several genetic defects in IL-12/IL-12R, IFN-γR, and Stat-1 having been found in patients who had unusually severe infectious diseases caused by poorly virulent mycobacteria and salmonellae [1, 2].

Acknowledgments

The support of The Netherlands Leprosy Relief Foundation (Amsterdam), The Netherlands Organisation for Scientific Research (The Hague), and the Commission of the European Communities (Brussels, Belgium) is gratefully acknowledged.

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More Experience, but Not a New Experience

Sir—We read with attention the article by Vuori-Holopainen and colleagues [1]. In their study, the use of PCR along with conventional methods to analyze samples obtained by transthoracic needle aspiration (TNA) provided the etiologic diagnosis for 20 (59%) of 34 children with community-acquired pneumonia.

We read with surprise the statement in the introduction that, “to our knowledge, this was the first time modern microbiological methods were used with this procedure” [1, p. 584]. In 1994, our group published a study of a series of 45 HIV-infected patients with pneumonia for whom no etiologic diagnosis was made by conventional methods and for whom PCR was used to detect Pneumocystis carinii DNA in samples obtained by TNA; this procedure had a sensitivity of 88.9% [2]. Again, in 1999, our group published a study of a series of 109 patients with community-acquired pneumonia for whom etiologic diagnoses were made by the combination of conventional methods and PCR detection of Streptococcus pneumoniae, Chlamydia pneumoniae, Mycoplasma pneumoniae, Legionella pneumophila, and P. carinii DNA in samples obtained by TNA [3]. In this series, conventional methods identified the etiologic diagnosis in 50% of cases. The use of TNA allowed us to determine the etiologic diagnosis in 65% of cases for which no etiology had been established and to identify a second infecting microorganism for 4 patients in whom a single etiologic agent had been identified by conventional methods.

In another study, which was designed to detect S. pneumoniae in whole blood samples by use of PCR, the technique of amplification of the genome to detect selected pneumolysin gene fragments of S. pneumoniae in TNA samples was added to the diagnostic work-up because it was judged to be the gold standard [4]. Other groups have focused their investigations in the usefulness of PCR for identification of S. pneumoniae DNA in lung parenchyma samples obtained by TNA [5]. Therefore, the study by Vuori-Holopainen et al. [1] describe another experience in the management of pulmonary infections by use of PCR to analyze TNA samples, but it is not the first such experience. The only novel aspect of the study is that TNA was performed in children.

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Pulmonary Tuberculosis Due to Multidrug-Resistant Mycobacterium bovis in a Healthy Host

Sir—Because of improvements in the hy-
Mycobacterium bovis products, the handling and production of dairy gien and practices of persons involved in the handling and production of dairy products, Mycobacterium bovis infection is quite unusual in developed countries of the Western hemisphere [1]. However, in recent years, outbreaks of multidrug-resistant M. bovis infection in patients with AIDS have been reported from Spain and elsewhere [2–5]. These reports have described the disseminated nature of M. bovis infection in HIV-infected patients and have emphasized the associated high mortality rate, which approaches 100% at 3 months after diagnosis [2]. Here, we describe an immunocompetent host who developed pulmonary tuberculosis caused by multidrug-resistant M. bovis.

A 32-year-old woman who was a social worker at Fundación Jiménez Díaz (Madrid) was admitted to the hospital because of fever and chest pain. One week before admission, she had developed a cough, malaise, fever, and sweats, and, later on, she experienced pleuritic chest pain. The findings of physical examination, blood tests, and biochemical studies were normal. The patient’s erythrocyte sedimentation rate was 41 mm/h. Radiography of the chest revealed infiltrates in the upper right lobe. The results of a tuberculin test were positive (15-mm induration). The patient started receiving antimicrobial therapy with rifampin and isoniazid until she completed a 12-month course. The findings of chest radiography and the erythrocyte sedimentation rate became normal. Three years after discontinuation of treatment, the patient is doing well, and there are no signs of residual tuberculosis.

In immunocompromised patients with AIDS, M. bovis infection is a very severe disseminated infection with an awful prognosis. However, the situation could be completely different in immunocompetent hosts. To the best of our knowledge, only 1 immunocompetent patient with multidrug-resistant M. bovis infection has been described to date [6]. That patient developed pulmonary tuberculosis that relapsed after cessation of treatment. Our patient had a mild course of infection in which symptoms rapidly subsided and pulmonary infiltrates abated, despite the patient having received treatment with a combination of drugs that were proven to be inactive in vitro against the isolate. Obviously, the immune integrity of this patient had to be of paramount importance to achieve the cure.

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Clinical Infectious Diseases 2002;35:212–3
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Intradermal Regimens for Rabies Postexposure Prophylaxis: More Confusion

Sir—We applaud the efforts of Dr. Henry Wilde and colleagues in publicizing the crisis in the global supply of rabies immunoglobulin (RIG) [1]. This is an old and familiar problem that seems to be getting worse. In 1998, the World Health Organization (WHO) reported that only 1.6% of 7.5 million patients who were given postexposure vaccination against rables also received RIG [2]. Because most postexposure treatments (PETs), by default, consist only of wound cleaning and vaccine administration, rapid induction of active immunity by vaccine is vital in the attempt to compensate for the lack of passive immunization in the early days after exposure to rables. The cost and, therefore, the amount of modern cell culture vaccine must be kept to a minimum in order that the vaccine can be used in developing countries.

Two economical, multiple-site, intradermal methods used for rables PET are currently recommended by the WHO: an 8-site regimen and a 2-site regimen that use the same amounts of vaccine [3]. In their article, Dr. Wilde and colleagues stated that, for the 2-site regimen, the intradermal dose per site “consists of 0.1 mL of any potent tissue culture vaccine” [1, p. 478]. This is clear and simple advice,