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Some epidemiological data on leprosy collected in a mass survey in Burma*

L. M. BECHELLI, P. GALLEGO GARBAJOSA, MG MG GYI, K. UEMURA, T. SUNDARESAN, C. TAMONDONG, V. MARTÍNEZ DOMÍNGUEZ, & J. WALTER

In the WHO Leprosy BCG Trial in Burma a mass survey was undertaken to determine whether children had been exposed to patients with leprosy and, if so, the form of the index case. This paper presents the most important epidemiological data collected in this survey. The prevalence rate was 31.6 per 1 000. It seems that even if the prevalence rate is very high the L rate does not increase accordingly. The high T rates in areas of high endemicity seem to be related mainly to the degree of spreading of leprosy, even to persons who react to lepromin. Comparison of the results with data available for the area before the survey was made shows that 87% of the L cases had already been detected and that 54% of the T cases had not. There was a tendency for high L rates to be associated with high prevalence rates. The results do not suggest that any particular age group has greater susceptibility or resistance; the prevalence rates seemed to be related mainly to the age when exposure occurred. A higher prevalence of leprosy in males started to appear in the 10–14-year age group, and after the age of 15 the difference became impressive. Biological, socio-economic, and environmental factors seem to be responsible for the level of endemicity, which does not seem to be essentially or primarily related to ethnic origin.

The study population and the methods used in the WHO Leprosy BCG Trial have been described by Bechelli et al. (1970, 1973a). The total population registered was 69 242 inhabitants, 87.5% of whom were examined during the initial survey; during subsequent visits over a 4-year period, however, a total coverage of 93.9% was achieved. Within each age group, coverage varied from 88.8% to 98.8%, and the number of females exceeded that of males (Table 1).

For the age groups <1, 1–4, and 5–9 years the coverage was about 95% in the initial survey and 98–99% at the end of the fourth follow-up. For the same periods it was 90% and 97%, respectively, for the 10–14-year age group and 79–85% and 89–93%, respectively, for the ⩾15-year age group. Attendance for examination was better among children than among adults.

For males the coverage was 84.6% (28 191 of 33 335 registered) in the initial survey and 91.9% at the end of 4 years. The comparable figures for females were 90.2% (32 401 of 35 907 registered) and 95.7%.

Table 1. Distribution by age and sex of the population examined in the initial survey

Age group (years)	Males	Females	Total
< 1	837	844	1 681
1–4	4 118	4 248	8 366
5–9	5 054	5 112	10 166
10–14	3 869	3 960	7 829
15–19	2 276	2 966	5 242
20–29	3 282	4 513	7 795
30–39	3 380	4 104	7 484
40–49	2 425	3 003	5 428
50–59	1 650	1 936	3 586
60–69	963	1 226	2 189
⩾70	337	489	826
Total	28 191	32 401	60 592

* From the World Health Organization, Geneva, Switzerland; the WHO Leprosy BCG Trial, Burma; and the Burmese National Leprosy Team, Mandalay. Requests for reprints should be addressed to Chief Medical Officer, Leprosy, World Health Organization, 1211 Geneva 27, Switzerland.

Table 2. Correlation between L rate and prevalence in groups of villages

L rate	Population	L cases		T cases		I cases		B cases		Total	
		No.	Prev.[a]	No.	Prev.[a]	No.	Prev.[a]	No.	Prev.[a]	No.	Prev.[a]
0	11 717	0	0.0	155	13.2	13	1.1	4	0.3	172	14.7
0.1–1.9	4 062	6	1.5	94	23.1	3	0.7	5	1.2	108	26.6
2–3.9	13 879	39	2.8	309	22.3	33	2.4	8	0.6	389	28.0
4–5.9	10 322	52	5.0	264	25.6	18	1.7	2	0.2	336	32.6
6–7.9	4 153	28	6.7	120	28.9	6	1.4	4	1.0	158	38.0
8–9.9	8 588	78	9.1	283	33.0	27	3.1	6	0.7	394	45.9
10–11.9	3 936	42	10.7	104	26.4	2	0.5	3	0.8	151	38.4
12–13.9	1 869	23	12.3	58	31.0	7	3.8	3	1.6	91	48.7
14–15.9	1 131	17	15.0	43	38.0	5	4.4	0	0.0	65	57.5
16–17.9	0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
18–19.9	325	6	18.5	3	9.2	1	3.1	0	0.0	10	30.8
20–21.9	0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
22–23.9	0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
24–25.9	39	1	25.6	0	0.0	0	0.0	0	0.0	1	25.6
26–27.9	439	12	27.3	19	43.3	4	9.1	0	0.0	35	79.7
28–29.9	69	2	29.0	3	43.5	0	0.0	0	0.0	5	72.5
⩾30	0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0

[a] Per 1 000.

