second patient were within the same subgroup, but similarity in nucleotide sequences was only 82.5%. This result suggests that this patient was infected with 2 different virus strains of HRV-A and a strain of HRV-C.

Comparison of the HRV-A strains with the HRV-C strain showed that they belonged to different subgroups and had low similarity for nucleotide sequences. The second patient had 3 distinct rhinovirus infections over 3 months, and each was associated with illness requiring hospitalization. Both patients had underlying diseases, reactive airway diseases, and repeated episodes of RTI that may have rendered them vulnerable to reinfection, compromising their immune responses.

Complete coding sequences of HRV-A and HRV-C have been determined (*4,7*). However, little is known about their involvement in the pathogenesis of recurrent wheezing in young children. According to recent reports, HRV-C has been detected in hospitalized children with lower RTI in the People's Republic of China (*5*). Possible association of novel infection with HRV and exacerbation of asthma in children has also been reported (*6*). We report HRV-A and HRV-C coinfections in conjunction with other respiratory viruses, such as RSV, as a potential cause of recurrent wheezing in infants with acute lower RTIs. Coinfections with HRV-A and HRV-C may contribute to increased virulence and subsequent pathogenesis of other respiratory viruses. Additional studies will be required to further explore the clinical role of novel HRVs.

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Extensively Drug-Resistant *Acinetobacter baumannii*

To the Editor: In the 1990s, patients infected with vancomycin-resistant *Enterococcus faecium* were successfully treated with new antimicrobial drugs. However, it is unlikely that new antimicrobial drugs will be available in the near future to treat patients infected with gram-negative pathogens such as *Acinetobacter baumannii* (*1*). No new antimicrobial drugs active against this organism are currently in clinical trials (www.clinicaltrials.gov). We report a patient infected with *A. baumannii* that lacked susceptibility to all commercially available antimicrobial drugs.

The patient, a 55-year-old woman, had a prolonged stay in an intensive care unit at the University of Pittsburgh Medical Center (Pittsburgh, PA, USA) after undergoing lung transplantation. In the tenth postoperative week, ventilator-associated pneumonia developed, which was caused by *A. baumanni* that lacked susceptibil-

ity to all antimicrobial drugs tested except colistin (MIC 0.5 μg/mL). Therapy with colistin and tigecycline was begun. Colistin was administered intravenously and by inhalation. Although the pneumonia showed radiographic response to the antimicrobial drug therapy, *A. baumannii* continued to be isolated from respiratory secretions on numerous occasions. Despite another course of therapy with colistin and cefepime, the patient never recovered from respiratory failure. She eventually died of sepsis caused by vancomycin-resistant *E. faecium*. An *A. baumannii* isolate obtained just before she died lacked susceptibility to all commercially available antimicrobial drugs (Table).

Multidrug-resistant *A. baumannii* has emerged as a substantial problem worldwide (*2*). Such strains are typically resistant to all β-lactams and fluoroquinolones and require salvage therapy with colistin, amikacin, or tigecycline. Unfortunately, notably high-level resistance to colistin and amikacin was found in the isolate we have described (Table). Tigecycline, a newly available glycylcycline antimicrobial drug, showed intermediate susceptibility. No randomized trials have been performed to specifically evaluate combination antimicrobial drug therapy for treatment of infection with *A. baumannii*.

Considerable media attention has been paid to extensively drug-resistant (XDR) strains of *Mycobacterium tuberculosis* (*3*). Infections with XDR strains are extremely difficult to treat and pose considerable infection control issues. We recently proposed that gram-negative bacilli lacking susceptibility to all commercially available antimicrobial drugs also be referred to as XDR because no therapeutic options are available (*4*).

Numerous outbreaks of *A. baumannii* infection have been reported worldwide (*5*). Unfortunately, multidrug-resistant *A. baumannii* strains have become endemic in some institutions. Experimental and clinical isolates lacking susceptibility to colistin, often considered the drug of last resort, are increasingly being reported (*6*–*8*). Therefore, we alert healthcare workers to the need for stringent care in adhering to infection control precautions when caring for patients infected with XDR *A. baumannii*. Use of contact isolation precautions, enhanced environmental cleaning, removal of sources of infection from the hospital environment, and prudent use of antimicrobial drugs can contribute to control of such outbreaks (*5*). Fortunately, no spread of the XDR strain affecting this patient occurred. A crisis is looming should XDR *A. baumannii* become established pathogens in hospitals.

