LETTERS

Religious Opposition to Polio Vaccination

To the Editor: In 1988, the Global Polio Eradication Initiative was formed, with the aim of reducing infection with poliomyelitis virus. Two decades later in 2008, a total of 1,625 children contracted acute flaccid paralysis caused by poliovirus infection (1). This finding represented a 150% increase over the number of cases in 2007 (1) and resulted in the reemergence of polio as one of the world’s deadliest infections. As of 2009, polio remains endemic to 4 countries (India, Nigeria, Pakistan, and Afghanistan); in 2008, cases were also detected in 14 other countries.

Religious opposition by Muslim fundamentalists is a major factor in the failure of immunization programs against polio in Nigeria (2), Pakistan (3) and Afghanistan (4). This religious conflict in the tribal areas of Pakistan is one of the biggest hindrances to effective polio vaccination. Epidemiologists have detected transmission of wild poliovirus from polio-endemic districts in Afghanistan, most of which are located in the southern region of this country bordering Pakistan, to tribal areas of Pakistan (4). This transmission has resulted in new cases of polio in previously polio-free districts. The local Taliban have issued fatwas denouncing vaccination as an American ploy to sterilize Muslim populations. Another common superstition spread by extremists is that vaccination is an attempt to avert the will of Allah. The Taliban have assassinated vaccination officials, including Abdul Ghani Marwat, who was the head of the government’s vaccination campaign in Bajaur Agency in the Pakistani tribal areas, on his way back from meeting a religious cleric (5). Over the past year, several kidnappings and beatings of vaccinators have been reported. Vaccination campaigns in Nigeria and Afghanistan have also been hampered by Islamic extremists, especially in the Nigerian province of Kano in 2003, which has resulted in the infection returning to 8 previously polio-free countries in Africa (2).

Before the Global Polio Eradication Initiative in 1988, a total of 1,000 persons/day were infected with a virus that would cripple them for the rest of their lives (6). To eradicate the disease, 1 major factor will be to gain support of those susceptible to fundamentalist propaganda. Islam is a progressive religion, and religious leaders should be asked to support polio eradication programs. The Imam of the Ka’aba and other influential religious figures should be asked to highlight the plight of children with polio. Vaccinators operating in conflict-ridden areas should be provided protection so that they are better able to perform their duties. Not only will children in these areas be safer, but the disease will not be exported to areas where wild polio transmission has been interrupted by vaccination. Further study of the attitudes of Muslim populations toward vaccination is needed.

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References

1. World Health Organization. Wild poliovirus weekly update. 2009 Jan 14 [cited 2009 Jan 17]. Available from http://www.polioeradication.org/casecount.asp
2. Kapp C. Surge in polio spreads alarm in northern Nigeria. Rumours about vaccine safety in Muslim-run states threaten WHO’s eradication programme. Lancet. 2003;362:1631–2. DOI: 10.1016/S0140-6736(03)14826-X
3. Ahmad K. Pakistan struggles to eradicate polio. Lancet Infect Dis. 2007;7:247. DOI: 10.1016/S1473-3099(07)70066-X
4. Centers for Disease Control and Prevention. Resurgence of wild poliovirus type 1 transmission and consequences of importation—21 countries, 2002–2005. MMWR Morb Mortal Wkly Rep. 2006;55:145–50.

Recurrent Human Rhinovirus Infections in Infants with Refractory Wheezing

To the Editor: Respiratory infections frequently cause illness among pediatric patients worldwide. Human rhinovirus (HRV) is a cause of acute respiratory tract infections (RTIs) (1); co-infections with other respiratory viruses such as respiratory syncytial virus (RSV) or influenza virus have also been reported. HRV strains have been subdivided into 2 genetic subgroups (HRV-A and HRV-B); a third genetic subgroup has been recently discovered (2–7). However, understanding of the epidemiology of novel HRV infection among Asian pediatric patients with respiratory illness (4,5) and association with recurrent wheezing and asthma has been limited (8–10).

We retrospectively analyzed 289 nasopharyngeal aspirates (NPAs) obtained from 286 pediatric patients admitted to Chulalongkorn Memorial Hospital in Bangkok, Thailand, during 2006–2007. The study was reviewed and approved by the Institutional Review Board, Chulalongkorn University,
Bangkok, Thailand. Each specimen was tested for common respiratory viruses such as RSV, HRV, parainfluenza 1–3, influenza A and B viruses, adenovirus, human metapneumovirus, and human bocavirus. On the basis of phylogenetic analysis of the VP4 region, we identified 2 patients who had been admitted with 5 episodes of acute RTIs and subsequent recurrent wheezing associated with HRV-A and HRV-C.

