Inquilinus limosus
and Cystic Fibrosis

To the Editor: Inquilinus limosus, a new multidrug-resistant species, was reported in 1999 as an unidenti-
fi ed gram-negative bacterium in a lung transplant patient with cystic fibrosis (CF) (1). This species was later char-
acterized by the description of 7 new isolates of I. limosus and 1 isolate of Inquilinus sp. (2). Infections and colo-
nizations by I. limosus have been docu-
mented mainly in adolescent or adult patients with CF. To date, 8 clinical cases have been described in Germany (3,4), 1 case in the United States (1), 5 cases in France (5), and 1 case in the United Kingdom (6) (Table). Only 1 isolate of Inquilinus sp. has been re-
covered from blood samples of a pa-
tient without CF who had prosthetic valve endocarditis (7).

Because this bacterium is not re-
corded in all commercial identification system databases currently available, a longitudinal study for I. limosus detection with a new real-time PCR assay with a Taqman probe (Applied Biosystems, Foster City, CA, USA), that targets the 16S rRNA gene, has been developed and compared with the culture isolation. Primers i1d (5′-TAATACGAGGAGGTCAGCCTT-GTTGCGTCAGATGTGAAAG-TAMRA), which were used in this study, were designed on the basis of multisequence alignment of all I. limosus 16S rDNA sequences available in the GenBank database.

To confirm specificity, the primers and probe were checked by using the BLAST program (www.ncbi.nlm.nih.gov/blast/Blast.cgi) and also by using suspension of several bacteria recovered habitually in patients with CF. For sensitivity of the Taqman PCR assay (Applied Biosystems), the minimal CFU detectable was 2 CFU/PCR. From January 2006 through June 2007, 365 sputum samples recovered from 84 children and 61 adults with CF and 71 sputum samples recovered from 54 patients without CF were screened blindly for I. limosus. Thus, in our study, the incidence of I. limosus was 2.8% (4.9% for adults with CF and 1.2% for children with CF). The incidence of Burkholderia cepacia complex during the same period and in the same patients was 2.1% (3 adults with CF were positive, data not shown).

The genus Inquilinus belongs to the α-Proteobacteria; the genus Azospirillum is the most closely re-
lated bacteria (2). This cluster of bacte-
teria contains several strains that are able to grow under saline conditions and in biofilms (8,9). The mucoid phe-
totype of I. limosus may contribute to its colonization and resistance to many antimicrobial drugs. Recently, the ex-
opoly saccharides (EPS) produced by I. limosus were studied. The authors indicated that I. limosus produces mainly 2 EPSs that exhibit the same charge per sugar residue present in alginate, the EPS produced by Pseudomonas aeruginosa in patients with CF. This similarity may be related to common features of the EPS produced by these 2 opportunistic pathogens that are re-
lated to lung infections (10). Trans-
mission of I. limosus between patients with CF is not known, but in the report from Chiron et al., 1 of the 5 patients with I. limosus had a brother who had never been colonized with this bacterium despite living in the same home (5). Schmoldt et al. reported that 3 patients were treated in the same outpatient CF clinic during overlapping time periods and each patient was in-
fected/colonized by an individual I. li-
osus clone, which suggests that there was no transmission among these pa-
tients (4). This bacterium has been re-
covered mainly from sputum of ado-
lescents (mean age 17 ± 6.47 years, range 8–35), except in our study with a 2-year-old boy, which suggests that this emerging bacterium may be hos-
pital acquired, as recently suggested (7). Because this bacterium is multiresis-
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tant to several antimicrobial drugs, particularly colistin, which is widely
used for treatment for P. aeruginosa colonization (as was the case for our 4 patients), we hypothesize that this bacterium is selected during the evolution of the disease.

We have developed a real-time PCR molecular method that is faster and easier than amplification-sequencing for prompt detection and accurate identification of *I. limosus* with good specificity and sensitivity. By using this screening assay, we identified 4 additional cases of patients with CF who were also infected with this bacterium, including a 2-year-old child. In addition, by using this technique, we were able to detect *I. limosus* in a patient with deteriorated respiratory function 3 months before the culture-based isolation, indicating that a low bacterial load, insufficient for being isolated in culture, can be detected by PCR in the lower respiratory tract of patients with CF.

