

Figure. Peripheral blood smears (buffy-coat preparation) showing variable-sized basophilic inclusions (arrows) in mononuclear cells from a 9-year-old boy with human monocytic ehrlichiosis, Carabobo, Venezuela. Dip Quick (Jorgensen Laboratories, Inc., Loveland, CO, USA) staining; magnification $\times 1,000$.

thrombocytopenia, hepatomegaly, and recent exposure to ticks. Although *Amblyomma americanum*, the main known vector of *E. chaffeensis*, has not been reported in Venezuela, *Rhipicephalus sanguineus* and *A. cajennense* are abundant in rural areas of Venezuela; their ability to be vectors should be investigated.

Acknowledgments

We are grateful to Jacqueline Dawson for providing the DH82 cell lines infected with *E. canis* and *E. chaffeensis*. We also thank Guillermo Comach for performing serologic testing, isolation, and PCR techniques for dengue virus.

This work was supported by a grant (no. FCS 96-014) from the Council for Scientific and Humanistic Development of the University of Carabobo.

María C. Martínez,*†
Clara N. Gutiérrez,*†
Franklin Monger,*

Johanny Ruiz,† Akemys Watts,*
Victor M. Mijares,* María G. Rojas,‡
and Francisco J. Triana-Alonso†

*University of Carabobo, Aragua, Venezuela; †Biomedical Research Institute, Maracay, Venezuela; and ‡Institute for Advanced Studies, Caracas, Venezuela

References

1. Paddock CD, Childs JE. *Ehrlichia chaffeensis*: a prototypical emerging pathogen. *Clin Microbiol Rev*. 2003;16:37–64.
2. Arraga-Alvarado C, Montero-Ojeda M, Bernardoni A, Anderson BE, Parra O. Human ehrlichiosis: report of the 1st case in Venezuela [in Spanish]. *Invest Clin*. 1996;37:35–49.
3. Gongora-Biachi RA, Zavala-Velasquez J, Castro-Sansores C, Gonzalez-Martinez P. First case of human ehrlichiosis in Mexico. *Emerg Infect Dis*. 1999;5:481.
4. Ripoll CM, Remondegui CE, Ordonez G, Arazamendi R, Fusaro H, Hyman MJ, et al. Evidence of rickettsial spotted fever and ehrlichial infections in a subtropical territory of Jujuy, Argentina. *Am J Trop Med Hyg*. 1999;61:350–4.
5. López J, Rivera M, Concha JC, Gatica S, Loeffholz M, Barriga O. Serologic evidence for human ehrlichiosis in Chile. *Rev Med Chil*. 2003;131:67–70.
6. da Costa PS, Valle LM, Brigatte ME, Greco DB. More about human monocytotropic ehrlichiosis in Brazil: serological evidence of nine new cases. *Braz J Infect Dis*. 2006;10:7–10.

7. Dawson JE, Biggie KL, Warner CK, Cookson K, Jenkins S, Levine JF, et al. Polymerase chain reaction evidence of *Ehrlichia chaffeensis*, an etiologic agent of human ehrlichiosis, in dogs from southeast Virginia. *Am J Vet Res*. 1996;57:1175–9.
8. Perez M, Rikihisa Y, Wen B. *Ehrlichia canis*-like agent isolated from a man in Venezuela: antigenic and genetic characterization. *J Clin Microbiol*. 1996;34:2133–9.
9. Perez M, Bodor M, Zhang C, Xiong Q, Rikihisa Y. Human infection with *Ehrlichia canis* accompanied by clinical signs in Venezuela. *Ann N Y Acad Sci*. 2006;1078:110–7.
10. Schutze GE, Buckingham SC, Marshall GS, Woods CR, Jackson MA, Patterson LE, et al. Human monocytic ehrlichiosis in children. *Pediatr Infect Dis J*. 2007;26:475–9.