The first patient was an infant girl whose first episode of breathing difficulty was at 5 months of age; a diagnosis of RSV bronchiolitis was made. She was hospitalized with respiratory failure and required mechanical ventilation for 3 days. At 6 months, she had pneumonia and wheezing. At 14 months, she had a low-grade fever, mild cough, breathing difficulty, and wheezing. While she was hospitalized for 7 days, a novel HRV-C (FJ435240) was identified by seminested PCR, and RSV was detected by reverse transcription–PCR. Seven months later, she had recurrent wheezing and respiratory distress. Virologic analysis indicated that she was co-infected with a divergent HRV-C strain (FJ435256) and influenza A virus. Nucleotide sequence identity score between the 2 isolated strains of HRV-C indicated a different cluster (identity score 70.1%).

The second patient was an infant boy with a diagnosis of acute bronchiolitis at 7 months of age. His underlying condition was congenital heart disease and an allergy to cow’s milk protein. Initial NPA showed HRV-A (FJ435274) and RSV by reverse transcription–PCR. Two months later, he had viral pneumonia and acute exacerbation of his reactive airway disease. He received systemic corticosteroids and a nebulized bronchodilator. His clinical course was complicated by 3 episodes of supraventricular tachycardia that were controlled with adenosine and cordarone. NPA was again positive for HRV-A (FJ435284). Three weeks later, he had upper respiratory tract symptoms, low-grade fever, and protracted cough; blood oxygen saturation was low and respiratory distress had rapidly increased. An NPA showed HRV-C (FJ435299). He received systemic corticosteroids and was discharged with corticosteroid inhalation. Comparison between 2 HRV-A strains isolated showed 82.5% nucleotide sequence identity. The sequence of the HRV-C strain also displayed 51.2% and 61.1% nucleotide identity to FJ435274 and FJ435284, respectively. Results of phylogenetic analysis are shown in the Figure.

A novel HRV-C infection in association with acute lower RTI was diagnosed in the first patient during her fourth and fifth hospitalizations. The 2 strains isolated are within the same genetic group and display 70% nucleotide similarity, which suggests that this infant was infected with 2 different virus strains. The second patient was infected with HRV-A during his first hospitalization. His condition subsequently progressed to refractory wheezing. Both patients were co-infected with RSV when a diagnosis of infection with lower RTIs was made. Two HRV-A strains detected in the

Figure. Phylogenetic analysis of nucleotide sequences of the virus capsid protein (VP4) region of 5 human rhinovirus (HRV) strains (shown in boldface) isolated from 289 nasopharyngeal aspirate specimens, including those of 2 infants with refractory wheezing (C1 and C2), on the basis of amplification of VP4/2 by seminested reverse transcription–PCR. The tree was constructed by using the neighbor-joining method and Kimura’s 2-parameter distance with bootstrap replicated from 1,000 trees by using MEGA 4.0 (www.megasoftware.net). Scale bar indicates number of nucleotide substitutions per site. Human enterovirus (HEV) was used as an outgroup for comparison.
second patient were within the same subgroup, but similarity in nucleotide sequences was only 82.5%. This result suggests that this patient was infected with 2 different virus strains of HRV-A and a strain of HRV-C.

Comparison of the HRV-A strains with the HRV-C strain showed that they belonged to different subgroups and had low similarity for nucleotide sequences. The second patient had 3 distinct rhinovirus infections over 3 months, and each was associated with illness requiring hospitalization. Both patients had underlying diseases, reactive airway diseases, and repeated episodes of RTI that may have rendered them vulnerable to reinfection, compromising their immune responses.

Complete coding sequences of HRV-A and HRV-C have been determined (4,7). However, little is known about their involvement in the pathogenesis of recurrent wheezing in young children. According to recent reports, HRV-C has been detected in hospitalized children with lower RTI in the People’s Republic of China (5). Possible association of novel infection with HRV and exacerbation of asthma in children has also been reported (6). We report HRV-A and HRV-C co-infections in conjunction with other respiratory viruses, such as RSV, as a potential cause of recurrent wheezing in infants with acute lower RTIs. Co-infections with HRV-A and HRV-C may contribute to increased virulence and subsequent pathogenesis of other respiratory viruses. Additional studies will be required to further explore the clinical role of novel HRVs.

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