

sequencing. This infection rate is within the range (13.5%–90%) that has been reported for *R. felis* infecting *Ctenocephalides* fleas in Brazil and Uruguay (2,3,7). Sixteen (72.7%) cats contained *R. felis*-reactive antibodies; 4 of them showed titers to *R. felis* at least 4-fold higher than those to the other 5 rickettsial strains, findings that enabled us to technically conclude that these cats were exposed to *R. felis* or a closely related organism (1,7,9). Our finding of 70% *R. felis* infection in fleas infesting the cats indicates that cats acquired the infection through infected fleas. However, the mechanism of *R. felis* transmission by fleas is yet to be demonstrated under experimental conditions.

To our knowledge, the presence of *R. felis*, or a spotted fever group *Rickettsia* species, has not been reported in Chile. Recent investigations have provided clinical and serologic evidence of canine (10) and human (K. Abarca and J. Lopez, unpub. data) infection by spotted fever rickettsia in Chile, confirmed by IFA that used *R. conorii* commercial antigen. Since substantial serologic cross-reaction occurs between *R. conorii* and *R. felis* antigens (1), *R. felis* could be causing infection in dogs or humans in Chile.

This research was financially supported by Clínica Veterinária Alcantara (Chile), Fundação de Amparo à Pesquisa do Estado de São Paulo (Brazil), and Conselho Nacional de Desenvolvimento Científico e Tecnológico (Brazil).

**Marcelo B. Labruna,\***  
**Maria Ogrzewalska,\***  
**Jonas Moraes-Filho,\***  
**Paulina Lepe,†**  
**Jose Luis Gallegos,†**  
**and Javier López†**

\*University of São Paulo, São Paulo, Brazil; and †Alcantara Veterinary Clinics, Santiago, Chile

## References

1. Parola P, Davoust B, Raoult D. Tick- and flea-borne rickettsial emerging zoonoses. *Vet Res.* 2005;36:469–92.
2. Horta MC, Pinter A, Cortez A, Soares RM, Gennari SM, Schumaker TTS, et al. *Rickettsia felis* (Rickettsiales: Rickettsiaceae) in *Ctenocephalides felis felis* (Siphonaptera: Pulicidae) in the State of São Paulo, Brazil. *Arq Bras Med Vet Zoot.* 2005;57:321–5.
3. Venzal JM, Perez-Martinez L, Felix ML, Portillo A, Blanco JR, Oteo JA. Prevalence of *Rickettsia felis* in *Ctenocephalides felis* and *Ctenocephalides canis* from Uruguay. *Ann N Y Acad Sci.* 2006;1078:305–8.
4. Pornwiroon W, Pourciau SS, Foil LD, Macaluso KR. *Rickettsia felis* from cat fleas: isolation and culture in a tick-derived cell line. *Appl Environ Microbiol.* 2006;72:5589–95.
5. Wedincamp J Jr, Foil LD. Infection and seroconversion of cats exposed to cat fleas (*Ctenocephalides felis* Bouche) infected with *Rickettsia felis*. *J Vector Ecol.* 2000;25:123–6.
6. Hawley JR, Shaw SE, Lappin MR. Prevalence of *Rickettsia felis* DNA in the blood of cats and their fleas in the United States. *J Feline Med Surg.* 2007;9:258–62.
7. Horta MC. Epidemiological study of *Rickettsia felis* in areas endemic and non-endemic for Brazilian spotted fever in the state of São Paulo [in Portuguese]. São Paulo: University of São Paulo (PhD thesis); 2006.
8. Guedes E, Leite RC, Prata MCA, Pacheco RC, Walker DH, Labruna MB. Detection of *Rickettsia rickettsii* in the tick *Amblyomma cajennense* in a new Brazilian spotted fever-endemic area in the state of Minas Gerais. *Mem Inst Oswaldo Cruz.* 2005;100:841–5.
9. Labruna MB, Horta MC, Aguiar DM, Cavalcante GT, Pinter A, Gennari SM, et al. Prevalence of *Rickettsia* infection in dogs from the urban and rural areas of Monte Negro Municipality, western Amazon, Brazil. *Vector Borne Zoonotic Dis.* 2007;7:249–56.
10. López J, Abarca K, Azocar T. Clinical and serological evidence of canine rickettsiosis in Chile [in Spanish]. *Rev Chil Infect.* 2007;24:189–93.

