may be available in the field and, when available, may not differentiate the nerve dysfunction due to a lepra reaction from that caused by the HIV in a patient infected with both *M. leprae* and HIV. The only direct confirmation of the fact that the HIV is responsible for the neuropathy will be by the demonstration or culture of the virus.

Another problem in these patients will be the treatment of lepra reactions. Systemic corticosteroids which are often needed for the control of reactions may have an adverse effect on the course of HIV infection by causing immunodeficiency.

The possible interaction of *M. leprae* and HIV in the peripheral nerves is a distinct possibility which will require careful study and follow up of the patients who may get infected with both of these agents.

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Quantitative Relationship Between Anti-PGL-I-specific Antibody Levels and the Lepromin Reaction

To the Editor:

The discovery the that phenolic glycolipid (PGL-I) from *Mycobacterium leprae* ([3]) was a species-specific antigen ([4, 9]) was one of the most important discoveries made in the last decade in the field of mycobacterial diseases, and it stimulated the already successful search for species-specific glycolipid antigens in other important human pathogens, notably *M. tuberculosis* ([1, 2, 5]). Although many studies were devoted to the PGL-I antigen (reviewed in [2]) and reasonable correlation was established between anti-PGL-I antibody titers and bacterial loads (bacterial index, BI [7]), to our knowledge an investigation to verify if there were quantitative relationships between antibody titers and the intensity of lepromin reactions has escaped the attention of previous workers.

For the purpose of this study, sera were collected from 53 first-time leprosy patients. After the blood was collected, 0.1 ml of lepromin-H was injected intradermally and the lepromin reaction was measured as millimeters of induration 21 days later. The patients were subsequently classified according to the Ridley-Jopling scheme ([6]). The sera were tested using ELISA, and IgG and IgM levels were scored as the absorbance obtained using a 1/250 dilution; the ELISA method was as described before ([1]). The lepromin test, ELISA, and the classification of the patients according to the Ridley-Jopling scheme were done blind by independent workers.

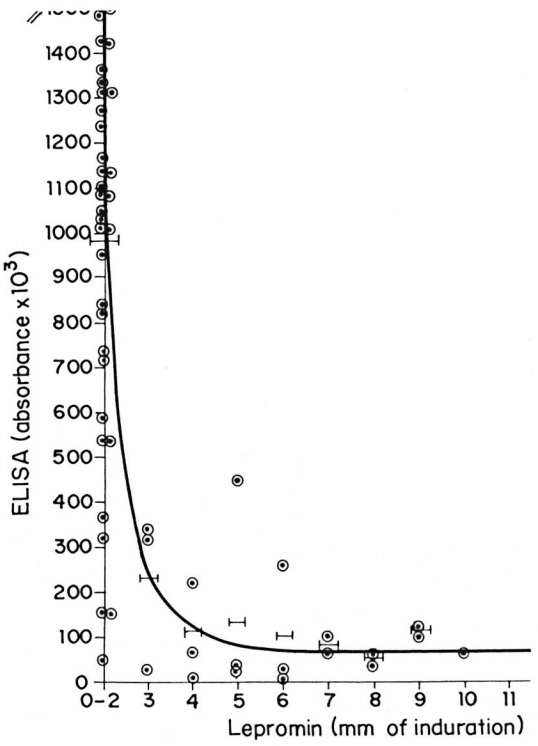
The quantitative relationships between the anti-PGL-I antibody levels and the lepromin reaction are shown in The Figure. The data neatly divided the patient population into three major groups: a) patients with a negative lepromin test (0–2 mm induration but antibody levels above 500); b) patients with a positive lepromin test (more

THE TABLE. *Relationship between the Ridley-Jopling classification and the lepromin/ ELISA classification of leprosy patients.*

Lepromin/ ELISA	Ridley-Jopling[a]						Total
	LL	BL	Ind	BB	BT	TT	
LL	21	2	1	1	0	0	25
Intermediate	1	5	2	2	4	5	19
TT	0	0	0	0	0	9	9
Total	22	7	3	3	4	14	53

[a] LL = lepromatous; BL = borderline lepromatous; Ind = indeterminate; BB = midborderline; BT = borderline tuberculoid, TT = tuberculoid.

than 6 mm of induration and antibody levels less than 150); and c) patients in an intermediate position in the two scales (either lepromin negative but with antibody levels below 500; or lepromin reactions between 3 and 6 mm with antibody levels below 150; or lepromin reactions between 3 and 6 mm with levels between 150 and 500). From the practical point of view it would therefore appear that the first category of cases were the truly lepromin-negative (LL cases), the second category were the truly lepromin-positive (TT cases), and the third category (intermediate) should include all borderline leprosy cases.



THE FIGURE. Quantitative relationships between ELISA (absorbance × 10^3) and the lepromin reaction (mm of induration).

When the patients classified according to the Ridley-Jopling scheme were arranged in respect to the corresponding lepromin/ ELISA data (The Table), good agreement was found between the two classifications. Therefore, the neat and simple quantitative relationship found in this study indicates that anti-PGL-I antibody assays may be useful for the classification of leprosy.

From the immunological point of view, the data indicate that *M. leprae* antigens must be equally well processed by macrophages at both ends of the leprosy spectrum, which contradicts the generally accepted hypothesis that macrophages in lepromatous patients carry a *M. leprae*-specific defect (reviewed in [8]). We propose that the simplest explanation to the observed relationship is to hypothesize that for each B-cell clone there must be a corresponding T-cell clone, their ratio being strictly regulated, and that during the protracted subclinical disease whichever of the clones is primed to proliferate the other must be repressed.

Finally, from the viewpoint of serodiagnosis, the observed relationship supports a previous conclusion ([2]) according to which the sensitivity of serological diagnosis in tuberculosis and leprosy using purified species-specific antigens must be necessarily low since it is dependent upon the immunopathogenesis in these mycobacterial diseases.

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Circulating Antispermatozoal Antibodies in Leprosy

To the Editor:

The presence of autoantibodies in leprosy, especially in lepromatous leprosy, has been widely investigated. Autoantibodies have been found against almost all body tissues, cellular and nuclear material, and immunoglobulins ([8]).

Autoantibodies reacting with testicular germinal cells and spermatozoa have been reported in both tuberculoid and lepromatous patients ([7, 9]). Antibodies may be formed because of antigenic similarity between *Mycobacterium leprae* and testicular tissue or due to an adjuvant-like action of *M. leprae*. Various studies have given widely varying figures, probably because of the various techniques employed to check the presence of antispermatozoal antibodies (ASA).

The present study was undertaken keeping in mind the widely variable results and tests employed not permitting a scientific comparison.

Subjects and serum samples. Sera from 30 healthy men of proven fertility and 68 male leprosy (bacillary-positive BL, LL) patients were obtained. The duration of disease varied from 1 to 14 years. None of the patients had or gave a history of erythema nodosum leprosum in the recent past, reasonably confirmed by taking a relevant history and asking leading questions.

Blood samples were collected in vacuum tubes, centrifuged at 1500 × *g* and the sera were separated and frozen at −70°C within 3 hr of collection. Decomplementation of the sera was done before performing the sperm agglutination test (SAT^4) and the sperm immobilization test (SIT^3). A titer of ≥ 1:8 was considered positive for the SAT. A sperm immobilization value of > 2.0 constituted a positive SIT result.