(bla_{PER-1} and bla_{VIM-2}) had been detected separately among clinical isolates, underscores the possibility of the emergence of new threatening combinations of resistance determinants among nosocomial pathogens. In fact, the recruitment of similar resistance determinants within a single *P. aeruginosa* strain can determine a resistance phenotype to virtually all the available antipseudomonal beta-lactams, an occurrence that can be particularly dramatic when, as in the present case, resistance to beta-lactams is associated with resistance against aminoglycosides and fluoroquinolones. In this case, only piperacillin (which appears to be a relatively poor substrate for both enzymes [3,5]) retained moderate activity in vitro and, administered at high dosage in combination with tazobactam, was apparently effective in vivo. Should a similar resistance phenotype disseminate, it might have strategic implications for the development of new beta-lactamase inhibitors and for selection of beta-lactam compounds to associate wth inhibitors.

Acknowledgments

This work was supported in part by grant no. FMRX-CT98-0232 from the European Training and Mobility of Researchers Network on metallo-beta-lactamases.

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Jamestown Canyon Virus: Seroprevalence in Connecticut

To the Editor: *Jamestown Canyon virus* (JCV), a member of the California serogroup, has a wide geographic distribution throughout much of temperate North America. It causes mild febrile illness and, rarely, aseptic meningitis or primary encephalitis (1). JCV has been isolated from mosquitoes each year that surveys have been done in Connecticut, and 28 positive pools from 10 mosquito species were found during 2000 (T. Andreadis, pers. commun.). In contrast, only 14 positive mosquito pools were found to contain *West Nile virus* (WNV), which has recently been introduced into Connecticut (2). JCV has been isolated from *Aedes* mosquitoes in Connecticut, and serologic evidence suggests it is widespread in deer (3,4). No recent seroprevalence surveys have been done in Connecticut, nor have any human cases of infection or disease due to JCV been documented.

We report the results of two seroprevalence surveys done with standard indirect fluorescent assays (IFA) to detect immunoglobulin G antibodies to JCV. One survey examined 1,086 sera collected in 1990 from blood donors. The second survey examined 1,016 sera submitted to the Connecticut State Public Health Laboratory in 1995.

The IFA used JCV-infected baby hamster kidney cells (BHK-21). Infected and uninfected cell suspensions were air dried and fixed onto Teflon-coated, 12-well slides. Prepared slides were stored at -70°C. Sera were tested at a minimum dilution of 1:16. After incubation and washing of the fluorescein-conjugated counterstain, slides were dried and examined by fluorescent microscope (American Optical, Buffalo, NY). The positive human control serum was designated as the 4+ baseline with which the test sera were compared. Selected sera were tested by a serum dilution plaque reduction neutralization test (PRNT) assay with JCV, *La Crosse virus*, and trivittatus virus.

Of the 1,086 sera collected from blood donors in 1990, 164 (15%) were positive by IFA at a minimum dilution of 1:16. Because IFA screening procedures are known to have poor specificity, a subset of 39 IFA-positive and 5 IFA-negative sera was tested by PRNT. None of the IFA-negative sera were positive, while 26 (67%) of the 39 IFA-positive sera were positive for JCV antibodies. Extrapolating the PRNT results to the 164 IFA-positive sera yields an overall positivity rate of 10.1%.

The second serosurvey, performed on 1,016 sera collected in 1995 from apparently healthy patients requesting immune status testing to viruses such as *Varicella zoster* or measles, had 57 IFA-positive specimens. Extrapolating addi-

tional PRNT results from 26 sera, of which 18 (69%) were positive, yields a 3.9% positivity rate.

In addition to our study, with crude seroprevalence rates ranging from 3.9% to 10.1%, another recent study demonstrated JCV antibodies in 2.9% to 13.3% of ill persons in Massachusetts (Tonry J et al., unpub. data). Although the screening results of our first serosurvey (10.1% positive) differed widely from those of the second serosurvey (3.9% positive), even the lower rate indicates substantial levels of human infection in Connecticut.

This report suggests that JCV infection is fairly frequent in Connecticut and that illness may occur, as corroborated by data from neighboring Massachusetts (Tonry J et al., unpub. data) and unpublished laboratory findings from the Connecticut State Public Health Laboratory. The interest in arboviral disease will continue unabated, spurred by the continued occurrence of WNV, and systematic testing for JCV infection may be timely, at least throughout the northeastern United States.

