INTRODUCTION

The story of the remarkable epidemic of leprosy on the little island of Nauru in the Central Pacific, where the disease was unknown in the first decade of this century but became rampant in the early 1920's, has been told only sketchily in the periodical literature. Two comprehensive reports have been written, but one is an official document of limited distribution while the other is an unpublished manuscript. The purposes of the present article are to review all of the available material, to bring information on the situation up to date, and to discuss certain outstanding features of the epidemic in retrospect.

The more accessible reports are those of Bray (3), who was assistant government medical officer (under Townsend) from 1925 to 1927, approximately; of Grant (7), who was government medical officer from 1932 to 1934; and of Clouston (4, 5), who held that position from 1935 to 1940. Actually the earliest one was written by Morgan, who as a bacteriologist of the Commonwealth Serum Laboratories in Melbourne was sent to Nauru early in 1922 to confirm the diagnosis of the few cases then in isolation and to examine suspects. His findings were first published as a government document, now rare (9); they were also presented in abbreviated form at a Pan-Pacific
Science Congress (10). Two years later Dew was also sent there to check on other cases which had not been found bacteriologically positive by ordinary means. His findings (6) were included in the annual report of the administrator of Nauru for 1924.

That document (11), prepared for the Mandates Commission of the League of Nations and rich in background material, is not cited by any of the later writers referred to although it contains a special report (15) which deals with the leprosy situation from various points of view as has no other which has been published. Although it is not signed, it was written by Townsend, the first government medical officer, who served from 1923 to 1928. The other comprehensive study referred to, which brings the story up to 1930, is a thesis written by Allan (1), who succeeded Townsend for a year in 1928-1929.

**ORIGIN AND COURSE OF THE EPIDEMIC**

The introduction of leprosy among the Nauruans is ascribed by Morgan and others primarily to a Gilbertese woman (Etsio) who had come to the island in 1911 or 1912, while the place was German territory. The doctor then there recognized her condition and objected to her landing, but the governor allowed her to stay. She settled near the abode of a young girl named Demau, then aged 13 years, and died late in 1914. Another imported case may also be implicated, a laborer from the Caroline Islands who, according to Townsend, had had close association with Nauruan natives and who, shortly before Etsio's arrival, had been found to have leprosy and was repatriated. According to Allan, this man entered in 1911 and was sent away early in 1912, so his sojourn was apparently not prolonged.

Indentured Chinese laborers of the Phosphate Commission have often been mentioned in this connection. Morgan told of one who, after eight years at Nauru, left there late in 1920 and

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1 This lengthy manuscript was prepared for presentation to the University of Melbourne as a thesis for the degree of M.D. The original copy has now been deposited in the U. S. Army Medical Library, Washington, D.C., from which photographic copies can be obtained. Another copy is in the library of the University of Melbourne, and two are in the possession of Dr. Allan's widow. The official report of 1924 referred to is also available in the Washington library.

2 In a cage slung over the side of a vessel, which seems to have been the practice of the German authorities. Morgan was told of "an unfortunate Chinese leper [who] was isolated in a cage, in which he was repatriated to China."
was found to be leprous while at Sydney, on his way to China. That the Chinese could have introduced the disease is generally discounted, however, for their contacts with the natives were strictly limited. They were quartered in a large fenced compound, and both they and the Nauruans were required to stay within their respective districts of residence at night. At other times there was no restriction except that the laborers were prohibited entry to any native house or dwelling (13), and they constituted a market for surplus products—pigs, fowl, fish, coconuts and other fruits—of the natives; but the actual intercourse was “very limited” (11) or “practically nil” (13). The numbers of natives of other Pacific islands have always been small.

The first recognized case among the Nauruans was discovered in June 1920 in Demau, who was then 22 years of age, by Dr. L. R. Clapp, medical officer of the British Phosphate Commission (who also served the Nauru government on a part-time basis until some time in 1923). Within a few months Clapp found three other cases, and all were isolated. Before bacteriological confirmation was received from Australia, late in 1920, there occurred a devastating epidemic of influenza which affected practically all of the Nauruans and killed off about 30 per cent of them (1, 5), including all of the leprosy patients except Demau. That leprosy had actually appeared earlier was indicated by Grant, who was told by a reliable patient—the trained hospital orderly in charge of the segregation station—that one of the other patients there had had an anesthetic macule before the outbreak of the war in 1914, and that he himself had developed such a lesion in 1916 while under training in the Ellice Islands.

When Morgan made his study early in 1922 there were 10 patients in the isolation station, and many others were regarded as suspects (1). He diagnosed 25 new cases, one of them a Caroline Islander, and all of them were isolated. With the system of monthly examinations of all the Nauruan population which Clapp introduced, the number of cases diagnosed and isolated was increased to 129 by the end of the year (7).

*At the end of 1924 there were only 11, all employees of the Phosphate Commission, although 100 had left during that year (11). In 1940 there were 49, increased in 1950 to 81 (see Table 3). The Chinese laborers of the Commission are now being replaced gradually by Gilbert Islanders, and late in 1951 there were 300 of them (7).
This number represents a prevalence rate of 12.5 per cent, since the Nauruan population was 1,113 at that time.

By 1924 almost every family had at least one member with leprosy (13). It was then decided that the least advanced and the clinically doubtful cases should be treated as outpatients, and a clinic was opened late in June. At the end of that year there were 284 patients under treatment, 189 in segregation and 95 attending the clinic. Assuming that the latter group included the 21 who, for the first time, had been released from segregation on parole in that year, and taking the population as 1,200 (given as 1,219 and 1,185 in different places), the prevalence was then 23.7 per cent.

A total figure of interest appears in the data of Allan. There had been no less than 438 cases among the Nauruans from 1922 to the middle of 1929, and he said that these represented 36.5 per cent of an average population of 1,200. Since, however, that was the population in 1924, and it had increased to 1,365 in 1929, it would probably be more correct to say that around 35 per cent of the entire community had been diagnosed as showing manifestations of leprosy.

Bray's tabulation shows 50 cases listed as clinic outpatients in 1921, 108 in 1922, and 144 in 1923, whereas according to both Townsend and Allan the clinic was not established until the middle of 1924, after Dew's visit. Chief Edoh of Buada states (personal communication) that a de facto outpatient clinic was started in 1921, although its operation was not made official until later. Morgan, however, says (personal communication to Prof. P. MacCallum) that there was no regular clinic at the time of his visit, and he suggests that the figures for 1921-1923 may represent the total of cases and suspects listed in the surveys of Clapp.