The coverage for children under 10 years of age was similar in both sexes. For other age groups coverage among females was always slightly higher than among males. The same was true up to the fourth follow-up.

FINDINGS [1]

Rates for different forms of leprosy

Among the 60 517 persons examined 1 914 cases were detected (31.6 per 1 000); they were classified as follows (rates per 1 000 are given in parentheses): [2] I, 119 (2.0); L, 306 (5.1); T, 1 454 (24.0); B, 35 (0.6). Singu township and the part of Shwebo district included in the mass survey therefore constitute an area of high leprosy endemicity.

Correlation between L rates and endemicity

Since lepromatous leprosy is the most contagious form of the disease,[3] it is to be expected that the higher the L rate the higher the prevalence and incidence rates. Detailed analysis of the L and prevalence rates for each village showed that this relationship obtained in the trial area.

For purposes of comparison, L and prevalence rates should be studied in different groups of villages of the same area, where biological, ecological, and socioeconomic factors are similar. Table 2 shows a correlation between the two rates: an L rate of 0.1–1.9 per 1 000 was associated with a prevalence rate of 26.6 per 1 000, while L rates of 26–29.9 were associated with prevalence rates of 79.7–72.5; the village group with an L rate of zero had the lowest prevalence (14.7). The population of the latter group of villages could have been exposed to L cases living in neighbouring villages; some of them may

[1] Space did not permit the inclusion of a large number of tables with this paper. These have been deposited in the WHO Library and professionally interested persons may obtain single photocopies on request to Chief Librarian, World Health Organization, 1211 Geneva 27, Switzerland.

[2] The following designations for forms of leprosy are used throughout this report: B, borderline; I, indeterminate; I-T ? indeterminate with a possible trend towards tuberculoid; L, lepromatous; T, tuberculoid; Tr, tuberculoid in reaction.

[3] For practical purposes and from the epidemiological point of view B cases could be grouped with L cases; the number of B cases in the trial area was small.

have had L patients who died or migrated. In a few villages the treated L cases, besides living rather isolated from the population, showed great improvement or became inactive. If L rates are plotted against leprosy rates (all forms) there is a wide scatter, but there is evidence of a positive correlation between the two rates.

Borderline patients could also be responsible for the spreading of leprosy in the villages with an L rate of zero; however, B cases were found in only two of these villages (with prevalence rates of 20.9 and 28.7 per 1 000) and each village had only one B case.

Tuberculoid patients, while in reaction and bacterially positive, might also be infectious to some extent and therefore contribute to the persistence or spread of the disease. In one village without L cases, 4 children had early leprosy. A bacterially positive (3+) Tr case was found and could be considered to be the source of infection.

L rates in the villages

As previously noted, the L rate was 5.1 per 1 000 in the initial survey. This could mean that, in a population with a prevalence rate over 30, only 5 per 1 000 are liable to develop L leprosy if all have been exposed to contagious cases. However, this does not seem to have occurred; although leprosy is highly endemic in the trial area, 11 707 inhabitants live in villages where there is not a single L case today (two villages each had a single B case).

The highest L rates were observed in villages with less than 450 inhabitants, as shown in the following tabulation.

Population	*L rate*	*Prevalence rate*
42	25.6	47.6
105	19.2	38.1
437	27	82
67	28.9	59.7

These figures indicate that, at least in such villages, about 3% of the inhabitants are prone to develop L leprosy.

Leprosy and age

The age-specific rates for the lepromatous, tuberculoid, indeterminate, and borderline forms of the disease are illustrated in Fig. 1.

Comparative prevalence among age groups. The prevalence rate was 1.6 per 1 000 in the 1–4-year age group; it then rapidly increased, being 13.6 in the 5–9-year age group, varying from 39.2 to 46.4 in the 10–29-year age groups, and reaching a peak of 48.11 in the 30–39-year age group. In the 49–69-year age groups it varied from 37.1 to 43.2, and at the age of 70 years and over it decreased to 24.1, possibly because persons who may have had incubating infections did not live long enough to present signs of the disease.

Age at detection and L rates. No L cases were diagnosed in children less than 10 years old, owing not to a higher degree of resistance in such children but to the long incubation period plus the time required for leprosy to develop into the L form. The L rate was 0.5 per 1 000 in the 10–14-year age group, varied from 6 to 10 in the 15–19-year age group, reached a peak of 12.5 in the 30–39-year age group, decreased to 6–10 in the 40–69-year age groups, and then decreased markedly at ages >70. The decreases after the age of 39 years could perhaps be explained by the relatively limited life span of these patients in the area.