Table. MICs and antimicrobial drug susceptibility for an extensively drug-resistant strain of *Acinetobacter baumannii**

Drugs	MIC, μg/mL	Interpretation
Carbapanems		
Imipenem	>32	Resistant
Meropenem	>32	Resistant
Penicillins		
Ampicillin/sulbactam	32	Resistant
Piperacillin/tazobactam	>256	Resistant
Cephalosporins		
Ceftazidime	48	Resistant
Cefepime	16	Intermediate
Aminoglycosides		
Gentamicin	>256	Resistant
Tobramycin	>256	Resistant
Amikacin	>256	Resistant
Others		
Ciprofloxacin	>32	Resistant
Tigecycline	2	Intermediate
Colistin	>1,024	Resistant

*Susceptibility testing was performed by using the Etest (AB Biodisk, Solna, Sweden), except for colistin, for which the standard agar dilution method was used. Interpretation was according to breakpoints provided by the Clinical and Laboratory Standards Institute (CLSI; Wayne, PA, USA). No tigecycline breakpoints for *Acinetobacter* spp. are provided by the CLSI, the European Committee on Antimicrobial Susceptibility Testing (Basel, Switzerland), or the US Food and Drug Administration (Silver Spring, MD, USA). Breakpoints of the British Society for Antimicrobial Chemotherapy (Birmingham, UK) are indicated for tigecycline.

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Cryptosporidium Pig Genotype II in Immunocompetent Man

To the Editor: Protozoan parasites from the genus *Cryptosporidium* have been described as a cause of diarrheal disease in immunodeficient and immunocompetent humans worldwide. Although *C. hominis* and *C. parvum* (cattle genotype) cause most infections, humans can be infected by several other *Cryptosporidium* species or genotypes: *C. meleagridis*; *C. felis*; *C. canis*; *C. suis*; *C. muris*; *C. andersoni*; *C. hominis* monkey genotype; *C. parvum* (mouse genotype); and *Cryptosporidium* rabbit genotype, deer genotype, skunk genotype, horse genotype, and chipmunk genotype I (*1*–*4*). Wild and domestic animals are sources of infection for humans (and other animals) and important contributors to contamination of food and drinking water; many nonhuman *Cryptosporidium* species or genotypes are detected in untreated water (*5*). We examined the diversity of *Cryptosporidium* spp. in immunocompetent persons in South Bohemia in the Czech Republic.

Diarrheal fecal samples (n = 457) from 203 anonymous immunocompetent patients ≤69 years of age with suspected cryptosporidiosis (at least 2 samples/patient/3-day period) were obtained from local health departments and public hospitals in South Bohemia during 2005–2007. Samples were examined for *Cryptosporidium* oocysts by using aniline-carbol-methyl violet staining and light microscopy at × 1,000 magnification (*6*). The microscopically positive samples were confirmed by DNA sequencing of the small subunit (SSU) rRNA gene. Total DNA was extracted from 200–300 mg stool by using the QIAamp DNA Stool Mini Kit (QIAGEN, Hilden, Germany), following the manufacturer's instructions, after previous homogenization and disruption of oocysts with the Mini-BeadBeater (Biospec Products, Bartlesville, OK, USA). An ≈830-bp fragment of the SSU rRNA gene was amplified by nested PCR according to Jiang et al. (*7*). Purified PCR products were sequenced in both directions on an ABI3130 sequencer analyzer (Applied Biosystems, Foster City, CA, USA) by using the secondary PCR primers and the BigDye Terminator v3.1 Cycle Sequencing Kit (Applied Biosystems). Sequences were assembled by using Chromas Pro (www.technelysium.com.au/chromas.html) and aligned with reference sequences using ClustalX (ftp://ftp-igbmc.u-strasbg.fr/pub/ClustalX). The BLAST server (www.ncbi.nlm.nih.gov/BLAST) was used for DNA database searches. The SSU rRNA gene partial sequences of the 7 patient isolates have been submitted to GenBank (Table).

Of the 203 patients, 7 (3.4%) (6 children and 1 adult) had positive results for *Cryptosporidium* spp. Moreover, all samples obtained from these persons during the 3-day period were *Cryptosporidium* spp. positive; partial sequences of the *Cryptosporidium* SSU rRNA gene were obtained from all positive samples identifying 3 different species or genotypes of *Cryptosporidium*. Five were *C. parvum* (bovine genotype), 1 was *C. hominis,* and 1 contained the *Cryptosporidium* pig genotype II (Table). *Cryptosporidium* pig genotype II was found in stool samples from a 29-year-old man who also was infected with *Giardia intestinalis* (assemblage A) (data not shown).

Only *C. parvum* (bovine genotype), *C. hominis*, and *Cryptosporidium* rabbit genotype have been implicated in waterborne outbreaks of cryptosporidiosis in humans. Further studies are needed to determine the potential of other cryptosporidia of animal origin. Recent genetic and biologic characterization studies have identified 2 distinct host-adapted cryptosporidia in pigs, *C. suis* and *Cryptosporidium* pig genotype II. Furthermore, both above-mentioned cryptosporidia have been identified in untreated water (*8*). Pigs could be sources of *Cryptosporidium* water and food pollution and a consequent risk to public health.

Although human infection with *C. suis* has been previously described (*9*), human infection with *Cryptosporidium* pig genotype II has been never reported. This genotype was found in diarrheal stool of 1 adult patient in this study. However, onset of diarrhea could have been caused by co-infection with *G. intestinalis* (assemblage A), which recently also has been described in pigs (*10*). Contact with infected animals and ingestion of contaminated food or water could be