Address for correspondence: Francisco J. Triana-Alonso, BIOMED-UC, Final Calle Cecilio Acosta, Cantarrana, Las Delicias, Maracay, Edo. Aragua, Venezuela; email: ftrianaalonso@yahoo.com

Resource Allocation during an Influenza Pandemic

To the Editor: Planning for pandemic influenza is accepted as an essential healthcare service and has included creation of national and international antiviral drug stockpiles and novel approaches to emergency vaccine development (1). The effectiveness of these strategies in a pandemic may be substantial but is unknown. More certain is that effective management of severe and complicated influenza will reduce deaths and that demand will exceed available treatment resources (2). Appropriate allocation of treatment resources is therefore essential, perhaps more important than any specific treatment such as administering antiviral medication to symptomatic patients. Re-

source allocation requires the following: 1) making clear societal decisions on the goals for healthcare resources; 2) conducting operational research to develop an evidence base to support the achievement of these goals; and 3) developing systems to capture and learn from new information in a pandemic to facilitate modification of the response as the characteristics of the pandemic emerge. Most societies have not yet addressed the first issue fully. In the United Kingdom, a Department of Health consultation on planning critical care during emergencies cites “the underpinning principle of providing the greatest good for the greatest number of people during the course of an emergency” and thus appears to support a capacity-to-benefit approach (3). Similarly, triage criteria developed in Canada, based on the Sequential Organ Failure Assessment (SOFA) score, exclude those persons believed to be too ill or otherwise unlikely to benefit from critical care (4). Even more important for most severely ill patients, however, will be deciding whether to admit them to the hospital at all. The UK pandemic-planning criteria currently recommend a scoring system for hospital admission based on an assessment of poor outcome rather than on capacity to benefit (2). Indeed, age >85 years and severe underlying cognitive impairment, which would rule out admission to critical care in Canada, would strongly favor admission to hospital care in the United Kingdom, the opposite of the situation for a younger cognitively intact person with similar disease severity. If tools are to be developed to support triage at all stages of the patient pathway in a pandemic, societies must consider the ethical issues raised (4,5), debate them, and take a position on the values that should underpin decision making in a pandemic.

Even when clear societal goals are established, much work remains to ensure that the healthcare community is equipped to steer healthcare

resources to deliver these effectively (6). Community-acquired pneumonia has been used as a surrogate for influenza to test predictive scoring systems for assessing severity and assisting triage decisions (7). Seasonal influenza epidemics would provide the most realistic setting available, in particular, if protocols were in place to test criteria when a relatively severe influenza season occurs. In addition to identifying criteria for setting priorities within influenza management, such testing will need to consider the balance of resources between influenza treatment and treatment of other usual noninfluenza conditions that will require emergency care during the pandemic. Decisions that must be made during a pandemic are complex, varying from when to stop major elective surgery so critical care capacity can be opened up, to how to triage those who have experienced major trauma and those with influenza. These decisions could differ from those same decisions made outside a pandemic, and an adequate evidence base is needed if they are to be of good quality.

The third component of our preparation for optimally deploying standard care in a pandemic is being able to change our approach quickly as new knowledge emerges. In the so-called Spanish influenza pandemic of 1918–19, the unfamiliar clinical course meant that influenza was not even considered when the first cases appeared (8), and expectations had to be revised concerning who was most vulnerable and at what stage in their clinical course they were most at risk. Therefore, healthcare professionals must develop and test the public health infrastructure to capture patient factors associated with outcome and treatment response during a pandemic and feed this information back into clinical practice rapidly and reliably, as occurred during the epidemic of severe acute respiratory syndrome (9). International collaboration will be important for sharing this work (10)

and developing useful tools early in a pandemic. Having recognized the risk for pandemic influenza, we must now complement the research into novel influenza treatments by addressing our knowledge gap on how best to use our resources to deliver optimal clinical care in the management of influenza guided by effective clinical surveillance.

