This issue of Leprosy Review has the theme of immigration and human rights for leprosy patients. Cassandra White (p. 17) has written a detailed review of the way in which leprosy has been reported in the US. Misinformation and misunderstood statistics have been used to claim that leprosy is now endemic in the USA. The number of imported cases actually remains stable but claims of leprosy being a new contagion have ‘gone viral’ on the Internet. It is striking how often this misinformation is repeated and it illustrates how the disease of leprosy continues to have a strong symbolic presence. Even today, when it is eminently curable, leprosy is still used to denote a contagion to be feared. Doug Soutar (p. 3) has explored the current legislation in several countries in relation to leprosy patients crossing borders, and finds that having leprosy is still a potential bar to migration to the UK. This dates from very old legislation, but shamefully the UK Department of Health has no plans to amend this legislation. Maybe the 2012 Olympics, which will be held in London, could prove a spur to amending this anachronism.

Vanaja Shetty and her group report (p. 41) some interesting and important data from Mumbai. They have followed a cohort of newly diagnosed patients with MB leprosy, doing nerve conduction studies and detailed bacteriological evaluations of these patients. In this paper they focussed on whether giving corticosteroids alters bacterial killing. They assessed bacteriological status by doing microscopic bacterial index assessment and also growing M. leprae in the mouse footpad model. Importantly, they found no difference in the bacterial levels fund whether or not patients had been treated with corticosteroids. This is very reassuring, and vindicates the policy of giving steroids alone to patients who have post-multidrug treatment reactions, and also means that there is no justification for stopping MDT whilst giving steroid therapy to patients. However, they also find that 15% of BL and LL patients still had viable bacterial growth in the MFP model after being treated with MDT for 12 months. Although these patients have not (yet) had clinical relapses with leprosy, this is further data to support the concern that patients with an initial high BI are at much higher risk of relapse. They, therefore, should be considered as an important subgroup of MB patients. This data also needs to be considered when developing new MDT regimens because it indicates that for these patients 12 months of MDT is probably insufficient. Any plans to further shorten the duration of MDT need to consider how these patients might be identified and given longer course of MDT to prevent relapse and re-infection and ultimately potential breakdown of leprosy control.

Another important finding in this paper is that using nerve conduction studies (NCS), the most sensitive test for assessing nerve function, there is little improvement in nerve function at 18 months after starting treatment. This study shows that treating leprosy nerve damage with 12 weeks of prednisolone does not improve nerve function impairment (NFI). Policy makers and clinicians should take note of this. There is no justification for continuing to use 12 weeks course of prednisolone to treat nerve damage. Detailed studies of the effect of 16, 20 and 24 week long courses of prednisolone should be undertaken urgently to see if they have a better effect. The evidence for 16 and 20 weeks long course of steroids is also rather weak with only the Sundar Rao study\(^1\) done in India doing a direct comparison of the effect of different duration courses.
This evidence is badly needed to guide policies. It is possible that using NCS we shall find that even 20 weeks treatment with prednisolone results in little improvement in nerve function when NCS are used. However it is very important to do this work.

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Reference