Again we are unable to correlate these data of Townsend, which appeared in the official report for 1924, with those published later by Bray, who is deceased. By 1924, Bray stated, 30 per cent of the entire native population had shown clinical manifestations, and he gave the following figures for that year: 193 in isolation and 153 outpatients, total 346, which would give a prevalence of 28.8 per cent. No attempt is made to calculate rates from his figures for later years.

*Regarding the accuracy of this figure, Austin (1) gives the following quotations from three of the annual medical reports: "By no means all the patients receiving treatment are lepers though classed as such. This is due to the difficulty of absolute diagnosis in early maculanaesthetic cases" (1926). "No significance is attached to the number of persons on the roll of the O. P. Clinic" (1927). "It must be borne in mind that a number of persons receiving treatment in the Clinic probably have no leprosy at all" (1927). Austin concludes that, since suspects as well as inactive cases discharged from the leprosy station were evidently on the clinic list, "the correct total of active cases at any time" must be assumed to lie between the number in segregation and the recorded total.
Beginning apparently in 1926, patients who had cleared up under treatment were discharged and dropped from the statistics. In 1928 Allan discharged no less than 119 such cases, which explains the abrupt drop to 218 (132 in isolation and 86 at the clinic) from 337 in the previous year. This gives a prevalence rate for 1928 of only about 16 per cent.

When Grant brought certain features of the story up to the end of 1933, there were only 66 cases in isolation while 115 were under treatment as outpatients. The total, 221, represents 14.2 per cent of 1,550 people. At the time of Clouston’s second report there were 57 segregated patients and 102 attending the clinics, the total of 159 constituting 10 per cent of the population at that time, just over 1,600. He noted that the number of new bacteriologically positive cases had decreased greatly but the negative early ones to a lesser extent.

Since that time no report of the situation has been published.

PRESENT REPORT: STATISTICS SINCE 1936
PRE-WAR PERIOD, 1937-1941

The available data for this period vary in form, but they suffice to show the trend of the situation. Those for 1937 to 1940 are authentic as far as they go; the copies of the detailed reports for those years on file at Nauru were destroyed dur-

<table>
<thead>
<tr>
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Prophylactic Clinic

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<td>10</td>
<td>6</td>
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<td>2</td>
<td>53</td>
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<tr>
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<td>4</td>
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<tr>
<td>TOTALS</td>
<td>32</td>
<td>36</td>
<td>63</td>
<td>30</td>
<td>6</td>
<td>167</td>
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ing the Japanese occupation. Those for 1941 are considered a close approximation.

Data for 1937.—At the end of this year there were 61 patients in the segregation station and 106 others attending the outpatient clinic, a total of 167. Their distribution by sex and age is shown in Table 1.

With regard to sex, the totals of 90 males and 77 females give a ratio of 1.17:1.0. The difference lies entirely in the segregated group (1.54:1.0), since the numbers of males and females attending the clinic were identical. A notable difference between the segregated and clinic groups is seen in the age data. Of the former group, only 16 per cent were below 20 years of age, whereas 55 per cent of the outpatients were of that age group; one-half of them were children below 10, infected long after the campaign had been started.

Data for 1938-1940.—In 1938 there was a reduction of 11 patients, with 55 in the segregation station and 101 attending the clinic, making a total of 156. There was a slight increase in the proportions of the younger groups because several adults had died or had been paroled. In 1939-1940 the number of isolated cases was reduced to 44, and the outpatients to 82. In 1939, 4 new cases were isolated and 2 were readmitted, while at the clinic 22 new cases and 5 readmissions were registered. In 1940—the first year after the outbreak of the war in Europe—only 1 new case was isolated, and 10 put on the clinic list; the available data show no readmissions at either place. During this period, then, the number of cases under treatment represented less than 8 per cent of the Nauruan population of 1,761 (see Table 2).

Data for 1941.—The data for this year have not been published, nor are they available at Nauru, but the Department of Territories of Australia (kindness of Mr. C. R. Lambert, secretary) has supplied them. At the end of the year there were 41 patients in the isolation station and 92 enrolled at the clinic, making a total of 133. Two patients had been admitted to the station from the clinic during the year, and an equal number released on parole to the clinic; 3 deaths had reduced the total of segregated patients from 44 at the first of the year. At the clinic, besides the changes noted, 2 new cases had been registered and 1 had died.
The Japanese occupation brought drastic changes for the leprosy patients of Nauru. About June 1943 all inmates of the segregation station were embarked in a leaky boat and towed to sea by a launch, on the pretext of being transferred elsewhere. Only three persons were allowed to see them off. Nothing is positively known of the fate of these patients, but not one of them was ever seen again. Those who had been attending the outpatient clinic dispersed themselves around the island, and some were included among the considerable numbers of people dispatched to Truk during the occupation; afterwards, no less than 759 Nauruans were brought back from there.

Population.—The population statistics for 1940 and 1950, given in Table 2, show that considerable changes took place in the interim. Whereas up to 1940 there had been a steady increase, a material decrease occurred thereafter. In 1940 the Nauruan males outnumbered the females only slightly, whereas

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<table>
<thead>
<tr>
<th>Year</th>
<th>Classification</th>
<th>Under 16 years</th>
<th>Over 16 years</th>
<th>Total</th>
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<tr>
<td></td>
<td>M.</td>
<td>F.</td>
<td>Total</td>
<td>M.</td>
</tr>
<tr>
<td>1940</td>
<td>Nauruan</td>
<td>471</td>
<td>428</td>
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</tr>
<tr>
<td></td>
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<td>21</td>
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<tr>
<td>Totals</td>
<td>482</td>
<td>438</td>
<td>920</td>
<td>455</td>
</tr>
<tr>
<td>1950</td>
<td>Nauruan</td>
<td>320</td>
<td>311</td>
<td>631</td>
</tr>
<tr>
<td></td>
<td>Others</td>
<td>14</td>
<td>8</td>
<td>22</td>
</tr>
<tr>
<td>Totals</td>
<td>334</td>
<td>319</td>
<td>653</td>
<td>498</td>
</tr>
</tbody>
</table>

a Excluding 39 persons absent at the date of census.
b Excluding 29 persons absent at the date of census.
c Other Pacific Islanders. Indentured Chinese laborers are not included.