Age at detection and I rates. The mean I rate was 2.0 per 1 000. In contrast to the absence of cases of the L form of leprosy, the I form was seen in children aged 1–4 and 5–9 years (the rates were 0.2 and 2.3 per 1 000, respectively). The I rate reached a peak of 5.7 in the 10–14-year age group and then decreased to less than 1 per 1 000 after the age of 40 years. Not a single I case was observed in persons aged 50–59 and >70 years.

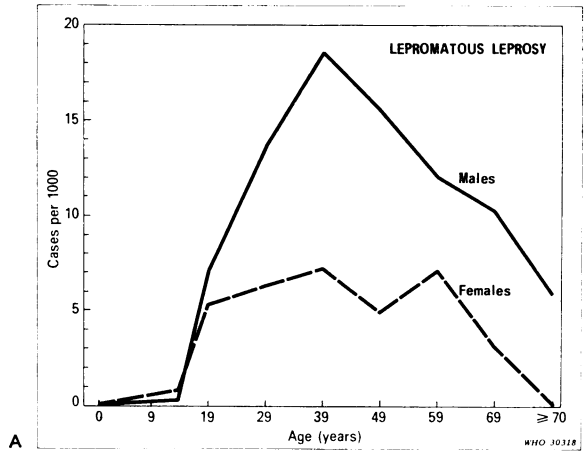
Age at detection and T rates. The largest proportion of cases registered was accounted for by the T form of leprosy, the mean rate being 24.0 per 1 000. The T rate was 1.3 per 1 000 in children aged 1–4 years and 11.1 in those aged 5–9 years; it then varied from 28.8 to a peak of 35.2 in the 15–19-year age group and dropped to 19.3 per 1 000 in persons aged >70 years.

Age at detection and B rates. The B form of leprosy appeared in children 5–9 years old, increased erratically in the subsequent age groups, and reached a maximum rate of 2.4 per 1 000 in persons aged >70 years.

Leprosy and sex

Prevalence in males and females. The mean prevalence rates were 40.4 per 1 000 for males and 24.0 per 1 000 for females, and the difference is statistically significant ($P>0.001$).

When prevalence is related to age, it is seen that the rates are similar in persons under 10 years of



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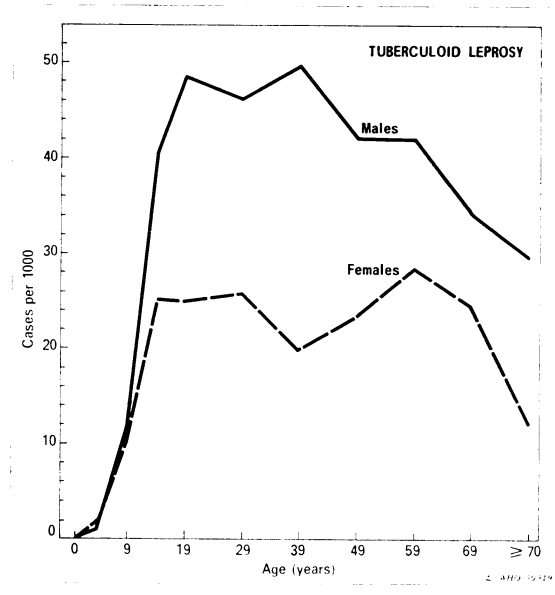

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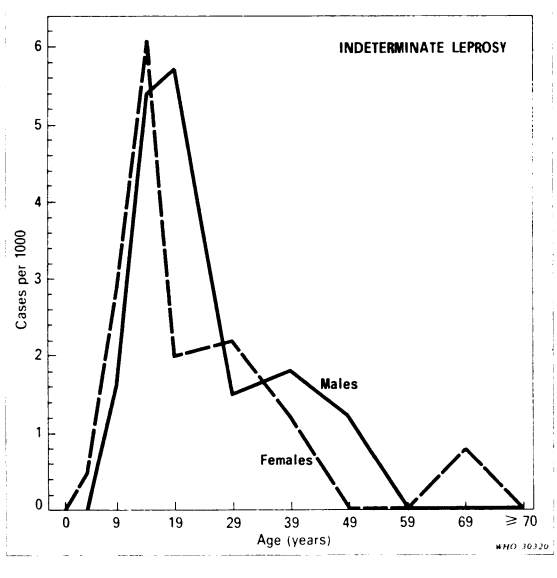

