Lepr. Rev. (1979) **50,** 99–106

Abstracts

1. SHEPARD, C. C., WALKER, L. L. & VAN LANDINGHAM, R. M. **Immunity to *Mycobacterium leprae* infections induced in mice by BCG vaccination at different times before or after challenge.** *Infect. Immun.,* Feb. 1978, Vol. 19, No. 2, 391–394.

The authors have previously reported that an intradermal injection of approximately 10^7 viable BCG given to mice about 1 month before challenge into the footpad with 5×10^3 *M. leprae*, results in effective protection. However the mechanism of this protection remained doubtful.

Either the BCG was causing macrophage activation via *BCG-recognizing* lymphocytes, which then non-specifically limited the growth of *M. leprae*, or the BCG was stimulating the development of a population of T-lymphocytes able to recognize antigens shared by the two organisms, which could then interact specifically with the antigens of the subsequently injected leprosy bacilli. Resolution of this dilemma is important, since only the latter antigen-specific mechanism is likely to give the very long-lasting protection which would be a prerequisite for a human vaccine. Thus the non-specific macrophage activation would not be expected to persist after the disappearance of the vaccinating organisms themselves.

In this paper the authors have attempted to clarify this point by varying the time of vaccination, relative to the *M. leprae* challenge. The numbers of organisms in challenged footpads were counted at 4 week intervals up to 300 days after challenge, and growth curves were plotted. There was clear protection whether the BCG was given as early as 168 days before challenge, or as late as 56 days afterwards. Only when vaccination was delayed until 91 days after challenge, was any reduction in protective efficacy seen. The authors noted that the ability of BCG to protect when given 168 days before the *M. leprae* did not, however, prove that the protection was due to the antigen-specific mechanism, since enlargement of the nodes draining the vaccination site persisted for at least 400 days. This implies the persistence of BCG, and, therefore, the possibility of continuing systemic macrophage activation, via the release of mediators from lymphocytes.

It seems likely that both of the mechanisms discussed are relevant to protection from *M. leprae* by BCG, but it is clearly exceedingly difficult to demonstrate the antigen-specific component unequivocally in the mouse model.

G. A. W. Rook

2. RIDLEY, M. J. & RIDLEY, D. S. **Surface markers on lymphocytes and cells of the mononuclear phagocyte series in skin sections in leprosy.** *J. Path.,* 1978, v. 125, 91–98.

The authors have attempted to identify the cells present in cryostat sections of lesions from the various forms of leprosy, and from other granulomatous and mycobacterial diseases. They have used a technique which is more often applied to cells in suspension than to intact tissue sections. It exploits the fact that lymphoid cells of different types can be distinguished by the presence or absence on the cell membrane of receptors for untreated sheep erythrocytes, or for the Fc portion of IgG, or for the third component (C_3).

The reagents used were

(1) untreated sheep erythrocytes, referred to as (E);
(2) erythrocytes which had been pre-incubated in a rabbit anti-sheep erythrocyte serum as a source of IgG. Such erythrocytes can attach to Fc receptors and are referred to as (EA);
(3) erythrocytes which had been preincubated in the same concentration of antiserum, and then in non-lytic mouse complement as a source of C_3. These cells were referred to as EAC. These EAC will have been able to bind to both Fc and C_3 receptors, so that only in sections where the EA *failed* to bind can one argue that adherence of EAC demonstrated the presence of C_3 receptors.

The E, EA, or EAC were allowed to settle onto fresh cryostat sections at room temperature for 1 h. The sections were then gently washed, fixed in paraformaldehyde, and stained in haematoxylin-eosin. The treated or untreated sheep erythrocytes remained attached to the sections in areas rich in the appropriate receptors.

Interpretation of the results in relation to lymphocyte subpopulations is difficult. It is generally accepted that adherence to *untreated* sheep erythrocytes is characteristic of T-lymphocytes, but may occur only if the T-cells are alive. This is in good agreement with the fact that the authors found no adherence of E, EA, or EAC to the lymphocytes found in TT or BT leprosy, or in sarcoid, and concluded that these were T-cells.