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a According to a statement by the administrator, Mr. Mark Ridgway (11), "Thirty-nine lepers were on Nauru when the Japanese occupied the island in August 1942. They received no treatment from the Japanese, who about a year later herded them into a barge which was towed out to sea and, according to reports, was then destroyed by gunfire. No trace has been found of any survivors."
b The following statement has been quoted (7) from the administrative report for 1948-1949: "The Nauruan population which stood at 1848 at the time of the invasion was reduced by malnutrition, starvation, disease and war atrocities to 1278 when the Australians returned."
in 1950 the ratio was 1.16:1.0. More striking is the change in the age groups: in 1940, 51 per cent were below 16 years of age, but in 1950 the younger group composed only 42 per cent of the diminished population.

Leprosy cases.—The first leprosy survey in the postwar period, made late in 1945, resulted in the segregation of 10 bacteriologically positive cases, 6 males under 20 years of age (2 under 10) and 2 males and 2 females who were older. Early in 1946 clinic treatment of 11 bacteriologically negative cases was begun; 9 of them were prewar outpatients and 2 were new; only 4 were below 20 years of age. In January 1947 the number of outpatients was 18; 11 were below 20 years, 12 were males and 6 were females. During that year, however, that number was increased to 62, of which 44 (61%) were of the lower age group; there were 33 males and 29 females. While this increment was taking place there was no increase in the number of bacteriologically positive cases in isolation. The situation saw no significant change in 1948 or 1949, nor in the early part of 1950.

The present statistics, as of September 1950, are given in Table 3. There are only 12 cases in the segregation station and 56 under treatment at the clinic; the total of 68 constitutes 4.3 per cent of the present Nauruan population (1,582).* Gen-

* See, however, the addendum at the end of this section.
oral examinations of the entire population are now made at intervals of three months, with a system of fines for unjustified nonattendance. Cases which become negative under treatment are discharged from the station and are systematically followed up. As of the middle of 1951, 5 have been discharged.

The segregation station.—During 1948 this station was rebuilt with two separate weatherboard cottages, one for males and the other for females, each with a capacity of ten beds and each with detached kitchen premises. In addition, there are now two native-fashioned huts, which permit more privacy. A new laboratory and an antiisolatorium have been provided, and an electric light plant has been installed. The patients are attended by a Nauruan, a senior medical orderly, who is housed in separate quarters immediately adjacent to the entrance of the enclosure. School is held for the four young inmates by a senior male patient.

Treatment.—Diagnoses treatment was begun in 1948 and continued for more than two years. Since then sulphone has been used, parenterally, in 50 per cent neutralized solution. It has been given only to the 12 bacteriologically positive cases, and only for about a year, but it is my opinion (V.L.) that this method has definite advantages with regard to simplicity of administration, ease of control, and economy. A maximum dosage of 3.5 gm. per week, given in two divided doses, has proved sufficient, and it can be used for indefinite periods without any painful reaction. There have been no toxic effects with respect to the hematopoietic system. In one case a Herxheimer-like reaction has been observed, quickly controlled by an antihistamin drug and calcium.

Satisfactory results have been obtained in all cases, although the clinical improvement has been relatively slow, perhaps because all of the cases were of long duration with well-established equilibrium between the parasitic activity of the leprosy bacilli and the biological defense mechanism. Granulation of bacilli is first seen after six months of treatment, and after a further considerable period—not less than six months—they disappear leaving the patient clinically and bacteriologically negative. Histological examinations have concerned only the dermal and underlying structures; the nerve terminals, which are probably the last locus of the bacilli, have not been examined.

Addendum.—Austin (2), in a rapid survey made in November 1951 for the South Pacific Health Service, at the instigation of one of us (V.L.), found no new cases. He examined all but 60 of the 1,621 Nauruan people, the absentees accounted for satisfactorily. Of the 56 patients attending the clinic only 4 had active lesions, all of the tuberculoid form and all bacteriologically negative. Among the others there were 3 obviously “burnt-out” cases with mild degrees of deformity. None of the rest, most of whom had received intradermal injections into suspicious areas, could have been diagnosed on sight, although a few of them showed limited areas of impaired sensation. The 11 patients in the leprosy station were all bacteriologically positive, three tuberculoid (T2), the others...
lepromatous (one L1, seven L2). Two had been born in the isolation station and removed soon after birth; 2 others had parents under treatment at the clinic; another 3 had been treated there themselves prior to the Japanese occupation; the remaining 4 had no recent family history of leprosy and had shown no previous evidence of the disease. Among these patients there was a striking absence of deformities or asthenia. The total of 15 active cases (7 tuberculoid, 8 lepromatous) represented 9.3 per thousand of the population. This number is by no means negligible, "but the satisfactory aspect of the situation is the evidence that no cases are being missed."

Austin also examined 332 of the 399 Gilbert Island employees of the Phosphate Commission (the others were engaged in phosphate loading operations), and found 1 bacteriologically negative case with neural manifestations.

EPIDEMIOLOGICAL FEATURES AND CONTROL MEASURES

The Nauru epidemic is unique in the history of leprosy in the extent to which the native population was affected within a relatively short time after the introduction of the infection, in the great predominance of the maculoanesthetic form of the disease, in the nature of the control measures employed, and in the apparently good results of treatment. Since an over-all picture of the significant background factors is not to be had from any readily accessible publication, they are recounted before taking up special features of the matter.

Physiography.—Nauru Island is an ovoid coral atoll some 12 miles in circumference and about 3.4 square miles in area (Text-fig. 1), although it does not conform to the usual idea of an atoll. The major part is an elevated plateau, some 6.6 square miles in extent, covered by phosphate rock derived from guano deposits, and it is this material which gives the island its commercial importance and special economic status. This central area is not cultivable, and hence unpopulated, except for a saucer-shaped depression (Buada District) around an inland lagoon—that being actually a sea-level lake of brackish water fed by seepage through the surrounding coral rock. The plateau drops off more or less abruptly for from 20 to 60 feet and more to a low foreshore, which varies from 100 to 450 yards in width and is heavily wooded with coconut and other fruit-bearing trees. On this encircling zone, totaling (by differ-
ence) about 1.8 square miles, are located all of the people except those in the lagoon district referred to.  

Austin writes that the place was well named “Pleasant Island” by John Fearn, captain of the “Hunter,” who first sighted it in 1798 while journeying from New Zealand to China; that the island represents the peak of an isolated upthrust of the ocean bed, with a fringing coral reef which slopes off so steeply that depths of 250 fathoms are found within a very short distance of the shore; and that removal of the embedded phosphate of the central area “leaves nothing but thickly strewn coral pinnacles giving the semblance of some primitive graveyard closely but irregularly studded with massive monoliths.”
For information regarding the climate, the people and their customs, and various other related matters, the 1924 reports and that of Allan should be consulted.

