

2. Falagas ME, Siakavella S. *Bacteroides*, *Prevotella*, and *Porphyromonas* species. A review of antibiotic resistance and therapeutic options. *Int J Antimicrob Agents* 2000;15:1-9.
3. Freifeld AG, Walsh TJ, Philip A. Infections in the cancer patient. In: Devita VT Jr, Hellman S, Rosenberg SA, editors. *Cancer. Principles and practice of oncology*. 5th ed. New York: Lippincott-Raven; 1997. p. 2659-04.
4. Bodey GP. Infections in patients with cancer. In: Holland JF, Frei F, editors. *Cancer medicine*. 2nd ed. Philadelphia: Lea and Febiger; 1982. p. 1339-72.
5. Brazier JS, Stubbs SL, Duerden BI. Metronidazole resistance among clinical isolates belonging to the *Bacteroides fragilis* group: time to be concerned? *J Antimicrob Chemother* 1999;44:580-1.
6. O'Donoghue MA, Potter J, Allen KD. Metronidazole-resistant *Bacteroides fragilis* infection. *J Infect* 1992;25:211-4.
7. Hickey MM, Davies UM, Dave J, Vogler M, Wall R. Metronidazole resistant *Bacteroides fragilis* infection of a prosthetic hip joint. *J Infect* 1990;20:129-33.
8. Brogan O, Garnett PA, Brow R. *Bacteroides fragilis* resistant to metronidazole, clindamycin and cefoxitin. *J Antimicrob Chemother* 1989;23:660-2.
9. Lamothe F, Fijalkowski C, Malouin F, Bourgault AM, Delornel L. *B. fragilis* resistant to both metronidazole and imipenem. *J Antimicrob Chemother* 1986;18:642-3.
10. Turner P, Edward SR, Weston V, Gazes A, Ispaham P, Greenwood D. Simultaneous resistance to metronidazole, co-amoxiclav, and imipenem in clinical isolate of *Bacteroides fragilis*. *Lancet* 1995;345:1275-7.
11. Snyderman DR, Jocubus NV, Dermott LA, Supran S, Cuchural CG, Finegold S. Multicentric study of in vitro susceptibility of the *Bacteroides fragilis* group 1995 to 1996 with comparison of resistance trends from 1990 to 1996. *Antimicrob Agents Chemother* 1999;24:17-22.
12. Aldridge KE, Gelfand M, Reller LB, Ayers CW, Pierson CL, Schoenknecht F, et al. A five year multicentre study of the susceptibility of the *Bacteroides fragilis* group isolates to cephalosporins, cephamicins, penicillin, clindamycin and metronidazole in the United States. *Diagn Microbiol Infect Dis* 1994;18:235-41.
13. Chaudhry R, Mishra B, Dhawan B, Sharma N. Anaerobic infections in an Indian tertiary care hospital with special reference to Bacteroidaceae. *J Infect* 1999;38:54-5.

Proper Nomenclature for the Human Granulocytic Ehrlichiosis Agent

To the Editor: In their recent article, "Antigenic variations in vector-borne pathogens," Barbour and Restrepo discuss the outer membrane protein components of *Anaplasma marginale* and related bacteria (1). Citing a reference by Zhi et al. (2), they state that *Ehrlichia granulocytophila* is the agent of human granulocytic ehrlichiosis (HGE).

The use of new names and combinations not widely recognized for genera and species lends increasing confusion to a group of bacteria already in taxonomic disarray. Several other species names have been suggested for the HGE agent since the initial description of the clinical illness caused by this agent and the in vitro technique used to isolate the agent in blood samples (3,4). Both *E. phagocytophila* and *E. equi* are genetically nearly identical to the HGE agent, and the three are probably conspecific. Thus, most scientists in the field today would support use of the name *Ehrlichia phagocytophila* to describe these bacteria.

Recent phylogenetic analyses show that *E. phagocytophila* strains align into a clade that includes *Anaplasma marginale*, the historical precedent in this grouping. Such phylogenetic analyses, which are also supported by comparative antigenic and biological studies, have resulted in a proposal for reclassification of several *Ehrlichia* spp., including *E. phagocytophila*, into the genus *Anaplasma* (5). Until a cogent reclassification based on objective criteria is firmly accepted, the creation and use of new scientific name combinations for a single bacterium yield clinical and laboratory confusion and should be avoided.

Johan S. Bakken* and J. Stephen Dumler†

*St. Mary's Duluth Clinic, Duluth, Minnesota, USA; and

†Johns Hopkins Medical Institutions, Baltimore, Maryland, USA

References

1. Barbour AG, Restrepo BI. Antigenic variations in vector-borne pathogens. *Emerg Infect Dis* 2000;6:449-57.
2. Zhi N, Ohashi N, Rikihisa Y. Multiple p44 genes encoding major outer membrane proteins are expressed in the human granulocytic ehrlichiosis agent. *J Biol Chem* 1999;274:17828-36.
3. Bakken JS, Dumler JS, Chen S-M, Eckman MR, VanEtta LL, Walker DH. Human granulocytic ehrlichiosis in the upper midwest United States: A new species emerging? *JAMA* 1994;272:212-8.
4. Goodman JL, Nelson C, Vitale B, Madigan JE, Dumler JS, Munderloh UG. Direct cultivation of the causative agent of human granulocytic ehrlichiosis. *N Engl J Med* 1996;334:209-15.
5. Dumler JS, Rikihisa Y, Dasch GA, Barbet AF, Palmer GH, Ray SC. Proposal for taxonomic reorganization of the order *Rickettsiales*, family *Rickettsiaceae*, and tribe *Ehrlichieae*. [abstract 75]. In: Program and Abstracts of the 15th Sesqui-Annual Meeting of the American Society for Rickettsiology; April 30-May 5, 2000; Captiva Island, Florida. American Society for Rickettsiology; 2000.

Single Nucleotide Polymorphisms in *Mycobacterium tuberculosis* Structural Genes

To the Editor: A recent article by Fraser et al. (1) discussed the frequency of single nucleotide polymorphisms (SNPs) in two genomes of *Mycobacterium tuberculosis*, strains H37Rv (2) and CDC1551 (unpublished). The article contains an inaccurate representation of our published *M. tuberculosis* data on SNP frequency. The authors state that "detailed comparison of strains H37Rv and CDC1551 indicates a higher frequency of polymorphism, approximately 1 in 3,000 bp, with approximately half the polymorphism [sic] occurring in the intergenic regions. In other words, 50% of the polymorphisms are in 10% of the genome. While this rate is higher than that suggested (3), it still represents a lower nucleotide diversity than found in limited comparisons from other pathogens."

On the basis of comparative sequence analysis of eight *M. tuberculosis* structural gene loci (open reading frames [orf]), we initially published an estimated average number of synonymous substitutions per synonymous site (K_s value) that indicated that this pathogen had, on average, approximately 1 synonymous difference per 10,000 synonymous sites (4). This finding was unexpected given the relatively large population size of *M. tuberculosis* and paleopathologic