CORRESPONDENCE

This department is for the publication of informal communications that are of interest because they are informative and stimulating, and for the discussion of controversial matters.

Bacteriuria, Chronic Pyelonephritis and Leprosy

To the Editor:

We would like to comment on two subjects which have figured in leprosy journals in recent years, namely bacteriuria (7-9, 11) and chronic pyelonephritis (2). In regards to bacteriuria, we can see no reason why this should be any more common in leprosy patients than in the general population from which they are drawn. During the period 1968-1971, we examined 229 adult leprosy patients attending the Leprosy Outpatient Clinic at this hospital and found bacteriuria in only 11 (4.8%). These 11 patients had normal renal tracts on radiological examination. There was no significant difference between lepromatous and nonlepromatous patients in regards to the incidence of bacteriuria and, as many of the 229 patients were middle-aged or older and there were almost as many females as males, we doubt if our figure of 4.8% can be considered abnormal (6).

On the question of chronic pyelonephritis, we would first of all ask for a definition of the condition. On this subject a recent Leading Article (5) in the British Medical Journal had this to say:

"The term 'chronic pyelonephritis' has been used in various senses, and it is difficult to define clearly a disease denoted by it. . . . The diagnosis of chronic pyelonephritis presents formidable difficulties. Beeson (1) considers that there are no direct symptoms, and that physical findings likewise are normal except for those caused by renal insufficiency. Microscopical examination of formed elements in the urine may disclose surprisingly little. Proteinuria is variable and never large in amount; bacteriuria alone is not diagnostic, while patients with unquestionable chronic pyelonephritis may have negative urine cultures. The provocation of urinary leucocyte excre-

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Fig. 1. Chronic interstitial nephritis showing heavy infiltration of chronic inflammatory cells, atrophied tubules, and one glomerulus with an intact tuft. The surviving tubules are dilated.

A study of renal biopsies and of post-mortem kidney specimens collected in England, Burma, and Malaysia, showed that the common renal conditions associated with leprosy are chronic glomerulonephritis, proliferative glomerulonephritis, amyloidosis, and interstitial nephritis (10). Heavy infiltration of chronic inflammatory cells in the interstitial tissue (interstitial nephritis, Fig. 1) was seen in 5 out of 24 specimens. These cases probably correspond to those reported as pyelonephritis by some workers. Such changes in leprosy may be due to consumption of large quantities of analgesic compounds, or to the intercurrent infections to which leprosy patients are prone. A recent study (3) of 49 leprosy patients and 30 normal controls revealed that 10 of the leprosy patients (20%) were unable to lower their urine pH below 5.5 in response to ammonium chloride stimulus, an acidification defect unrelated to the type of leprosy. As there is a relationship between renal tubular acidosis and peritubular lymphocytic infiltration (8) the finding of this type of infiltration in some of our renal specimens could be due to this acidification defect.

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REFERENCES

Absence of Enhancing Effect of Trimethoprim on the Activity of
a Sulfonamide Against Mycobacterium leprae

To the Editor:

The activity against Mycobacterium leprae of the sulfonamides has been well
documented (Pattyn 1965, Gaugas 1967, Languillon 1964), although the minimal
inhibitory concentration (MIC) of these substances is much closer to the blood
concentration attainable than is the case with dapsone (Ellard, et al, 1972).

Since trimethoprim (TMP) is known to enhance the activity of sulfonamides on a
wide variety of microorganisms, we tested the effect of TMP on the activity of a
sulfonamide against M. leprae in the mouse model.

The continuous method (Shepard 1967) was applied, using strain 12445 previously
found to be fully sensitive to dapsone (Pattyn, et al, 1972). At the end of the observation period acid-fast bacilli were counted in foot pad harvests from two to three mice individually, and in one or more pools of three foot pads; furthermore, three to six foot pads were examined histologically.

The sulfonamide used was sulfadimethoxazole (Grüenthal). The results can be
summarized as follows:

<table>
<thead>
<tr>
<th>Concentrations of chemotherapeutics in food</th>
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<tr>
<td>% Sulf</td>
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<td>1</td>
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<td>0.1</td>
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<tr>
<td>0.01</td>
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<td>0.001</td>
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<td>DDS 0.01</td>
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A = active on M. leprae
I = inactive

The MIC of this sulfonamide is higher than those found for other sulfonamides by
Ellard, et al (1970). It thus appears that TMP does not enhance the activity of sul-
fadimethoxazole against M. leprae. Knowing that TMP alone was inactive (Shepard 1972), this result was not entirely unexpected.

Apart from the importance of this finding for the treatment of human leprosy, this result might also indicate—together with the previous finding of Shepard (1967) that the antibacterial activity of dapsone is only partially antagonized by para-aminobenzoic acid—that the mode of action of sulfones and sulfonamides is not on the pathway of the folic acid synthesis in M. leprae.

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