**Karthikeyan Paranthaman,*
Christopher P. Conlon,†
Claire Parker,‡
and Noel McCarthy§**

*Public Health Department, Oxford, UK;

†John Radcliffe Hospital, Oxford, UK;

‡Jericho Health Centre, Oxford, UK; and

§Thames Valley Health Protection Unit, Oxford, UK

References

1. Mounier-Jack S, Coker RJ. How prepared is Europe for pandemic influenza? Analysis of national plans. *Lancet*. 2006;367:1405–11.
2. UK Health Departments. Pandemic flu: UK influenza pandemic contingency plan. October 2005 [cited 2007 Nov 20]. Available from <http://www.dh.gov.uk/assetRoot/04/12/17/44/04121744.pdf>
3. UK Health Departments. The NHS emergency planning guidance 2005: underpinning materials. Critical care contingency planning in the event of an emergency where the number of patients substantially exceeds normal critical care capacity. Best Practice Guidance. August 2006 [cited 2007 Nov 20]. <http://www.dh.gov.uk/assetRoot/04/13/76/19/04137619.pdf>
4. Christian MD, Hawryluck L, Wax RS, Cook T, Lazar NM, Herridge MS, et al. Development of a triage protocol for critical care during an influenza pandemic. *CMAJ*. 2006;175:1377–81.
5. Torda A. Ethical issues in pandemic planning. *Med J Aust*. 2006;185(Suppl):S73–6.
6. Reilly BM, Evans AT. Translating clinical research into clinical practice: impact of using prediction rules to make decisions. *Ann Intern Med*. 2006;144:201–9.
7. Challen K, Bright J, Bentley A, Walter D. Physiological-social score (PMEWS) vs. CURB-65 to triage pandemic influenza: a comparative validation study using community-acquired pneumonia as a proxy. *BMC Health Serv Res*. 2007;7:33.

8. World Health Organization. Avian influenza: assessing the pandemic threat. January 2005 [cited 2007 Nov 20] Available from <http://www.who.int/csr/disease/influenza/H5N1-9reduit.pdf>
9. Leung GM, Rainer TH, Lau FL, Wong IO, Tong A, Wong TW, et al. A clinical prediction rule for diagnosing severe acute respiratory syndrome in the emergency department. *Ann Intern Med.* 2004;141:333–42. Epub 2004 Aug 23.
10. Naylor CD, Chantler C, Griffiths S. Learning from SARS in Hong Kong and Toronto. *JAMA.* 2004;291:2483–7.

Address for correspondence: Karthikeyan Paranthaman, Specialist Registrar in Public Health, Public Health Department, Room 9 Richards Building, Old Road Campus, Oxford OX3 7LF, UK; email: karthik.paranthaman@oxfordshirepct.nhs.uk

Novel Relapsing Fever Spirochete in Bat Tick

To the Editor: Tick-borne relapsing fever in western North America is a zoonosis caused by spirochetes in the genus *Borrelia* that are transmitted by argasid ticks of the genus *Ornithodoros* (1). Human disease occurs in many focal areas and is associated with infections of *Borrelia hermsii*, *B. turicatae*, and possibly *B. parkeri* (2,3). Although the ecologic parameters that maintain *B. hermsii* and *B. turicatae* differ, human infections usually occur in rustic cabins (*B. hermsii*) and caves (*B. turicatae*) inhabited by ticks and their terrestrial vertebrate hosts (1). Recently, Gill et al. (4) provided evidence that the argasid bat tick, *Carios kellei*, feeds upon humans. Subsequently, Loftis et al. (5) used PCR analysis and DNA sequencing to detect in *C. kellei* an unidentified *Borrelia* species that was closely related to *B. turicatae* and *B. parkeri*. We report the partial molecular char-

acterization of another novel tick-borne relapsing fever spirochete in *C. kellei*, which expands our knowledge for this group of pathogenic spirochetes and their potential vertebrate hosts and tick vectors.

