plant, and pulmonary infection with M. kansasii developed in 2 recipients of lung transplants (5). There is also a case report of systemic M. bovis infection developing in a patient with relapsing B chronic lymphocytic leukemia after administration of alemtuzumab (10).

Although we believe that alemtuzumab is responsible for the severe immunosuppression that predisposed these patients to M. haemophilum infection, other explanations are plausible. For example, patient 1 had received rituximab and cyclophosphamide for 6 months. These drugs, in addition to his underlying disease of chronic lymphocytic leukemia, may have predisposed him to M. haemophilum infection. However, his lesions did not appear until he received alemtuzumab. In patient 2, the immunosuppression associated with his transplant may have predisposed the patient to M. haemophilum infection.

This report identifies M. haemophilum as an opportunistic pathogen in patients who have received alemtuzumab. We recommend that all patients who have received at least 1 dose of alemtuzumab, and who have undiagnosed tender skin lesions located over the extremities, be evaluated by using appropriate techniques to isolate M. haemophilum. Communication with microbiology laboratory staff concerning appropriate methods for detection of the organism is crucial.

Mini Kamboj, Eddie Louie, Timothy Keihn, Genovefa Papanicolaou, Michael Glickman, and Kent Sepkowitz

Author affiliations: Memorial Sloan-Kettering Cancer Center, New York, New York, USA (M. Kamboj, T. Keihn, G. Papanicolaou, M. Glickman, K. Sepkowitz); and New York University, New York (E. Louie)

DOI: 10.3201/eid1411.071321

References


Address for correspondence: Mini Kamboj, Department of Medicine, Memorial Sloan-Kettering Cancer Centers, 1275 York Ave, Box 9, New York, NY 10021, USA; email: kambojm@mskcc.org

Prior Evidence of Putative Novel Rhinovirus Species, Australia

To the Editor: Briese et al. (1) are to be congratulated for their delineation of the global geographic presence of human rhinovirus (HRV) strains similar to those reported in 2006 from one third of cases of an otherwise pathogen-negative respiratory outbreak in New York. Of equal importance is the temporal occurrence of these strains. Although it is intriguing to suggest, on the basis of limited sequence data, that these strains were circulating at least 2 centuries earlier (1), Briese et al. neglect to acknowledge empirical evidence that what we now call HRV-C strains circulated before 2004–2005. Unculturable PCR-positive rhinoviruses were reported in 1993; however, more compelling is the fact that subgenomic sequence and phylogenetic data were reported from Belgium (2), Australia (3), and then New York (4).

The Belgian noncoding sequences were reported in 2006 but originated from specimens collected in 1998–1999. Australian coding sequences from 2003 to 2004 were assigned, for the first time, to a novel clade called HRV-A2, reflecting both their phylogenetic isolation and branching from the known HRV-A strains (3).

It can be deduced that NY-041 and NY-060, strains from the 2004 New York winter outbreak, are variants (>98% amino acid identity) of the first characterized HRV-A2 strain, HRV-QPM (4,5). More recently, we proposed that the HRV-A2 strains diverged sufficiently to meet several of the International Committee on Taxonomy of Viruses criteria for classifying a putative new species, HRV-C (6).

It is an exciting time for those interested in rhinoviruses. With increased implementation of multiplexed screening approaches (such as the MassTag PCR), or by simply including a specific
and sensitive PCR for all known strains (7), further details of the geographic and temporal extent of the neglected rhinoviruses should soon be available. Better identification may finally enable accurate characterization of the clinical, economic, and social impact (8) of HRV infection.

Ian M. Mackay,
Stephen B. Lambert,
Peter K. McErlean,
Cassandra E. Faux,
Katherine E. Arden,
Michael D. Nissen,
and Theo P. Sloots

Author affiliations: Sir Albert Sakzewski Virus Research Centre, Herston, Queensland, Australia; University of Queensland, Brisbane, Queensland, Australia; email: ian.mackay@uq.edu.au

DOI: 10.3201/eid1411.080725

References


Address for correspondence: Ian M. Mackay, Sir Albert Sakzewski Virus Research Centre, Queensland Paediatric Infectious Diseases Laboratory, c/o Royal Children’s Hospital, Herston Rd, Herston, Queensland 4029, Australia; email: ian.mackay@uq.edu.au

In Response: We appreciate the enthusiasm for our recent publication highlighting the global distribution of a long-unrecognized third clade of rhinoviruses. Robust, sequence-based clock estimates with associated confidence limits indicate that these viruses have been circulating for hundreds of years (1), consistent with the presence of such viruses in historic samples. As isolates from various collections are analyzed in informative regions (e.g., virus protein [VP] 4/2 or VP1), we will undoubtedly find examples in which human rhinoviruses (HRVs) could have been classified as members of the new species HRV-C but were not because the characteristics that define HRV-C were not yet appreciated or because only noncoding sequences had been analyzed. Indeed, we anticipate that waxing interest in HRVs may well lead to the discovery of additional clades.