Administration and sanitation.—Following immemorial native custom the island is divided into 14 districts by boundaries radiating from points in the interior to the sea except for the Buada district, which alone is entirely inland. It is governed by an appointed administrator, who has no connection with the British Phosphate Commission, which operates as a private company. Again according to ancient custom, the people of each district have an elective chief, each assisted by a constable, and over them is a head chief. These chiefs have a certain degree of authority—and responsibility—for local affairs, and they constitute a group consultative to the administrator.

The powers of the administrator are broad, and they are exercised for the well-being of the people in many ways, including economic affairs, education and health. Much more has been done in such matters than in most Pacific island communities because of the unusual economic situation. A part of the royalties on the phosphate shipped goes to the administration; a larger part goes to the owners of the land being mined, which makes them relatively well off.

As for the administrative activities, mention will be made only of matters pertaining to sanitation and health as recounted in the 1924 reports. Personal hygiene was regarded as of vital importance, especially because of the leprosy problem. Among other things, periodical house inspections were made. The houses—simple rectangular, pandanus-thatched structures mostly without walls, protection being provided by removable mat screens—were on the whole kept in a clean and sanitary condition except for the floors. These consisted mostly of raised platforms of loose coral “shingle” which could not be properly cleansed, and a program was undertaken to convert them to concrete. The wells were cemented over and provided with pumps to minimize danger of pollution, and each district was provided with a cement cistern to catch rainwater against times of shortage. Many small natural waterholes were filled in to reduce the breeding places of mosquitoes; fly-proofed, deep-pit latrines were built; and the disposal of other refuse was controlled.

Protection of the natives went as far as to prohibit supplying them with intoxicating liquors; and, although they were
encouraged to produce "toddy" (coconut sap) because of its nutritive value, they were not allowed to ferment it. It was prohibited for children under 16 years of age to use or possess tobacco. When beriberi appeared because of the use of white rice, steps were taken to prohibit the sale of any but the unmilled grain. For the care of the sick there was a hospital separate from that of the Phosphate Commission, and with it an active outpatient department.

Antileprosy campaign.—Early in the epidemic, before a full-time medical officer was appointed, there was instituted a system of monthly examinations of the entire population, and this was continued. For each inspection the chief of the district concerned was required to assemble all his people, which would not be difficult because of the limited areas of jurisdiction, the small numbers of persons living in each, and their evident spirit of cooperation. At the assemblies, which were checked by roll-call, the people appeared dressed in the native costume, which consisted only of a knee-length coconut-leaf skirt (ridi) or a cloth of similar length (lava-lava).

Anyone found to have a lesion suggestive of leprosy was taken to the government hospital for a more thorough examination. Diagnosis was mainly clinical, since few bacteriologically positive cases were found. The clinically doubtful cases were recorded as leprous and treated accordingly; even those found clinically negative were nevertheless regarded as suspects and closely observed. This system doubtless exaggerated the statistics to some extent, but on the other hand leprosy cases were undoubtedly discovered in the earlier stages with a frequency unparalleled in any other such campaign.

During the first few years, all cases were segregated. In 1922, because of the number of new ones found during Morgan’s visit, the isolation station was removed to a location on the southeast coast, where the land varied in width from 100 to 200 yards between sea and cliff. Twice the location had to be extended, so that in 1924 it was over a mile in length. Because of the nature of the terrain, little fencing was needed except at the two ends of the strip.

The clinic for outpatient treatment established in 1924 was located near the government hospital and in Townsend’s time was operated six mornings a week. Later a second such clinic was opened, to save the patients long journeys (7). It has been emphasized (10, 1) that only the “very earliest” and “mild-
est" cases with repeated negative bacteriological findings were so dealt with; those with multiple or progressive lesions, even though negative for bacilli, were segregated and any outpatient who showed even the slightest aggravation of symptoms was transferred to the isolation station. By 1929 only 23 cases had been so transferred out of 201 which had commenced treatment at the clinic (1).

Under the original regulations, the patients in isolation long outnumbered those treated at the clinic. After segregation was applied only to bacteriologically positive cases, about 1929, the clinic patients gradually became the more numerous. From that year to 1933 (7), only 4 or 5 cases were admitted to the station annually except in 1932, when a relieving medical officer isolated all new cases, most of which were released to the clinic when they were examined bacteriologically (5).

Patients paroled from the segregation station were transferred to the clinic for continued treatment. The numbers registered there were relatively large because, to be discharged and dropped from the statistics, a patient had to have been with no active sign of leprosy for two years and should have been under treatment for at least five years (7). Later, apparently these periods were shortened, especially for the cases of uncertain diagnosis (9). Patients discharged from treatment as "apparently cured" were kept under observation at the monthly general examinations and were required to attend the clinic every three months for more thorough examination, to watch for relapses (1).

According to Townsend, the early and doubtful cases treated as outpatients were required to have their own separate food, utensils, mats and clothing, and to prevent laxity occasional "surprise visits" were made. Ten years later Grant said that these patients were made to live in houses separate from the healthy community, that their children lived apart from them, and that they could be employed only for certain types of work.

Babies born in the station—of which there had been 43 in 1936, 34 still alive—were removed at birth and given to foster-mothers not attending the leprosy clinic, and their welfare was guarded by attendance at a weekly baby clinic. It was regarded as unlikely that they had any contact with infectious cases, or if so for more than a short time. Nevertheless, 5 of the 24 such children above the age of three years had developed the disease, 3 in an infectious form at the ages of nine, seven and four years (4).
Sex.—The sex ratios in this epidemic have shown, in total, no material predominance of males. In 1924 (13) there had been 157 males and 127 females (1.2:1.0), but by 1929 (1) this difference had disappeared, with totals of 217 males and 226 females. The situation is somewhat different with respect to the segregated patients. In 1928 (1) there were 75 males and 57 females in isolation, the ratio 1.3:1.0; and of all the cases which had been segregated up to 1936 (5), there had been 155 males and 129 females (1.2:1.0), “the males when once infected [exhibiting] a slightly greater tendency to develop the infectious type of the disease.” In 1937 there was no sex-ratio difference in the clinic patients, but a considerable predominance of males among those in isolation (Table 3). At present this difference is more marked, whereas at the clinic there is an insignificant predominance of females.

