C. psittaci, *C. pneumoniae*, *C. abortus*, and *C. suis* (*6*–*10*). To our knowledge, this member of the family *Chlamydiaceae* has been seen in amphibians, but not in other vertebrate hosts. The 16S rRNA analysis showed this taxon to belong to a clade with *Candidatus* Clavochlamydia salmonicola, a taxon found in fish. The phylogenetic position of the novel taxon in the family *Chlamydiaceae* thus roughly reflects the phylogenetic relation between the host species, providing evidence for host–bacterium co-evolution in the family *Chlamydiaceae*.

Although the results obtained are not conclusive with regard to the pathogenic potential of this novel genus and species of Chlamydiales, we were not able to attribute the clinical signs to any known disease. We therefore suggest that we discovered a novel bacterial taxon with possible considerable impact on amphibian health.

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Novel Variant of Beilong Paramyxovirus in Rats, China

To the Editor: In 2003, two cDNA strands were identified in a human mesangial cell line during experimental screening for genes upregulated by angiotensin II (*1*). Sequence analysis showed that the strands were homologous to the matrix, fusion, and phosphoprotein genes of paramyxoviruses, suggesting the possibility of a novel paramyxovirus (*2*,*3*). Subsequent research found that these sequences, believed to originate from human kidney mesangial cell lines, were not amplifiable from such cell lines or human kidney samples but were amplifiable from a rat kidney mesangial cell line (*4*). Isolation and complete genome sequencing of the virus confirmed that it was a novel paramyxovirus of the subfamily *Paramyxovirinae*, named Beilong virus (BeV).

BeV is most closely related to J virus, discovered in autoculture of kidney tissue from a moribund house mouse, and Tailam virus from Sikkim rats (*5*,*6*). Because J virus and Tailam virus were found to originate in rodents and BeV was amplifiable from a rat kidney mesangial cell line, we hypothesized that BeV was a novel paramyxovirus originating in rats. To test this hypothesis, we conducted a territorywide molecular epidemiologic study of rats and other mammals to evaluate this novel paramyxovirus.

We tested 4,130 samples from 1,398 animals collected from various locations in Hong Kong, People's Republic of China, during September 2008–August 2009 (Table). These included 480 kidney, spleen, respiratory swab, and anal swab samples from 120 asymptomatic rats (105 brown rats [*Rattus norvegicus*] and 15 black rats [*R. rattus*]). To

prevent cross contamination, we used disposable scalpels, decontaminated the work surface, and used sterile gloves for each tissue sample. We performed RNA extraction and reverse transcription PCR by using strategies we previously published for discovery and epidemiologic study of paramyxoviruses (*6*–*9*).

We performed BeV screening by PCR amplification of a 440-bp fragment of the large (L) gene, located at the 5′ end of the genome and used specific primers (LPW9739 GGAGGATTCCCTCATAGAG AA-3′ and LPW9741 5′-CTCATAT GTATTTACATTTAAACCA-3′). The PCR mixture (25 μL) contained cDNA, PCR buffer (10 mmol/L Tris-HCl, pH 8.3, 50 mmol/L KCl, 3 mmol/L $MgCl_2$, and 0.01% gelatin), 200 μM of each dNTP, and 1.0 units of *Taq* polymerase (Applied Biosystems, Foster City, CA, USA). The mixtures were amplified in 60 cycles at 94°C for 1 min, 48°C for 1 min, and 72°C for 1 min; and a final extension at 72°C for 10 min in an automated thermal cycler (Applied Biosystems).

BeV in the positive samples was confirmed by amplifying a 318-bp fragment of the nucleocapsid (N) gene of BeV, located at the 5′ end of the genome; by using specific primers (LPW10723 5′-TATATGGTTGAGAT YCTNATHGA-3′ and LPW10408 5′-CCATKGCRTAGCTCCADAG-3′) and experimental conditions described above. We confirmed the specificities of the primers by testing samples positive for Tailam virus (*6*), which all showed negative results.

Results of reverse transcription PCR for a 440-bp fragment in the large gene of BeV were positive for 40 kidney and 9 spleen samples from 43 rats (40 brown rats and 3 black rats). Sequencing and phylogenetic analysis showed 6–13 base differences between the sequences and the corresponding region in the large gene of BeV (GenBank accession no. NC_007803), suggesting that this is a novel variant of BeV in our locality.

Results of reverse transcription PCR for a 318-bp fragment in the N gene of BeV were positive in the same 40 kidney and 9 spleen samples from the 43 rats. Sequencing and phylogenetic analysis showed 1–9 base differences between the sequences and the corresponding region in the N gene of BeV. The kidney and spleen samples were positive in 4 brown rats and 2 black rats. The L and N gene sequences amplified from the kidney and spleen samples were identical in 5 of the 6 rats. However, in 1 brown rat, L and N gene sequences from the kidney and spleen samples differed by 4 and 6 bases, respectively, suggesting the possibility of 2 strains of BeV in the same rat. None of the samples from the other mammals were positive. The authenticity of the results was supported by identical results from 2 independent genes of the BeV genome, sequence variations in the L and N genes from the positive samples, and negative results from all other mammals tested.

