Lepr. Rev. (1973) **44**, 96-98

Abstracts

1. **Mycobacteria on vegetation in Uganda,** by D. J. P. BARKER, J. K. CLANCEY and S. K. RAO. *East Afr. med. J.* 1972, **49**, 667-671.

The authors isolated in pure culture 27 fast-growing and 17 slow-growing mycobacteria from vegetation found in marshy areas in Uganda. Although none of the isolates had the cultural characteristics of *M. ulcerans,* the presence of these organisms on 49 out of 322 samples of grasses and sedges investigated suggests that the epidemiological indications that Buruli-ulcer infections could be implanted intradermally from such a source, may eventually be microbiologically confirmed.

S. G. Browne

2. **Leprosy VI. The treatment of leprosy patients with intravenous infusions of leukocytes from normal persons,** by SOO DUK LIM, R. FUSARO and R. A. GOOD. *Clin. Immun. Immunopath.* 1972, **1**, 122-139.

The authors treated 4 leprosy patients with weekly intravenous infusions of leucocytes obtained from a pint of citrated blood (from healthy donors having had no contact with leprosy) and suspended in 150 ml of normal saline. The infusions were given very slowly. Three of the patients had lepromatous, and one tuberculoid leprosy; all were intolerant of drug treatment.

All the patients responded rapidly to the infusions. Full case-notes are provided, together with clinical and histopathological photographs showing the appearance of lymphocytes and multiple small epithelioid cell follicles in the lymph nodes, and the rapid disappearance of *Myco. leprae.* After treatment, all the patients were able to tolerate standard drug therapy.

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3. **Clofazimine therapy of lepromatous leprosy caused by dapsone-resistant *Mycobacterium leprae,*** by L. LEVY, C. C. SHEPARD and P. FASAL. *Am. J. Trop. Med. Hyg.* 1972, **21**, 315-321.

In this investigation, carried out in San Francisco, USA, leprosy bacilli recovered from skin biopsy specimens on 11 patients with lepromatous leprosy who had not responded to sulphone therapy were tested for susceptibility to dapsone in the mouse footpad. The organisms from 5 patients were found fully susceptible. and it was later confirmed that these patients had defaulted on treatment. The organisms from the other 6 patients were resistant to dapsone, and 5 of these patients were treated with clofazimine (Lamprene; B663) in a dosage of 100 to 200 mg daily. The patients with susceptible bacilli were given 50 mg of dapsone daily, and in both groups the infectivity of the organisms for the mouse footpad was tested at intervals during treatment. It was found that bacterial killing began at the start of treatment with dapsone, but only after 50 days in those treated with clofazimine. Once started, however, the rate of bacterial killing was the same in both groups of patients.

W. H. Jopling

4. **Dapsone acetylation and the treatment of leprosy**, by G. A. ELLARD, P. T. GAMMON, H. S. HELMY and R. J. W. REES. *Nature*, Lond., 1972, **239**, 159-160.

Dapsone (DDS) is converted in man to monoacetyldapsone (MADDS) by the same enzyme system that acetylates isoniazid, but MADDS is rapidly deacetylated in man. The half-life of dapsone in the body, unlike that of isoniazid, seems to be unrelated to the speed at which it is acetylated. A study was made of 6 patients with leprosy who had received 1 mg dapsone daily and responded well initially, and of 23 patients from whom dapsone-resistant *Mycobacterium leprae* were isolated. The acetylator phenotype of each patient was judged on the capacity to acetylate isoniazid. Two of the 6 patients in the first group were found to be rapid acetylators but they "responded to treatment during the first 4.5 months as satisfactorily as the slow acetylators". In the second group 6 were slow and 17 were rapid acetylators. The authors conclude that "the rate of acetylation of DDS is likely to be without prognostic effect in the treatment of leprosy".

C. S. Goodwin

5. **The death rate of *Mycobacterium leprae* during treatment of lepromatous leprosy with acedapsone (DADDS)**, by C. C. SHEPARD, L. LEVY and P. FASAL. *Am. J. Trop. Med. Hyg.* 1972, **21**, 440-445.

