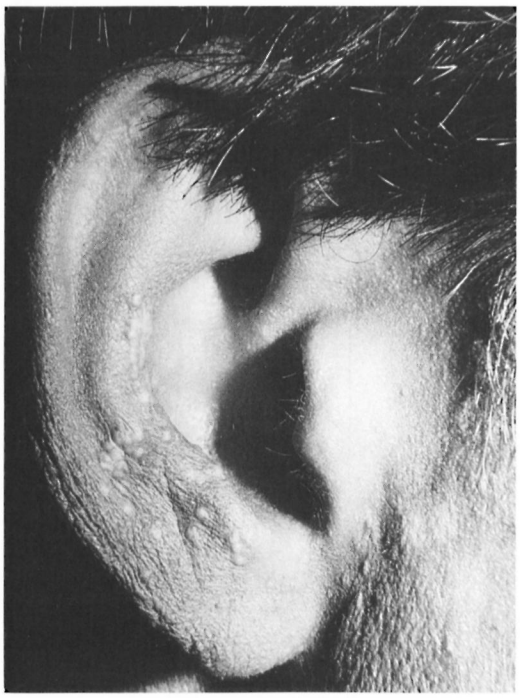

FIG. 2. Milia over the ears.

reaction could lead to epidermal damage, and the subsequent repair could be responsible for the development of milia. Secondary milia often is seen in diseases associated with subepidermal bullae, such as bullous pemphigoid, dystrophic epidermolysis bullosa, porphyria cutanea tarda and lichen sclerosus et atrophicus, after dermabrasion, and following trauma ([2]). It also has been noted after topical steroid therapy ([4]) and topical 5 fluorouracil therapy ([1]).

—Acharya Sandhya, M.D.
C. Balanchandran, M.D.
C. R. Srinivas, M.D.
Shruthakirthi D. Shenoi, M.D.
L. Sabitha, M.D.
P. Satish, M.D.

Department of Skin and STD
Kasturba Medical College
Manipal 576119, Karnataka, India

REFERENCES

1. ARNOLD, H. L., JR., ODOM, R. B. and JAMES, W. D. Epidermal nevi, neoplasms and cysts. In: *Andrews' Diseases of the Skin: Clinical Dermatology.* 8th edn. Philadelphia: W. B. Saunders and Company, 1990, p. 806.
2. LEEPARD, B. and SNEDDON, I.B. Milia occurring in LSEA. Br. J. Dermatol. **92** (1975) 711–714.
3. LEVER, W. F. and SCHAUMBERG-LEVER, G. *Histopathology of the Skin; Tumors and Cysts of the Epidermis.* 6th edn. Philadelphia: J. B. Lippincott Company, 1983, 484–485.
4. TSUJI, T., KADOYA, A., TANAKA, R., KONO, T. and HAMADA, T. Milia induced by corticosteroids. (Letter) Arch. Dermatol. **122** (1986) 139–140.

HIV-1 Infection and Leprosy

TO THE EDITOR:

This letter is to call attention to mounting evidence that HIV infection is a risk factor for leprosy. We published in 1989 that SIV (simian immunodeficiency virus), the sooty mangabey monkey equivalent of the human AIDS virus (which is virtually identical to the human AIDS virus, HIV-2), increases the susceptibility of rhesus monkeys (*Macaca mulatta*) to experimental leprosy, and that there is an increased incidence of multibacillary cases in SIV-coinfected monkeys. Subsequently, we reported on the pathology of such dually infected rhesus monkeys. Our publications are as follows: B. Gormus, *et al.*, Interactions between simian immunodeficiency virus and *Mycobacterium leprae* in experimentally-inoculated rhesus monkeys. J. Infect. Dis. 160:405–413 (1989) and G. Baskin, *et al.*, Pathology of dual *Mycobacterium leprae* and simian immunodeficiency virus infection in rhesus monkeys. Int. J. Lepr. 58:358–364 (1990).

In recent years, two reports of which we are aware published observations dealing with the same subject matter in humans. The first, by J. Ponnighaus, *et al.* (Is HIV infection a risk factor for leprosy? Int. J. Lepr. 59:221–228, 1992) observed no increased risk for leprosy among HIV-posi-

tive patients in Malawi. They did, however, observe an increased risk of relapse among HIV-positive patients after chemotherapy.

More recently, Borgdorff, *et al.,* (HIV-1 infection as a risk factor for leprosy: a case-control study in Tanzania. Int. J. Lepr. 61: 556–562, 1993) reported that HIV-1 infected persons in Tanzania had more than a twofold increased risk for leprosy, compared to HIV-1 negative persons, and that the increased risk was for the lepromatous form of leprosy. These observations are consistent with our previously reported results in the rhesus monkey model.

Neither the Ponnighaus nor the Borgdorff paper acknowledged our publications. Perhaps this was due to the fact that our studies were done in monkeys. We hasten to point out that the monkey model is very similar to man due to phylogenetic similarities, which makes monkey disease models much more relevant to humans than any other model.

Together, the three reports dealing with the risk factor in AIDS-virus-infected individuals suggest that there will be an increase in leprosy cases worldwide secondary to the AIDS epidemic. First, based on the Ponnighaus report, it can be expected that an increase in relapse rates will contribute to such an increase. Secondly, based on our observations and those of Borgdorff, *et al.,* an increase in the number of primary multibacillary cases of leprosy can be expected as a result of the AIDS epidemic. The increased number of multibacillary cases of leprosy will itself then ultimately lead to further increases in the rate of exposure and infection among contacts due to increases in the number of *M. leprae* shed into the environment.

We hope that appropriate notice and concern will be taken by health authorities. We also hope that authors of future publications in the area of leprosy risk among HIV-positive and -negative persons will be so gracious as to acknowledge our 1989 and 1990 manuscripts, which were, to our knowledge, the first to suggest increased leprosy risk among AIDS-virus-positive individuals.

—Bobby J. Gormus, Ph.D.

Research Scientist
Tulane University Medical Center
Tulane Regional Primate
Research Center
18703 Three Rivers Road
Covington, LA 70433-8905, U.S.A.

Dr. Pönnighaus Replies

TO THE EDITOR:

The comments by Dr. Gormus are most welcome. However, I would like to emphasize that we did not observe any evidence of an association between incident leprosy and HIV infection in northern Malawi ([6]). In the whole of Malawi detection rates continue to decline as shown in The Table while disability and lepromatous ratios are stable, although HIV infection rates are known to be on the increase. This observation also argues against the likelihood of "an increase in leprosy cases worldwide secondary to the AIDS epidemic."

The authors' conclusion in a recent study from Tanzania indeed suggests that the HIV epidemic may lead to an increase in the number of multibacillary cases ([2]). However, this conclusion hinges on a single (!) slit-skin smear-negative, "multibacillary," HIV-seropositive leprosy patient and should, at this stage, not be given more credence than it is worth.

We did suggest that there might be an association between HIV infection and relapses ([6]). Indeed, it would be surprising if the clinical course of leprosy would never be changed by co-infection with the HIV ([3]). However, given that relapse rates after multidrug therapy seem to be extremely low ([4, 5]), even a moderate increase due to HIV infection would not change the nearly