Letters to the Editor

Parasitologic Infestation in Hospital Foodhandlers and Risk to AIDS Patients

To the Editor:

Parasitologic infestation in hospital foodhandlers presents a risk of transmission to patients. To evaluate this risk, we studied the results of parasitologic coprology of foodhandlers in a 2,000-bed French hospital over a six-year period.

See also page 457.

Among 56 foodhandlers annually examined for parasitologic infection from 1985 to 1990, 27 (48%) had at least one positive examination. One foodhandler was positive twice, one three times, and two four times. Three harbored two types of parasites. Thus, the total number of positive examinations was 39 (Table). Among these 39 positive results, 31 involved protozoa with direct transmission: Blastocystis hominis. Endolimax nana, Entamoeba coli, Lumblia intestinalis, Entamoeba histolytica, Entamoeba hartmanni, and Sarcocystis hominis.

In 16 cases (41% of positive tests), the parasite was *B hominis*. *The* role of *B hominis* as a human pathogen is subject to debate. Some investigators¹ believe that *B hominis* is not really a pathogen. Senay and MacPherson² found no correlation between the presence

of *B hominis* and symptoms. On the other hand, several investigators^{3,4} consider it a pathogen, particularly if found in large quantity in stool specimens. According to Qadri et al,⁵ among 647 patients harboring *B hominis*, 239 (46.4%) had symptoms including abdominal pain, constipation, and diarrhea. In acquired immunodeficiency syndrome (AIDS) patients, *B hominis* may produce mild persistent or recurrent diarrhea.⁶⁸

In this study, the number of protozoa with direct transmission found in systematic parasitologic coprology of hospital foodhandlers was high. Some of the parasites found were well-known pathogens. For others, such as *B hominis*, pathogenicity is subject to debate. However, according to several investigators, the latter appears to be pathogenic in immunocompromised patients. Because the num-

ber of immunocompromised patients is increasing in hospitals, the risk of nosocomial infection cannot be neglected. Thus, it appears valid to perform such examinations routinely.

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TABLE
ANALYSIS OF 39 POSITIVE RESULTS AFTER PARASITOLOGIC COPROLOGYIN
HOSPITAL FOODHANDLERS, 1985 TO 1990

		No. of
	Parasites	Cases
Pathogenic parasites	Cysts of Entamoeba histolytica	1
	Larvae of Strongyloides stercoralis	2
	Eggs of Sckistosoma mansoni	4
	Cysts of Lumblia intestinalis	2
Parasites with questionable	Blastocystis hominis	16
pathogenicity	Cysts of Entamoeba hartmanni	1
	Cysts of Entamoeba non-kistolytica	3
	Cysts of Endolimax nana	8
Parasites nonpathogenic	Eggs of Trichiuris trichiura	1
under standard conditions	Oocysts of Sarcocystis kominis	1
Total		39

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TB and HIV in Healthcare Settings

To the Editor:

Drs. Castro and Dooley (1993;14:65-66) raised some questions about our interpretation of the findings concerning the retrospective comparative evaluation we made of the occupational risk of tuberculosis in healthcare workers (HCWs) assisting HIV-infected and uninfected tuberculosis patients.¹

As stated in the editorial, some of the points already have been clarified,² particularly the most potentially confounding one: that concerning the HIV status of those HCWs who developed tuberculosis. Unlike in the United States, in Italy HIV-infected patients are assisted in the hospitals by regularly employed nurses and HCWs who, in this investigation, have been considered to be HIV-seronegative and had no other immunosuppressive condition.

Drs. Castro and Dooley have calculated the rate of active tuber-culosis based on the total number of HCWs. This approach ignores the striking difference in the cumulative number of tuberculosis patients between HIV-infected (85) and uninfected ones (1,079), which represent the source of infection.

In our opinion, this difference must be taken into account when comparing the incidence rates of tuberculosis in the two groups of HCWs. If we consider both the personyears due to HCWs (606 among those caring for HIV-infected patients and 486 among those caring for HIV-uninfected patients) and the cumulative number of tuberculosis patients in calculating the denominator of the two rates, the expected number of HCWs with active tuberculosis is 0.81 among those caring for HIVinfected patients (seven were observed), so that an estimate of the relative risk is $35.4 (7 \times 8.20) \div$ (2×0.81) . Using the procedure described by Breslow and Day,³ a 95% confidence interval for the relative risk is 6.8 to 351.5, which is considerably different from that reported by Drs. Castro and Dooley. This result doesn't change if the number of infected patients are given a weight much lower than that given to the number of HCWs; for example, using a square root weight, the 95% confidence interval for the relative risk is 1.9 to 98.7.

Drs. Castro and Dooley also state that there are no reasons to believe that HIV-infected tuberculosis patients may disseminate Mycobacterium tuberculosis at a greater extent than expected. They support this notion by quoting only those reports that confirm their view⁴⁻⁶ and avoiding any mention of the papers that describe opposing evidence, such as those of Standaert,⁷ Brodt,⁸ and Franchini,⁹ who found a convincing association between cases of HIV-associated tuberculosis and an unexpectedly high spread of tuberculous infection and disease. Along with clinical and epidemiological reports, it could be useful to consider also the "lepromatous-like" pattern shown by HIVassociated tuberculosis in several anatomic areas, 10,11 including the lungs, 12,13 where a multibacillary

picture often is seen in a background of aspecific and poorly granulotamous inflammatory reaction, specifically when the most immunosuppressed patients are investigated. In the case of leprosy, another airborne mycobacterial disease, infectiousness is associated rigorously with patients suffering from the lepromatous form of the disease, in whom the specific cellular immune defect makes the affected patient unable to limit bacterial growth.¹⁴ Because severely immunosuppressed patients with HIV infection and tuberculosis display the same histopathological picture seen in cases of lepromatous leprosy, it appears not too hazardous to consider the hypothesis that these (deeply immunosuppressed) patients may disseminate M tuberculosis to a greater extent than immunocompetent tuberculosis patients.

Drs. Castro and Dooley also state that there are reports describing a lower rate of sputum smear positivity for acid-fast bacilli (AFB) in HIV-infected patients compared with seronegative controls. In these reports, however, no information was available on the immune status of those with HIV infection. We believe that, in order to provide the readers with a more comprehensive view of the subject, we also should consider that among HIV-infected patients with tuberculosis, the most immunosuppressed have a higher frequency of positive sputum smears for AFB than those with greater immunity (75% versus 45%), probably reflecting a higher bacillary burden in the setting of greater immunodeficiency. 15 These findings indirectly confirm what we observed in terms of individual immune status of the HIV-infected patients who were identified as the source cases of occupational tuberculosis in our investigation'; all had signs of