

Detection of Newly Described Astrovirus MLB1 in Stool Samples from Children

To the Editor: We read with interest the article by Finkbeiner et al. describing an epidemiologic survey of newly described astrovirus MLB1 (AstV-MLB1) conducted in the United States in 2008 (1). This study was an extension of recently published reports of characterization of AstV-MLB1 from a fecal sample obtained in Australia in 1999 (2,3). These studies provide evidence of a divergent group of astroviruses and their etiologic association with human disease.

However, the occurrence of a MLB1-like AstV in humans has already been documented. Walter identified a novel AstV in an 8-month-old child with diarrhea in Mexico in 1991 (4). In that study, Walter screened fecal samples for AstVs by using a variety of techniques. Sequencing of selected PCR products identified a unique virus that had typical AstV morphologic appearance, but was nonreactive with human AstV-specific monoclonal or polyclonal antibodies. Phylogenetic analysis of fragments of open reading

frame 1a (ORF1a) and ORF2 genome regions of this virus strain (M3363) showed that it was only distantly related to other mammalian AstVs, including human AstVs (4). This sequence divergence from canonical human AstVs suggested that M3363 might have been transmitted from an animal reservoir (4,5).

When we reanalyzed ORF1a of M3363, we found that this strain was actually an MLB1-like AstV with >98% amino acid similarity to prototype and strains from the United States (Figure) that dated back to 1991. The fact that such closely related viruses were found in a scattered temporal and spatial pattern in children may indicate that MLB1-like AstVs represent a true human enteric virus, which was probably overlooked in the absence of adequate diagnostic reagents and protocols.

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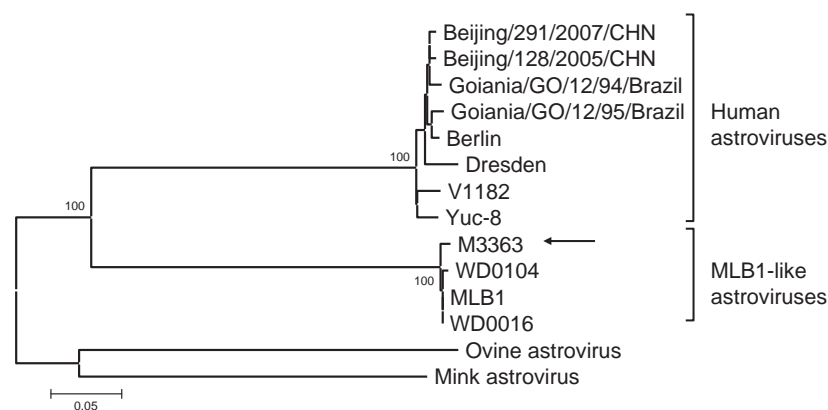


Figure. Neighbor-joining tree based on partial sequences of open reading frame 1a protein of human and animal astroviruses. Amino acid sequences of the Mexican M3363 strain (arrow) were obtained from Walter (4); other sequences were obtained from GenBank. Bootstrap values ≥ 90 are indicated. Scale bar is proportional to genetic distance and indicates nucleotide substitutions per site.

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In Response: We thank Bányai et al. (1) for drawing attention to the unpublished data of Walter (2), which was not part of the peer-reviewed literature at the time we described the complete genome of astrovirus MLB1 (3) or when we described our epidemiologic survey of stools collected in St. Louis, Missouri, USA (4). The results of Walter extend the known geographic range of astrovirus MLB1 to include Mexico, thus supporting our recent proposal that astrovirus MLB1 is likely to be globally widespread (4). We look forward to including the partial sequence generated by Walter in future analyses of astrovirus MLB1 genetic diversity. We strongly encourage Bányai et al. to submit their sequence data to any

of the publicly accessible and searchable international sequence databases such as GenBank, the European Molecular Biology Nucleotide Sequence Database, or the DNA Database of Japan so that it can be readily accessed by the scientific community.

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Optimal Therapy for Multidrug-Resistant *Acinetobacter baumannii*

To the Editor: I read with interest the article by Doi et al. about a lung transplant patient presumed to have *Acinetobacter baumannii* ventilator-associated pneumonia (1), but some points deserve comment. *A. baumannii* is a relatively avirulent organism that frequently colonizes body fluids. For multidrug-resistant strains, antimicrobial drug selection is limited. Resolution of this patient's pulmonary infiltrates suggests that they were not caused by *A. baumannii* that persisted in respiratory secretions. Because *A. baumannii* persisted in this patient's respiratory secretions, colistin and cefepime were given. Colistin is an antimicrobial drug with low resistance potential; but when given by inhalation, it may lead to drug resistance (2,3).

Doi et al. stated that the patient's *A. baumannii* strain lacked susceptibility to all available antimicrobial drugs but that cefepime and tigecycline were intermediately susceptible (MICs 16 µg/mL and 2.0 µg/mL, respectively) (1). Intermediate susceptibility may also be interpreted as relatively susceptible when achievable serum or tissue concentrations exceed the MIC. The article did not mention the dosages of colistin, tigecycline, and cefepime. A 2-g dose of cefepime given intravenously results in peak serum levels of ≈163 µg/mL with a relatively low volume of distribution (0.29 L/kg), which would not be expected to eradicate *A. baumannii* in respiratory secretions. High-dose intravenous tigecycline (initial dose of 200 mg followed by 100 mg daily) has been used to treat *A. baumannii*, achieving peak concentrations of ≈3 µg/mL, which exceed the isolate's MIC of 2 µg/mL, and a high volume of distribution (8 L/kg), which would be expected to eradicate *A. baumannii* in respiratory secretions.

Optimal treatment for *A. baumannii* depends on susceptibility, pharmacokinetic principles, and site of infection. For optimal effectiveness, cefepime and tigecycline should have been given at high doses. To prevent potential resistance, antimicrobial drugs should not be given by inhalation (3). The alleged advantage of inhalation therapy is high local drug concentrations, but concentrations in some alveoli may be subtherapeutic (3). If possible, tigecycline should not be used to treat *A. baumannii* infections; however, if it is used, high doses should be given to optimize its pharmacokinetic attributes (4,5).

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