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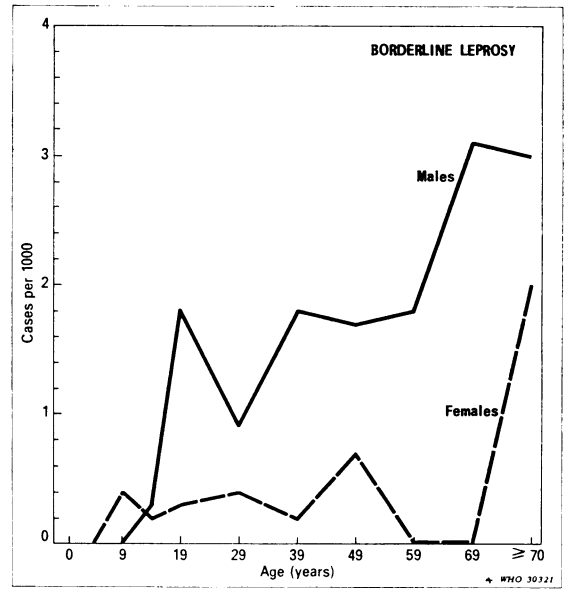

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Fig. 1. Age–specific rates for four forms of leprosy in males and females.

age and that the higher prevalence in males starts to appear in the 10–14-year age group; with increasing age the difference is impressive, the rate for males usually being twice that for females.

T rates, sex, and age at detection. The mean T rates for males and females were 30.6 and 18.3 per 1 000, respectively, the difference being parallel to that in the prevalence rates for the two sexes. Up to the age of 9 years the T rates were similar; the difference was observed in subsequent age groups.

I rates, sex, and age at detection. The I rates were similar for males and females (2.0 and 1.9 per 1 000, respectively) for all age groups with the exception of persons aged 15–19 years.

B rates, sex, and age at detection. The mean B rates for males and females were 0.9 and 0.3 per 1 000, respectively, and this difference was also observed in most of the age groups.

L rates, sex, and age at detection. The mean L rates for males and females were 6.9 and 3.4 per 1 000, respectively. The rates were similar in persons aged under 20 years; from that age onwards, however, the average L rate for males was twice that for females.

Incidence in children 0–14 years old at the initial survey

Data on the incidence in children have been presented by Bechelli et al. (1973a). The incidence rate for the total person-years of observation in a 6-year period was 6 per 1 000. The annual incidence, which was approximately the same, was fairly stable, indicating that there was no appreciable change in the epidemiological situation. This incidence rate appears to be too high for the total prevalence (31.6 per 1 000), when it is considered that prevalence is incidence times the duration of the disease. However, in the follow-up study of cases in children detected in the BCG trial it was observed that skin lesions regressed relatively rapidly, even without treatment or with irregular intake of dapsone (Bechelli et al., 1973a). In fact, the majority of these cases presented only single skin lesions of the T type.

Rates after 6 years of follow-up in relation to sex and age at initial survey

The data of Bechelli et al. (1973a, Table 4) show that in children aged 0–14 years at the time of the initial survey the incidence in total person-years of observation was slightly higher in males (6.7 per 1 000) than in females (5.4 per 1 000).[1] When considered separately for each age group, the rates are found to be similar in males and females of the 0–4-year group, slightly higher in males of the 5–9-year group, and even higher in males of the 10–14-year group.

Rates after 6 years of follow-up in relation to sex, age, and tuberculin reaction at initial survey

From the data of Bechelli et al. (1973a, Table 5) it appears that the degree of tuberculin reactivity did not influence the attack rates, either in males and females of different age groups or in the entire sample, and that tuberculosis infection did not reduce the incidence rates in males and females followed up for a 6-year period.

From a study of the findings in relation to tuberculin reactions of 5–9 mm, which could result from an infection with nonpathogenic mycobacteria, it appears that the latter did not affect leprosy attack rates.[2]

Leprosy rates in children exposed and not exposed at home

In 5 657 child household contacts and 48 368 other children followed up for 6 years the number of cases and the rate per 1 000 person-years were 78 and 13.8, respectively, for the former and 247 and 5.1 for the latter.

As expected the proportion of cases was much higher in children exposed to L and B cases (11.5% and 10.1%, respectively) than in those exposed to T (3.8%), Tr (2.7%), and I (2.9%) cases. The proportions of cases among contacts of T, Tr, and I cases were similar and rather high. It is likely that the majority of the child contacts of T, I, and Tr cases acquired leprosy because they were also exposed to the L or B patient who infected their relatives.

Forms of leprosy detected after initial survey

The incidence of different forms of leprosy in children aged 0–14 years at the time of the initial survey is shown in Table 3.