Age.—The data of Townsend for 1924, of Allan for the patients in segregation in 1928, of Clouston for all new cases registered from 1929 to 1936, together with those of 1950, are shown in Table 4 in somewhat consolidated form. Townsend

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<tr>
<td></td>
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Table 4. Age distribution at different periods.
remarked that only approximations of age could be made, but he held that "to all intents and purposes" his figures were correct; the oldest patient must be close to 80 years of age, he said, while the youngest was only 3 years old. No later writer has spoken of difficulty in this matter.

Townsend's data are of particular interest because the epidemic was still so recent. They show that 47.2 per cent of the patients were above 20 years of age in 1924; indeed, 24.3 per cent were 30 years or more (not shown in Table 4). As for sex-age relationship, there was a marked predominance of males in the 10-20 year group, whereas in all of the others the proportions of females were slightly the higher. For reasons not apparent, there was a decidedly smaller proportion of younger patients among those in isolation in 1928 (Allan) than in the earlier years (Townsend), and the percentage of older females was extraordinarily high.

More recently the predominance of younger cases has increased. This is seen in Clouston's data, which are directly comparable with those of Townsend's total group in that they comprise all new cases diagnosed over a period of several years. In the present group (Ledowsky) the proportion of younger patients is still larger, almost 70 per cent, but as shown in Table 3 this pertains only to the clinic patients.

Treatment.—Antileprosy treatment was with one or another form of chaulmoogra. Townsend, in 1924, was depending almost entirely on intravenous injections of the oil, although later it was given largely by mouth (1). Allan used alepol to some extent, and also potassium iodide. Chaulmoogra ethyl esters, given intramuscularly, was the sheet-anchor of Grant's therapy.

The treatment given seems to have been highly beneficial. Townsend reported that 70 per cent of the patients in the station were improved, and 31 of them had been paroled. Allan's later figures are more striking: Of the total of 265 segregated cases which had been treated—most of them maculonesthetic—no less than 110 (41.5%) had been paroled, and he regarded another 45 (17%) as fit for parole, making a total of nearly 60 per cent. The cases discharged from further treatment at the clinic totaled 168 (38%), and adding another 60 (18.5%) regarded as fit for discharge makes 51 per cent of all patients treated in either place. Practically all patients still undergoing treatment showed varying degrees of improve-
ment "according to the severity of the disease in them, and the duration of their treatment." Even Demau, who was nodular when discovered in 1920 and had progressed to an advanced stage, was regarded as almost cured. Only 8 (4.8%) of the discharged cases had relapsed.

Grant reported that 83 cases had been paroled from the station in the five years from 1929 to 1933, while 11 relapsed cases had been readmitted. Clouston ascribed the relatively low relapse rate largely to the close check kept on the patients for attendance at the clinic and the long period of after-treatment insisted upon.

**PREVALENT FORM OF THE DISEASE**

A striking and peculiar feature of the Nauru epidemic is that an overwhelming majority of the cases were of the kind called maculoanesthetic. The findings of the various observers have been consistent, and no reason whatever is seen to doubt the validity of this type-diagnosis as applied.

**Summary data.**—The first four cases recognized by Clapp were evidently all "nodular," since smears sent to Australia were reported positive, and presumably they were well advanced since they were noticed where the disease had not been known to exist. Of the 25 new cases diagnosed by Morgan, however, 17 were classified as "anaesthetic," and biopsy specimens were taken from the centers of the lesions without pain.

Of the 284 cases reported on by Townsend in 1924, no less than 268, or 94.4 per cent, had been classed as maculoanesthetic; only 16 were called "nodular" or "mixed." He described four distinct types of macules, of which more will be said later. Anesthesia could be demonstrated in practically all macules. His description of the development and distribution of sensory changes in the lesions (loss of temperature discrimination first, followed by loss of pain sense, and lastly sensation of light touch) indicates careful examination.

Of the total of 443 cases which had occurred to the time of Allan's report (these including 2 Caroline Islanders and 3 Chinese), 412 had been maculoanesthetic, or 93 per cent. Bray said that about 90 per cent of the cases had been of that form. Grant gave no over-all figure, and Clouston did not deal with this point.

Even among the cases in segregation at different periods, maculoanesthesias predominated. They constituted 92 per cent
in 1924, 85 per cent in 1927, and 79 per cent in 1929. In 1936 they were still 68 per cent.

Peripheral nerve involvement.—Neural manifestations, apart from anesthesia of the macules, seem always to have been slight. Townsend remarked that affection of the larger nerves was rare. Two patients had marked unilateral enlargement of the ulnar, without macules or anesthesia corresponding to the distribution of the nerve, and one showed marked nodulation of a superficial cutaneous nerve of the forearm; in none did the peroneal, great auricular or other nerves show abnormality. No deep anesthesias, paralyses, deformities or other trophic complications had appeared.

The number of Allan’s patients showing involvement of the large nerves was not large, although more frequent than in Townsend’s earlier experience and affecting more nerves. In some instances thickened nerves could be traced from the areas of macules, while in others there were no lesions in the regions of affected nerves. A few cases with trophic changes had been seen by that time, but the number was remarkably small. Later writers did not deal with this matter.

Bacteriological examinations.—The methods which were employed to demonstrate bacilli were often drastic, involving the removal of biopsy specimens. With such material Morgan got positive results in 9 of his 17 maculoanesthetic cases, bacilli “scanty” in 5 instances, more numerous in the others. In 3 cases, only, were “squeeze” smears made from the macules, and they were all negative although the excised-tissue smears from the same lesions revealed bacilli in small numbers. Nasal smears were all negative. Dew later remarked on the uselessness of that examination, whereas he had demonstrated bacilli in all of his cases from tissue excised from macules, mostly in the nerves.

Townsend had also failed to find acid-fast bacilli in the nasal mucus of maculoanesthetic cases, or lesions of the nasal mucosa. The most common finding in skin-tissue smears was a large round cell with faintly acid-fast cytoplasm containing strongly acid-fast particles, mostly cocciform, but no typical bacilli; they could be demonstrated in some of the more advanced cases when a little underlying superficial nerve was secured.

Bray said that the examination of suspicious cases involved “... nasal swabbing, clamping and pricking or sectioning of nodules, dissection of lesions—if small, total resection—and preparation of smears and
stained paraffin sections, and finally—the most important and accurate procedure—the dissection for, and the staining of sections of, subcutaneous nerves entering the lesions."

Allan noted, as no one else had, that while it was often impossible to discover bacilli in tissue excised from the anesthetic portion of a macule, they could often be demonstrated in specimens taken from the nonanesthetic edge of the same macule, especially if it was thickened and indurated.

