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Picobirnaviruses in the Human Respiratory Tract

To the Editor: Picobirnaviruses (family *Picobirnaviridae*) are nonenveloped, double-stranded RNA viruses of vertebrates with a bisegmented genome. Segment 1 (2.2–2.7 kb) encodes the capsid protein, and segment 2 (1.2–1.9 kb) encodes the RNA-dependent RNA polymerase. On the basis of sequence diversity in segment 2, picobirnaviruses are classified into 2 genogroups (1–4). Picobirnaviruses have been detected in fecal samples from humans with and without gastroenteritis; in patients co-infected with known enteric pathogens, including rotaviruses, caliciviruses, and astroviruses (1,4); and in a wide range of animals, such as pigs, calves, dogs, monkeys, and snakes. The pathogenicity of picobirnaviruses largely remains to be determined, but studies in immunocompromised persons suggest that picobirnaviruses may be opportunistic enteric pathogens (5,6).

Recently, we identified picobirnaviruses in the respiratory tract of pigs in Asia, and this identification expanded the knowledge on the tropism and host range of picobirnaviruses (7). No respiratory or other clinical signs were observed in these pigs at the time of sampling, making it unclear whether picobirnaviruses are indeed respiratory pathogens (7). To determine whether picobirnaviruses could also be present in the human respiratory tract, we performed a diagnostic genogroup I picobirnavirus PCR, with degenerated primers, that targeted the RNA-dependent RNA polymerase coding region (1,4,8) on 309 bronchoalveolar lavage specimens collected from 309 patients with respiratory disease of unknown origin in the Netherlands during 2003–2006. (All study procedures were performed in compliance with relevant laws and institutional

guidelines and in accordance with the Declaration of Helsinki.)

Samples from 3 patients were confirmed by sequencing to be positive for genogroup I picobirnaviruses. To determine genetic relationships between human genogroup I picobirnaviruses from the respiratory tract and genogroup I picobirnaviruses detected in wastewater and in human and porcine fecal samples, we constructed a phylogenetic tree on the basis of a ≈165-nt fragment of the RNA-dependent RNA polymerase gene as described (8) (Figure, Appendix, wwwnc.cdc.gov/EID/article/18/9/12-0507-F1.htm). Before tree construction, 75 groups were created from the ≈300 available picobirnavirus sequences by using FastGroup II (10). Because the average pair-wise Jukes-Cantor distance was 0.46, a neighbor-joining tree was created by using the Jukes-Cantor model, with a bootstrap replication of 1,000 (Figure). One of the 3 genogroup I picobirnavirus sequences found in this study, PBVI/Homo sapiens/VS2000057/2003, showed <95% sequence identity with previously described picobirnavirus sequences and is shown as a separate branch in the phylogenetic tree. The genogroup I picobirnavirus nucleotide sequences from the respiratory tracts of persons in the Netherlands showed 58% to 97% similarity with each other. They belonged to different phylogenetic clades and did not group with other picobirnaviruses according to year of isolation or host species.

In conclusion, the identification of new picobirnaviruses in respiratory tract samples from pigs (7) prompted us to look for the presence of picobirnaviruses in the respiratory tracts of humans. Genogroup I picobirnaviruses could be identified in some of the bronchoalveolar lavage specimens obtained from patients with unexplained respiratory disease in the Netherlands. This observation expands our knowledge of picobirnaviruses in humans

and provides a clear example of how epidemiologic baseline information on virus host range and tropism in animals may provide indications for the presence of similar viruses in the same organ system of humans. To clarify the epidemiology and pathogenicity of picobirnaviruses in humans, additional surveillance should be carried out in persons with and without respiratory and enteric disease.

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New Delhi Metallo- β -Lactamase 4-producing *Escherichia coli* in Cameroon

To the Editor: The metallo- β -lactamase (MBL) group of enzymes inactivates many β -lactam antimicrobial drugs. First identified from a *Klebsiella pneumoniae* strain recovered from a patient hospitalized in India, the New Delhi metallo- β -lactamase-1 (NDM-1), particularly in *Enterobacteriaceae*, is now the focus of worldwide attention (1). Whereas India and Pakistan were considered as the main reservoirs of the *bla*_{NDM-1} gene (2) that produces this MBL, several NDM-1-producing *Enterobacteriaceae* isolates have been reported from the Balkan states and the Middle East, suggesting that those areas might be secondary reservoirs (2).

Since 2010, 3 NDM-1 point-mutation variants have been described (3–5). The first variant, NDM-2, was identified from an *Acinetobacter baumannii* isolate collected from a patient transferred from a hospital in Egypt to Germany (4). Subsequently, a clonal dissemination of NDM-2-producing *A. baumannii* was described in Israel (6). The second variant, NDM-4, which was identified in *Escherichia coli* from a patient hospitalized in India, possessed a higher carbapenemase activity compared with NDM-1 (5). The most recent variant, NDM-5, was identified in *E. coli* from a patient who had a history of hospitalization in India (3).

As recommended for the detection of carbapenemase producers (7), a rectal swab specimen was collected from a patient transferred from Cameroon to France. The *E. coli* strain FEK was isolated from the specimen.