This study suggests that BeV and its variants are endemic in brown rats and black rats, but it is not known whether transmission is vertical or horizontal. Detection of BeV and Tailam virus in kidney and spleen samples, but not respiratory or anal swabs, suggested that they are probably systemic viruses excreted in urine. Phylogenetic and genomic evidence support the grouping of BeV, Tailam virus, and J virus into a new genus of *Paramyxovirinae*. Distinctly, the genomes of all 3 viruses contain 8 genes (3′ -N-P/V/C-M-F-SH-TM-G-L-5′). We speculate that the ancestor of these closely related paramyxoviruses infected the common ancestor of rats and mice, with subsequent co-evolution and divergence with the host.

Table. Mammals screened for Beilong virus, People's Republic of China, September 2008–August 2009

Animal	Sample type*
Bats, n = 502	Throat swab, rectal swab
Cats, n = 130	Nasal swab, rectal swab, urine, blood
Cattle, n = 100	Nasal swab, rectal swab, liver, buffy coat, plasma
Dogs, n = 149	Nasal swab, rectal swab, urine, blood
Hamsters, n = 49	Throat swab, intestinal swab, kidney
Pigs, n = 100	Nasal swab, rectal swab, liver, blood
Wild urban rodents, n = 120	Rectal swab, throat swab, rectal swab, kidney, spleen
Wild rural rodents, n = 248	Throat swab, rectal swab

*All sample types listed for each animal were collected except wild rodents: 237 throat swab and 246 rectal swab samples were collected from 248 wild rodents.

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Pneumococcal Serotype–specific Unresponsiveness in Vaccinated Child with Cochlear Implant

To the Editor: Approximately 100,000 persons worldwide have received cochlear implants for hearing loss, and more children now receive them than ever (*1*). Such children have a >30-fold increased risk for pneumococcal meningitis than the background rate (*1*,*2*). During 2006–2010, children born in the United Kingdom were offered the 7-valent pneumococcal conjugate vaccine (PCV7) at 2, 4, and 13 months of age (*3*). Those at high risk for invasive pneumococcal disease (IPD) were additionally offered the 23-valent pneumococcal polysaccharide vaccine (PPV23) at 2–5 years (*3*). We describe a fully vaccinated child with a cochlear implant in whom recurrent pneumococcal meningitis developed, caused by a vaccine serotype (i.e., vaccine failure). The child continues to have nonprotective antibody concentrations against the infecting serotype, despite further pneumococcal vaccination.

A previously healthy, appropriately vaccinated 23-month-old girl (Table) had a cochlear device implanted in the right ear after receiving (through the universal newborn hearing screening program) a diagnosis of profound, bilateral, sensorineural deafness. Two weeks later, she exhibited fever, lethargy, and drowsiness. On hospital admission, she had a peripheral blood leukocyte count of 19.3 × 10^9 cells/L, a neutrophil count of 17.0 × 10^9 cells/L, and C-reactive protein level 75 mg/L. Meningitis was diagnosed, and she received intravenous ceftriaxone but was too ill for a lumbar puncture. Blood cultures subsequently grew fully sensitive *Streptococcus pneumoniae*, later confirmed as serotype 4 by the national reference laboratory. She was discharged after 14 days of receiving intravenous antimicrobial drugs without complications.

At 24 months, she received a fourth dose of PCV7. Blood tests 1 month later showed good antibody responses to 6 PCV7 serotypes but not to serotype 4, which did not reach the putative protective level of ≥0.35 μg/mL antibody threshold (Table). At 28 months, she received 1 dose of PPV23 per national guidelines (*3*). Four months later, she was brought to the hospital with fever, rigors, drowsiness, and vomiting. Blood tests showed a leukocyte count of 24.4 × 10^9 cells/L, neutrophil count of 21.6 × 10^9 cells/L, and C-reactive protein level of 272 mg/L. Lumbar puncture performed the next day showed 890 leukocytes/mL (predominantly polymorphs), cerebrospinal fluid glucose level <1.1 mmol/L, protein level of 1.0 g/L, gram-positive diplococci on Gram staining, and positive PCR results for pneumococci, although cerebrospinal fluid culture was negative.

A blood culture grew fully sensitive *S. pneumoniae*, also confirmed by the national reference laboratory as serotype 4. She recovered after receiving intravenous ceftriaxone and oral rifampin for 2 weeks, followed by 4 weeks of oral amoxicillin and rifampin. She then received prophylactic oral penicillin for maintenance. Subsequently, an abdominal ultrasound confirmed the presence of a spleen, and her immunoglobulin concentrations were in the normal range. At 35 months, she received another dose of PCV7, and a blood test 1 month later showed variable but high responses to 6 of the PCV7 serotypes and no response to serotype 4 (Table). Moreover, nasopharyngeal swab specimens, obtained when the patient was 39 months old and receiving penicillin prophylaxis, were positive for serotype 4.