

Drs. Scollard, Lathrop and Truman Reply

TO THE EDITOR:

It is reassuring to learn that Dr. A. C. McDougall and his colleagues, examining a larger number of specimens in a different laboratory, made observations similar to ours concerning nerve involvement in experimental *Mycobacterium leprae* infections in the armadillo. We share his view that the reasons for the individual differences in extent and intensity of infection re-

main obscure. A number of factors, including the source of the bacilli, viability, variation between isolates, and individual host resistance, could all contribute and merit additional investigation. However, the question of asymmetry may be more immediately informative.

Nerve lesions in human leprosy are notably asymmetric, and the finding of similar

asymmetry in armadillo infections thus accurately parallels the human disease. More importantly, this appears to be a characteristic feature of the natural pathogenesis of leprosy, and suggests that random events play a major role in the localization of *M. leprae* to nerve. This has some potentially helpful corollaries, cautioning, for example, against theories of neural localization (or injury) which depend upon assumptions of prior nerve injury for which there is no substantive evidence in leprosy, clinically or experimentally.

Our working hypothesis is that *M. leprae* reach nerves through blood vessels. Vascular dissemination plays a prominent role in many major diseases, a few examples include primary tuberculosis, toxoplasmosis, syphilis, malaria, and the metastasis of many malignant tumors. All of these also show tropism for particular organs within the context of random localization due to hematogenous dissemination. Thus, although Dr. McDougall correctly observes that the reasons for *M. leprae*'s asymmetric involvement of nerves remain obscure,

thoughtful recognition of this asymmetry may offer clues to its means of dissemination within the body.

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