**DID THE TUBERCULOID FORM EXIST?**

With the evidence of the mildness of the disease contained in the reports of Bray and Grant, the question was raised (14) whether there occurred cases of the tissue-reactive tuberculoid form. Clouston (5) thereupon made a search of recent cases and older records and concluded that "the tuberculoid form of the disease appears to be absent." Since he presumably sought the more conspicuous "major" variety as described in the literature, the question remains whether the lesser, or "minor," variety occurred.

**Clinical descriptions of macules.—**Morgan described the macular lesions of his cases as sometimes with "reddish papules . . . in the affected area, frequently arranged as a ring at the periphery." This is suggestive, as are some of the cited statements about enlarged nerves, but more evidence is seen in Townsend's description of the macules, which Allan quoted and confirmed. Besides (a) simple, flat hypopigmented areas, and (b) groups of pale spots which tended to enlarge and coalesce, there were (c) flat pale areas surrounded by a raised, almost papular, erythematous edge which extended peripherally with increase in size of the central depigmented areas, these lesions closely resembling ringworm, and (d) rarely depigmented areas with edges formed by tiny, raised, colorless, somewhat transparent elevations, resembling a herpetiform eruption. The third class is obviously of the minor tuberculoid kind, as perhaps the fourth may also be.11

An unusual case described in detail by Allan is highly suggestive of tuberculoid reaction, and his notes are summarized:

The patient, a woman then 45 years of age, was admitted to the clinic in 1924 with two small lesions, on the chin and left forehead, partially anesthetic and bacteriologically negative. These cleared up under treatment and she was discharged as apparently cured in 1926. At the monthly inspection in February 1929 she was found to have developed "a sudden

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11 As has been seen, Austin has classified as tuberculoid all of the four active cases now attending the clinic, and also three of the 11 cases in the isolation station.
outbreak of nodular leprosy in rather severe form," with large nodules surrounded by erythematous areas on the face, arms, legs and trunk, partially or totally anesthetic. Numerous acid-fast bacilli were found in sections of two lesions of the face; nasal smears were negative. The response to alepil was "almost miraculous." After a few injections the lesions began rapidly to decrease; in three months they had almost disappeared, with return to normal sensation in most of them; and in 6 months they had all gone.

Morgan's biopsy specimens.—At about the time Clouston's first report appeared, one of us (H.W.W.) was given by Morgan sections of nine of his biopsy specimens. They were of formalin-fixed tissue and somewhat faded, and the material was not favorable since they had come from centrally in the lesions, where most resolution would have occurred. Later he supplied information regarding the status of his cases as of late 1938, nearly 16 years after he had examined them.

Only one of the six specimens from patients who had been classified as anesthetic is definitely tuberculoid, and that is of rather slight degree. That patient (Adiefor) died of pulmonary tuberculosis in 1925. Of the three specimens from cases which had been classed as nodular or mixed, only one shows typical lepromatous changes. In 1938 that patient (Zaramartin) was still alive and the disease advanced (C3-N2).

Two of the sections from anesthetic cases are virtually negative. Both of these patients (Noa and Domi) were ultimately discharged and had had no further developments in 1938; one was then under training at Suva as a native medical practitioner.

The other sections, from two nodular and three anesthetic cases, permit no definite decision. In certain of them there are groups of cells of epithelioid aspect, but without characteristic tuberculoid formalization. For one, however, the histological diagnosis of "leprid, tuberculoid, rather slight" was ventured at the time; it turned out that that specimen was from a patient (Dekanea) classed as "mixed," who in 1938 was still alive and advanced nodular (C3-N2). The specimen from the other nodular case is more indefinite; that patient (Adip) was still in the isolation station when he died of "heart failure (?)" in 1934. Of the three maculoanesthetic patients, one (Arabarab) was discharged from the station in 1930 but soon died of appendicitis. Another (Johannes) developed nodules in 1932 and became an advanced case (C3-N2). The third (Obed) was discharged in 1934 but continued under clinic treatment because of occasional slight clinical activity with continued negative bacteriological findings; during the occupation he was sent to Truk, and he died there while hiding because of the advanced condition of his lesions.

Lepromin testing.—This test has been employed only by Clouston, and by him to a limited extent. He first (4) tested 16 children above the age of 5 years who were born in the segregation station and removed at birth, including 5 with
symptoms of the disease. All gave negative or negligible re­
sults except one of the latter group (2+ response). Later (5) he told of applying the test to eight relapsed cases, all of which proved negative.

DISCUSSION

The most remarkable feature of the Nauru epidemic is the contrast between the very high proportion of the population which became affected and the overwhelming predominance of the maculoanesthetic form of the disease. The high prevalence bespeaks an extraordinary susceptibility of these isolated people to the newly-introduced infection, whereas the predominating form signifies a generally high degree of resistance to progression of the disease once symptoms had appeared. This is contrary to the prevailing idea that among a newly infected people leprosy is prone to be particularly severe until the most susceptible individuals are weeded out and the populace as a whole develops resistance. The severity in this instance pertains only to the extent of the epidemic, not the type of the disease.

It is certain that a remarkably large proportion of the population was affected, regardless of what the real percentage may have been. If the figure derived from Allan’s data, 35 per cent up to 1929, is too large because of inclusion in the clinic lists of doubtful cases not actually leprous, it may still be that 30 per cent were actually affected.

Evidence of great susceptibility is also seen in the rate of development of the epidemic once it was well started. The peak period apparently extended from 1924 to 1927; the figures diminished materially thereafter because of discharges. Nevertheless, on the average 48 new cases a year were registered in the four-year period between 1929 and 1932 (7).

The explosiveness of the outbreak has often been ascribed to debilitation of the survivors of the disastrous influenza epidemic which occurred in 1920. It seems open to question, however, if that factor could have had as great an influence as has been supposed. How many people may have been infected by then is problematical. The after-effects of that epidemic may indeed have caused some latent infections to become active, and

"It appears (5) that the medical officer wrote in 1922, "The extraordinarily rapid spread of the disease from the small focus existent in 1920 I believe to be due in notable degree to the 100% incidence of the pneumatic influenza epidemic in September 1920, with its resultant severe debility."
it may have left the people not already infected temporarily more susceptible than they would have been otherwise, but that factor would hardly be expected to be operative for long (14). Indeed, we have Morgan's description of the Nauruans, as seen a year and one-half later, as a happy, well-nourished people who quickly forgot any minor disturbances of their usual good health. The principal factor was probably an extremely high innate susceptibility.

