origin, I would again urge that the organism name *P. carinii* be retained for the organism that infects humans, because this will allow continuity of the medical literature.

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**References**


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**Pneumocystis carinii**

**Nomenclature: 2 Misnomers Are Not Better Than 1**

To the Editor—Some of the finer points of *Pneumocystis carinii* taxonomy made by Gigliotti [1] and Cushion and Stringer [2] may not be clear to those unfamiliar with the confusing history and biology of this atypical fungus. The proposed reclassification of *P. carinii* forma specialis *hominis* (*P. carinii* f. *sp. hominis*) as a new species (“*Pneumocystis jirovecii*”) [3, 4] is not compelling, and it does not “foster scientific understanding and communication” [5, p. 278]. I think that this debate can best be framed by 2 questions: what criteria are required for taxonomic revision, and if revision is required, should historic, widely used taxons be altered?

First, with respect to the criteria for taxonomic revision, there is no question that there are differences in nucleotide sequences between genes of the different special forms of *P. carinii* that are found among mammalian hosts [6]. However, very few—if any—data on the replication mechanisms and lifestyle (i.e., sexual or asexual reproduction) of the organism have been published since 1994 that would allow one to differentiate evolutionary divergence of *Pneumocystis* strains infecting separate hosts from true speciation [7, 8]. In fact, recent data suggest that genetic differences among primate-derived *P. carinii* isolates do, indeed, vary with phylogenetic differences in the hosts (i.e., they are the result of coevolution) [9, 10], but they do not fit the proposed speculation scheme on the basis of the “phenotype” of host specificity or the “genotype” of genetic divergence [9]. At what point does speciation begin and clonality end [7]? How do we determine this point in the absence of a valid, reproducible system for in vitro culture of the organism? Whether phenotypes or genotypes should “drive” taxonomy is not a problem unique to *P. carinii*; bacteriologists and eukaryotic biologists alike have noted the difficulties of applying molecular genotype differences to phenotypic speciation [11–13]. Until further data on life cycles, horizontal gene flow in nature, genomic DNA-DNA hybridization, and other traditional taxonomic characteristics are available to complement the newer molecular data [8, 11–13], we should continue using (i.e., conserving) the trinomial nomenclature spelled out by the 1994 Third International Workshop on *Pneumocystis* [14], as has been successfully done over the past decade.

Second, even if we agree that phylogenetic information demands the renaming of the special forms of *P. carinii*, that renaming has been done incorrectly. Although Cushion, Stringer, and Frenkel followed the International Code of Botanical Nomenclature rules of Latin and English grammar in describing “*P. jirovecii*” [3–5], they have incorrectly ignored the portion of the code (chapter II, section 4, article 14) that clearly discusses the need for “conservation of names … which best serve stability of nomenclature … [in order to] avoid disadvantageous nomenclatural changes entailed by the strict application of the rules” [15]. It is disadvantageous (i.e., destabilizing) to the scientific literature and confusing to physicians and patients alike to change the name of *P. carinii* f. *sp. hominis* to “*P. jiroveci*.” Furthermore, Jirovec’s name should not take historic precedence over van der Meer or Brug, who are recognized as having described human-derived *P. carinii* a decade before Jirovec [16–19]. Even Jirovec’s colleague Vanek published his work on human *P. carinii* in 1951, a year before Jirovec [16–18]; Vanek’s prior work, along with that of van der Meer and Brug, was clearly cited by Jirovec himself [20]. Perhaps the most widely disseminated work of that decade was the landmark review of D. Carleton Gajdusek, from 1957 [18]. If historical priority is to be ignored, one could easily argue for his name to be used instead, because he is the one who brought *Pneumocystis* to general attention, although this would cause more taxonomic confusion. Thus, the most accurate, least controversial, and most obvious solution to this debate (as Gigliotti [1] points out) is to retain the name given to the pathogen as clearly described by the Delanoès in 1912 [21] (reprinted in [17]): *Pneu-
mocystis carinii. If someday enough data accrue that demand speciation, we should rename the many animal-derived forms of *Pneumocystis* with either the names of the host species or of deserving scientists and physicians from past and present (e.g., Brug, Chagas, Delanoe, Gadjusek, Hughes, Jirovec, van der Meer, Vanek, and Wakefield) and leave the clinically relevant (i.e., human-derived) *P. carinii* name alone as a colorful reminder of the past that remains instantly recognizable by all. Indeed, *Histoplasma capsulatum* is neither a protozoan “plasma” nor encapsulated, nor does *Haemophilus influenzae* cause influenza, but both names have been maintained throughout the past century without undue distress. Such names remind us of the progress science has made and provide teaching moments for students and trainees. And, as our mothers taught us, “2 wrongs do not leave you with a right.”

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**Pneumocystis Nomenclature**

To the Editor—The recent viewpoint article by Dr. Francis Gigliotti [1] discussing the name change of *Pneumocystis carinii* and the accompanying editorial commentary by Drs. Melanie Cushion and James Stringer [2] clearly delineate the skirmish lines of this ongoing battle concerning the appropriate nomenclature for *Pneumocystis* and related opportunistic fungal organisms. The genus *Pneumocystis* represents a medically important group of related fungi that colonize healthy hosts and cause lethal pneumonia in immunocompromised humans, particularly those with AIDS or malignancies or those receiving immune-modulating medications [3]. However, despite recent proposals to change the name of the human variant to *Pneumocystis jiroveci* and the rat-based organism to *P. carinii*, considerable controversy remains over appropriate approaches for the taxonomy of these poorly understood opportunistic pathogens.

The basis of the controversy stems from our continued inability to successfully culture *Pneumocystis* organisms from any host species, as well as the lack of any means to manipulate the genetics of this fungus [3]. In lieu of classical methods to isolate the organism to homogeneity, to biochemically characterize the fungi, and to reproducibly propagate these isolates, alternate approaches to delineate *Pneumocystis* species have been proposed, largely based on genetic variations. Although this approach is initially attractive, relatively few genes have been characterized for *Pneumocystis* across multiple laboratory and clinical isolates (other than a handful of genes derived from organisms obtained from rats). This is also problematic, in that genotyping performed on rat-derived *Pneumocystis* strains representing colonies cultured at different institutions exhibit considerable genetic sequence diversity (5%–15%) at single, otherwise-conserved genetic loci [3, 4]. Thus, it remains impossible to clearly determine