225 mg acedapsone (DADDS) was injected every 77 days into each of 10 patients with untreated lepromatous leprosy, and material from biopsy specimens taken "at intervals" was inoculated into the footpads of mice. Biopsy specimens from 14 patients receiving dapsone "orally in a dosage rising over a period of 4 weeks to 50 mg daily" were similarly processed. "Infectivity for mice was not detectable after 100 days" in 3 patients receiving acedapsone, and in 12 patients receiving dapsone. Of the other patients receiving acedapsone, this effect was found in 3 patients before 200 days, and in 3 others before 300 days. The clinical response in the 2 treatment groups "appeared to be the same". Four of the patients developed erythema nodosum leprosum although their dosage of acedapsone was equivalent to only 2.2 mg dapsone daily. The isolates of *Mycobacterium leprae* showed no resistance to dapsone, and no explanation can be found for the results obtained with the specimens from patients receiving acedapsone.

C. S. Goodwin

6. **Rapid bactericidal effect of Rifampin on *Mycobacterium leprae***, by C. C. SHEPARD, L. LEVY and P. FASAL. *Am. J. Trop. Med. Hyg.* 1972, **21**, 446-449.

600 mg rifampicin (Rifampin) daily was given to 5 patients with untreated lepromatous leprosy. Material from biopsy specimens lost its infectivity to mice before the first specimen was taken, after 7 days' treatment in 4 patients and 14 days in 1 patient. In mice one dose of rifampicin 10-40 mg/kg "produced a bactericidal-type effect". The authors state that patients are being given rifampicin 1500 mg once every 11 weeks with acedapsone.

C. S. Goodwin

7. **The risk of transmission from lepromatous leprosy cases under therapy**, by R. M. WORTH. *Bull. Wld Hlth Org.* 1972, **46** (6), 853-854.

Bechelli and Guinto (*Trop. Dis. Bull.* 1971, **68**, abstr. 1014), in discussing the implications of experimental infections with *Mycobacterium leprae* in the mouse footpad, said that "final proof" of the relationship between the morphological index and contagiousness could come only from a controlled study of children exposed to infection. Worth [*ibid.*, 1969, **66**, abstr. 1365] and Worth and Wong [*ibid.*, 1972, **69**, abstr. 2203] reported on such a study in Hong Kong and on the situation at follow-up after 3 years. In the present communication, the author analyses and discusses these papers in relation to the observations by Bechelli and Guinto. He concludes that "the Hong Kong study and follow-up come very close to meeting the criteria

proposed by Bechelli and Guinto for 'final proof'. It is very desirable, however, that these observations should be repeated in another population to obtain confirmation". Mention is also made of the important study by Shepard *et al.* (*Trop. Dis. Bull.* 1969, **66**, abstr. 2158) on biopsy specimens from patients with leprosy, in which it was shown that *Myco. leprae* lost their ability to multiply shortly after the start of therapy, the morphological index falling at about the same time. The closely reasoned argument should be read in the original.

F. I. C. Apted

8. **The relationship between surface temperature and dermal invasion in lepromatous leprosy,** by S. A. ANISH. *Int. J. Lepr.* 1971, **39** (4), 848-851.

The skin temperature of 12 patients with active lepromatous leprosy at Carville, Louisiana, was studied, and biopsy specimens of skin were taken from the hairy scalp, the forearm and the axilla. The Biopsy Index of Ridley (*Trop. Dis. Bull.* 1958, **55**, 525) was estimated. The temperatures of the scalp ranged from 35.2 to 37.3°C, of the axilla from 34.0 to 37.1°C, and of the forearm from 32.2 to 37.3°C. The Biopsy Index in the 3 areas was 0.17, 0.09, and 0.81, and the area with the lowest temperature apparently had the highest Biopsy Index. The author concludes that "the scalp and axilla were significantly warmer and had significantly less bacilli than the cooler forearm".

C. S. Goodwin

9. **A study of clofazimine in the rat,** by H. L. F. CURREY and P. D. FOWLER. *Br. J. Pharmacol.* 1972, **45** (4), 676-681.

Clofazimine in oil, 50 mg/kg daily, was given to rats, and control rats were fed with oil only. Widespread arthritis was induced by one injection of heat-killed *Mycobacterium tuberculosis* into a footpad. 350 mg clofazimine suppressed the arthritis while 200 mg and 100 mg produced partial suppression. The suppression lasted for 2 weeks. Inflammatory swelling of the footpad was produced by an injection of 0.05 ml Freund's complete adjuvant. Pre-treatment with clofazimine, 50 mg/kg for 7 days, produced a 40% reduction of paw swelling. Antibody response to sheep erythrocytes and the tuberculin skin test were not suppressed by clofazimine. The authors conclude that "clofazimine exhibits anti-inflammatory (but not immunosuppressive) activity, and that it should be tested in patients with rheumatoid arthritis".

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