rived, antigens was seen on X-ray films after long exposures. Also, no leprosy serum contained antibodies capable of neutralizing the HTLV-III_B, Ma or RF isolates of HIV-1 in the cytopathic effect inhibition assay ([4]). Similarly, all control sera from Agra and Surabaya regions were negative for anti-HIV antibodies.

Since mycobacterial infections are not unusual in HIV-infected persons ([2]), a possible explanation for the discrepant results could be that ShivRaj and colleagues, in fact, observed dual (HIV-1 plus mycobacterial) infections in their cohort of patients. It is also conceivable that these authors observed reactions with crossreacting non-HIV antigens, possibly associated with the virus-producing cell line. This latter explanation is supported by the fact that some of the leprosy sera we tested contained antibodies reacting with antigens present in the lysates of uninfected H9 cells. In any case, we conclude on the basis of our data that sharing common determinants antigenic for humans is not a characteristic feature of HIV-1 and *Mycobacterium leprae.*

—Miroslav Malkovský, M.D., Ph.D., M.R.C.Path.

Senior Scientist
Xenova Ltd.
545 Ipswich Road
Slough
Berkshire SL1 4EQ, England

—Paula Dilger, B.Sc.

Research Technician
NIBSC

—Robin Thorpe, Ph.D.

Head, Division of Immunobiology
NIBSC
Blanche Lane, South Mimms
Potters Bar
Hertfordshire EN6 3QC, England

Acknowledgment. We thank Dr. Juraj Ivanyi (MRC Tuberculosis Unit, Hammersmith Hospital) for proposing this investigation and for the supply of serum samples from previous serological studies [Lepr. Rev. **59** (1988) 195–199; Lancet **1** (1987) 1098].

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Serum Ferritin in Lepra Reactions

To the Editor:

During an inflammatory process in the body, there is a fall of serum iron levels and a release of various acute phase reactants ([4]). Ferritin behaves as an acute phase reactant, and it is a measure of the body's iron stores ([5]). Hyperferritinemia has been observed in inflammatory conditions, such as Hodgin's disease, carcinomas, osteomyelitis, tuberculosis ([1]), and AIDS ([5]). Serum iron and ferritin levels were comparable in lepromatous patients and control subjects ([9]), while their levels were remarkably decreased in lepromatous patients co-infected with pulmonary tuberculosis ([12]). Here we report the serum ferritin levels in type 1 and type 2 lepra reactions. These complications are often encountered even after the intro-

TABLE 1. *Serum ferritin levels in normal subjects and leprosy patients.*

Group	Serum ferritin μg/l mean ± S.D. (N) (range)	
	Males	Females
I. Normal controls[a]	96.7 ± 72.66 (10) (44–300)	43 ± 30.79 (10) (14–120)
II. Lepromatous leprosy patients[a]	120.8 ± 91.34 (10) (26–380)	54.8 ± 31.96 (10) (13–125)
III. Lepra reaction patients		
a) Type 1 (at onset)	187.33 ± 93.69 (9) (76–360)	322.2 ± 313.62 (5) (76–850)
b) Type 1 (at remission)	269.44 ± 237.3 (9) (30–650)	552 ± 299.6 (5)[b] (180–930)
c) Type 2 (at onset)	643.5 ± 425 (10)[c] (85–1400)	392 ± 379.12 (5) (115–900)
d) Type 2 (at remission)	169.7 ± 143.9 (10) (2.0–440)	247 ± 312.4 (5) (55–800)

[a] Rao, *et al.* 1987 (9).
[b] Significantly higher than female lepromatous leprosy patients, $p < 0.01$.
[c] Significantly higher than male lepromatous leprosy patients, $p < 0.01$, and male type 2 reaction patients at remission, $p < 0.001$.

duction of multidrug therapy (MDT) in India (15).

