low incidence and restriction to sharply localized foci, presents particular advantages for epidemiologic studies of the sort proposed. Leprosy persists in New Brunswick, Canada, and in Louisiana among the Acadian French; in Galveston, Texas, predominantly in those of German blood; until recently in a small focus among the Norwegians in Minnesota; and in Key West, Florida. It is endemic in Corrientes in Argentina and in Paraguay, and is an important public health problem in Brazil and Colombia today.

Leprosy in New Brunswick.—The whole range of the coast of the Gulf of the St. Lawrence was partially settled by Norman immigrants, no doubt many of whom had fled from the Acadian expulsion of 1755, crossed the Nova Scotia isthmus and scattered along the shores, forming settlements at intervals as far north as the St. Lawrence. Tracadie, one of the older settlements, was composed almost entirely of French Acadians of Norman descent, and it was here that the disease was first detected.

There had been rumors of cases for several years, but no official notice was taken until 1844, when the leprosarium in Tra-

cadie was opened. Over thirty cases were discovered. The disease had been lingering in the settlement for many years and was considered to be confined to two families. It was deemed contagious, however, when three or four instances were reported in individuals not connected to these families by blood relationship (5).



TEXT-FIG. 2. Map of Nova Scotia and adjacent part of New Brunswick, showing the places of origin of 292 cases of leprosy arising between 1815 and 1933.

The records of leprosy in New Brunswick, unfortunately, go back only to 1815. At about this time the disease was occurring in some four or five families who had come to Tracadie together after the Acadian expulsion, and were probably already interrelated. From 1844 to the present time, 311 cases have been admitted to the leprosarium in Tracadie, practically all of whom

came from a few villages in the east of Northumberland and Gloucester counties, comprising an area bounded by Chaleur Bay, Miramichi Bay and the Bay of Fundy (Text-fig. 2).

An analysis of the records from the point of view of familial occurrence reveals interesting relationships. The 311 cases bear only 87 different surnames. These figures, however, may be appreciably altered when more closely examined from the point of view of the present study.

1. Eighteen immigrant cases admitted from other parts of Canada may be excluded, leaving 293 cases with 69 surnames.

2. Variations in spelling in branches of some of the older families account for 12 additional designations, bringing the number of family names to 57.

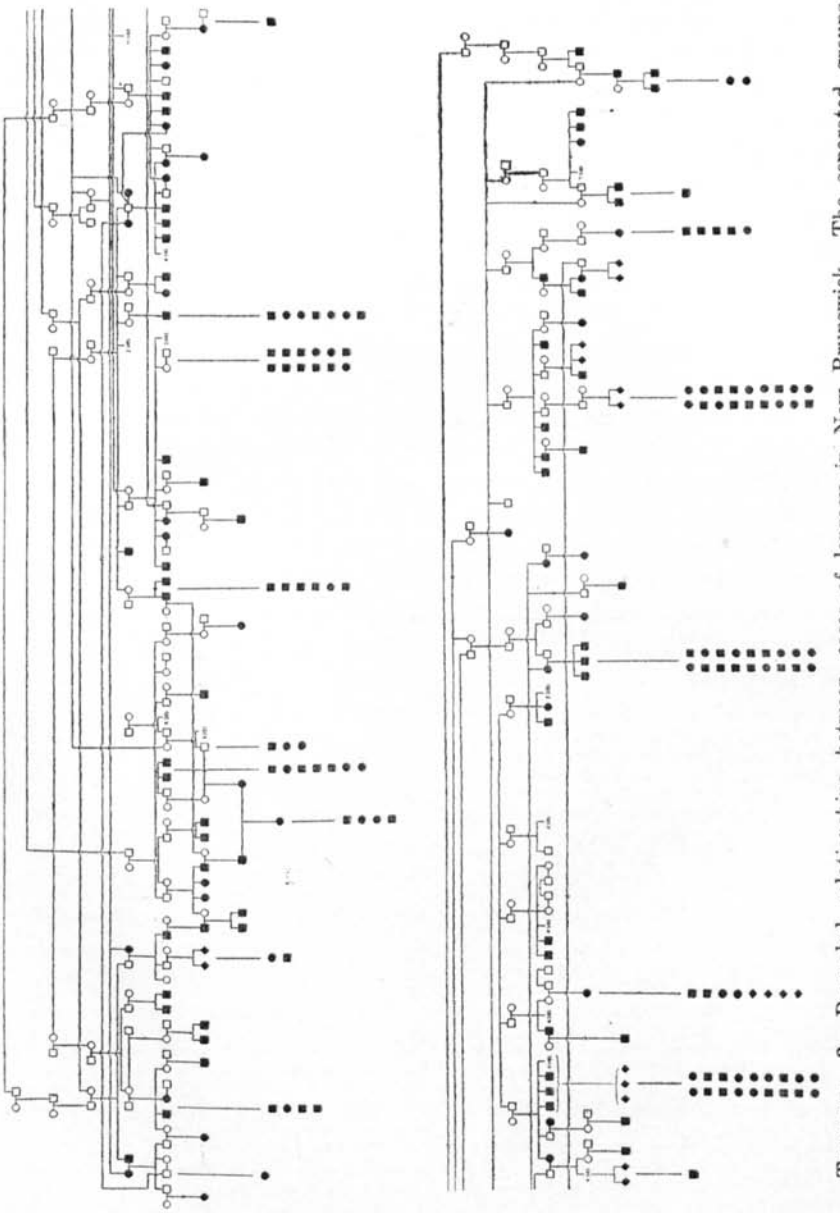
3. The list includes 58 cases whose 18 surnames are foreign to the community, but who are nevertheless members of the local families, leaving 39 family lines to account for 293 cases.

