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.

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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

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.

—Jorg M. Pönnighaus,
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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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