A NOTE ON FAMILIAL RELATIONSHIP AND THE RISK OF DEVELOPING LEPROSY

By

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In the Philippine Islands the risk of developing lepromatous leprosy following exposure to a lepromatous case in the household has been shown to be more than eight times that for those not known to have had household exposure (1). The probability of developing leprosy is also influenced by the age at which household exposure takes place; the earlier the age at exposure the greater the risk (2). Little is known, however, regarding the influence of familial relationship to the first patient on the risk to others in the household. Manalang (3) has stated that the chance of attack is greater for children if the primary case occurs in the mother rather than in the father.

For evidence concerning the relative importance of familial relationship recourse was had to data previously collected in Cordova and Talisay, Cebu, P. I. (4, 5). Unfortunately this series is not large; the records of only 283 families with a primary case of lepromatous leprosy were considered sufficiently complete for a study of this problem.

The modified life-table was selected as the method of analysis. The working rules adopted were essentially those used by Doull *et al* (1). The life experience of all individuals following household exposure to lepromatous leprosy was expressed in person-years, that is, each year of life following exposure was counted as a year at risk. The life experience was then divided according to the familial relationship of the first patient to each individual exposed.

After preliminary study of the material it was decided to include only those households in which the primary case was in a member of the immediate family. Households in which the primary case was in other relatives were excluded only because the cases of leprosy developing after exposure were too few to justify calculation of rates. Step-children, step-parents, half-brothers and half-sisters were considered the same as blood relatives.

The comparatively small size of the group available for analysis likewise did not permit study of the effect of exposure to more than one case in the family. Observation was therefore terminated for all exposed individuals

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TABLE 1. *Person-years of risk following exposure in the household to lepromatous leprosy and cases of lepromatous leprosy developing in the group classified according to age at exposure, sex, and relationship to primary case. Cordova and Talisay, Cebu, P. I.*

Age in Years	Exposed to Father				Exposed to Mother				Exposed to Brother				Exposed to Sister			
	Male		Female		Male		Female		Male		Female		Male		Female	
	Person-Years	Cases	Person-Years	Cases	Person-Years	Cases	Person-Years	Cases	Person-Years	Cases	Person-Years	Cases	Person-Years	Cases	Person-Years	Cases
0–4	418	0	329	0	92	0	49	0	208	0	157	0	79	0	115	0
5–9	435	3	359	0	109	2	56	0	337	5	325	2	175	0	139	1
10–14	390	6	288	4	127	0	48	2	453	6	516	5	213	2	197	1
15–19	327	2	215	4	109	0	47	0	475	3	490	1	238	4	233	0
20–29	309	2	276	0	265	0	48	0	669	3	651	1	310	3	310	1
30–39	155	0	201	0	64	0	26	1	331	0	263	0	146	0	174	0
40–49	63	0	101	0	23	0	0	0	113	0	122	0	83	0	87	0
50+	11	0	22	0	0	0	0	0	28	0	41	0	63	0	41	0
TOTAL	2108	13	1791	8	789	2	274	3	2614	17	2565	9	1307	9	1296	3

when a second case developed, if this case was lepromatous. If, however, the second case was neural in type the period of observation was not terminated since it has been shown that the risk of attack following household exposure to neural leprosy is no greater than that for the general population (1).

Inasmuch as the analysis is based on historical records only secondary cases of lepromatous type are considered. This follows from the assumption that neural cases are more likely to have been omitted in an historical study.

The risk of attack for respective groups, that is, for those exposed to a lepromatous mother, father, brother, or sister are expressed as incidence rates per 1,000 person-years, or the average number of cases per 1,000 persons observed for one year. It should be borne in mind that the rates are not applicable to the present but reflect what has occurred during the period covered by the lives of those included on the schedules.

In the 283 selected families there were 1,450 individuals, with a total life experience following exposure of 17,230 person-years. Included in this total are 1,840 person-years following exposure to a son, 1,012 person-years following exposure to a daughter, and 1,634 person-years following exposure to husband or wife. Among these persons only one case of leprosy developed, and that followed exposure to a son. The balance of the life experience together with the cases of lepromatous leprosy are shown in table 1, classified according to relationship to primary case, sex, and age. The data are not sufficiently large to justify calculation of age-specific incidence rates. Incidence rates for all ages combined, crude and adjusted for age differences, are given by sex and relationship to primary case in table 2.

TABLE 2. *Crude and age-adjusted* secondary attack rates for lepromatous leprosy following household exposure to lepromatous leprosy by sex, according to relationship to primary case.*

Rate per 1,000 person-years	Exposed to father			Exposed to mother			Exposed to brother			Exposed to sister		
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
Crude	6.17	4.47	5.39	2.53	10.95	4.70	6.50	3.51	5.02	6.88	2.32	4.61
Age-adjusted	4.81	3.71	4.31	1.95	8.10	3.57	4.88	2.46	3.61	5.16	1.86	3.60

*Adjusted for age, using the indirect method on the basis of corresponding age-specific rates for the total exposed population.

Considering both sexes together, the highest attack rate, 4.31 per 1,000, is found in the group exposed to a father. The rates following exposure to mother, brother, and sister are practically equal, being 3.57, 3.61, and 3.60 per 1,000 respectively.

The incidence rates for males are higher than for females for all familial relationships except where the primary case is a mother. Here the female rate of 8.10 per 1,000 person-years is considerably higher than the male rate of 1.95. It should be noted, however, that the female rate is based on 3 cases

of leprosy and the male rate on only 2 cases. Consequently the difference can not be considered significant.

The data are obviously too limited to warrant final conclusions regarding the effect of familial relationship. Intimacy of contact is certainly not identical for all members of a family. If degree of intimacy be an important factor in the risk of developing leprosy, incidence following exposure should not be the same for all types of familial relationship. Even in the same relationship, variations in intimacy may occur. A closer bond may exist between mother and child in one family than in another or at one period in the child's life than at another. Such variations are important but very detailed studies are necessary if they are to be considered. This note is presented in the hope that other workers with more abundant opportunity for familial investigations may be stimulated to make further contributions to this aspect of the epidemiology of leprosy.

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