using Vero and RD-18S cells, was unsuccessful. This result is consistent with previous EV-104 reports, wherein the virus could not be grown or isolated (*1,2*).

To determine the presence of other respiratory viruses in this patient, the EV-104–positive specimen was tested by using real-time PCR for any of 17 other viruses (human metapneumovirus, respiratory syncytial virus, human parainfluenza virus types 1–4, human bocavirus, human coronavirus [229E, OC43, HKU1, NL63], influenza virus [A, pandemic (H1N1) 2009, B, C], human adenovirus, and HRV). No other viruses were detected (data not shown). This result indicates that EV-104 was associated with upper RTI in this patient. During the 2 months in which the EV-104–positive sample was collected, influenza A virus, HRV, and respiratory syncytial virus were most frequently detected in other patients, and no enterovirus was observed in other specimens from persons with RTI.

EV-104 detection is rare (5/1,500 [0.3%] for a 1-year study in Italy [*1*]; 8/1,592 [0.5%] for a 10-year study in Switzerland [*2*]). As part of a virus surveillance program in Osaka City, Japan, during November 2010–October 2011, a total of 645 respiratory tract specimens were collected from children with RTI (360 male, 285 female; age 0–59 months, mean ± SD 18.9 ± 13.8 months) and subjected to PCR by using EVP4 and OL68-1 primers. No EV-104 was detected. In 2 previous studies in Japan, we detected no EV-104 in 764 specimens from patients with RTI during November 2008 and October 2010 (*3,7*); therefore, we have found EV-104 in only 1 (0.07%) of 1,410 samples tested.

Infrequent detection and insensitivity to cell culture contribute to the rarity of EV-104 identification. However, given the lack of contact between EV-104–positive patients in Italy and Switzerland, more RTI patients might actually be carrying EV-104 than testing has indicated. The collection of additional EV-104 strains and associated epidemiologic and virologic information will help clarify the role of this virus in RTI.

This work was supported in part by a grant-in-aid for Young Scientists (B) (23790720) from The Ministry of Education, Culture, Sports, Science, and Technology of Japan.

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DOI: http://dx.doi.org/10.3201/eid1805.111890

References

1. Tapparel C, Junier T, Gerlach D, Van-Belle S, Turin L, Cordey S, et al. New respiratory enterovirus and recombinant rhinoviruses among circulating picornaviruses. Emerg Infect Dis. 2009;15:719–26. http://dx.doi.org/10.3201/eid1505.081286
2. Piralla A, Rovida F, Baldanti F, Gerna G. Enterovirus genotype EV-104 in humans, Italy, 2008–2009. Emerg Infect Dis. 2010;16:1018–21.
3. Kaida A, Kubo H, Sekiguchi J, Kohdera U, Togawa M, Shiomi M, et al. Enterovirus 68 in children with acute respiratory tract infections, Osaka, Japan. Emerg Infect Dis. 2011;17:1494–7.
4. Ishiko H, Miura R, Shimada Y, Hayashi A, Nakajima H, Yamazaki S, et al. Human rhinovirus 87 identified as human enterovirus 68 by VP4-based molecular diagnosis. Intervirology. 2002;45:136–41. http://dx.doi.org/10.1159/000065866
5. Katayama K, Shirato-Horikoshi H, Kojima S, Kageyama T, Oka T, Hoshino F, et al. Phylogenetic analysis of the complete genome of 18 Norwalk-like viruses. Virology. 2002;299:225–39. http://dx.doi.org/10.1006/viro.2002.1568
6. Yozwiak NL, Skewes-Cox P, Gordon A, Saborio S, Kuan G, Balmaseda A, et al. Human enterovirus 109: a novel interspecies recombinant enterovirus isolated from a case of acute pediatric respiratory illness in Nicaragua. J Virol. 2010;84:9047–58. http://dx.doi.org/10.1128/JVI.00698-10
7. Kaida A, Kubo H, Takakura K, Togawa M, Shiomi M, Kohdera U, et al. Molecular epidemiology of human rhinovirus C in patients with acute respiratory tract infections in Osaka City, Japan. Jpn J Infect Dis. 2011;64:488–92.

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Clonal Spread of *Geomyces destructans* among Bats, Midwestern and Southern United States

To the Editor: Bat geomycosis (white nose syndrome) is caused by the psychrophilic fungus *Geomyces destructans*, which has rapidly spread in the United States and Canada since it was first reported from Albany, New York (*1*,*2*). In 2011, a single genotype of *G. destructans* was found in bats with geomycosis in different parts of New York (*3*). The findings raised the possibility of clonal spread of a new pathogen with serious implications for the survival of the affected bat populations (*4*). To provide information for devising conservation measures, we explored whether this emerging infectious disease is caused by a novel pathogen (*5*). To do so, we genotyped *G. destructans* isolates from the midwestern and southern United States.

