these ticks suggest that human habitation near bats and their associated tick colonies could pose a public health risk. Growth in laboratory animals or culture could help isolate these novel organisms for further studies to establish the distribution and public health implications of this newly identified *Borrelia* sp.

Acknowledgments

We thank M.J.R. Gilchrist and the University of Iowa Hygienic Laboratory for support, and G. Hettrick and J. Gathany for help with the figure.

This work was supported in part by the Division of Intramural Research, National Institute of Allergy and Infectious Diseases, National Institutes of Health.

James S. Gill,*
Amy J. Ullmann,†
Amanda D. Loftis,‡
Tom G. Schwan,§
Sandra J. Raffel,§
Merry E. Schrumpf, §
and Joseph Piesman†

*Iowa State University, Ames, Iowa, USA; †Centers for Disease Control and Prevention, Fort Collins, Colorado, USA; ‡Centers for Disease Control and Prevention, Atlanta, Georgia, USA; and §Rocky Mountain Laboratories of National Institutes of Health, Hamilton, Montana, USA

References

1. Dworkin MS, Schwan TG, Anderson DE. Tick-borne relapsing fever in North America. Med Clin North Am. 2002;86: 417–33.
2. Schwan TG, Raffel SJ, Schrumpf ME, Porcella SF. Diversity and distribution of *Borrelia hermsii.* Emerg Infect Dis. 2007;13:436–42.
3. Schwan TG, Raffel SJ, Schrumpf ME, Policastro PF, Rawlings JA, Lane RS, et al. Phylogenetic analysis of the spirochetes *Borrelia parkeri* and *Borrelia turicatae* and the potential for tick-borne relasping fever in Florida. J Clin Microbiol. 2005;43:3851–9.
4. Gill JS, Rowley WA, Bush PJ, Viner JP, Gilchrist MJ. Detection of human blood in the bat tick *Carios* (*Ornithodoros*) *kelleyi* (Acari: Argasidae) in Iowa. J Med Entomol. 2004;41:1179–81.
5. Loftis AD, Gill JS, Schriefer ME, Levin ML, Eremeeva ME, Gilchrist MJ, et al. Detection of *Rickettsia, Borrelia*, and *Bartonella* in *Carios kelleyi* (Acari: Argasidae). J Med Entomol. 2005;42:473–80.
6. Zeidner NS, Burkot TR, Massung R, Nicholson WL, Dolan MC, Rutherford JS, et al. Transmission of the agent of human granulocytic ehrlichiosis by *Ixodes spinipalpis* ticks: evidence of an enzootic cycle of dual infection with *Borrelia burgdorferi* in northern Colorado. J Infect Dis. 2000;182:616–9.
7. Bacon RM, Pilgard MA, Johnson BJ, Raffel SJ, Schwan TG. Glycerophosphodiester phosphodiesterase gene (*glpQ*) of *Borrelia lonestari* identified as a target for differentiating *Borrelia* species associated with hard ticks (Acari: Ixodidae). J Clin Microbiol. 2004;42:2326–8.
8. Johnson BJ, Happ CM, Mayer LW, Piesman J. Detection of *Borrelia burgdorferi* in ticks by species-specific amplification of the flagellin gene. Am J Trop Med Hyg. 1992;47:730–41.
9. Porcella SF, Raffel SJ, Anderson DE Jr, Gilk SD, Bono JL, Schrumpf ME, et al. Variable tick protein in two genomic groups of the relapsing fever spirochete *Borrelia hermsii* in western North America. Infect Immun. 2005;73:6647–58.

Address for correspondence: James S. Gill, 313 N Mt Vernon Dr, Iowa City, IA 52245, USA; email: bugmangill@yahoo.com

KI and WU Polyomaviruses in Children, France

To the Editor: Two new members of the *Polyomaviridae* family, provisionally named *Karolinska Institutet virus* (KIPyV) and *Washington University virus* (WUPyV), have been recently discovered (*1*,*2*). These new polyomaviruses were identified by screening human respiratory secretions with molecular tools. KIPyV and WUPyV are genetically related to the BK virus and the JC virus, the 2 known members of the family *Polyomaviridae* that affect humans.