Address for correspondence: Marcelo B. Labruna, Laboratório de Doenças Parasitárias, Departamento de Medicina Veterinária Preventiva e Saúde Animal, Faculdade de Medicina Veterinária e Zootecnia, Universidade de São Paulo, Av Prof Dr. Orlando Marques de Paiva 87, São Paulo, SP, Brazil 05508-270; email: labruna@usp.br

## Possible Typhoon-related Melioidosis Epidemic, Taiwan, 2005

**To the Editor:** Melioidosis is a severe infection caused by *Burkholderia pseudomallei*. This organism is present in tropical and subtropical regions where melioidosis is endemic. Before 1995, melioidosis was rare in Taiwan. In 2001, when the annual number of cases of melioidosis in Taiwan was determined to be 1–3 per year from 1996 to 2000, the idea was first proposed that the disease was endemic (1).

From July 21 through August 24, 2005, an unusually large number (54) of melioidosis cases occurred in Taiwan. This number exceeded the average case number of 9.4 per year from 2001 to 2004. Since this outbreak appeared to be a common-source epidemic, all persons were suspected of becoming infected from this source at the same time.

To determine this common source, we investigated the role of Typhoon Haitang, which hit Taiwan on July 18 and 19, 2005, and resulted in heavy rainfall. Because the date of this typhoon overlapped the incubation period (1–21 days in most cases) (2) and rain is a factor in outbreaks of melioidosis (3), Typhoon Haitang may have been the cause.

All 57 clinical strains of *B. pseudomallei* isolated during this outbreak were typed by pulsed-field gel electrophoresis (PFGE) DNA macrorestriction analysis (4). A higher incidence rate (8.86% per million) and clonal diversity (9 PFGE types) of *B. pseudomallei* were observed in the subtropical zone (south of 23.5°N) of Taiwan than in the temperate zone (north of 23.5°N) (0.18% per million and 2 PFGE types) (Table). Because clonal diversity in outbreaks of melioidosis is characteristic of extreme weather (5), these data support possible involvement of the typhoon in this outbreak.

Table. PFGE patterns of clinical isolates of *Burkholderia pseudomallei* obtained before and after Typhoon Haitang, Taiwan, 2005\*

PFGE types	No. clinical isolates					
	Before (Jan–Jun)			After (Jul–Sep)		
	Subtropical zone, no.	Temperate zone, no.	Total, no. (%)	Subtropical zone, no.	Temperate zone, no.	Total, no. (%)
S1	0	0	0	31	0	31 (57.4)
S1a	0	0	0	1	0	1 (1.9)
S2	0	0	0	0	1	1 (1.9)
S3	0	0	0	3	1	4 (7.4)
S3a	2	0	2 (66.6)	10	0	10 (18.5)
S3b	0	0	0	2	0	2 (3.7)
S3c	0	0	0	2	0	2 (3.7)
S4	0	0	0	1	0	1 (1.9)
S5	1	0	1 (33.3)	0	0	0
S6	0	0	0	1	0	1 (1.9)
S7	0	0	0	0	1	1 (1.9)
Total	3	0	3 (100)	51	3	54 (100)
Incidence rate†	0.52	0	0.13‡	8.86§	0.18§	2.38‡

\*Typhoon Haitang hit Taiwan on July 18, 2005. Logistic regression analyses evaluating the associations were conducted with SAS software version 6.12 (SAS Institute, Cary, NC, USA). PFGE, pulsed-field gel electrophoresis.

†Per million population. At the end of June 2005, the population of subtropical counties was 5,753,647, and the population of temperate zone counties was 16,936,127. In 2005, the population at risk for melioidosis in Taiwan was 22,689,774. Data obtained from the Department of Taiwan Internal Affairs.

‡Odds ratio (OR) 17.99, 95% confidence interval [CI] 5.63–57.54,  $p = 0.0001$ .

§OR 0.019, 95% CI 0.006–0.060,  $p = 0.0001$ .

Because *B. pseudomallei* can grow at a temperature as low as 4°C (6) and the possible spread of melioidosis into temperate zones has been reported (7), the epidemic distribution of *B. pseudomallei* in the temperate zone of Taiwan is still not clear. Determining the role of Typhoon Haitang in exposing microbes distributed in the soil, as described by Thomas et al. (8), may provide evidence of differences in the distribution of *B. pseudomallei* in the soil of subtropical and temperate zones of Taiwan.