4. Lack of familial data makes it necessary to omit 25 families with 41 cases. Thus, there remain actually 14 families in which 252 cases occurred.

5. Furthermore, 128 of the 252 cases actually bear the same surnames as 8 of the cases admitted to the leprosarium in 1844. Studies show that intermarriage between these original families has been extensive up to the present time. An interesting note is that the four most recent patients admitted to the leprosarium actually bear the same surnames as cases admitted in 1844. (Text-fig. 3).

Leprosy in Louisiana.—Leprosy was not uncommon in the delta of the Mississippi 150 years ago. In the course of some years, however, the number appears to have diminished gradually and the disease disappeared almost entirely. Subsequent sporadic cases attracted little public health interest, and it was not until 1872 that the increasing importance of leprosy in certain parts of Louisiana was noticed. The cases were principally of French descent, many being offsprings of the Acadians who had been deported from the neighborhood of New Brunswick in 1755. Blanc, in 1888, reported 42 cases seen by him in New Orleans within a period of five years. The histories which he obtained indicated that many of these lepers were epidemiologically related to the stray cases which came with the Acadians from Nova Scotia.

From data available, a sort of first approximation is seen in the suggestive figures of the occurrence of leprosy in Germans



TEXT-FIG. 3. Recorded relationships between cases of leprosy in New Brunswick. The separated groups in the lower part of the chart are cases in the vicinity bearing the same surnames, but concerning which actual data are not available.

of New Orleans. Thus, of a total of 370 lepers reported at present in the United States, 34 are of German blood; and of these, 12 were born in New Orleans. Seven of the 12 cases were admitted from New Orleans, and one each from Lutcher, La., Detroit, Mich., Biloxi, Miss., St. Louis, Mo., and Norfolk, Va. A total of 27 cases are recorded as born in New Orleans. Thus, not only is the ratio of German lepers born in New Orleans to the total number of cases of German extraction in the United States far out of proportion to the population distribution, but there is also a lack of correlation between the number of New Orleans born lepers who are German and the population composition of that city.

Leprosy in Minnesota.—Leprosy was introduced into the northern part of the United States, especially Minnesota, by 160 Norwegians who migrated there either when suffering from leprosy or when in the incubation period of the disease. Hansen pointed out at the time of his visit to America in 1888 that not one of the descendents of these cases had developed the disease. Such a conclusion, however, was apparently premature, since leprosy has continued in Minnesota to the present day. Though at this writing no actual data are available concerning the familial occurrence of the disease, it was stated in 1912 that "no case of leprosy has arisen in Minnesota in an individual who did not have leper relatives."

The elucidation of the phenomenon of hereditary predisposition to leprosy, which would appear to constitute a necessary adjunct to the completion of the cycle of infection, might serve to bring into sharper focus the actual mode of transmission by redirecting the search, hitherto fixed on peculiarities of exposure in those infected, to a study of undifferentiated and perchance simpler circumstances of exposure shared by both susceptible and insusceptible persons. The accomplishment of this objective might lead to more effective and immediate measures for the control of contagion. Coincidentally, an understanding of the Mendelian behavior of susceptibility might create a consciousness of the biologic importance of heredity, and this knowledge might be expected in turn to exert, through the eugenic control of autarceologic susceptibility, a deterrent effect on the leprosy of the future.

SUMMARY

Two concepts of the epidemiology of leprosy were evolved in

earlier times from essentially the same observations. On the one hand, the frequency with which cases could be traced to exposure resulted in the theory of contagion, while the fact that contagion was largely restricted to relatives seemed to point to hereditary transmission. The latter theory is discrepant in that leprosy is not congenitally transmitted. Nor does contagion appear to be the sole etiologic factor, since the disease usually fails to spread to nonrelated persons equally exposed. Perhaps largely due to the discovery of the Hansen bacillus, the theory of contagion has taken precedence, although it has been necessary to qualify it by the doctrine of predisposing causes.

Webster's demonstration of inborn susceptibility to experimental infection, as well as epidemiologic findings in poliomyelitis which indicate that the paralytic disease is determined in large measure by individual susceptibility, often familial, suggest a similar concept of leprosy. Such an interpretation may harmonize the validities and adjust the discrepancies in the opposed theories.

A preliminary study of the available records of the occurrence of leprosy in a restricted focus on the American continent, where it has persisted for many years, indicates blood relationship between the majority of individuals affected. In addition, there is evidence of frequent intermarriage within the affected lines.

At present, epidemiologists concerned with the problem of leprosy are emphasizing as most important factors in the transmission of the disease the degree, duration and closeness of contact. The type of contact which young children experience with their elders appears to represent the degree and duration of intimacy which would satisfy this concept; that which occurs between adults is of another character and does not provide the factor of duration. One observes in various parts of the tropics, for example, the mother or father holding the naked child for hours in his or her arms. Many of these children develop their first lesions of leprosy on the buttocks or thighs and it has been suggested that this represents a skin-to-skin contact transmission from the older person to the child as a result of prolonged and repeated opportunity for such transmission. Be that as it may, there must still be many instances of this nature where degree, duration and closeness of contact have existed and no leprosy has resulted. This angle of the problem has received little attention, and might it not be that those children who acquire

leprosy in this manner are those who have inherited susceptibility to the disease?

While no conclusions can be drawn, further investigations are proposed with a view to elucidating the suggested concept of the epidemiology of leprosy, namely, that both exposure to the infection and a predisposition are essential to the development of the disease.

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