There has been discussion in the field as to whether the novel sequences represent a sublineage HRV-A2 of the classified species HRV-A (2,3), as Mackay et al. had proposed, or whether they should be considered as representatives of a third species of HRV (4,5). The International Committee on Taxonomy of Viruses (ICTV) is charged with the recognition and naming of taxonomic entities. Thus, we provisionally designated our sequences as a novel clade distinct from HRV-A and HRV-B (4) and submitted a proposal to ICTV with data supporting the recognition of HRV-C as a third species of rhinovirus. The proposal was recently approved by the ICTV Study Group on Picornaviruses (Europic May 2008 meeting in Sitges, Spain). Irrespective of taxonomic discourse, we agree with Mackay and colleagues that molecular analyses of as-yet-uncultured HRVs are fascinating and have potential to reveal unexpected insights into the role of HRVs in disease.

Thomas Briese,
Gustavo Palacios, W. Ian Lipkin,
Neil Renwick, Marietjie Venter,
Richard G. Jarman,
Samuel R. Dominguez,
Kathryn V. Holmes,
and Edward C. Holmes

Author affiliations: Columbia University, New York, New York, USA (T. Briese, G. Palacios, W.I. Lipkin); Rockefeller University, New York (N. Renwick); University of Pretoria, Pretoria, South Africa (M. Venter), Armed Forces Research Institute of Medical Sciences, Bangkok, Thailand (R.G. Jarman); University of Colorado Denver School of Medicine, Aurora, Colorado, USA (S.R. Dominguez, K.V. Holmes); Pennsylvania State University, University Park, Pennsylvania, USA (E.C. Holmes); and Fogarty International Center, Bethesda, Maryland, USA (E.C. Holmes)

DOI: 10.3201/eid1411.081088

References

LETTERS


Address for correspondence: Thomas Briese, Center for Infection and Immunity, Mailman School of Public Health, Columbia University – Epidemiology, 722 W 168th St, New York, NY 10032, USA; email: thomas.briese@columbia.edu

Errata—Vol. 14, No. 9

In Forest Fragmentation as Cause of Bacterial Transmission among Primates, Humans, and Livestock, Uganda (T.L. Goldberg et al.), 2 errors occurred. In Table 3, the numerical values are not in the right positions. The corrected table is available from www.cdc.gov/EID/content/14/09/1375-T3.htm. In the same article, Figures 3 and 4 were inadvertently reversed. This has also been corrected in the online version of the article (available from www.cdc.gov/EID/content/14/9/1375.htm).

In Neurobrucellosis in Stranded Dolphins, Costa Rica (G. Hernández-Mora et al.), the name of co-author Elias Barquero-Calvo was misspelled. Several other editing changes to the online version of the article (available from www.cdc.gov/eid/content/14/9/1430.htm) have also been made upon the authors’ request.

In Texas Isolates Closely Related to Bacillus anthracis Ames (L.J. Kenefic et al.), 3 author names were inadvertently omitted from the submitted article. They are Carla P. Trim, Jodi A. Beaudry, and James M. Schupp; each is from Northern Arizona University, Flagstaff, Arizona, USA. The complete author list as it should have appeared on the article: Leo J. Kenefic, Talima Pearson, Richard T. Okinaka, Wai-Kwan Chung, Tamara Max, Carla P. Trim, Jodi A. Beaudry, James M. Schupp, Matthew N. Van Ert, Chung K. Marston, Kathy Gutierrez, Amy K. Swinford, Alex R. Hoffman, and Paul Keim. The corrected article is available online from www.cdc.gov/EID/content/14/9/1494.htm.

In Clindamycin-Resistant Clone of Clostridium difficile PCR Ribotype 027, Europe (D. Drudy et al.), the Figure contained errors. The correct version appears in the online version of this article (available from www.cdc.gov/EID/content/14/9/1485.htm) and is reprinted below.

We regret any confusion these errors may have caused.