In some circumstances T-lymphocytes can carry Fc or C_3 receptors, but these are unlikely to be demonstrable by the technique used. Therefore the authors suggested that lymphocytes binding EA or EAC were B-cells. Here again difficulties arise, because as explained above the EAC used were not specific for Fc receptors. This point is critical, because although it was generally believed that B-cells carry both Fc and C_3 receptors, in which case this lack of specificity would be unimportant, it is becoming clear that peripheral blood B-cells carry C_3 receptors, but *not* Fc receptors. The latter are characteristic of a third heterogeneous group of cells, the majority of which are neither B-cells, nor T-cells, and some of which may be Killer cells. (Ref. Horwitz, D. A. *et al.* (1978), *J. Immun.* **121,** 678–684.)

Thus both EA and EAC bound to the lymphocytes shown to be paradoxically present in BL cases, and also to cells in *M. ulcerans* and atypical mycobacterial lesions. The authors consider these to be B-cells, but as discussed above, the EAC binding must be considered suspect, and EA binding may not indicate B-cells. This raises fascinating questions as to the identity of these cells, which will require further study, particularly in view of the fact that EA and EAC did *not* bind to the few lymphocytes present in LL cases.

Interpretation of the results in relation to the macrophage/epithelioid cell system is easier because giant cells, and the epithelioid cells of TT patients bound EAC strongly, but not EA, suggesting genuine C_3 receptors without Fc receptors.

Macrophages in BB, BL and *M. ulcerans* infection had both Fc and C_3 receptors (though not necessarily on the same cells) whereas epithelioid cells and giant cells in TT, BT, and sarcoid had C_3 receptors only. The foamy macrophages in LL had neither receptor. The functional significance of these changes invites exciting speculation.

In cases of ENL, or BT in reaction, EA and EAC adherence was seen in areas of polymorph infiltration around blood vessels, and in the intercellular spaces. The authors suggest adherence to immune complexes.

Further studies of this type will obviously be immensely rewarding.

G. A. W. Rook

3. **Leprosy: cultivation of the etiologic agent, immunology, and animal models.** Proceedings of the workshop on future problems in the microbiology of *M. leprae*. Scientific Publication No. 342 of the Pan American Health Organization, Washington, D.C. 20037, 1977.

This is a report on the first of three workshops on problems in the microbiology of leprosy, sponsored by the Pan American Health Organization and held at Johns Hopkins University School of Hygiene and Public Health in Baltimore, Maryland, from 12–15 October 1976. In its 74 pages which include 13 Figures and 2 Tables it deals with three major topics.

Topic I is on the problems involved in the cultivation of obligate intracellular mycobacteria, especially on the conditions that must be controlled, such as oxygen tension, oxidation–reduction potential, and temperature. A paper by Dr Lane Barksdale, of New York, questioned the assumption that acid-fast mycobacteria found in leprosy patients are a single organism, namely, *M. leprae*.

Topic II deals with immunology of leprosy, and opens with papers by Dr Quentin Myrvik of Wake Forest University, North Carolina, and by Dr Hubert Sansarricq of WHO, Geneva. Dr Myrvik discusses the role of cell-mediated immunity, interpretation of the lepromin test, the role of BCG as a prophylactic vaccine, and possible approaches to developing a specific vaccine. Dr Sansarricq describes the objectives of the WHO IMMLEP Programme: finding a test for subclinical leprosy, developing an effective vaccine, and methods of immunotherapy.

Topic III is on the subject of animal and rodent models in leprosy research, and contains papers by Dr Jacinto Convit on experience with various species of armadillo, and by Dr Charles Shepard on the role of rodents and armadillos.

The Discussions at the end of each Topic add their quota of stimulating and informative reading. No microbiologist or immunologist will fail to find something of value in this volume, and for all those who are engaged in leprosy work in general, and in leprosy research in particular, it is essential reading.

