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 cyclophosamide for 6 months. These drugs, in addition to his underlying disease of chromic 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.

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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 Belgium 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.

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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.

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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 Elías 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. Hoffmaster, 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.

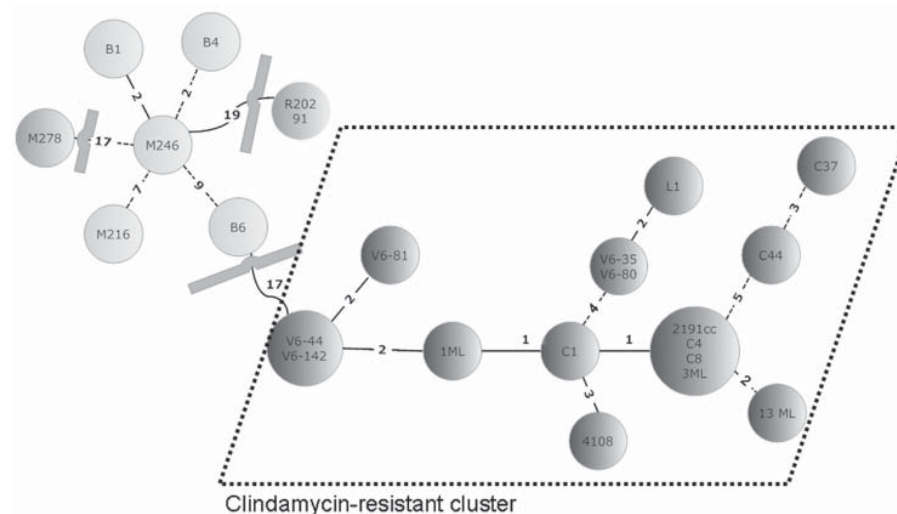We regret any confusion these errors may have caused.

Figure. Minimal spanning tree of 23 *Clostridium difficile* isolates. In the circles, the individual isolates are mentioned. The numbers between the circles represent the summed tandem repeat differences (STRDs) between multiple-locus variable-number tandem-repeat analysis types. Straight lines represent single-locus variants, dashed lines double-locus variants. Curved lines represent triple-locus variants. Two related clusters can be discriminated: the light gray cluster (isolates B1, B4, M246, B6, and M216) and the cluster within dotted lines (isolates V6–44, V6–142, V6–80, L1, 2191cc, C4, C8, 3ML, C44, C37, and 13ML) The isolates in the light gray cluster are sensitive to clindamycin; isolates in the cluster surrounded by dashed lines are resistant. Two isolates (M278 and R20291) did not belong to a cluster but were more related to the sensitive cluster than to the resistant cluster. Genetically related clusters were defined by an STRD ≤10.