In France, from November 2006 through June 2007, nasopharyngeal aspirates were obtained from 537 children who were <5 years of age and who had acute respiratory tract disease. The aspirates were tested for respiratory syncytial virus (RSV); influenza virus types A and B; parainfluenza virus types 1, 2, and 3; and adenoviruses (AdVs) by direct immunofluorescence assay. The aspirates were also tested for human metapneumovirus (HMPV) by an enzyme immunoassay (HMPV EIA, Biotrin, Lyon, France) and for the human bocavirus (HBoV) by PCR (*3*). Samples were placed on MRC5 cell monolayers for virus isolation.

Nucleic acid extracts were tested for KIPyV and WUPyV DNA by PCR. KIPyV detection was performed by using a nested PCR approach that targeted the VP1 capsid gene as described by Allander et al. (*1*). For WUPyV detection, primers targeted the predicted 3′ end of the large T antigen coding region as described by Gaynor et al. (*2*). The amplification specificity was assessed by sequencing the PCR product; sequences were deposited in GenBank (WUPyV isolates, accession no. AM778536–48; KIPyV isolates, accession no. AM849808–10).

At least 1 type of virus was identified for 271 (50.5%) children. The viruses found were RSVs in 175 (32.6%), HBoVs in 54 (10.0%), HMPVs in 50 (9.3%), rhinoviruses/enteroviruses in 11 (2%), influenza A viruses in 8 (1.5%), human AdVs in 6 (1.1%), and parainfluenza type 3 viruses in 4 (0.7%) samples. Aspirates were not tested for coronaviruses; detection of rhinoviruses/enteroviruses was likely low because cell culture is less sensitive than molecular assays.

A total of 13 (2.4%) samples were positive for WUPyV; of these 4 (30.8%) were co-infected with another virus. The 13 children with samples positive for WUPyV had a median age

of 11.2 (2–48) months and the male/female sex ratio was 2.2. KIPyV DNA was detected in samples from 3 (0.6%) boys (ages 10, 18 and 30 months); 1 of those samples was co-infected with RSV and HMPV.

Sequences of WUPyV and KIPyV isolates varied little from each other and from other GenBank sequences, which suggests that these polyomaviruses are genetically conserved viruses. Clinical characteristics of children infected with WUPyV and KIPyV are retrospectively recorded (Table). All children recovered and were able to return home within 1 to 10 days, with the exception of 1 child. This child had been hospitalized since birth for congenital myopathy; nosocomial acquisition or vertical transmission of the WUPyV is suspected.

Our data are in agreement with the 2 original reports that show that the new KI and WU polyomaviruses may be detected in respiratory secretions from patients with respiratory diseases (*1*,*2*). WUPyV was detected in 2.4% of children <5 years of age who were hospitalized with respiratory tract disease, which is in accordance with the 2% incidence reported by Gaynor et al. (*2*). The 0.6% prevalence observed for KIPyV PCR is also in agreement with data reported from Sweden (*1*) and Australia (*4*). A seasonal change in the presence of WUPyV was not observed; however, all KIPyV isolates were found only during January.

KIPyV and WUPyV were mainly detected in samples from children with lower respiratory tract disease, such as bronchiolitis or atypical pneumonia, and in samples from children with exacerbated asthma. These preliminary data on the likely role of these viruses as respiratory pathogens need to be interpreted with caution. Aspirates were obtained only from those with observed symptoms; no asymptomatic controls were tested. Detection of WUPyV and KIPyV in respiratory samples may simply reflect a respiratory transmission route as previously suggested for BK virus and JC virus (*5*). Another virus was in aspirates from 31% of the children with KI and WU polyomaviruses. Substantial rates of codetection were also reported in the initial descriptions of both WUPyV and KIPyV (*1*,*2*). More recently, Bialasiewicz et al. reported a 25% rate of codetection of KIPyV with another pathogen (*4*). These high rates of co-