W. H. Jopling

The abstracts which follow are reprinted from the Tropical Diseases Bulletin, *June 1978, through the courtesy of the Director, Bureau of Hygiene and Tropical Diseases, London. They are classified according to subject.*

1. MICROBIOLOGY

4. RIGHTSEL, W. A., SAWYERS, M. F. & PETERS, J. H. **Comparative effects of sulfones and rifampin on growth of *Mycobacterium lepraemurium* in macrophage diffusion chamber cultures.** *Antimicrob. Agents Chemother.,* 1978, v. 13, No. 3, 509–513.

"A cell-impermeable diffusion chamber technique has been developed that lends itself to growth studies of *Mycobacterium lepraemurium.* This technique, in which the organism grows within macrophage cultures inside the chambers that are maintained on monolayer cultures of macrophages, provides a method for a strict *in vitro* evaluation of antileprosy drugs without the influence of a multiplicity of host factors. This system was used to compare the effect of three sulfone derivatives and rifampin on the growth of *M. lepraemurium* within these diffusion chamber cultures. Two sulfones, 4,4′-diaminodiphenyl sulfone and 4,4′-diacetamidodiphenyl sulfone, as well as rifampin, suppressed the growth of *M. lepraemurium*, but monoacetyl sulfone 4-amino-4′acetamidodiphenyl sulfone had no effect. The results indicate that the diffusion chamber technique can be used to evaluate the inhibitory effect of antileprosy drugs on the growth of *M. lepraemurium.* Also, the method provides for the first time a relatively rapid *in vitro* method for directly comparing the effects of drugs or their analogs when outside the metabolic influence of an animal host. This technique may be a useful tool for chemotherapy studies with other antileprosy compounds."

2. IMMUNOLOGY, PATHOLOGY

5. LEFFORD, M. J. & MACKANESS, G. B. **Suppression of immunity to *Mycobacterium lepraemurium* infection.** *Infection & Immunity,* 1977, v. 18, No. 2, 363–369.

"After injection of 10^8 live *Mycobacterium lepraemurium* (MLM) into the left hind footpad of mice, there is development of local swelling attributable to a granuloma of the cell-mediated immunity type. Concomitant intravenous inoculation of live MLM delays and may even suppress footpad swelling, the effects being proportional to the intravenous dose of organisms. Concomitant footpad infection and intravenous inoculation of 10^9 dead MLM also delays footpad swelling, but over a period of months the feet become excessively swollen. The excessive swelling is due to the local enhancement of infection as evidenced by an increase in the number of MLM per footpad. Attempts were made to prevent such immunosuppression by splenectomy or treatment with BCG. Splenectomy was entirely without effect, but 10^7 live BCG administered intravenously 2 to 4 weeks before dead MLM prevented enhancement of infection. The mediator of the immunosuppressive mechanism that results in enhanced infection remains to be elucidated, but it is unlikely to be antibody or immune complexes."

6. LEFFORD, M. J., PATEL, P. J., POULTER, L. W. & MACKANESS, G. B. **Induction of cell-mediated immunity to *Mycobacterium lepraemurium* in susceptible mice.** *Infection & Immunity,* 1977, v. 18, No. 3, 654–659.

"A mouse strain (CB6) that is highly susceptible to *Mycobacterium lepraemurium* was infected with 10^8 bacilli into the hind footpad. These mice developed cell-mediated immunity to *M. lepraemurium,* as expressed by the development of a granulomatous lesion at the site of inoculation in normal but not in T-lymphocyte-depleted mice, a proliferative response in the paracortical zone of the draining lymph node, delayed-type hypersensitivity to a sonic extract of *M. lepraemurium,* and immunopotentiation of the delayed hypersensitivity response to sheep erythrocytes. Resistance to a second challenge infection with *M. lepraemurium* was not demonstrated."

7. McADAM, K. P. W. J., ANDERS, R. F., AIKEN, G. & TAKITAKI, F. F. **Secondary amyloidosis and the serum amyloid precursor in leprosy: geographical variation and association with leukocytosis.** *Int. J. Lepr.,* 1977, v. 45, No. 2, 150–157.