Of 325 cases detected in children subsequent to the initial survey (in a 6-year period of annual

[1] Males and females had been allocated to the groups according to their age, whether or not they were exposed to leprosy in the home, and tuberculin reaction.

[2] Reactivity to PPD-B, a purified protein derivative antigen prepared from the "Battey" atypical mycobacterium (*M. intracellulare*), was found in a greater proportion of subjects than was reactivity to PPD-S, the international standard for mammalian-type purified protein derivative tuberculin.

Table 3. Classification of leprosy cases detected after initial survey in relation to tuberculin reaction at first examination

Form of leprosy	Tuberculin reaction [a]		Total
	0–9 mm	⩾10 mm	
I	68 (24.5)	15 (31.2)	83
I-T?	31 (11.2)	7 (14.6)	38
T	171 (61.7)	26 (54.2)	197
Tr	7 (2.5)	0	7
L	0	0	0
B	0	0	0
total	277	48	325

[a] Percentages are given in parentheses.

follow-ups for the first group of villages included in the investigation and a shorter period for the others), only I (37.2%) and T (62.8%) cases were diagnosed. Not a single L case was found among the early cases or in subsequent years, confirming the view that the abrupt appearance of the L form is exceptional.

The late lepromin reaction was negative or doubtful in 6.8% of the I cases and in 2.6% of the I-T? cases and was weak (1+) in 38% and 18.4%, respectively (Bechelli et al., 1973b).

Table 3 also shows that the T form was seen in the majority of the cases.

Site and evolution of early lesions in children

The site of single early leprosy skin lesions has been studied by Bechelli et al. (1973c), who found that in 469 cases it was most frequently the thighs (19.4% of the cases) and buttocks (18.3%), followed by the arms (13.2%), the forearms (11.5%), and the legs (8.3%).

The evolution of early leprosy lesions in children has been discussed by Bechelli et al. (1973a). At the first yearly follow-up 42.3% of the cases had become inactive, with local atrophy in half of them. At the fourth yearly follow-up the proportion of inactive cases was 83.8%. None had evolved to the L form.

Bacterial positivity in Tr cases

Bacteria were found in patients with Tr leprosy with multiple lesions, and sometimes globi were found. Bacteria were also observed in nasal smears and more often in the ear lobes, even in the absence of noticeable skin lesions. In one case that was initially Tr bacilli (1+) were found in the ear lobes after a 2-year period of inactivity; at that time the patient showed no leprosy lesions, but many lesions appeared a few months later.

Form of leprosy in relation to tuberculin reaction at initial survey

The reaction to a dose of 2 tuberculin units of PPD RT23 diluted with Tween 80 was normally read after 2 days. Table 4 shows that the proportion of tuberculin reactions of >10 mm was similar in patients with L and T leprosy and slightly lower in those with the I form.

Table 4. Classification of leprosy cases in relation to tuberculin reactivity [a]

Form of leprosy	Tuberculin reaction [b]			Total
	0–4 mm	5–9 mm	⩾10 mm	
I	25 (28.1)	30 (33.7)	34 (38.2)	89
L	79 (35.3)	26 (11.6)	119 (53.1)	224
T	285 (26.3)	276 (25.4)	524 (48.3)	1 085
B	10 —	4 —	9 —	23
total	399	336	686	1 421

[a] Cases for which the results of turberculin reaction are unknown (38 I, 90 L, 426 T and 13 B) are not included.

[b] Percentages are given in parentheses.

When the results of tuberculin reactivity in T cases are considered in relation to age, 61% (379 of 618) of those aged 20 years or more showed reactions of ⩾10 mm. However, the proportion of reactions of ⩾10 mm was 37% (131 of 350) for T patients 10–19 years old and only 11% (13 of 114) for children 0–9 years old. Moreover, 54% (106 of 196) of L patients aged 20 years or more had tuberculin reaction of ⩾10 mm.

DISCUSSION

In a random sample survey carried out in Shwebo by the WHO Leprosy Advisory Team (unpublished data) in 1964, a prevalence rate of 32.16 and an L rate of 7.1 per 1 000 were found. The rate for Shwebo in 1964 was similar to that later obtained for Singu and part of Shwebo.

The L rate of 5.1 observed in the initial survey of the Burma trial is similar to that observed in

Khon Kaen, Thailand (4.6 per 1 000) in a WHO random sample survey. The prevalence rate for that area was 12.4, the T rate 4.8, and the I rate 3.0 (Bechelli et al., 1966). These and other data suggest that even if the prevalence rate is very high there is no corresponding increase in the L rate. In fact the high prevalence rates are due mainly to the increase of T rates. Even when prevalence rates are high, L rates usually do not exceed 10 (Doull et al., 1942; Newell, 1966) at the country or township level. In villages, however, L rates may exceed 10 and may even be as high as 30 per 1 000 (Bechelli & Martínez Domínguez, 1972), as was observed in the trial area.

