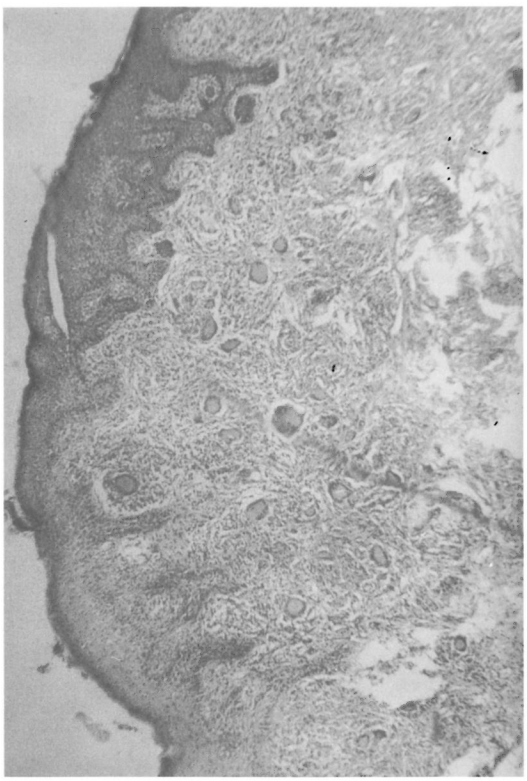

FIG. 2 Histology showing epithelioid cell granulomas in dermis with many giant cells (hematoxylin-eosin ×100).

involvement of the genitalia in leprosy ([2, 3, 5]). Arora, *et al.* ([2]) in their study on 450 males with leprosy reported genital lesions in 2.9% of their patients; the lesions were seen in borderline, borderline lepromatous, and lepromatous cases. Parikh, *et al.* ([3]) described scrotal and penile lesions in six patients with borderline leprosy (BT-BL). These reports suggest that lesions on genitalia are not so uncommon as believed. However, to the best of our knowledge, lesions occurring on the prepuce involving the mucosal aspect have not been reported earlier. Under-reporting of these patients is due either to reluctance on the part of patients to expose or the physician to examine the genitalia. Our patient was unaware of his skin lesion, and the lesions were detected only after a thorough physical examination.

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Investigation of Anti-*Mycobacterium leprae* Antibodies in Leprosy Patients' Sera by an A60 Antigen Immunoassay

TO THE EDITOR:

Antigen 60 (A60) described by Cocito and Van Linden ([5]) in 1986 is a cytoplasmic antigen purified from *Mycobacterium bovis* BCG.

Several authors, including Ladron de Guevara, *et al.* ([6]) and Sanchez Monton and Martin Luengo ([7]), have employed A60 in the serodiagnosis of leprosy with different results.

The object of our study, in light of the limited existing studies on leprosy with A60 and given the relative important number of cases in our community, is to demonstrate the presence of immunoglobulin G (IgG) against A60, as well as the possible utility of this technique in the diagnosis of leprosy.

A total of 121 leprosy patients were studied and classified according to the Riddley-Jopling criteria as follows: 90 lepromatous leprosy (LL), 7 borderline lepromatous (BL), 6 borderline borderline (BB), 1 borderline tuberculoid (BT) and 17 tuberculoid (TT) leprosy. The study also included 23 immediate family members. Neither patients nor healthy contacts included in our study had been previously vaccinated for tuberculosis. Those individuals included as healthy contacts and leprosy patients had

tion or active disease. The majority of the sera studied were from Cordoba and its province. The Sanitorium of San Francisco de Borja of Fontilles supplied 10 sera which had come from other provinces in Spain.

An indirect ELISA technique using the commercialized Anda-Tb test kit ([4]) was employed in our study.

Of the 90 LL patients, 43% were positive, 15% intermediate, and 42% negative. Of the 7 BL, 71% were positive and 29% intermediate. Of the 6 BB, 50% were positive, 33% intermediate and 17% negative. The only BT patient was negative. Of the 17 TT, 18% were positive, 6% intermediate and 76% negative. We found significant statistical differences in the proportion of positivity in the different clinical forms of leprosy ($p < 0.01$).

With respect to the detection of IgG in the 23 family members of leprosy patients, 16 were negative, 3 intermediate and 4 positive (of which one had high levels of positivity).