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A Newly Discovered Variant of a Hantavirus in *Apodemus peninsulae*, Far Eastern Russia

To the Editor: Hemorrhagic fever with renal syndrome (HFRS) is caused by *Hantaan virus* (HTNV) or *Seoul virus* (SEO) in Asia and *Puumala virus* (PUUV) or *Dobrava virus* (DOBV) in Europe (1). Each of these hantaviruses is predominantly associated with a single rodent species as its primary natural reservoir: HTNV with the striped field mouse *Apodemus agrarius*, SEO with *Rattus norvegicus*, PUUV with the bank vole *Clethrionomys glareolus*, and DOBV with the yellow-necked mouse *Apodemus flavicollis*. An additional rodent reservoir of DOBV, *A. agrarius*, was reported recently (2).

The first HFRS cases (then called "hemorrhagic nephroso-nephritis") were clinically described in the Amur River basin during the 1920s by Russian scientists (3). Serologic studies suggest that numerous hantaviruses are present in humans and rodents in the far east of Asian Russia (4-5). Serologic evidence of hantavirus infection in *A. agrarius*, *A. peninsulae* (Korean field mouse), *R. norvegicus*, *Cl. rufocanus*, *Cl. rutilus*, and *Microtus fortis* has been reported (5). Only *Khabarovsk virus* (KBR), isolated from *M. fortis*, has been characterized in detail, and no association with human disease was established (6).

To genetically characterize hantaviruses in *A. peninsulae*, we studied samples from rodents captured in July and August 1998 in the same region of the forest near Khabarovsk. Lung-tissue samples were screened by enzyme-linked immunosorbent assay for HTNV/SEO/PUUV-related antigen. Samples from four hantavirus-positive rodents were tested by reverse transcription and nested polymerase chain reaction (PCR). Four M-segment PCR products (nt 2639-3000) and two S-segment PCR products (nt 592-945) were produced and directly sequenced (GenBank accession numbers AF332569-AF332573). All sequences were closely related to each other, with nucleotide diversity between strains not exceeding 0.6% for M segments and 1.3% for S segments. Comparative analysis of the M segments showed that hantaviral nucleotide sequences from *A. peninsulae* were very similar to those we identified earlier in HFRS patients (diverging 3.1% to 6.6%), which we term the Amur genotype of HTNV (7). The S-segment sequences of the AMR genotype from human patients were not available for comparison. The nucleotide sequence (the M and S segments, respectively) of the hantavirus detected in *A. peninsulae* diverged substantially from those of other hantaviruses (15% and 19% for HTNV, 21% to 28% for SEO, 22% and 29% for DOBV, 38% and 39% for PUUV, and 36% and 37% for KBR).

Neighbor-joining phylogenetic analysis based on partial sequences of the S segment indicated that the hantaviral sequences from *A. peninsulae* form a separate lineage on the phylogenetic tree, and together with HTNV virus strain 76-118, which originates from *A. agrarius*, constitute a well-supported group. A phylogenetic tree based on partial M segment sequences placed all hantavirus strains originating from *A. peninsulae* or from HFRS patients apart from all HTNV sequences recovered from *A. agrarius* (strain 76-118) and HFRS patients from Korea (strains HoJo, Lee). The taxonomic placement of this hantavirus (Amur genotype) as a distinct hantavirus or a distinct genetic lineage of HTNV remains to be determined. In addition, the finding of distinct DOBV genetic lineages in *A. flavicollis* and *A. agrarius* raises the same question of whether the two DOBV variants represent distinct hantaviruses (2).

A. peninsulae is widely distributed throughout eastern Asia, from Altai and south Siberia to the Russian far east, northeastern and eastern parts of China, and Korea. A survey of hantavirus antigens in rodent populations in the far east of Russia demonstrated the presence of HTNV-like antigen in 8% to 16% of *A. peninsulae* (5). Whether pathogenic AMR genotype of virus exists in *A. peninsulae* throughout far eastern Asia, from Russia to China and Korea, requires further study. Comparing hantaviral genome sequences available from GenBank shows that the M segment nucleotide sequence recovered from an HFRS patient from China (strain H8205, GenBank accession number AB030232) was very similar to the AMR genotype from *A. peninsulae* (94% to