"The prevalence of the amyloid-related serum component, protein SAA, was investigated in two groups of leprosy patients from different areas of Papua New Guinea. Protein SAA was more prevalent in coastal leprosy patients (49% positive) than in highland patients (21% positive). Paradoxically, many more cases of amyloidosis were diagnosed in the highland group (17 of 199) than in the coastal group (3 of 112).

"In the highland patient group, SAA was found to correlate with the leprosy disease spectrum, being more prevalent in patients toward the lepromatous pole. Borderline and tuberculoid patients who had detectable SAA usually had neurotrophic ulcers. No such relationships were observed in the coastal patient group, probably because other infections, more common on the coast, were also responsible for causing increased concentrations of SAA which is known to behave as an acute phase reactant.

"A correlation was observed between SAA positivity and neutrophil leukocytosis. This suggests that various inflammatory stimuli, such as *erythema nodosum leprosum* reactions, neurotrophic ulcers and intercurrent infections, all contribute to the prevalence of SAA in leprosy patients."

[This is a valuable paper which would repay reading in full.]

M. F. R. Waters

8. ATTA, A. G., FLEURY, R. N., MARINGONI, R. L., TRINDADE, A. S. JR, RUFINO, C. B. F. & FILHO, B. S. **Renal amyloidosis in leprosy. Functional and histopathologic studies.** *Int. J. Lepr.,* 1977, v. 45, No. 2, 158–166.

"Seven cases of renal amyloidosis secondary to lepromatous leprosy are reported. . . . One patient had only mild tubular involvement, three were in a far advanced stage and the other three were moderately affected. Five had a previous history of repeated episodes of *erythema nodosum leprosum* (ENL), three of ENL and plantar ulcers, and one of plantar ulcers without episodes of ENL. None were in active phase of ENL during the renal studies.

"Renal function was evaluated by the clearances of inulin (to measure the glomerular filtration rate), *p*-amino-hippuric acid (to measure the effective renal plasma flow) and by the maximal capacity of the tubules to raise the urine osmolarity after water deprivation.

"The patient with only slight deposits of amyloid in tubules showed excellent functional reserve. The three advanced cases presented serious impairment of the glomerular filtration rate, effective renal plasma flow and tubular capacity to concentrate the urine. The three cases with intermediate type involvement showed an increase of the filtration fraction suggesting a greater vascular involvement or this associated with a deficient capacity of the tubules to transport the dye. . . ."

9. GUPTA, J. C., DIWAKAR, R., SINGH, S., GUPTA, D. K. & PANDA, P. K. **A histopathologic study of renal biopsies in fifty cases of leprosy.** *Int. J. Lepr.,* 1977, v. 45, No. 2, 167–170.

"Renal biopsies from 50 cases of leprosy, including 45 cases of lepromatous and 5 cases of tuberculoid, have been studied in detail histopathologically with special reference to any specific leprosy lesion such as the presence of leproma or granuloma, the presence of acid-fast bacilli and the occurrence of amyloid deposit. Leproma or granuloma, acid-fast bacilli and amyloid deposit could not be detected in any of these cases. Pathologic features of nephritis of various types were seen in only 40% of cases. . . ."

10. BULLOCK, W. E., EVANS, P. E. & FILOMENO, A. R. **Impairment of cell-mediated immune responses by infection with *Mycobacterium lepraemurium*.** *Infection & Immunity,* 1977, v. 18, No. 1, 157–164.

"The effect of chronic infection with *Mycobacterium lepraemurium* upon cell-mediated immune responses was studied in Lewis rats. Rats infected for 40 to 175 days were completely protected from attempted induction of experimental adjuvant disease, and the severity of experimental allergic encephalomyelitis in leprous rats was markedly attenuated. Full manifestations of each autoimmune disease were expressed in littermate control groups. Skin homograft rejection by infected rats was significantly impaired ($P < 0.001$) as was the delayed-type hypersensitivity response to sheep erythrocytes ($P < 0.02$). It is suggested that chronic infection with *M. lepraemurium* exerts a nonspecific inhibitory effect on cell-mediated immunity by perturbation of normal lymphocyte recirculation and by induction of immuno-suppressor cell activity."