The ELISA was positive in 41% of the patients and in 17% of the family members. There was an intermediate response in 15% of the patients and 13% of the family members; while 44% of the patients and 70% of the family members had negative responses. These results were statistically significant ($p < 0.001$).

In a study by Ladron de Guevara, *et al.* ([6]), 20.8% of the clinically healthy contacts had antibodies against A60, with 12.5% being very positive. This level of positivity is very similiar to that observed by other authors such as Buchanan, *et al.* ([3]) in 1983 against phenolic glycolipid-I (PGL-I) in family members of patients in Mexico, in which leprosy is as endemic as in Spain. In highly endemic countries the levels are higher ([1, 2]).

We observed also a direct relationship between antibody levels and bacillary load ($p < 0.0l$).

We conclude that the detection of IgG against A60 by the ELISA could be useful as a complementary test in the diagnosis of leprosy, especially in suspected cases of lepromatous leprosy. Independent of the clinical form, a direct relationship exists between the presence of anti-IgG antibodies against A60 and the bacillary load. A prospective study on the evolution of family members with an immunologic status of lepromatous leprosy (Mitsuda negative and ELISA positive) would be of value in that family members are at high risk of developing the disease.

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Antibodies to Phenolic Glycolipid-I and Sulfatide-I in Leprosy and Tuberculosis

To the Editor:

Following the discovery of phenolic glycolipid-I (PGL-I) by Hunter and Brennan ([11]) in *Mycobacterium leprae*-infected tissues and once being established that the material was specific to *M. leprae* ([12]), several assays for the detection of that lipid antigen have been developed with the intention of applying them in the serological diagnosis of leprosy ([5, 18]) to identify those household contacts with an incipient disease ([4]) and to monitor the response of the patients subjected to chemotherapy ([2]). A similar glycolipid has been isolated and characterized from *M. tuberculosis* by Daffe, *et al.* ([7]) and has been used by some authors for the serological diagnosis of tuberculosis with variable results ([3, 15, 16]).

In this study, we measured the reactivity of the sera from 34 tuberculous patients, 33 patients with lepromatous leprosy, and 38 healthy individuals to PGL-I of *M. leprae* and to sulfolipid-I of *M. tuberculosis* H37Rv. Each lipid has been considered to be species-specific, and in the case of PGL-I, this specificity has been the basis for its use as an antigen for the serological diagnosis of leprosy. Although a similar consideration of specificity has been given to the sulfolipid-I (sulfatide-I, SL-I) of *M. tuberculosis,* its use as an antigen for the diagnosis of tuberculosis has not been a common practice, perhaps because of the more extensive information on protein antigens ([1, 9, 14]) and other lipids ([6–8]). PGL-I was isolated from *M. leprae*-infected armadillo tissue by the techniques of Vemuri, *et al.* ([17]) and Hunter, *et al.* ([13]). SL-I was purified from *M. tuberculosis* H37Rv using the method of Goren, *et al.* ([10]). Although the patients studied were under treatment at the time of sampling and most leprosy patients were old multitreated cases, all of the patients still had active disease. Patients and control groups included both male and female individuals whose ages ranged from 16 to 72 years.

Antibodies to the mycobacterial lipids were measured using an enzyme-linked immunosorbent assay (ELISA) adapted for lipid antigens. From the results, it could be concluded that: a) lepromatous (LL) sera and tuberculous (Tb) sera contain similar amounts of IgG antibodies to PGL-I [0.154 ± 0.101 (mean OD 492 nm of triplicates ± S.D.) in LL vs 0.104 ± 0.052 in Tb, $p = 0.5$]; b) LL sera contain higher levels of IgM antibodies to PGL-I than Tb sera (0.164 ± 0.227 vs 0.046 ± 0.035, respectively; $p = 0.01$); c) LL sera and Tb sera show similar amounts of IgG antibodies to SL-I (0.144 ± 0.072 vs 0.096 ± 0.050, $p = 0.5$); d) LL sera and Tb sera contain similar, very low amounts, if any, of IgM antibodies to SL-I (0.019 ± 0.034 vs 0.026 ± 0.020, respectively; $p = 0.5$), and e) although low, the levels of IgG and IgM antibodies to PGL-I and to SL-I in LL and Tb sera were still higher than those levels in the control group, with the numerical values not always reaching statistical significance.