The leprosy problem in the South Pacific

I am a New Zealander living in London. From 1966 to 1968, I was a medical officer, specialising in leprosy, in the northern region of Nigeria. Patients were treated here in villages by auxiliaries, using weekly doses of dapsone. In 1967, I carried out a population survey and found that the prevalence of leprosy had declined from 67 per 1000 to 2 per 1000 after 15 years of dapsone monotherapy.\(^1\) This information, together with other evidence, indicating that the disease can be eradicated, has recently been published.\(^2\)

Despite this optimism, leprosy remains a serious problem in the South Pacific. Recent data from the World Health Organization (WHO) shows that there have been 94 new cases in Kiribati, including 21 children.\(^3\) The incidence of childhood leprosy is very significant, as an indicator of ongoing transmission. The percentage of 22%, next to Micronesia and the Marshall Islands, is the highest in the world.

In contrast, in Vietnam, in a population of some 80 million and after a horrific civil war there were only 10 new children with the disease. It appears that the Pacific Leprosy Foundation continues to focus on managing patients after they have become crippled rather than providing early treatment with multidrug therapy, which would prevent the spread of the disease.

I produced maps of the area in Kaduna Province (Nigeria) where domiciliary treatment could be obtained.\(^1\) No such maps appear to be available in Kiribati, or where the focus of infection remains. Mobile clinics could be introduced through nautical means of transport. The successful decline in Nigeria proves that field workers should be only employed to treat leprosy and not ‘integrated’ with other diseases. This would stop workers being diverted to treat tuberculosis.

It appears that some workers have not been trained to recognise leprosy, although the ‘diagnosis and treatment is easy’ (WHO). It does not need highly qualified people to work in leprosy. Any school leaver can be trained. In fact, the best leprosy auxiliary I worked with in Nigeria could barely read or write, but he was always on time for the weekly administration of dapsone and knew all the patients. There should be a good response to treatment provided that the patients are not segregated.

Other countries listed in the Foundation’s website are Tonga, Fiji, Western Samoa and Vanuatu. In *The Weekly Epidemiological Record* (a WHO publication),\(^3\) there were three new cases in Fiji with no children; Samoa had eight with one child, but there were no returns for Vanuatu and Tonga. Surely it is the Foundation’s responsibility to ensure that all new cases are recorded, especially in children. In Tonga, there are apparently no new infections, but this has to be confirmed by examining the contacts of new child and multibacillairy cases.

As there is now a Centre of International Health in Dunedin, I would suggest a collaboration with the Pacific Leprosy Foundation, especially as professionals are conducting surveys for tuberculosis.
A recent publication entitled *A strategy to halt leprosy transmission*, reinforced these points. For example, ‘Few countries now have a surveillance-response system that could provide the epidemiological data to map high-risk areas for leprosy, to monitor the changing epidemiological pattern of the disease and to implement the required interventions’ and ‘School surveys, too, might provide clues: the finding of school-age children with leprosy is a strong indicator of ongoing transmission’.

This approach should also be adopted in Micronesia and the Marshall Islands where the Pacific Foundation has recently taken over responsibility for leprosy. The high prevalence of leprosy has been recognised since 1971, but little appears to have been done here to prevent the spread of the disease. The Pacific Leprosy Foundation has been given an award which could be put to good use and I am sure that the New Zealand public would donate generously.

It has been claimed that the incubation period for leprosy is very long; at least 4–6 years and sometimes longer, but there is no evidence for this. Instead the decline in the Karamui study suggests that it is quite short. The authors of the *Lancet* publication write ‘A serious obstacle, however, to gaining the full potential of contact tracing is the absence of a diagnostic test for early-stage or sub-clinical infection in contacts’.

As you can see from the details in the chapter, we have shown that it is possible to reproduce the features of tuberculoid leprosy as a result of an autoimmune response to an antigen in peripheral nerve rather than a direct response to *Mycobacterium leprae*. A specific positive skin test will produce an epithelioid cell granuloma, thus reproducing the pathology of this form of the disease. This will define the incubation period and determine whether there is a subclinical infection. It will also determine whether transmission has ceased in a previously endemic area.

Money for research projects is available from the leprosy charities at info@leprosyresearch.org – I would strongly encourage New Zealand neuroscientists to apply for a grant to isolate the non-myelin antigen involved. Details of the procedure are available.

Nerve damage is the main reason why leprosy is a serious disease. I have emphasised that patients with non-lepromatous leprosy may develop acute sensory loss in all four limbs.

On the basis of this clinical finding, rabbits were injected with a homogenate of human sensory peripheral nerve plus adjuvant and electrophysiological recordings were taken from the hind limb by Jim Pascoe at University College London. There was a specific diminution of C fibre action potentials with preservation of A delta fibres.

This is also a good model to study pain mechanisms and diabetic neuropathy as well as leprosy, so physiologists should also consider applying for a grant to continue this work.

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References:


