LETTER TO THE EDITOR

Emerging human respiratory syncytial virus genotype ON1 found in infants with pneumonia in Beijing, China

Guanglin Cui, Yuan Qian, Runan Zhu, Jie Deng, Linqing Zhao, Yu Sun and Fang Wang

Emerging Microbes and Infections (2013) 2, e22; doi:10.1038/emi.2013.19; published online 24 April 2013

Dear Editor,

Human respiratory syncytial virus (HRSV) is the leading viral agent that causes acute lower respiratory infections (ALRIs) in infants, young children, and vulnerable adults. HRSVs have been classified as subgroups A and B by both antigenic and genetic analyses. Both HRSV subgroups A and B have been circulating in China and have caused large outbreaks of severe ALRIs in young children.1,2 Studies on the molecular epidemiology of HRSVs have mainly focused on the second hypervariable region (HVR2), which contains multiple epitopes, in the ectodomain of the major attachment (G) protein.

To date, 11 HRSV-A genotypes (GA1-GA7, SAA1, NA1-NA2, and ON1) and 20 HRSV-B genotypes (GB1-GB4, BA1-BA10, SAB1-SAB4, URU1, and URU2) have been identified based on the sequence analyses of HVR2.3,4 Genotype BA of subgroup B, with a 60-nucleotide duplication in HVR2, was first identified in Buenos Aires and rapidly became the predominant strain of subgroup B.5 Recently, a new genotype, ON1, for subgroup A, with a 72-nucleotide duplication in HVR2, was first reported in Canada.4 However, it is not known whether this new genotype could replace the current genotype and become the predominant one or whether this emerging ON1 could show more pathogenesis, which is information that would be of high interest. This letter reports the first identification of genotype ON1 sequences in clinical specimens from children with ALRI in Beijing in 2012 during the investigation of the subgroup distribution and genetic variability of HRSV in paediatric patients. The rapid global transmission and genetic variation of the ON1 genotype are also discussed.

A total of 3391 nasal pharyngeal aspirates (NPAs) were collected from children with ALRIs hospitalised at the Affiliated Children’s Hospital, Capital Institute of Pediatrics in Beijing, China, during the period from July 2012 to December 2012. Of these NPA samples, 285 (8.4%, 285/3391) were positive for HRSV by direct fluorescent assay (Diagnostic Hybrids, Ohio, USA), including 11 samples for which the virus was successfully isolated in Hep-2 cells. A total of 95 of the 285 HRSV-positive NPA samples were randomly selected for subtyping with a genotype ON1 sequence emerged in Beijing, China, in 2012.5,6

The alignment of the deduced amino acids of the ON1 HVR2 sequences available in GenBank revealed that amino acid substitutions have occurred in the duplicated segment (Figure 1). For example, three strains from Japan (Chiba-C24031, JPN/P6540/2012, and 12221/AN/2012) and one German strain (WUE/16397/12) contained 3 to 4 amino acid substitutions in the duplicated region and are, thus, less closely related to the original ON1 strain than the 3 Beijing ON1 strains. This difference reflects the high genetic variability of the ON1
strains to escape herd immunity in different populations. It can be predicted that, as found for the BA genotype, several branches of the ON1 genotype will form in the future due to the rapid accumulation of changes in the sequence.

The virulence and immunogenicity of the ON1 strains may change because of the 72 nucleotide G gene duplication. Ongoing surveillance of this genotype around the world is necessary to understand not only the evolution of this important virus but also the relationship between epidemic progress and pathogenesis.

This study was reviewed and approved by the Institutional Review Board of the Capital Institute of Pediatrics.

ACKNOWLEDGMENTS

This work was supported by grant no. Z111107056811041 from the Beijing Municipal Science and Technology Commission.

1 Deng J, Qian Y, Liu C et al. [Etiological study on an outbreak of epidemic asthma-like pneumonia of infants and children in Ruyang county, Henan province, China.] Chin J Pediatr 2003; 39: 75–75. Chinese.
2 Deng J, Qian Y, Zhu R, Wang F, Zhao L. [Surveillance for respiratory syncytial virus subtypes A and B in children with acute respiratory infections in Beijing during 2000 to 2006 seasons.] Zhonghua Er Ke Za Zhi 2006; 44: 924–927. Chinese.

3 Arnott A, Yong S, Mardy S et al. A Study of the Genetic Variability of Human Respiratory Syncytial Virus (HRSV) in Cambodia Reveals the Existence of a New HRSV Group B Genotype. J Clin Microbiol 2011; 49: 3504–3513.

4 Eshaghi A, Duvvuri VR, Lai R et al. Genetic variability of human respiratory syncytial virus a strains circulating in ontario: a novel genotype with a 72 nucleotide G gene duplication. PLoS One 2012; 7: e32807.

5 Trento A, Casas I, Calderon A et al. Ten years of global evolution of the human respiratory syncytial virus BA genotype with a 60-nucleotide duplication in the G gene. J Virol 2010; 84: 7500–7512.

6 Shobugawa Y, Sairo R, Sano Y et al. Emerging genotypes of human respiratory syncytial virus subgroup A among patients in Japan. J Clin Microbiol 2009; 47: 2475–2482.

7 Khor CS, Sam IC, Hooi PS, Chan YF. Displacement of predominant respiratory syncytial virus genotypes in Malaysia between 1989–2011. Infect Genet Evol 2013; 13: 357–360.

8 Lee WI, Kim YJ, Kim DW, Lee HS, Lee HY, Kim K. Complete genome sequence of human respiratory syncytial virus genotype a with a 72-nucleotide duplication in the attachment protein gene. J Virol 2010; 84: 7500–7512.

9 Valley-Omar Z, Muloiwa R, Hu NC, Eley B, Hisao NY. Novel Respiratory Syncytial Virus Subtype ON1 among Children, Cape Town, South Africa, 2012. Emerg Infect Dis 2013; 19: 668–670.

10 Trento A, Viegas M, Galvano M et al. Natural history of human respiratory syncytial virus inferred from phylogenetic analysis of the attachment (G) glycoprotein with a 60-nucleotide duplication. J Virol 2006; 80: 975–984.

This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivative Works 3.0 Unported License. To view a copy of this license, visit http://creativecommons.org/licenses/by-nc-nd/3.0