Most clones of *B. pseudomallei* in this study were isolated in the subtropical zone of Taiwan, but 2 clones (S2 and S7) that each caused 1 case of melioidosis were found in the temperate zone. The 2 patients infected with the S2 and S7 clones lived ≈200 km north of the boundary between the subtropical and temperate zones and had not crossed this boundary for ≥3 years. Although the incubation period for *B. pseudomallei* may be as long as 62 years (9), and the presence of this organism in the temperate zone before Typhoon Haitang cannot be excluded, we believe that these 2 patients are newly infected cases in the temperate zone.

The 2 predominant clones in this outbreak, S1 and S3a, caused 30 and 10 cases of melioidosis, respectively. Since the appearance of predominant clones, a case-cluster of melioidosis been regarded as an indicator of contamination of an environmental source (5). This clustering suggests contamination of soil in the subtropical zone of Taiwan with the S1 and S3a clones.

Patients in this outbreak had severe symptoms of melioidosis, including fever (38/54), cough (16/54), pneumonia (12/54), septic shock (9/54), shortness of breath (4/54), and chest pain (2/54). Eleven of the 54 patients died. Because few patients had skin injuries and most (32/54) had a short incubation period of 1–9 days, inhalation may have been the route of transmission. Increased inhalation of *B. pseudomallei* has been reported in cases of melioidosis during heavy monsoonal rain and wind (3).

In conclusion, Typhoon Haitang likely had a role in an outbreak of melioidosis in the subtropical zone of Taiwan that showed high incidence rates and clonal diversity of isolates of *B. pseudomallei*. Our findings showed differences in distribution of *B. pseudomallei* in the soil of subtropical and

temperate zones of Taiwan. *B. pseudomallei* clones found only in the temperate zone warrant further study to help prevent their spread. Some clones predominant in the subtropical zone may be suitable for vaccine development.

Hsun-Pi Su,\*†  
Chen-Ying Chou,†  
Shin-Chan Tzeng,†  
Tien-Lin Ferng,† Ya-Lei Chen,‡  
Yao-Shen Chen,§  
and Tung-Ching Chung\*

\*National Chung-Hsing University, Taichung, Taiwan, Republic of China; †Centers for Disease Control, Taipei, Taiwan, Republic of China; ‡National Kaoshiung Normal University, Kaoshiung, Taiwan, Republic of China; and §Kaoshiung Veterans General Hospital, Kaoshiung, Taiwan, Republic of China

## References

1. Hsueh PR, Teng LJ, Lee LN, Yu CJ, Yang PC, Ho SW. Melioidosis: an emerging infection in Taiwan? *Emerg Infect Dis.* 2001;7:428–33.
2. Currie BJ, Fisher DA, Howard DM, Burrow JN, Selvanayagam S, Snelling PL. The epidemiology of melioidosis in Australia and Papua New Guinea. *Acta Trop.* 2000;74:121–7.

3. Currie BJ, Jacups SP. Intensity of rainfall and severity of melioidosis, Australia. *Emerg Infect Dis.* 2003;9:1538–42.
4. Popovic T, Schmink S, Rosenstein NA, Ajello GW, Reeves MW, Plikaytis B. Evaluation of pulsed-field gel electrophoresis in epidemiological investigation of meningococcal disease outbreak caused by *Neisseria meningitidis* serogroup C. *J Clin Microbiol.* 2001;39:75–85.
5. Cheng AC, Jacups SP, Gal D, Mayo M, Currie BJ. Extreme weather events and environmental contamination are associated with case-clusters of melioidosis in the Northern Territory of Australia. *Int J Epidemiol.* 2006;35:323–9.
6. Chen YS, Chen SC, Kao CM, Chen YL. Effects of soil pH, temperature and water content on the growth of *Burkholderia pseudomallei*. *Folia Microbiol (Praha).* 2003;48:253–6.
7. Perret JL. Melioidosis: a tropical time bomb that is spreading. *Med Trop (Mars).* 1997;57:195–201.
8. Thomas AD, Forbes Faulkner J, Parker M. Isolation of *Pseudomonas pseudomallei* from clay layers at defined depths. *Am J Epidemiol.* 1979;110:515–21.
9. Athan E, Allworth AM, Engler C, Bastian I, Cheng AC. Melioidosis in tsunami survivors. *Emerg Infect Dis.* 2005;11:1638–9.