11. MELSOM, R., NAAFS, B., HARBOE, M. & CLOSS, O. **Antibody activity against *Mycobacterium leprae* antigen 7 during the first year of DDS treatment in lepromatous (BL-LL) leprosy.** *Lepr. Rev.,* 1978, v. 49, No. 1, 17–29.

"A specific radioimmunoassay was developed for demonstration and quantitation of antibodies against *Mycobacterium leprae* antigen 7 which cross-react extensively with a similar antigen in many species of mycobacteria, including BCG-antigen-60.

"The antibody activity against *M. leprae* antigen 7 showed only a slight tendency to decrease in 15 patients with lepromatous leprosy during their first year of treatment with dapsone associated with marked clinical improvement."

12. McDOUGALL, A. C. **The work of the Leprosy Study Centre in London: a review over 13,000 biopsies.** *Proc. R. Soc. Med.,* 1977, v. 70, No. 10, 731–732.

That leprosy has taken its rightful place in the main stream of medical research is due in no small measure to R. G. Cochrane, whose scientific approach is evident, not only in his writings but in the incomparable collection of histological material matched by clinical records at his consulting rooms in London. It was his dream that this should form the nucleus of an international focal point of leprosy study, and the Leprosy Study Centre is the fulfilment of that dream. Standards of excellence in patient care, in training and in histopathology have given the Centre a high reputation. Biopsy material has been sent from many parts of the world, and now, in magnitude, range and in detailed records the histological collection is unique.

This article, the result of much careful study of the great wealth of material, concentrates more on the results than on the techniques used, already well described by Harman (*Lepr. Rev.,* 1975, v. 46, 125). Unusual aspects of differential diagnosis are mentioned, as is the value of serial sections in indeterminate cases. There is particular reference to microfilariasis [see *Trop. Dis. Bull.,* 1977, v. 74, abstr. 2792] and the contribution of the Centre to elucidating exit routes of leprosy bacilli from the body.

T. F. Davey

3. CLINICAL

13. PALANDE, D. D., DE SEVERY, C. & RAJAGOPALAN, M. S. **Plantar ulcers with osteomyelitis underneath. A bacteriological study.** *Lepr. India,* 1977, v. 49, No. 3, 322–329.

"Thirty-nine consecutive cases of plantar ulcers with underlying chronic osteomyelitis admitted in the Sacred Heart Hospital during 1975/76 were studied for the infecting organisms and their sensitivity to easily available antibiotics. A single organism was isolated in only 10 cases, the infection in the rest being a mixed one. The commonest organisms were *Staphylococcus, Streptococcus* and *Proteus mirabilis.* In a few cases *Pseudomonas* and *E. coli* were also isolated. Chloramphenicol was the most effective antibiotic in general and streptomycin the least. 70% of the staphylococcus strains isolated were found to be resistant to penicillin. Empirical use of ntibiotics especially penicillin and streptomycin is hence deprecated."

14. RAI, V., SINGH, G., SINGH, R. H. & UDUPA, K. N. **Blood histamine and histaminase in leprosy patients — a short communication.** *Indian J. Med. Res.,* 1977, v. 66, No. 6, 978–982.

"In 91 patients of different types of leprosy, blood levels of histamine and histaminase were studied and compared to matched normal controls. The leprosy patients showed markedly raised levels of both histamine and histaminase as compared to controls. This rise was more pronounced in cases of leprosy with a history of longer duration. Patients of leprosy in reaction showed the maximum levels whereas tuberculoid, borderline and lepromatous cases showed moderate levels and others minimum changes."

[See *Trop. Dis. Bull.,* 1975, v. 72, abstr. 511.]