Table. Clinical findings for 13 children infected with WU polyomavirus and 3 children infected with KI polyomavirus*

Sex/age, mo	Copathogen	Fever, °C	CRP, mg/L	SaO_2, %	WBC, x 10^3 cells/μL	Chest radiograph findings	Diagnosis	Clinical signs and symptoms
WU polyomavirus								
M/2	–	37.9	8.3	NA	9.4	Hyperinflation	Pneumonia	Cough, dyspnea
M/12	–	39.2	112.0	NA	18.2	Hyperinflation, interstitial infiltrate	Bronchiolitis	Cough, dyspnea
F/20	–	38.7	56.0	NA	13.7	Hyperinflation	Atypical pneumonia	Cough, respiratory distress, diarrhea
F/12	–	37.8	15.0	96	12.3	Hyperinflation	Bronchiolitis	Cough, dyspnea, pharyngitis
M/10	–	39.0	90.3	NA	18.0	Normal	Idiopathic fever	Fever
F/14	–	38.9	20.8	95	11.0	Hyperinflation, interstitial infiltrate	Atypical pneumonia	Cough, wheezing, dyspnea
F/10	RSV	38.0	14.0	NA	17.2	Hyperinflation, interstitial infiltrate	Bronchiolitis	Cough, respiratory distress, dyspnea
M/6	RSV	38.0	NA	89	NA	Hyperinflation	Bronchiolitis	Respiratory distress, wheezing
M/48	HMPV	36.8	15.0	94	13.0	Hyperinflation	Asthma	Cough, respiratory distress
M/24	–	37.0	23.0	98	13.9	Hyperinflation	Asthma	Cough, respiratory distress
M/2	–	38.5	38.5	70	16.0	Normal	Idiopathic fever	Cough, fever
M/11	HBoV	39.0	5.5	94	6.2	Interstitial infiltrate	Atypical pneumonia	Cough, fever
M/10	–	37.0	10.0	97	18.0	Alveolar syndrome	Congenital myopathy	Respiratory distress
KI polyomavirus								
M/10	RSV, HMPV	39.0	NA	NA	NA	Hyperinflation	Bronchiolitis	Cough, wheezing, otitis, diarrhea
M/30	–	39.2	<5.0	NA	11.0	Interstitial infiltrate	Influenza-like pneumonia	Cough, dyspnea, otitis, sore throat
M/18	–	38.9	23.0	96	14.8	Hyperinflation, atelectasis	Asthma	Cough, dyspnea, wheezing

*CRP, C-reactive protein; SaO_2, oxygen saturation; WBC, white blood cells; NA, not available; HMPV, human metapneumovirus; RSV, respiratory syncytial virus; HBoV, human bocavirus.

infection raise questions about the real pathogenic role of these viruses.

As with other polyomaviruses, WUPyV and KIPyV could establish persistent and latent infections with likely asymptomatic reactivations (*5*), and detection of these viruses could also reflect a long-term shedding from previous acute episode. Recently published studies have not shown a pathogenic role for these new polyomaviruses in respiratory tract disease (*6*,*7*); however, more comprehensive studies are needed to elucidate whether both KIPyV and WUPyV have any clinical relevance.

Vincent Foulongne,* Natalie Brieu,* Eric Jeziorski,* Amandine Chatain,* Michel Rodière,* and Michel Segondy*

