LEPROSY AND PREGNANCY

Sir,

Duncan *et al.* have recently reported that onset, relapse or deterioration of clinical leprosy is especially liable to occur during pregnancy, in particular during the third trimester (*Lepr Rev*, 1981; **52**: 245–62). They suggest this could be associated with depression of cell-mediated immune responses during that period.

Whereas the accumulated evidence (*cf.* references in Duncan *et al.* paper) is consistent with pregnancy as an important risk factor in leprosy, it may be useful to point out a methodological problem which makes interpretation of the data very difficult. Women generally have more frequent contact with health services when they are pregnant or lactating, than at other times. Given this situation, even if there were no true association between leprosy and pregnancy, an apparent or observed association between them is to be expected. This is especially true in areas with high fertility, where women spend much of their lives either pregnant or lactating. A proper control group is required, consisting of non-pregnant and non-lactating women followed as closely as is the pregnant or lactating group. I am aware of no study which has included such a control group. Furthermore, the observation that clinical onset or deterioration is especially liable to occur during the third trimester could also be due to the fact that pregnant women are most liable to contact health services during the latter stages of pregnancy (eg. Table 1 in Duncan *et al., op. cit.*). The way to avoid this bias is by presenting risks in terms of incidence rates by person months under observation, rather than by trimester of observed onset.

This letter is not intended to be unduly critical of Duncan *et al.*'s valuable contribution, but to point out methodological issues which might be addressed by future investigators of this important subject.

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