15. HUIKESHOVEN, H. *et al.* **Demonstration of dapsone in urine and serum by ELISA inhibition.** [Correspondence.] *Lancet,* 1978, Feb. 4, 280–281.

The authors produced anti-dapsone antibody in rabbits, and showed that it could be used to detect dapsone by a micro-scale enzyme-linked immunosorbent assay (micro-ELISA). They coated the wells of polystyrene microtitre trays with a dapsone-haemocyanin conjugate, added the anti-dapsone antibody serum so that it reacted with the dapsone, and then detected the antibody by the addition first of anti-rabbit IgG antiserum conjugated to horseradish peroxidase, and then of an amino-salicyclic acid/H_2O_2 solution, whereupon a brown colour developed. When diluted urine or serum containing dapsone was initially added to the coated wells, followed by the anti-dapsone antibody, immunosorption of the latter to the walls of the wells was completely inhibited, so that on completion of the test no brown colour developed (test of inhibition of ELISA (ELISIT) .

From the published results, it is claimed that dapsone solutions containing as little as 0.3 µg/ml could be detected with ELISIT, and that positive and negative solutions could be clearly distinguished when diluted 1/100. Full technical details are available on request to the senior author at the Department of Tropical Hygiene, Royal Tropical Institute, Amsterdam.

[If the authors' results are confirmed, the method will be considerably more sensitive and more specific than the Bratton-Marshall technique. However, although it is claimed that the method is simple enough to be performed in many tropical laboratories, the questions of cost and of the shelf-life of the reagents under tropical conditions are not discussed.]

M. F. R. Waters

16. HUIKESHOVEN, H. & BIJLEVELD, I. **Encouraging results from DDS urine analysis among registered leprosy patients in the Wangas, Kenya: an exception that challenges the rule.** *Lepr. Rev.,* 1978, v. 49, No. 1, 47–52.

"From previous research among the Wangas (Kenya), it appeared to be the standard of medical services, and in particular the leprosy fieldworker's approach, rather than sociocultural factors, which accounts for failure of leprosy control.

"The present investigation adds weight to these findings. Urine samples were taken from 39 patients of one highly reputable leprosy fieldworker, and analysed for DDS/creatinine ratios. Comparison with data from elsewhere demonstrates their scrupulousness in weekly DDS-taking at home."

17. BEDI, T. R., KAUR, S., SINGHAL, P. C., KUMAR, B. & BANERJEE, C. K. **Fatal proliferative glomerulonephritis in lepromatous leprosy.** *Lepr. India,* 1977, v. 49, No. 4, 500–503.

Acute glomerulonephritis is a well known manifestation of ENL reaction in lepromatous leprosy but is usually a transient and self-limiting condition, and this case is reported because of the rarity of a fatal outcome. An Indian male aged 50 years, suffering from lepromatous leprosy, had received irregular treatment with dapsone for 2 years and during this time had experienced a number of ENL reactions which were treated with salicylates and prednisolone. He was admitted to hospital in Chandigarh (Punjab) with signs of renal failure and poorly controlled leprosy, and renal biopsy revealed acute proliferative crescentic glomerulonephritis. He died of uraemia 8 weeks after admission, and post-mortem examination confirmed the biopsy finding. Immunofluorescent studies showed deposition of immune complexes at the glomerular sites.

W. H. Jopling

18. SINHA, S. N., GUPTA, S. C. & BISHT, D. **Serum calcium and magnesium in different types of leprosy.** *Lepr. India,* 1978, v. 50, No. 1, 54–56.

"Serum calcium and magnesium were studied in 200 leprosy patients and 25 apparently healthy individuals. Serum calcium was found to be significantly decreased in all types of leprosy except tuberculoid. The decrease in serum magnesium was highly significant in tuberculoid, lepromatous and borderline lepromatous cases."

19. GANAPATI, R., REVANKAR, C. R., CHRISTINA & ROMANO. **Associated cases in the families of school children with leprosy.** *Lepr. Rev.,* 1978, v. 49, No. 1, 43–46.

