THE TABLE. *Detection rates of new Malawian leprosy patients, 1984–1993.*

	Year									
	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993
No. patients detected during the year	1197	1135	1029	1093	907	810	781	638	693	618
Detection rate/10,000	1.7	1.6	1.4	1.4	1.1	1.0	0.9	0.70	0.73	0.63
Annual reduction/increase in detection rate (%)	−14	−8.5	−12	+2.4	−20	−14	−7.0	−21	+4.7	−14
Percentage of patients with a disability > 1 (WHO grading 1960)	12.9	12.5	11.1	9.8	11.7	7.9	11.3	11.9	10.2	13.2
Percentage of patients with multibacillary leprosy	17.4	21.1	20.7	17.7	19.7	14.8	18.3	21.2	21.5	20.7

worldwide pattern of declining leprosy detection rates ([4]).

The relevance of the Gormus, *et al.* and Baskin, *et al.* findings (based on three out of five rhesus monkeys) thus still awaits confirmation. But time will tell.

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REFERENCES

1. BOERRIGTER, G., PONNIGHAUS, J. M., FINE, P. E. M. and WILSON, R. J. Four-year follow-up results of a WHO-recommended multiple drug regimen in paucibacillary leprosy patients in Malawi. Int. J. Lepr. **59** (1991) 255–261.
2. BORGDORFF, M. W., VAN DEN BROEK, J., CHUM, H. J., KLOKKE, A. N., GRAF, P., BARONGO, L. R. and NEWELL, J. N. HIV-1 infection as a risk factor for leprosy; a case-control study in Tanzania. Int. J. Lepr. **61** (1993) 556–562.
3. BWIRE, R. and KAWUMA, H. J. S. Type 1 reactions in leprosy, neuritis and steroid therapy: the impact of the human immunodeficiency virus. Trans. R. Soc. Trop. Med. Hyg. **88** (1994) 315–316.
4. FINE, P. E. M. Reflections on the elimination of leprosy. (Editorial) Int. J. Lepr. **60** (1992) 71–80.
5. PONNIGHAUS, J. M. and BOERRIGTER, G. Are 18 doses of WHO/MDT sufficient for multibacillary leprosy—results of a trial in Malawi. Int. J. Lepr. (in press)
6. PONNIGHAUS, J. M., MWANJASI, L. J., FINE, P. E. M., SHAW, M.-A., TURNER, A. C., OXBORROW, S. M., LUCAS, S. B., JENKINS, P. A., STERNE, J. A. C. and BLISS, L. Is HIV infection a risk factor for leprosy? Int. J. Lepr. **59** (1991) 221–228.

Dr. Borgdorff Replies

TO THE EDITOR:

The papers of Gormus, *et al.* ([3]) and Baskin, *et al.* ([1]) suggest that SIV infection increases the risk for the development of leprosy in experimentally inoculated rhesus monkeys, although their results were not statistically significant (3/5 SIV-infected and 6/29 non-SIV-infected monkeys developed leprosy; Fisher's exact test $p > 0.05$). The papers by Pönnighaus, *et al.* ([4]) and Borgdorff, *et al.* ([2]), on the other hand, aimed at estimating the risk of HIV-1 infection for the development of leprosy in humans.

Once cannot simply extrapolate statements on SIV in rhesus monkeys to those on HIV-1 in humans. However, if both SIV in rhesus monkeys and HIV-1 in humans increase the risk for developing leprosy (as some, although not all, of the evidence suggests), the former may be a good model for the latter.