Address for correspondence: Tung-Ching Chung, Department of Veterinary Medicine, National Chung-Hsing University, Taichung, Taiwan, Republic of China; email: tchung@dragon.nchu.edu.tw

## Instructions for Authors

### Letters

Letters commenting on recent articles as well as letters reporting cases, outbreaks, or original research are welcome. Letters commenting on articles should contain no more than 300 words and 5 references; they are more likely to be published if submitted within 4 weeks of the original article's publication. Letters reporting cases, outbreaks, or original research should contain no more than 800 words and 10 references. They may have one Figure or Table and should not be divided into sections. All letters should contain material not previously published and include a word count.

## Human Bocavirus in Infants, New Zealand

**To the Editor:** In 2005, a parvovirus, subsequently named human bocavirus (HBoV), was discovered in respiratory samples taken from infants and children hospitalized at Karolinska University Hospital, Sweden, with lower respiratory tract infection (1). HBoV has since been identified in infants and children with respiratory illness in >17 countries, at frequencies ranging from 1.5% to >18.0%.

In the past decade New Zealand has experienced increasing bronchiolitis hospitalization rates, currently >70 admissions per 1,000 infants. To determine the contribution of HBoV to New Zealand's bronchiolitis disease prevalence, we tested samples collected from infants hospitalized with community-acquired bronchiolitis (2) during 3 consecutive winter epidemics (June to October, 2003; July to October, 2004; and June to October, 2005) in Wellington, NZ, for HBoV by PCR. The Central Regional Ethics Committee approved the study. Written, informed consent was obtained from the parent or guardian.

Demographic, clinical, and laboratory data were collected during hospitalization. Ethnicity of those who ascribe to >1 group was determined by using a national census method that prioritizes ethnicity as follows: Māori>Pacific>Other>New Zealand European. Oxygen requirement was determined to be the best measure of bronchiolitis severity (2). Infants who needed assisted ventilation or continuous positive airway pressure were classified severe; those who required oxygen supplementation moderate; and infants who were hospitalized but did not require supplemental oxygen mild.

Nucleic acid was extracted from thawed nasopharyngeal aspirates (stored at 80°C) by using a High Pure

Viral Nucleic Acid kit (Roche Diagnostics, Auckland, NZ). The HBoV nonstructural protein (NP-1) gene was amplified by using primers 188F (5'-GAGCTCTGTAAGTACTATTAC-3') and 542R (5'-CTCTGTGTTGACTGAATACAG-3') (1) with Expand High Fidelity DNA Polymerase (Roche Diagnostics, Basel, Switzerland) for 35 cycles. Products (354 bp) were purified and sequenced from primers 188F and 542R on an ABI3730 Genetic Analyzer by using a BigDye Terminator version 3.1 Ready Reaction Cycle Sequencing kit (Applied Biosystems, Foster City, CA, USA). Sequences were submitted to GenBank under accession nos. EF686006–13.

Alignments of NP-1 gene sequences from nucleotides (nt) 2410–2602, and NP-1 predicted amino acid sequences from amino acids (aa) 1–97 were constructed by using ClustalW version 1.83 (available from [www.ebi.ac.uk/tools/clustalw/index.html](http://www.ebi.ac.uk/tools/clustalw/index.html)) and compared with HBoV prototype sequences from GenBank (DQ00495–6). Nasopharyngeal aspirates were also screened for respiratory syncytial virus (RSV) by reverse transcription–PCR (RT-PCR) and nested PCR (3) and for human metapneumovirus (4), influenza A (H1, H3), and influenza B by RT-PCR (5).

Eight (3.5%) of 230 samples, collected from infants hospitalized with bronchiolitis during the 2003–2005 winter epidemic seasons, were positive for HBoV. In 5 HBoV-positive infants no other pathogens were identified, but RSV was detected in 3 (Table). The 8 HBoV-positive infants had a median age of 9.5 months, and the male:female ratio was 1:1. The median length of hospital stay was 5.5 (range 1–16) days.

As expected, because HBoV NP-1 is highly conserved, sequence variation among New Zealand isolates and the prototype Stockholm ST-1 and ST-2 (1) NP-1 sequences was limited. Alignments of the partial NP-1 sequence (nt 2410–2602) of New Zea-