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Abstracts

1. GIDOH M, TSUTSUMI S, NARITA M, FUKUSHI K. **Influences of several anti-leprosy drugs on immunological state of experimental animals.** (1979) *Jap J Lepr,* 1979, **48**, 4–169.

In a previous paper (Gidoh *et al, Jap J Leprosy* **48**, 1979, 7–8) these authors provided evidence that DDS can suppress acute inflammation and cellular exudation in several experimental systems, while exacerbating the chronic inflammation involved in adjuvant-induced arthritis. In contrast, B663 was able to suppress this type of chronic inflammation. They speculated that the effect of B663 might be due to the activation of suppressor T-cells, leading to the normalization of cell-mediated immunity.

In the present paper the authors have investigated the effects of these and other drugs, including levamisole, on the antibody and delayed skin-test responses to sheep erythrocytes of mice and guinea-pigs. They also looked at the percentage of guinea-pig peripheral blood lymphocytes which formed rosettes with rabbit erythrocytes. Such cells are assumed to be mainly T-lymphocytes. Dapsone was given daily for 10 days at between 10 and 100 mg/kilo, whereas B663 was used daily at 30 mg/kilo. The use of such large doses must cast doubt on the relevance of the results to man. Nevertheless the findings were interesting.

There was no convincing effect of either drug on antibody or delayed skin-test responsiveness of normal animals unless the dose was so large that the animals were ill. On the other hand dapsone significantly decreased the percentage of guinea-pig lymphocytes which formed rosettes with rabbit erythrocytes, whereas B663 did not, or sometimes, like levamisole, caused a slight increase. These effects were enhanced in thymectomized animals. The authors suggest therefore, that thymectomized animals may provide a sensitive assay for any immunomodulatory effects of these drugs.

Comment

The efficacy of dapsone in the treatment of several conditions not known to be caused by an infectious agent has prompted the suggestion from many authors that in the treatment of leprosy it may have effects on the immune response of the host, as well as effects on the organism. Indeed the rather low serum levels which can be achieved with dapsone have led some authors to doubt the existence of a direct antibacterial effect. The demonstration of apparently dapsone-resistant *Mycobacterium leprae* by the mouse foot-pad technique can never completely refute this argument, since it does not exclude the possibility that such organisms are resistant to a dapsone-induced change in the immune response, rather than to the drug itself. However the need for such a tortuous hypothesis has now diminished, because mycobacterial species have been found (Portaels, *personal communication*) which are sensitive to very low dapsone levels comparable to those achieved in the patient's serum.

Nevertheless it remains theoretically possible that direct effects of dapsone on the host are also important and therefore, that failure of dapsone therapy could sometimes be due to host factors.

G A W Rook

2. TEN DAM HG, HITZE KL. **Determining the prevalence of tuberculoiis infection in populations with non-specific tuberculin sensitivity.** *Bulletin of the WHO* 1980 **58**, 475–83.

A simple and interesting concept is presented in this paper based on tuberculin testing before and after BCG vaccination. Basically the authors say that whereas positive responses due to infection with tubercle bacilli or previous vaccination are not enhanced after BCG, tuberculin reactions due to sensitization by non-tubercle bacilli are increased in size after BCG vaccination. Their explanation for this is that only *Mycobacterium tuberculosis* and its variants including BCG can sensitize to the species specific antigens of this species. Thus a reaction to shared mycobacterial antigens will be increased in size by the addition of sensitization to the species specific antigens.

This simple hypothesis enables the authors to estimate the prevalence of tuberculosis infection by measuring the percentage of persons in a given age group who do not have previous BCG scars, but do have positive responses to tuberculin that are not increased in size by subsequent BCG. Applying their method to Dahomey and Mauritania the authors arrive at the figures of 10.7% and 17.5% as the prevalences of tuberculosis in the two countries respectively. In each case this is less than half of the prevalence calculable on the basis of a single tuberculin test result. The same was true when the authors applied their principles to the published work of others on Burundi.

An omission from this work was detail on how soon after BCG the second tuberculin test was performed, although this would not undermine the hypothesis. More serious today was their lack of consideration of the probability that not all positive responses to tuberculin are by the same immunological mechanism. If this is finally proven to be the case then perhaps the assessment of disease prevalence are erroneous or only fortuitously correct and the authors' system might prove better as a measure of susceptibility.

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