L cases accounted for 15.9% and T cases for 75.9% of the 1 914 cases of leprosy detected. The proportion of L cases found in mass surveys or random sample surveys is always smaller than that found in control projects, because a greater number of early cases (T or I) are detected. Similarly, the more active the case finding, involving surveillance of contacts and also certain groups of the population, the larger the number of early cases detected and the lower the proportion of L cases.

The results of the mass survey showed that of the total existing cases the following proportions had not previously been detected: L cases, 13%; I cases, 43%; T cases, 54%; and B cases, 48%. Thus, a high proportion (87%) of L cases had already been detected and a high proportion (54%) of T cases had not; this is a common observation in many areas of the world. From the practical and epidemiological point of view, control projects that detect almost all L cases and I cases liable to evolve to the L form and that keep them under regular treatment are reaching the heart of the problem. In fact, a high proportion of undetected early T cases are expected to show spontaneous regression of their lesions, as observed by Lara & Nolasco (1956) and Dharmendra (unpublished data). Table 5 shows the proportions of L and T cases in the trial area and in other countries where WHO random sample surveys have been made.

Although the leprologists involved in these surveys used slightly different criteria for classifying cases, these were related mainly to certain transitional cases, and the comparison is valid.

The L rates were significantly higher in Thailand (Khon Kaen) and Burma (trial area) than in Cameroon and northern Nigeria. The degre of endemicity was lower in Thailand than in the other three countries.

The fact that T rates are higher in areas of high endemicity than in those of lower endemicity seems to be related mainly to the degree of spreading of leprosy, which also affects lepromin reactors, with consequent high prevalence rates; it should not be interpreted as being caused by higher resistance of the population.

Table 5. Prevalence, proportion, and rate of L and T cases in the trial area and in some other areas of the world (WHO random sample survey)

Country	No. of leprosy cases	Prevalence [a]	Lepromatous cases		Tuberculoid cases	
			Proportion [b]	Rate [a]	Proportion [b]	Rare [a]
Argentina						
Chaco	42	5.6	26.2	1.5	23.8	1.3
Entre Rios	11	1.1	—	0.3	—	0.5
Burma (trial area)	1 914	31.6	15.9	5.1	75.9	24.0
Cameroon [c]	374	25.8	11.2	2.9	55.6	14.4
Nigeria (Katsina) [c]	705	28.7	7.2	2.1	48.3	13.9
Philippines (Ilocos Sur)	69	6.7	39.2	1.6	39.5	2.7
Thailand (Khon Kaen) [c]	205	12.4	37.1	4.6	38.5	4.8

[a] Per 1 000.
[b] As a percentage of the number of leprosy cases.
[c] Data from Bechelli et al. (1966).

Leprosy and age

The rates in the 0–4-year age group (comparable with the total prevalence rate in many South American countries) and the 5–9-year age group (higher than that of any Central or South American country) give an idea of the severity of the endemic in the trial area.

The constancy of rates in persons aged 10–59 years does not mean that T leprosy appears at similar rates in all age groups. Taking into account the duration of the disease, it seems that there is an increasing number of old T cases with neurotrophic lesions or skin sequelae among persons over 30 years of age. The proportion of such old T cases increases with age and probably many of them had been inactive for many years; if detected earlier they would perhaps have received enough treatment to be released from control on the occasion of their registration.

A high proportion of children in the trial may show spontaneous healing of the lesions, therefore appearing to be healthy in a subsequent survey by another leprologist. However, neural and neurotrophic lesions persist until death.

The prevalence and rates of each form of leprosy confirm that the disease may appear at any age. The prevalence rate in children 0–4 years old is a result of the long incubation period. The prevalence in the different age groups suggests that in areas of high endemicity exposure may occur at any time of life, and that children are not more susceptible than adults. The difference in prevalence in most of the age groups is not substantial and does not suggest that any particular group has greater susceptibility or resistance. Moreover, it appears that all persons, regardless of their age, have similar potentiality to develop the L, T, I, and B forms of leprosy and thereby similar susceptibility or resistance to *M. leprae*.

Thus, it appears that the prevalence rates in the different age groups are mainly related to an earlier or later exposure, as concluded by Bechelli & Rotberg (1949), Doull (1961), and Bechelli et al. (1966).