*Montpellier University Hospital, Montpellier, France

References

1. Allander T, Andreasson K, Gupta S, Bjerkner A, Bogdanovic G, Persson MAA, et al. Identification of a third polyomavirus. J Virol. 2007;81:4130–6.
2. Gaynor AM, Nissen MD, Whiley DM, Mackay IA, Lambert SB, Wu G, et al. Identification of a novel polyomavirus from patients with acute respiratory tract infections. PloS Pathog. 2007;3:e64.
3. Foulongne V, Olejnik Y, Elaertz S, Perez V, Rodière M, Segondy M. Human bocavirus in French children. Emerg Infect Dis. 2006;12:1251–3.
4. Bialasiewicz S, Whiley DM, Lambert SB, Wang D, Nissen MD, Sloots TP. A newly reported human polyomavirus, KI virus, is present in the respiratory tract of Australian children. J Clin Virol. 2007;40:15–8.
5. Randhawa P, Vats A, Shapiro R. The pathobiology of polyomavirus infection in man. Adv Exp Med Biol. 2006;577: 148–59.
6. Abed Y, Wang D, Boivin G. WU polyomavirus in children, Canada. Emerg Infect Dis 2007;13:1939–41.
7. Norja P, Ubillos I, Templeton K, Simmonds P. No evidence for an association between infections with WU and KI polyomaviruses and respiratory disease. J Clin Virol. 2007;40:307–11.

Address for correspondence: Vincent Foulongne, Laboratory of Virology, Hôpital St-Eloi, 34295 Montpellier, Cedex 5, France; email: v-foulongne@chu-montpellier.fr

Milk Replacers and Bovine Spongiform Encephalopathy in Calves, Japan

To the Editor: Milk replacers produced from a specific feed factory in Japan were suspected of being associated with a cluster of bovine spongiform encephalopathy (BSE) infection in calves. We conducted a case–control study to test this association.

In Japan, BSE infection has been confirmed in 32 calves as of the end of May 2007; 13 of these calves were born between December 1995 and August 1996. One BSE-infected calf was born in 1992 and had an atypical BSE phenotype (*1*). Because no BSE-infected calves were born in 1997 and 1998, we considered that those born in 1995 and 1996 formed an independent temporal cluster (Figure). Epidemiologic investigation showed that all 13 calves were fed milk replacers produced by a specific factory. Ten calves were born in Hokkaido, and 3 were born in the Kanto region, which is ≈800 km away from Hokkaido.

In the case–control study, all farms where the 13 BSE-infected calves were born and raised for at least 1 year were defined as case farms. Control farms were defined as dairy farms where no BSE calves had been reported. Candidates for control farms comprised 200 randomly selected farms, which were located in 23 prefectures where the milk replacers were distributed. We used a national cattle identification database for random selection. Veterinary officers from the local government interviewed farmers in November and December 2006 and requested that they complete a questionnaire on farming practices in 1996, including herd size and use of milk replacers and calf concentrates. For the case farms, information previously obtained from outbreaks was used. Of the 200 potential control farms, 154 farms were used as controls. Forty-six farms were excluded; 24 farmers did not respond or could not specify the use of milk replacers; and 22 farms had either closed or farmers did not respond for miscellaneous reasons.

Among the 154 control farms, 36 farms (23%) used the milk replacers from the specific factory, 89 farms (58%) used other milk replacers, and 29 farms (19%) did not use milk replacers. Since 1 case farm lacked

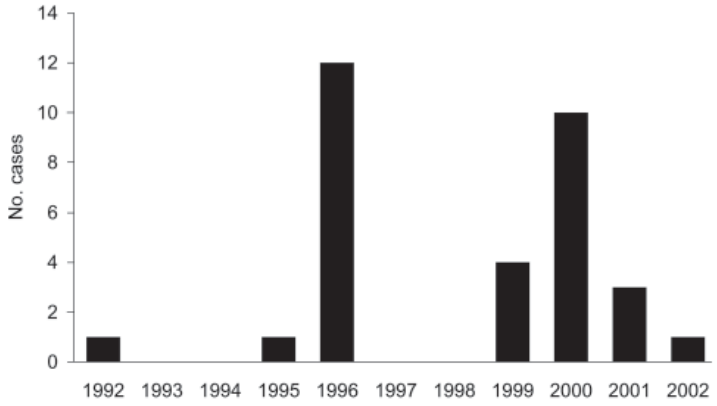

Figure. Number of cases of bovine spongiform encephalopathy by calves' birth year, Japan.