"The screening of 190 families in which children suffering from leprosy discovered through school surveys were present, yielded a total of 41 cases. Though the prevalence rate among the contacts was 44 per thousand, only in 14% of the families visited, another associated case could be found, and only in 2 instances out of 27 families, the associated case belonged to **L** type. The school surveys as well as contact examination yielded predominantly cases belonging to non-lepromatous type mostly with single lesions whose contribution to the pool of infection in the community is questionable."

20. MALIK, R., KHANDPUR, R., CHANDRA, K. & SINGH, R. **A clinicopathological study of 244 cases of leprosy with special reference to histoid variety.** *Lepr. India,* 1977, v. 49, No. 3, 400–405.

The authors give brief details of 8 cases of histoid leprosy diagnosed amongst a total of 60 patients with lepromatous leprosy who were studied clinically and histopathologically between 1972 and 1976. Six of the 8 were treated with dapsone and all save 1 patient showed an initial response to the drug, although details of their regimens are not given. Therefore, they conclude that drug resistance is not a significant factor in the pathogenesis of histoid leprosy. However, it is generally agreed that histoid leprosy is usually related to relapse of treated lepromatous leprosy, and this report does not exclude either relapse through failure to take treatment or relapse due to partial (low grade) dapsone resistance.

M. F. R. Waters

21. SINGHAL, P. C., CHUGH, K. S., KAUR, S. & MALIK, A. K. **Acute renal failure in leprosy.** *Int. J. Lepr.,* 1977, v. 45, No. 2, 171–174.

"Three patients having lepromatous leprosy developed acute renal failure. Two patients completely recovered and one was left with a moderate degree of renal insufficiency. Renal tissue obtained by percutaneous biopsy revealed acute tubular necrosis in two and diffuse crescentic glomerulonephritis in the third case."

4. THERAPY

22. SHESKIN, J. **Study with nine thalidomide derivatives in the lepra reaction.** *Int. J. Derm.,* 1978, v. 17, No. 1, 82–84.

"In our studies, 3 out of the 9 thalidomide derivatives used to treat lepra reaction of lepromatous leprosy were effective. All 3 are known to be teratogenic in animal studies. This suggests that the teratogenic and the lepra reaction suppressive properties may be related."

5. EPIDEMIOLOGY

23. WHITE, S. J., STONE, M. M. & HOWLAND, C. **Genetic factors in leprosy: a study of children in Uganda.** *J. Hyg.,* Cambridge, 1978, v. 80, No. 2, 205–216.

"A group of 20,990 children in Uganda was examined for leprosy over a period of 8 years. There was no evidence that the incidence of leprosy varied according to a child's genetic relationship to a leprosy patient, once allowance had been made for the grade of physical contact."

24. DÍAZ ALMEIDA, J., FERNANDEZ BAQUERO, G., MENÉNDEZ GARCIA, V. G., SAGARO DELGADO, B., MUÑOZ, H. & TOLEDO, G. Estudio clinico-epidemiológico de los enfermos ingresados en el hospital "El Rincón". **[Clinicoepidemiological study of (leprosy) patients admitted to the "El Rincón" hospital.]** *Revta Cub. Med. Trop.,* 1976, v. 28, No. 3, 143–155. English summary (5 lines).

Data were collected under the following headings in response to a questionnaire to be completed in respect of 207 leprosy patients: province and area of birth; age-groups; civil status (single, married, etc.); sex distribution, generally and in relation to clinical disease forms [lepromatous (179 cases), tuberculoid (27)]; colour; domicile of relatives (urban or rural); habitual residence of patients; whether working or not and type of employment; time of diagnosis of leprosy (before 1950 to after 1970); bacillary status of patients; Mitsuda test results; previous use of BCG vaccine; previous incidence of pulmonary tuberculosis; results of screening; analysis of initial symptoms; localization of primary symptoms; previous contact with leprosy sufferers; family and other contacts before contracting the disease; and time of contact with a possible source of infection. The survey has been conducted to study clinical symptoms and possibilities of epidemic.

M. de O. Tollemache