We have measured serum ferritin levels by radioimmunoassay (9) in leprosy patients at Singhbhum Navjivan Niketan, Bihar; Gandi Memorial Leprosy Foundation, Wardha; and the Dermatology Department of Maulana Azad Medical College, New Delhi, India. Age- and sex-matched controls from the same areas were also included for comparison. Twenty patients (male and female) with nonreactional lepromatous leprosy (10) with a mean age of 40.5 years (Table 1) and another 29 patients of both sexes with type 1 and type 2 lepra reactions (7) (Table 2) were studied. The duration of illness in all of these patients varied from 3 months to 25 years. In the type 1 reaction cases, there were 1 upgrading BL, 1 BT and 2 BB downgrading cases; the remaining 10 cases were either unstable or uncertain. The clinical details of patients in lepra reaction are given in Table 2. The control group consisted of 20 normal male and female subjects, with a mean age of 36.5 years. All patients received MDT (15). The patients in lepra reaction were managed with steroids, clofazimine, chloroquin and/or aspirin (8). Paired serum samples were collected from the reactional patients, one at onset prior to the antireaction therapy and the other at clinical remission of the reaction.

The serum ferritin levels in the different groups of subjects are given in Table 1. The differences in mean serum ferritin levels between males and females in both the normal controls (Group I) and lepromatous patients (Group II) were not significant ($p > 0.1$). However, in the males the mean serum ferritin levels were twice that found in the females. None of the control subjects or nonreactional lepromatous patients had a deficiency of serum ferritin (< 12 μg/l).

At the onset of type 1 reaction, only 4 of 14 (29%) cases showed elevation of serum ferritin levels; at the start of type 2 reaction, 11 of 15 (73%) cases demonstrated elevated levels. Values > 320 μg/l in the males and > 125 μg/l in the females, i.e., the upper limits of nonreactional lepromatous patients, were taken as raised (Table 1). At the onset, the rise of the mean serum ferritin level was more in type 2 than in type 1 lepra reaction. However, this difference is not significant. At the clinical remission of type 1 reaction, the serum ferritin levels declined to the normal range in 1 BL case and 1 BB case while, curiously, in 5 other cases showing no rise at onset, the levels were raised (Table 2). At the clinical subsidence of type 2 reaction, only four patients still had elevated serum ferritin levels. The fall of the mean serum ferritin level at remission of the type 2 reaction was remarkable (Table

1), its level being significantly less than that at onset ($p < 0.001$).

Reactions in leprosy are consequent to the disregulation in the immunological homeostasis. Differentiation of upgrading and downgrading reactions is possible histologically, provided a previous biopsy report is available ([2]), which is not always the case. In our previous studies, we had tried to differentiate type 1 and 2 lepra reactions by studying the levels of serum cortisol ([15]) and serum-β-2-microglobulin ([11]) and also by the solubilizing capacity of preformed immune complexes (an indicator of complement activation) by the sera of lepromatous patients ([3]), at the clinical onset and remission of lepra reactions. Some overlapping of these parameters prompted us to search further for means of delineating type 1 and 2 lepra reactions by laboratory tests.

We found that a larger number of cases (73%) showed a sudden and spectacular elevation of serum ferritin levels at the onset of the type 2 reaction and demonstrated an accelerated fall at remission in most cases. On the other hand, in type 1 reaction only 29% of cases showed elevation at the onset and, surprisingly, at remission 50% of the cases showed elevated levels. The rise in the serum ferritin levels during reaction is perhaps due to damage to reticuloendothelial cells, leading to the release of stored ferritin into the circulation. This indicates that the possibility of acute systemic involvement and tissue destruction may be more common in type 2 lepra reaction, which is quickly extinguished by antireactional drugs. To the contrary, type 1 reaction might be a localized immunologic struggle, which perhaps gradually intensifies. However, very high levels of serum ferritin in some cases of type 1 reaction (Table 2) possibly point to immunological instability and a gradual worsening or downward shift of cellular immunity in these patients. The fall of serum ferritin at the remission of type 2 reaction suggests a settling of the immunoinflammatory processes. It may be further noted that only 4 out of 15 patients with type 2 reaction had minimally elevated serum ferritin levels. These patients had perhaps milder Gell and Coombs type III hypersensitivity, with marginal systemic involvement. To the contrary, hyperferritinemia at the onset of type 1 reaction indicates a Gell and Coombs type IV hypersensitivity reaction at the site of the lesions. The increased level of serum ferritin in 7 out of 14 cases of type 1 reaction at clinical remission either indicates a downward shift of the cellular immunity or, alternatively, a relapse. Leprologists are aware of the difficulty in distinguishing relapse in paucibacillary patients from late reversal reaction ([14]).

