peripheral nerve trunks from the upper and lower armadillo limbs, from which we cut a minimum of 30 sections for examination with hematoxylin and eosin (H&E) and the Fite-Faraco stain for acid-fast bacilli (AFB). In all of the animals with disseminated infection, the commonest finding in nerve was of AFB in large numbers in all four nerves submitted, usually affecting the epineurial, perineurial, and endoneurial areas. However, this was not invariably the case, particularly in view of the heavy, often massive bacillary multiplication seen in other tissues where it was remarkable that AFB were absent in some of the nerve specimens examined. In several instances, this produced obvious asymmetry, i.e., positive findings on one side but negative on the other, in either the upper or lower limbs. The cellular responses in these animals were typically lepromatous (LL); borderline (dimorphous) changes were not seen in tissues submitted to Oxford.

In view of the main priority of the TDR project briefly described above, it was not possible to consider allocating time or money to the further investigation of a histopathological and essentially incidental finding, unlikely to be of relevance to the development of a bank of *M. leprae* for research purposes. No obvious correlation was seen between the presence or absence of bacilli in nerves and 1) the origin (source) of the inoculum (animal or human), 2) the route of inoculation, or 3) the time between inoculation and sacrifice. It was difficult then, and remains difficult now, to see what further line of investigation could reasonably be pursued by way of explanation. Clearly, it has to be recognized that there is a considerable difference between the time interval from inoculation to sacrifice in the armadillo (12–24 months) compared with the interval between infection and the development of lepromatous leprosy in the human being. However, while this might explain the differences in the extent or intensity of involvement of the peripheral nerves, it would not account for the lack of involvement and asymmetry referred to above.

For reasons which remain obscure, the findings reported by Scollard, *et al.* and our experience in Oxford seem to indicate that peripheral nerve involvement in the armadillo model does not invariably correlate with the extent of ultimate bacillary dissemination.

—A. Colin McDougall, M.D., F.R.C.P.

Department of Dermatology
The Churchill Hospital
Headington, Oxford OX3 7JH, U.K.

Reprint requests to: 87 Lower Radley, Near Abingdon, Oxfordshire OX14 3BA, U.K.

REFERENCES

1. McDougall, A. C., Rees, R. J. W. and Lowe, C. The histopathology of experimental leprosy in the armadillo; *Dasypus novemcinctus* Linn. and *Dasypus sabanicola* Linn. (Abstract) Int. J. Lepr. **47** Suppl. (1979) 343.
2. Scollard, D. M., Lathrop, G. W. and Truman, R. W. Infection of distal peripheral nerves by *M. leprae* in infected armadillos; an experimental model of nerve involvement in leprosy. Int. J. Lepr. **64** (1996) 146–151.
3. World Health Organization. Report of the first meeting of IMMLEP Project Group. Geneva: World Health Organization, 1974. TDR/SWG/IMMLEP/IMM/74.3.

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 remain 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.

—David M. Scollard, M.D., Ph.D.

Chief, Pathology Research
Laboratory Research Branch
GWL Hansen's Disease Center at
Louisiana State University
P. O. Box 25072
Baton Rouge, LA 70894, U.S.A.

—George W. Lathrop, D.V.M.

OIC, Small Animals
Division of Veterinary Medicine
USAMRID
Fort Detrick, MD 21701, U.S.A.

—Richard W. Truman, Ph.D.

Chief, Microbiology Research
Laboratory Research Branch
GWL Hansen's Disease Center at
Louisiana State University
P. O. Box 25072
Baton Rouge, LA 70894, U.S.A.

Dapsone-Induced Motor Polyneuropathy in a Patient with Leprosy

TO THE EDITOR:

Dapsone neuropathy is not a common occurrence in spite of the widespread use of this drug for the treatment of a variety of unrelated disorders. It was first reported by Saqueton, *et al.* ([4]) in a patient with pyoderma gangrenosum treated with dapsone 400 mg daily. Most other cases have been reported in patients with chronic skin diseases, such as dermatitis herpetiformis ([2]), subcorneal pustular dermatosis ([1]) and herpes gestationis ([5]), treated with high doses of dapsone. It has not been reported in leprosy cases ([3]). With a dapsone dose of 100 mg daily as given for treatment of leprosy, neuropathy is unlikely to occur, and even if it develops may remain unrecognized in the presence of the associated neuropathy caused by leprosy itself.

A 22-year-old male developed an asymptomatic, hypopigmented, dry, anesthetic, partly ill-defined bordered plaque of 3 × 2 cm on the lower left leg. There was no nerve thickening. Slit-skin smears from the patch and earlobe smears did not show acid-fast bacilli (AFB). A histopathological study of the biopsy specimen from the plaque revealed tuberculoid granuloma in the upper and mid-dermis. The epidermis was not eroded by the granuloma. Blood hemoglobin was 13 gm%, total leukocyte count 7000/cmm, and differential count neutrophils 60%, lymphocytes 34%, and eosinophils 6%. ESR was 12 mm/1st hr (Westegren). Blood sugar fasting level was 80 mg% and postprandial 140 mg%. The liver function and renal function tests were within normal limits.

A diagnosis of borderline turberculoid Hansen's disease was made and the patient was prescribed dapsone 100 mg daily and rifampin 600 mg once a month. Two months later while on multidrug therapy