Leprosy and sex

At first sight, the prevalence figures for the two sexes might suggest that males are more susceptible than females. However, other aspects, mainly the distribution of the different forms of leprosy, must also be taken into account. From the results of the random surveys in Cameroon, northern Nigeria, and Thailand, it could be said that the differences in rates could depend on the degree of exposure to the disease, living conditions, habits, etc., which vary in different areas, and not on a greater susceptibility of males or females to contract the disease.

When the difference between the mean T and L rates for males and females is analysed, several factors may be taken into account. First, lepromin reactivity results tend to indicate that males and females, children and adults, have similar resistance to *M. leprae*. If these data are confirmed by further studies, it could be deduced that the degree of exposure, living conditions, way of life of males and females, and other factors are responsible for the differences in prevalence and L and T rates. Second, the higher prevalence in males could be caused by greater exposure to leprosy after they are 10 years old, and probably before, if the incubation period is taken into account. Thus, if leprosy is more prevalent in males, a higher proportion of them would also be prone to develop the L and T forms. In fact, the T rates were similar in children aged less than 15 years, as were the L rates in those aged less than 20 years. After these ages, however, the males developed higher L and T rates. The differences would appear to be the function of prevalence rates for each age group.

Duration of the disease

The short duration of the disease in children aged 0–14 years at the time of the survey as compared with that in L and B cases (which account for only a small number of the total existing cases) induces one to think that the average duration of leprosy (for I, T, L, and B cases together) could be about 5 years in the trial area. Determination of the average duration of each form of leprosy is hindered by the difficulty of determining when a patient is cured, even for T cases (not to mention L or B cases). The same difficulties exist for tuberculosis, syphilis, deep mycoses, and many other diseases. Criteria for inactivity (or arrest) of the disease have been established, even if they do not meet general agreement on certain aspects, but none has yet been established for cure. For administrative purposes, therefore, criteria for releasing patients from control had to be proposed (WHO Expert Committee on Leprosy, 1960, 1970).

Infections with other mycobacteria

When considered in relation to tuberculin reactivity at the time of the initial survey, the leprosy attack rates for the different age groups and for the whole sample seem to indicate that neither tuberculosis infection nor an infection with nonpathogenic

mycobacteria affected the incidence rates in males and females followed up for a 6-year period.

Evolution of leprosy

If it is accepted that the disease starts in the I form (which later may evolve to the L or mainly the T form), and if the 1-year interval between follow-ups is taken into account, the evolution of I to T seems to take place in less than 1 year in a large number of cases. The data also indicate that the evolution from I to L takes several years. The early diagnosis of I cases and their regular treatment is the golden rule in the control of leprosy, although it is difficult to implement in field projects, no matter whether the area is one of high or low endemicity. The difficulties are related to (*a*) the near impossibility of detecting annually all I cases in the population at high risk, in which the exposure is unknown, (*b*) the fact that only a relatively small proportion of I cases progress to the L form (30% to perhaps 50% of the nonreactors to lepromin), and (*c*) the long duration of the treatment required.

The data in Table 3 also indicate that a probable tuberculosis infection (as indicated by a tuberculin reaction of $\geq$10 mm) did not seem to favour the appearance of T leprosy, which was slightly more frequent among those with reactions of 0–9 mm. More years of observation are needed to determine whether tuberculosis infection would prevent the development of L leprosy.

The findings with respect to the evolution of early lesions in children show that such lesions exhibit a relatively rapid regression. A high proportion of such cases would not be detected in ordinary leprosy control projects and, in fact, their public health importance seems to be limited unless they again show new lesions or some of them later present some type of disability. This has not occurred in the period of observation, but it cannot be excluded that inactive cases might show a reactivation of the disease in future years. Even T cases treated for a few years and then released from control may have a relapse.

Lepromin reactivity

The late lepromin reactions in children and in a random sample of the population (Bechelli et al., 1970, 1973a) indicate that most of the inhabitants of the trial area have the potential to become reactive to lepromin and have a certain degree of resistance against leprosy. Among them a certain number have acquired leprosy, but of the T type. The proportion of lepromin-reactive persons was low (and the reaction was only 1+) in those aged up to 1 year; after this age there was a rapid increase in the proportion of reactive persons and in the intensity of reaction, less than 5% of the sample showing a negative or doubtful reaction after the age of 15 years.

Bacterially positive cases

In several studies the proportion of Tr cases with bacterial positivity was found to be high, and even higher when histological examination was undertaken. The number of bacilli was also high in many patients. The finding of bacteria in ear lobes even in the absence of noticeable lesions is also worthy of attention. Such positivity in apparently inactive L cases was known, but the findings in active or apparently inactive Tr cases were rather unexpected.