C. kellei were collected August 18, 2005, from a house in Jones County, Iowa, built in 1857. Bats had been excluded from the attic since 1992. Nine months before ticks were collected, bats were prevented from roosting under the eaves. DNA was extracted from 31 nymphal *C. kellei*, as described previously (6). For each tick, regions of the *glpQ*, *flaB*, and *16S rRNA* genes were amplified and sequenced as described (3,7,8). Sequences were assembled by using the SeqMan program in the Lasergene software package (DNASTAR, Madison, WI, USA).

Fourteen (45.1%) of 31 ticks were positive by PCR for ≥ 1 of the genes tested. Partial DNA sequences were determined from tick no. 16, for which amplicons for all 3 genes were obtained. The partial *flaB* sequence had 4 bases different from the 300-base sequence (98.66% identity) reported previously (GenBank accession no. AY763104) for another *Borrelia* sp. found in *C. kellei* (5). We constructed a 1,992-bp concatenated sequence that contained 1,273 bp of the *16S rRNA*, 351 bp of *flaB*, and 368 bp of *glpQ*. This concatenated sequence was aligned with homologous, trimmed

DNA sequences of the same length obtained from representative full-length sequences determined previously for *B. hermsii*, *B. turicatae*, and *B. parkeri* (3,9) (Figure). This *C. kellei* spirochete was more closely related to *B. turicatae* and *B. parkeri* than to *B. hermsii* but was clearly distinct from all 3 species (DNA sequence identities of 98.89%, 98.75%, and 95.98% to *B. turicatae*, *B. parkeri*, and *B. hermsii*, respectively).

A *glpQ* amplicon from another nymphal tick (no. 3) was sequenced (GenBank accession no. EF688578) and was unique in the database; it was also considerably different from the *glpQ* sequence determined from tick 16, with 325 of 368 bases matching (88.3% identity). The *Borrelia glpQ* sequence from tick 3 had 85.1%–89.1% identity compared with *glpQ* sequences from *B. hermsii*, *B. turicatae*, and *B. parkeri*. This finding suggests the presence of at least 2 relapsing fever group spirochetes in *C. kellei* that await further characterization.

We found a novel *Borrelia* in bat ticks that is closely related to, but distinct from, the other known species of tick-borne relapsing fever spirochetes in North America. The human health implications of the new relapsing fever group spirochete are not yet known. The willingness of *C. kellei* to feed on humans and the fact that infection with bacteria closely related to true relapsing fever spirochetes occurs in

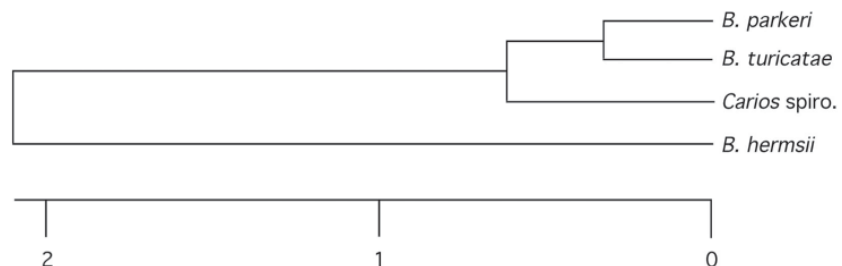


Figure. Phylogram comparing the novel spirochete in the bat tick *Carios kellei* with *Borrelia parkeri*, *B. turicatae*, and *B. hermsii* based on the concatenated partial *16S rRNA-flaB-glpQ* DNA sequences in the *Carios* spirochete (1,992 bp total) (produced with ClustalV software from DNASTAR [Madison, WI, USA]). Scale bar represents the number of base substitutions per 100 aligned bases. GenBank accession numbers for the *C. kellei* spirochete sequences used to construct the tree are EF688575, EF688576, and EF688577. Spiro, spirochete.