In this epidemic females appear to have been practically as susceptible as males, contrary to the usual situation in places where the disease has been long established, although in the males the disease as usual showed a tendency to greater severity. This lack of a sex differential is not ascribable to an overwhelming preponderance of patients below the age of puberty, among whom boys are usually no more susceptible than girls, for in the earlier period of the epidemic the proportion of older individuals affected was high.

It is obvious that large numbers of the cases must have been infected after early childhood, and even in adult life. There has not been even a hearsay record of an indigenous case until about 1914, ten years previous to the time of Townsend's report. As has been noted, no less than 47 per cent of his 284 cases were 20 years old or more in 1924, and so at least 8 years of age in 1912; and a large majority of them must have been considerably older when actually exposed to infection. Or, starting with the next ten-year group, 24 per cent were at least 18 years of age in 1912. Clouston, taking 1917 as the time when the first generalized opportunity for infection might have occurred, said that 15 per cent of the cases must have been over 25 years old when first exposed.

With respect to the predominance of the maculoanesthetic type, it is highly probable that when the first four cases were discovered in 1920—they presumably all "nodular," as Demau, the first case, is said definitely to have been—there must also have existed more numerous ones of the less noticeable form. Grant was told of two cases with anesthetic macules existing in 1914 and 1916. After Clapp had begun to examine the population systematically, and especially after Morgan had confirmed the diagnosis in 25 new cases, 17 of which he classed as "anaesthetic," the number of such cases increased rapidly. Then and thereafter many times more were diagnosed as maculoanesthetic than as "nodular"; for years the former constituted
over 90 per cent of the whole. It seems highly doubtful that early diagnosis and active treatment could have been responsible for the infrequency of the more serious form of the disease. Nor is it likely that many early lepromatous cases with erythematous macular lesions could have been mistakenly called maculoanesthetic, as evidenced both by the drastic methods that usually had to be employed to find bacilli and by the infrequency of change to the nodular form.

Relatively high resistance on the part of the infected persons is also indicated by their response under treatment. The cited data all bespeak marked benefit from chaulmoogra oil and its derivatives, and chaulmoogra is not rated as highly effective even by its defenders; and in most places the maculoanesthetic form is not particularly amenable to it. Although the cases were mostly early and the treatment was doubtless active and regular, we hold it as doubtful if it could have given the results that it evidently did had not, so to speak, the terrain in the patients been particularly favorable.

The resistance thus indicated cannot, it would seem, be related to a particularly high degree of tissue reactivity to the infecting agent. Clouston was unable to find cases or records suggestive of the tuberculoid form of leprosy as he understood it, which presumably would have been of the major variety. That reactional tuberculoid cases actually occurred seems evident from the one of Allan which has been cited, but the fact that he described it in detail suggests that it was unusual. That he designated it as "nodular" suggests that others of the segregated cases so classified may have been of that kind, but they could not have been numerous. The detailed descriptions of macules clearly include the minor variety of the tuberculoid form. There is no indication of how frequent such cases were, but from the priority given the simple flat macules in the descriptions it seems probable that the latter predominated. The evidence of Morgan’s biopsy specimens is not very helpful, for only one of six specimens taken from (the centers of) maculoanesthetic lesions shows a definite tuberculoid picture; on the other hand, there is interest in the fact that only one of three specimens from "nodular" cases is frankly lepromatous. It is unfortunate that Clouston applied the lepromin test to only a few selected individuals, but his results suggest a low tendency to reactivity.

On the whole, it would seem probable that the high resist-
ance to the progression of the disease, among a people so highly susceptible to infection, must have been related to some factor less evident and more fundamental than tissue reactivity. Pertinent to this question is a conclusion of Lurie (8), drawn from studies of tuberculosis in his specially bred rabbits, certain strains of which are genetically very susceptible while others show relatively high resistance: "Resistance to infection . . . is two-phased. Resistance to attack . . . is distinct from resistance to the progress of an already engrafted disease."

The fact that the disease became so prevalent despite the small proportion of definitely lepromatous cases raises another important question, namely, whether it was spread entirely by those cases, or whether those rated as "noninfectious" and not subjected to segregation may also have been implicated. This point was raised only by Allan. None of the 13 surviving children who had been born in the isolation station and transferred at birth to the care of nonleperous foster parents had (as yet) shown evidence of the infection, whereas "many parents suffering from leprosy in its early stages who are treated at the leper clinic without isolation because considered to be noninfectious, are found to have children also suffering from the disease." It is not known, he pointed out, how early in the disease a person with leprosy may be infective, and the number of such infected children suggested that many cases were infective in the earliest stages.

There are those today who do not agree with the prevailing belief that "closed" cases are relatively harmless, and who consequently disagree with the common practice of exempting from segregation or other control all that are found bacteriologically negative by the usual simple methods of examination. The control measures applied at Nauru to maculoanesthetic generally, and even to suspects, were relatively severe. The fact remains that the prevalence of the disease decreased steadily after the first few years. From a maximum annual prevalence of about 24 per cent of the population in 1924, the cases diagnosed and under treatment—those discharged as apparently cured having been dropped from the statistics—had decreased to 16 per cent in 1928, and to less than 8 per cent in 1940. The present rate is a little over 4 per cent—obviously somewhat less than it would have been had the segregated patients not been eliminated as they were in 1943—although less than one-fourth of the cases are active ones.
These results may be held amply to justify the measures that were employed. Progress has been slow, for it has taken thirty years to effect the present rate, which in most localities would be regarded as very high. It is evident that new infections are still occurring, so that it will yet be some time before the disease is eradicated. This experience should give pause to anyone who would say that in any endemic region leprosy could be cleaned up in a few years by any control measures which it would be possible to employ.

Whether or not the trying conditions which existed between 1942 and 1945 will have an adverse effect on the situation cannot yet be said with assurance. However, after more than five years there is no evidence that they caused the appearance of manifestations in any material number of individuals in whom the infection may have been present but latent at the time.

SUMMARY

A study of the unparalleled leprosy epidemic at Nauru which began somewhat before 1920 is presented, drawn in large part from reports which are not readily accessible, and the statistics of recent years are recorded. The significant epidemiological factors existing and the control measures applied are reviewed and discussed.

The earlier figures show that from the time the first cases were recognized in 1920, up to 1929, a total of 35 per cent of the population had been diagnosed as having leprosy, although some of the recorded cases were evidently only suspects. The prevalence rates for individual years, not counting cases which had been discharged as apparently cured, ranged from an approximate maximum of about 25 per cent in the period from 1924 to 1927, down to only 4 per cent at the present time. During the Japanese occupation the bacteriologically positive cases were eliminated, but it does not appear that the conditions which prevailed in that period have resulted in an increased rate of development of new cases.