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REFERENCES

1. BASKIN, G. B., GORMUS, B. J., MARTIN, L. N., MURPHEY-CORB, M., WALSH, G. P. and MEYERS,

W. M. Pathology of dual *Mycobacterium leprae* and simian immunodeficiency virus infection in rhesus monkeys. Int. J. Lepr. **58** (1990) 358–364.
2. BORGDORFF, M. W., VAN DEN BROEK, J., CHUM, H., KLOKKE, A. N., GRAF, P., BARONGO, L. R. and NEWELL, J. N. HIV-1 infection as a risk factor for leprosy: a case-control study in Tanzania. Int. J. Lepr. **61** (1993) 556–562.
3. GORMUS, B. J., MURPHEY-CORB, M., MARTIN, L. N., ZHANG, J., BASKIN, G. B., TRYGG, C. B., WALSH, G. P. and MEYERS, W. M. Interactions between simian immunodeficiency virus and *Mycobacterium leprae* in experimentally inoculated rhesus monkeys. J. Infect. Dis. **160** (1989) 405–413.
4. PONNIGHAUS, J. M., MWANJASI, L. J., FINE, P. E. M., SHAW, M.-A., TURNER, A. C., OXBORROW, S. M., LUCAS, S. B., JENKINS, P. A., STERNE, J. A. C. and BLISS, L. Is HIV infection a risk factor for leprosy? Int. J. Lepr. **59** (1991) 221–228.

Experimental Transmission of Human Leprosy Bacilli in Foot Pads of Severe Combined Immunodeficient Mice

TO THE EDITOR:

After the discovery of *Mycobacterium leprae* as the etiologic agent of human leprosy, it soon became clear that this mycobacterium cannot be grown *in vitro*. Hence, the search for a suitable animal model began. Animal models of leprosy used by investigators between 1879 and 1986 have been reviewed by Johnstone ([1]). Of the several animal models so far employed, only armadillos and nude mice are currently used for the production of *M. leprae* to be used in all fields of leprosy research. After infection, the maintenance of these animals for 12–18 months under controlled conditions is quite expensive. Recently, a mouse with severe combined immunodeficiency (SCID) reconstituted with human peripheral blood leukocytes has been developed ([4]). In an attempt to determine if SCID mice are susceptible to human leprosy and whether higher yields of *M. leprae* can be obtained in a relatively shorter period of time, studies on the transmission of human leprosy to SCID mice were carried out.

A bacillary suspension of *M. leprae* containing 1×10^8/ml acid-fast bacilli (AFB) was prepared from a foot-pad lesion of nude mice previously infected with human leprosy bacilli. Three groups of 10 SCID mice (females, 6 weeks of age) were inoculated in the hind foot pads with a 20 μl bacillary suspension containing 1×10^5, 1×10^6 and 1×10^7 bacilli. In parallel, three groups of 10 nude mice (as controls) were also infected the same way. Both types of mice were kept at 22°C in the same specific pathogen free vinyl plastic isolator. Food, water (*ad libitum*) and bedding after sterilization were provided under aseptic conditions. Following the inoculation of the foot pads with *M. leprae* both SCID and nude mice were sacrificed at various time intervals and AFB were counted according to the method of Shepard and McRae ([5]).

Regardless of the number of bacilli used in the inocula, about 5 months' postinfection a slight swelling in all foot pads of both types of mice started to appear; although more visible in SCID mice. The swelling gradually continued and became quite apparent after 7 to 8 months of infection. Our results have shown that in the foot pads of SCID mice infected with 1×10^5, 1×10^6 and 1×10^7 AFB maximum yields of 1.2×10^8, 4.3×10^8 and 9.0×10^8 bacilli were found after 11, 9 and 8 months of infection, respectively. Thereafter, the number of bacilli gradually decreased upon further incubation, and only some degenerated bacilli were found at the inoculation site after 15 months of incubation. In the foot pads of nude mice infected with 1×10^5 and 1×10^6 bacilli, at 10 months' postinfection 7.8×10^7 and 2.5×10^8 bacilli/foot pad were obtained, respectively. These results show that up to 10 months postinfection the total number of bacilli in the foot pads of nude mice were lower than estimated in the foot pads of SCID mice. However, in the foot pads of nude mice multiplication of *M. leprae* continued progressively at all three inocula used and about 12 months postinfection remarkable swelling of the infected foot