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Splenic Rupture and Malignant Mediterranean Spotted Fever

To the Editor: Mediterranean spotted fever (MSF) is a Rickettsia conorii infection endemic to the Mediterranean. In this case, a 55-year-old man was referred to the Necker-Enfants Malades Hospital, Paris, France, for fever, myalgia, and hypotensive shock. The patient had been in Southern France (Montpellier) 6 days before symptom onset and had been bitten by a tick on the left hand. Four days later, he reported fatigue, fever (39°C), and myalgia. His medical history showed polycystic kidney disease, which had necessitated hemodialysis and a kidney transplant. He was receiving ongoing treatment with an immunosuppressive regimen of cyclosporine, prednisolone, and tacrolimus; his baseline hemoglobin level was 15 g/dL, and creatinine level was 230 μmol/L.

At admission, the patient’s temperature was 39.5°C, blood pressure 55/40 mm Hg, and heart rate 104 beats/min. Physical examination showed a diffusely tender abdomen with guarding, no hepatosplenomegaly, a nontender renal transplant, and no lymphadenopathy. Results of cardiovascular, respiratory, and neurologic examinations were unremarkable. A diffuse maculopapular cutaneous eruption was noted on the lower limbs; no eschar was detected.

Laboratory analyses showed the following values: hemoglobin 7.9 g/dL, platelet count 115 × 10⁹/L, leukocyte count 6.7 × 10⁹/L (neutrophils 5.2 × 10⁹/L, lymphocytes 1.4 × 10⁹/L); serum creatinine 466 μmol/L, and C-reactive protein 156 mg/L. Blood cultures were negative. Serologic study results were negative for HIV, hepatitis viruses, Epstein-Barr virus, cytomegalovirus, Legionella, Mycoplasma, Coxiella, Bartonella, Leishmania, and Toxoplasma spp. Serologic testing obtained at day 1 was negative for spotted fever group (SFG) rickettsiosis.

A computed tomographic scan showed hemoperitoneum secondary to a ruptured subcapsular splenic hematoma (online Appendix Figure, available from www.cdc.gov/EID/content/14/6/995-appG.htm), and an emergency splenectomy was performed. Histopathologic evaluation of the spleen showed white pulp atrophy; the red pulp indicated congestion and ill-defined nodules, varying in size and comprising macrophages, polymorphonuclear neutrophils, and necrotic cells (Figure, panels A, B). Skin biopsy of the macular eruption on day 2 demonstrated a leukocytoclastic vasculitis with nonocclusive luminal thrombi in the dermal capillaries (Figure, panel C).

Universal 16S rRNA gene PCR amplification on spleen and skin tissue samples and direct sequencing identified an R. conorii–specific 16S rRNA sequence match. We confirmed this by using primers for gltA and ompA specific for R. conorii. Immunohistochemical staining demonstrated Rickettsia in endothelial cells and macrophages in the spleen and skin (Figure, panels D–F). Blood culture, skin biopsy specimens, and splenic tissue cultures were subsequently R. conorii positive.

Doxycycline therapy (100 mg intravenously twice a day) was instituted at day 2 because rickettsiosis was suspected. The patient dramatically improved within 72 hours and remained well 36 months after diagnosis.

MSF is a rickettsiosis belonging to the tick-borne SFG caused by R. conorii, an obligate intracellular bacteria transmitted by the dog tick Rhipicephalus sanguineus. Endemic to Mediterranean countries, MSF generally results in a benign febrile illness accompanied by a maculopapular rash, myalgia, and local black eschar at a tick bite inoculation site. A minority of persons seeking treatment display a malignant form, which results from disseminated vasculitis associated with increased vascular permeability, thrombus-mediated vascular occlusion, and visceral perivascular lymphohistiocytic infiltrates (1). Focal thrombi have been identified in almost all organs of patients with fatal cases. Manifestations of MSF include neurologic involvement, multi-organ failure, gastric hemorrhage, and acute respiratory distress syndrome; the case-fatality rate is 1.4%–5.6%.

Splenic rupture has been reported in the course of infection with several microbial agents, including Epstein-Barr virus (2), HIV, rubella virus, Bartonella spp. (3), Salmonella spp., mycobacteria (4), and Plasmodium spp. (5). Splenomegaly as a result of MSF has also been documented previously (6);