The epidemiological factors considered are the physical characteristics of the island, where the population is located almost entirely on a narrow foreshore encircling a largely uninhabited phosphate-bearing plateau; the administration of the place and the general measures that were applied to promote sanitation and health after the outbreak of the leprosy epidemic; and the factors of age and sex among the affected individuals. With
regard to sex, the figures show no material or even constant predominance of males, except latterly among the more serious cases. The data on age show on the whole large proportions of children and adolescents, greater in recent years than in the earlier period. It is evident, however, that a considerable proportion of the cases known in 1924 must have been infected in adult life.

The main features of the special measures to control the disease were monthly inspections of the entire population, special examinations of suspects, segregation of all but the very earliest and mildest of maculoanesthetic cases regardless of the bacteriological findings (that during the earlier period, later modified to segregation of only the cases found bacteriologically positive by the rigorous methods of examination employed), and outpatient treatment of the unsegregated cases and of those paroled from segregation until regarded as ready for discharge. Various other measures were also applied.

The most remarkable feature of this epidemic is the contrast between the high susceptibility of the people to infection and the high resistance to progression of the disease evidenced by the mild form which most of them exhibited, more than 90 per cent of the cases having been classified as maculoanesthetic. Innate resistance is also indicated by the highly beneficial effects of the treatment given, based almost entirely on chaulmoogra oil and its derivatives.

This condition is held to be not wholly ascribable to early diagnosis and active treatment. Nor can it be ascribed to or correlated with high reactivity of the tissues to the infecting agent, for the recorded clinical descriptions and the findings in several biopsy sections here reported indicate that the tuberculoid form of the disease was uncommon, and was mostly of the minor variety. The few lepromin tests that have been made suggest a low tendency to reactivity. The facts in the case suggest that some other inherent and fundamental factor was operative, and are in keeping with Lurie's view with respect to tuberculosis that resistance to attack is distinct from resistance to the progress of an already engrafted disease.

The question of whether or not the early maculoanesthetic cases which were found bacteriologically negative by relatively drastic examinations and were treated as outpatients may have had a part in spreading the infection is considered, without conclusion. However that may be, the results of the control
campaign carried out since 1922—interrupted only during the war and the earlier post-war period—are held amply to justify the measures employed.

ACKNOWLEDGEMENTS

Our thanks are due the authorities of the Department of Territories, Commonwealth of Australia, Canberra, for numerous courtesies, in the first place for supplying requested information on the Nauru situation since 1936 (prepared by V.L.), originally intended for use in the news section of The Journal. Examination of the manuscript of Dr. R. Tennyson Allan in the possession of one of us, and of the official Nauru report of 1924 referred to in that document, revealed how inadequate are the reports in the periodical literature. The present review has been cleared with the authorities concerned in Australia and Nauru.

Thanks are also due Dr. V. Grantley Morgan, director of the Commonwealth Serum Laboratories, for supplying copies of his reports and the sections of several of his biopsy specimens. It cannot be said whether Allan’s manuscript was received from him or someone else. Mrs. Allan, and at her request Professor P. MacCallum of the University of Melbourne, have supplied certain information. After the manuscript of this review was prepared Dr. C. J. Austin, superintendent of the Makogai Leprosy Hospital, Fiji, who visited Nauru late in 1951, supplied a copy of his report containing certain data which have been inserted as footnotes and an addendum.

RESUMEN

La extraordinaria epidemia de lepra en Nauru, donde la enfermedad fue introducida cerca del 1912 y donde fueron reconocidos los primeros casos de lepra en el 1920, ha sido inadecuadamente tratada en los reportes publicados accesibles al autor. Esto se hizo aparente al examinar el reporte anual de Townsend en 1924, quien sirvió de oficial médico del gobierno de 1923 a 1928, al cual los autores posteriores no hacen referencia, y una tesis manuscrita por Allan, quien sirvió durante el año 1928-29, y que fue escrita en 1930. Todos los reportes accesibles son reportados en este trabajo, y datos que hasta la fecha no habían sido publicados son también presentados por el autor.

Para los fines del 1922 ya había 139 casos diagnosticados, o sea el 12.5% de la población en esa época, y en 1924 el número ascendió a 264, o sea el 23.7%. Ya en el 1929 el 35% de la población había sido diagnosticada como leprona. Debido a altas después de tratamiento la frecuencia en record indica una disminución al 16% en 1926, el 14% en 1928, el 10% en 1933 y el 8% en 1940. En 1950 se notó una frecuencia de 4.3%. La mayor parte de estos datos son exagerados pues no hay duda que muchos casos sospechosos fueron ingresados y tratados como definitivamente leprones.

Después de hacer un recuento de la fisiografía peculiar de Nauru, la distribución de la población y las actividades administrativas que en relación a la sanidad creó el problema de la lepra, las medidas especiales necesarias para el control de la lepra son discutidas por el autor. Estas constaban exámenes mensuales a toda la población; segregación, al principio de todos los casos, luego de todos menos los más iniciales sin
considerar los hallazgos bacteriológicos, después de aquellos que eran bac-
teriológicamente positivos solamente, y tratamiento en las clínicas de pa-
ciente ambulatorios a todos los demás casos. En cuanto a sexo, los varones
no han arrojado predominancia, excepto posteriormente entre los casos más
avanzados. Los datos relativos a la edad demuestran que muchas personas
deben haber sido infectadas en vida adulta. El tratamiento, basado exclu-
sivamente en el aceite de chalumgra y sus derivados hasta muy reciente-
mente, parece haber sido de efectividad poco usual.

La severidad de esta epidemia concierne la gran proporción de la
población que fue afectada, no a la forma en que ocurrió la enfermedad.
Por lo menos el 90% de los casos fueron clasificados, aparentemente cor-
rectamente, como maculo-anestesicos. De la información disponible, incluso
algunas biopsias reportadas aquí, y la sospecha de que la reacción a la
lepromina en algunas pruebas fue de poca intensidad, parece ser que la
forma tuberculoide no fue prominente; y de aquí se deduce que la forma
benigna de la enfermedad y el buen resultado del tratamiento, no se
deieron a una alta reactividad tisular, sino más bien a alguna otra forma
de resistencia innata. Esta situación está de acuerdo con la opinión de
Lurie que la resistencia al ataque de una enfermedad es diferente de la
resistencia al progreso de la ya establecida enfermedad.

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