Form of leprosy in relation to tuberculin reaction

The findings do not support the view that patients with the L form have a lower capacity of reacting to tuberculin. Tests of 1 421 patients showed that there was no substantial difference in the occurrence of tuberculin reactions of 0–4 mm, 5–9 mm, and $\geq$10 mm in the three forms of leprosy. The conflicting results obtained by other workers may be explained by several factors—e.g., the use of different tuberculin and of different criteria for reading the reaction and the fact that some workers studied patients living in towns or institutions while others studied farmers. Differences in the age of the patients should also be taken into account.

It appears that the high proportion of positive tuberculin reactions in T patients in the older age groups was merely the result of an increased reactivity with age and that T leprosy manifests itself in a very high proportion of children in the absence of a positive tuberculin reaction.

Leprosy and ethnic group

On the basis of the data gathered in this and other surveys, it appears that high prevalence rates may occur in any population, no matter what its ethnic origin. Biological, socioeconomic, and environmental factors seem to be responsible for the level of endemicity, which does not seem to be essentially or primarily related to ethnic origin. The potentiality to develop T leprosy is likely to be the same in the local ethnic groups studied.

RÉSUMÉ

QUELQUES DONNÉES ÉPIDÉMIOLOGIQUES RELATIVES À LA LÈPRE RECUEILLIES AU COURS D'UNE ENQUÊTE DE MASSE EN BIRMANIE

Une enquête épidémiologique a été menée dans le cadre de l'essai de prévention de la lèpre par le BCG organisé par l'OMS en Birmanie.

Dans la région de Singu et de Shwebo, l'endémicité de la lèpre est élevée; 1914 cas (31,6 pour 1000) ont été diagnostiqués parmi les 60 517 personnes examinées, se répartissant en: lèpre indéterminée (I): 119 (2,0 pour 1000); lépromateuse (L): 306 (5,1 pour 1000); tuberculoïde (T): 1454 (24,0 pour 1000); borderline (B): 35 (0,6 pour 1000). Dans certains petits villages, la prévalence de la lèpre L dépasse 10 par 1000 habitants et peut atteindre près de 30 par 1000 habitants. En comparant les chiffres obtenus lors de la présente enquête et ceux d'études antérieures, on a relevé les proportions suivantes de cas non dépistés par rapport au total des cas existants: L: 13%; I: 43%; T: 54%; B: 48%. Il existe une corrélation positive entre la prévalence de la lèpre L et la prévalence de la lèpre toutes formes.

La prévalence de la lèpre est maximale dans le groupe d'âge 30-39 ans (48,1 pour 1000); elle varie de 37,0 à 46,4 pour 1000 dans les autres groupes d'âge de 10 à 69 ans. Les formes L sont les plus fréquentes dans le groupe d'âge 30-39 ans (12,5 pour 1000); dans les autres groupes de 15 à 69 ans, leur prévalence est de 6 à 10 pour 1000. La prévalence de la lèpre I est maximale entre 10 et 15 ans (5,7 pour 1000); celle de la lèpre T entre 15 et 19 ans (35,2 pour 1000). L'examen des données relatives à la prévalence et aux formes de la lèpre selon l'âge ne fait apparaître aucune réceptivité ou résistance particulières dans un groupe donné. La fréquence plus élevée de la lèpre dans le sexe masculin, manifeste dès l'âge de 10 à 14 ans, se marque très nettement dans les groupes d'âge supérieur où l'on compte en général deux hommes atteints pour une femme.

L'infection par le bacille tuberculeux ou par des mycobactéries non pathogènes n'a pas diminué l'incidence de la lèpre chez des sujets des deux sexes âgés de 0 à 14 ans au début de l'enquête et suivis pendant 6 ans. Des épreuves tuberculiniques pratiquées sur 1421 malades n'ont pas montré de différences sensibles de la répartition des réactions de 0-4 mm, 5-9 mm et $\geq$10 mm selon la forme de lèpre. On n'a pas constaté de réactivité moindre à la tuberculine chez les malades L.

Chez les enfants âgés de 0 à 14 ans lors de l'enquête initiale, l'incidence de la lèpre a été d'environ 6 pour 1000 et elle est restée relativement stable durant les 6 années d'observation. Dans beaucoup de cas, l'évolution des formes I vers la forme T s'est faite en moins d'un an.

Ces données, et celles d'études antérieures, montrent que la prévalence de la lèpre peut être élevée dans n'importe quel type de population. Le degré d'endémicité est fonction de facteurs biologiques, socio-économiques et mésologiques et non de l'origine ethnique.

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