During 2010 and 2011, a total of 11 cultures of *G. destructans* were isolated and identified: 1 each from Pennsylvania and Ohio, 3 from North Carolina, and 6 from West Virginia

(Figure). The cultures came from 8 little brown bats (*Myotis lucifugus*) and 3 tricolored bats (*Perimyotis subflavus*). Two recent *G. destructans* isolates from New York and 1 *G. pannorum* isolate were included as controls. Genomic DNA was prepared from fungal growth by the conventional glass bead treatment, phenol–chloroform extraction, and ethanol precipitation. PCR amplifications of 8 *G. destructans* gene fragments (*ALR*, *Bpntase*, *DHC1*, *GPHN*, *PCS*, *POB3*, *SRP72*, and *VPS13*) were performed as described (*3*). The amplicons were sequenced and nucleotides were aligned by Sequencher 4.8 (www.genecodes.com); phylogenetic analyses were done using PAUP*4.0 software (www.sinauer.com).

A total of 4,722 nt sequences were obtained from 8 gene fragments of 13 *G. destructans* isolates (GenBank accession nos. JQ029780–JQ029883) and 1 *G. pannorum* isolate (GenBank accession nos. HQ834330, HQ834347, HQ834364, HQ834381, HQ834398, HQ834415, HQ834432, and HQ834449). Multiple alignments of these sequences showed 100% identity, and the aligned nucleotides matched perfectly with those of earlier *G. destructans* sequences for the same gene fragments analyzed from New York isolates (*3*). The nucleotide alignments of 8 sequences showed differences from those obtained from the closely related fungus, *G. pannorum*. Maximum-parsimony trees were generated by using sequences from each gene fragment. These trees showed a single clade of *G. destructans* strains distinct from *G. pannorum*; similar topologies were obtained when different phylogenetics methods were used for analysis (details not shown). A consensus maximum-parsimony tree derived from the 8 concatenated gene fragments also showed a single clade of *G. destructans* isolates from New York and the midwestern and southern United States (Figure).

The data obtained in this study strongly indicate further clonal spread of *G. destructans* from its origin near Albany, New York. The locations in which *G. destructans* was detected in the current study were spread across 5 states, which were >800 miles from Albany. The test isolates were compared with a New York isolate from 2008, which provided a 4-year temporal variation in our sampling. Bats of 2 species were positive for *G. desctructans* in the current samples, and they yielded the same *G. destructans* genotype. Thus, there is evidence for host-independent spread of a single clone of *G. destructans*.

These data would support the novel-pathogen hypothesis for the origin of bat geomycosis (*5*). However, these conclusions are based on limited sampling because isolations of *G. destructans* from affected bats are uncommon. The demonstration of pure fungal culture in the affected animals is still not the standard for geomycosis diagnostics, and most geomycosis is confirmed by bat morphologic appearance or histopathologic examination. Additionally, our phylogenetics analyses were limited to ≈5 kbp of fungal genomes, which could lead to sampling bias (*3*). Ideally, a large number of *G. destructans* isolates, including isolates from Europe, and additional polymorphic markers would be needed to determine the novel or local origin of this pathogen (*6,7*).

The environmental factors that led to introduction or reemergence of *G. destructans* in mines and caves remain unknown, and their contribution in the spread of the fungus through air, water, and soil is yet to be determined (*8*). Although no direct evidence has emerged, a role for anthropomorphic activities (occupational or recreational) in this spread is a distinct possibility (*9*). We provide genetic evidence for further spread of a single genotype of *G. destructans* from Albany, New York, to locations in the midwestern and southern United States. Experimental transmission of geomycosis from infected bats to healthy bats by direct contact has recently been confirmed (*10*). Therefore, *G. destructans* might

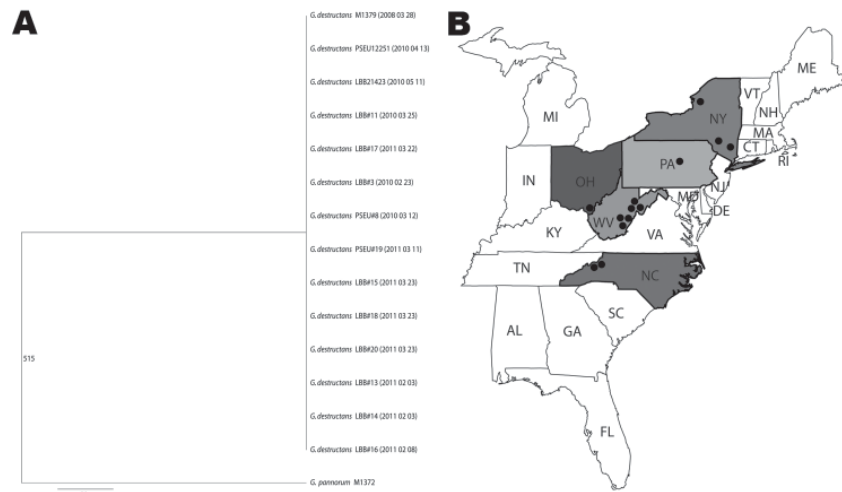

Figure. A) Consensus maximum-parsimony tree of 8 concatenated gene fragments of *Geomyces destructans*. Data were derived from 13 *G. destructans* test isolates. *G. destructans* M1379 and *G. pannorum* M1372 were used as controls in this study; they were described in an earlier report (*3*). The number 515 on the branch indicates the total number of variable nucleotide positions (of 4,722 nt) separating *G. pannorum* M1372 from the clonal genotype of *G. destructans*. Isolation dates are shown in parentheses (YYYY MM DD). Scale bar indicates nucleotide substitutions per site. B) States where *G. destructans* isolates were found; dots indicate locations of positive test results. A color version of this figure is available online (wwwnc.cdc.gov/EID/article/18/5/11-1711-F1.htm).

be rapidly spreading along summer and winter migration routes of bats, which present ample opportunities for mixing of healthy and diseased animals.

