

Comments on Immuno-Epidemiology of Leprosy

TO THE EDITOR:

You kindly asked me to comment on your editorial "Immuno-Epidemiology of Leprosy" (IJL **43** [1975] 145-148), in relationship to the findings of our epidemiometric model (Lechat *et al.*, Bull. WHO: **51** 361-373).

In fact, both your review of declining incidence trends in such countries as Norway

and Hawaii, and the computer-simulated predictions we generated for South India, are approaches to the same end, using different methodologies. What the epidemiometric model tries to achieve is to predict the trends of incidence (that is new leprosy cases) under base line conditions over a long-term period on the basis of the number (prevalence) of infecting cases over the

successive years. In the model, baseline conditions are the current control methods (early detection and large scale treatment) as presently applied in a reference area in South India. Then, other control methods are simulated by modifying the parameters, that is manipulating, for example, the numbers of susceptible individuals, the number and respective proportions of infective cases (treated, nontreated, abandoning treatment, etc. . . .) in order to see how it affects subsequent annual incidences.

What the model amounts to is to simulate longitudinal studies whereby populations submitted to a given set of epidemiologic conditions would be submitted to different control measures. While it would manifestly be preposterous to think of an experiment in which two or several population groups given different treatments (chemotherapy, segregation, or no treatment at all) would be followed during let us say 10 or 20 years, modeling does just that provided the parameters have been defined and quantified. The model also enables to simulate control measures which do not or do not yet exist (such as specific vaccination).

The observations your editorial mentions of leprosy declines constitute in some way the output of longitudinal studies whose conditions were not, as you rightly pointed out, and for obvious reasons, controlled. In this case, as often, the epidemiologist has to make do with the results of unplanned experiments. Although uncontrolled, these *in vivo* experiences present however the advantage of referring to real life situations, since they lack the simplifying assumptions which are central to the model approach. Both methods have as their objectives, to quote the words of the editorial, "to predict the general length of time it will take to reduce leprosy to a minor problem." In both approaches, incidence is taken as the index of effectiveness for leprosy control.

The examples given in the editorial, that is Hawaii, Norway, Okinawa, Taiwan, refer to trends observed under different conditions, such as physical segregation, chemotherapy, a mix of both, and other unidentified factors possibly involved in the dynamics of leprosy, among which socioeconomic improvement could play an important role. In every one of these countries one observes a decline in the number of new cases, which could or could not be due to the

control measures implemented. The similarity with the trends observed in the model is of course striking. In the model, with current control measures as described, leprosy incidence in South India is expected to be reduced by 60% in 20 years. No attempt has been made to extend predictions until eradication or virtual eradication is achieved (which according to your review took some 60 years in Norway and 85 years in Hawaii). It surely calls for long-term vision when planning leprosy control. Reducing leprosy to a minor health problem is not by any way an affair for over a couple of years, at least with present control measures.

A difficulty that you mention is that case detection is often confused with incidence, the peak of new cases appearance reflecting some intensified leprosy related effort. In the model, this difficulty has been circumvented by assuming a logarithmic function constant over the years for the delay between onset and detection, all cases being finally detected. What would happen to the values predicted for incidence when detection is not complete has not been tested.

It should be stressed that simulations of our model have yielded striking results with respect to actual or potential control measures such as segregation and vaccination. Segregation of infective cases has been shown to be remarkably inefficient, showing virtually no gain in decrease of incidence over a 20 year horizon. By contrast, specific vaccination aiming at preventing leprosy of any type has been shown to be the most efficient among the methods which were simulated, vaccination of 100% of the population resulting in incidence zero after 13 years. This justifies present efforts invested in microbiologic and immunologic research.

It should be stressed that the observations reviewed in the editorial, which induce optimism in the long-term for the future of leprosy, as well as the results of our model, do not consider drug-resistance. It seems as though even a slight increase in the prevalence of resistance could dramatically affect and possibly reverse the present declining trends. And that is another tale that the model, we hope, will help us to decipher.

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