Patients near the lepromatous end of the

TABLE 2. *Serum ferritin levels (μg/l) in 29 reactional patients at onset and at clinical remission of type 1 and type 2 lepra reactions.*

Age/Sex (yr)	Leprosy type	Serum ferritin[a] At onset	Serum ferritin[a] At clinical remission
Type 1 lepra reaction (N = 14)			
45/M	BL	360 (I)	30
45/M	BT	140	120
30/M	BT	110	55
45/M	BT	80	110
35/M	BL	210	210
50/M	BL	240	650 (II)
17/M	BL	250	380 (I)
40/M	BT	220	230
30/M	BB/BL[b]	76	640 (II)
45/F	BB	390 (III)	180
45/F	BB	850 (III)	950 (III)
40/F	BB	105	340 (III)
40/F	BB	76	640 (III)
20/F	BB	240 (II)	650 (III)
Type 2 lepra reaction (N = 15)			
21/M	LL	760 (II)	170
?/M[c]	LL	1400 (III)	180
30/M	LL	1000 (III)	440 (II)
?/M[c]	LL	320 (I)	50
40/M	LL	1100 (III)	120
50/M	LL	390 (I)	400 (I)
40/M	LL	35	20
55/M	LL	550 (I)	85
25/M	LL	650 (II)	180
50/M	LL	180	52
19/F	LL	700 (III)	180 (II)
?/F[c]	LL	120	55
40/F	LL	900 (III)	800 (III)
40/F	BL	115	100
25/F	BL	125 (I)	100

[a] Underlined figures indicate raised levels of serum ferritin. Roman numerals in parentheses show grade of rise of serum ferritin levels: I = less than twice the upper range in lepromatous patients without reaction; II = more than twice upper range and less than three times the upper range; III = more than three times upper range.

[b] Unstable.

[c] Age not known accurately.

leprosy spectrum may also undergo overlapping type 1 and 2 reactions. Thus, the complexity of the clinical picture may make the distinction between these two types of reaction difficult ([7]). We had two such patients grouped in type 2 reaction in our series. Admittedly, these cases could not be differentiated by serum ferritin estimations.

So far as we know, until now there is no laboratory method except histology to gauge the severity of lepra reactions. Although the elevation of the serum ferritin level in lepra reactions is an indicator of nonspecific inflammatory processes, our preliminary data warrant a further in-depth study on the rise and fall of this acute phase reactant during lepra reaction.

—Kunal Saha, M.Sc., M.B.B.S.,Ph.D.
K. N. Rao, Ph.D.
Avind Kashyap, M.Sc.

Immunology Department
Vallabhbhai Patel Chest Institute
Delhi 110007, India

—V. N. Sehgal, M.D., F.N.A.Sc.
S. K. Agrawal, M.D.

Department of Dermatology and Medicine
Maulana Azad Medical College
New Delhi 110002, India

Acknowledgment. The authors are grateful to the Indian Council of Medical Research, New Delhi, for financial assistance.

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Interleukin-2 Receptors in the Sera of Leprosy Patients

To the Editor:

A variety of immunological changes occur in borderline tuberculoid leprosy progressing toward the lepromatous pole ([4]). A particular deficit in the number and functions of T cells has been seen in step-ladder fashion from the tuberculoid to the lepromatous end. On the other hand, B-cell ac-