Acknowledgments

We thank Xiaojiang Li and Sudha Chaturvedi for fungal characterization and comments on the manuscript.

This study was supported in part with funds from the US Fish and Wildlife Service.

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DOI: http://dx.doi.org/10.3201/eid1805.111711

References

1. Blehert DS, Hicks AC, Behr M, Meteyer CU, Berlowski-Zier BM, Buckles EL, et al. Bat white-nose syndrome: an emerging fungal pathogen? Science. 2009;323:227. http://dx.doi.org/10.1126/science.1163874
2. Chaturvedi V, Springer DJ, Behr MJ, Ramani R, Li X, Peck MK, et al. Morphological and molecular characterizations of psychrophilic fungus *Geomyces destructans* from New York bats with white nose syndrome (WNS). PLoS ONE. 2010;5:e10783. http://dx.doi.org/10.1371/journal.pone.0010783
3. Rajkumar SS, Li X, Rudd RJ, Okoniewski JC, Xu J, Chaturvedi S, et al. Clonal genotype of *Geomyces destructans* among bats with white nose syndrome, New York, USA. Emerg Infect Dis. 2011;17:1273–6. http://dx.doi.org/10.3201/eid1707.102056
4. Frick WF, Pollock JF, Hicks AC, Langwig KE, Reynolds DS, Turner GG, et al. An emerging disease causes regional population collapse of a common North American bat species. Science. 2010;329:679–82. http://dx.doi.org/10.1126/science.1188594
5. Rachowicz LJ, Hero J-M, Alford RA, Taylor JW, Morgan JAT, Vredenburg VT, et al. The novel and endemic pathogen hypotheses: competing explanations for the origin of emerging infectious diseases of wildlife. Conserv Biol. 2005;19:1441–8. http://dx.doi.org/10.1111/j.1523-1739.2005.00255.x
6. Puechmaille SJ, Wibbelt G, Korn V, Fuller H, Forget F, Muhldorfer K, et al. Pan-European distribution of white-nose syndrome fungus (*Geomyces destructans*) not associated with mass mortality. PLoS ONE. 2011;6:e19167. http://dx.doi.org/10.1371/journal.pone.0019167
7. Archie EA, Luikart G, Ezenwa VO. Infecting epidemiology with genetics: a new frontier in disease ecology. Trends Ecol Evol. 2009;24:21–30. http://dx.doi.org/10.1016/j.tree.2008.08.008
8. Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, Ostfeld RS, et al. Climate warming and disease risks for terrestrial and marine biota. Science. 2002;296:2158–62. http://dx.doi.org/10.1126/science.1063699
9. Lebarbenchon C, Brown SP, Poulin R, Gauthier-Clerc M, Thomas F. Evolution of pathogens in a man-made world. Mol Ecol. 2008;17:475–84. http://dx.doi.org/10.1111/j.1365-294X.2007.03375.x
10. Lorch JM, Meteyer CU, Behr MJ, Boyles JG, Cryan PM, Hicks AC, et al. Experimental infection of bats with *Geomyces destructans* causes white-nose syndrome. Nature. 2011;480:376–8. http://dx.doi.org/10.1038/nature10590

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Electronic School Absenteeism Monitoring and Influenza Surveillance, Hong Kong

To the Editor: Potentially useful public health interventions, such as school closure, need to be introduced in a timely manner during the evolution of an ongoing epidemic to substantially affect community transmission (*1*,*2*). In most traditional surveillance systems that include health care use data, however, considerable delays occur between data collection and feedback, which leads to suboptimal and untimely information for guiding evidence-based public health decisions. Newer syndromic surveillance approaches have been attempted to improve timeliness by targeting earlier events in the health-seeking pathway and by promoting real-time collection and processing of surveillance data by using modern information technology (*3*,*4*). Building on an existing platform of an electronic school management system, we developed an automated school absenteeism surveillance system for influenza-like illness (ILI) in Hong Kong and evaluated its performance using data collected from March 2008 through June 2011. The Institutional Review Board of the University of Hong Kong/Hospital Authority, Hong Kong West Cluster, approved the study.

We collaborated with a commercial vendor that develops and provides online learning platforms and management systems for educational institutions, including 337 primary and secondary schools in Hong Kong, attended by children 6–18 years of age. Invitations to participate in the new absenteeism system were sent to all schools subscribing to the electronic school management system, and 62