of the clinical specimens were aligned with sequences from other poxviruses available in the public database (GenBank). They showed 100% nt identity with their orthologs of SWPV Nebraska strain. Concatenated amino acid alignments were used for phylogenetic inference (Figure). The clinical isolates and SWPV branched together in the phylogenetic tree with high bootstrap support. No amplification of the hemagglutinin gene was obtained, demonstrating that the animals were not infected with VACV. Samples were also negative for *Erysipelothrix* spp. (by PCR and ELISA) and porcine circovirus-2 (by PCR).

Outbreaks of swinepox disorders have been frequently reported in Europe, North America, and Oceania, and special attention has been given to congenital cases, which usually lead to high case-fatality rates (*2*,*8*,*9*). Our data identified SWPV as the cause of a recent outbreak in Brazil and suggest that previous outbreaks in the neighboring municipality of Campinas in 1976 and 1980 (*3*) may have been caused by SWPV as well because pigs are the only host and reservoir of the virus. Further sequencing analysis of the virus isolates will be necessary to characterize the strain of SWPV circulating in Brazil.

Recently, an outbreak of VACV-related disease in horses was reported in southern Brazil, which alerted the scientific community to the possible spread of this disorder to animal hosts other than dairy cattle (*10*). However, our data clearly demonstrate that this outbreak in pigs does not represent a spread of VACV infection, despite frequent reports of VACV-related outbreaks in dairy cows in São Paulo State (*6*). Therefore, the differential diagnosis of skin diseases of pigs might be a useful tool in epidemiologic surveys to access VACV spread and host range in Brazil.

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Maria Luiza G. Medaglia, Adriana de Cássia Pereira, Tânia R.P. Freitas, and Clarissa R. Damaso

Author affiliations; Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil (M.L.G. Medaglia, C.R. Damaso); Centro de Patologia Animal, Campinas, Brazil (A.C. Pereira); and Laboratório Nacional Agropecuário de Minas Gerais–Ministério da Agricultura, Pecuária e Abastecimento, Pedro Leopoldo, Brazil (T.R.P. Freitas)

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Address for correspondence: Clarissa R. Damaso, Instituto de Biofísica Carlos Chagas Filho, Universidade Federal do Rio de Janeiro, Av. Carlos Chagas Filho, 373-CCS, Ilha do Fundão, 21941-590, Rio de Janeiro, RJ, Brazil; email: damasoc@biof.ufrj.br

Plasmodium vivax Seroprevalence in Bred Cynomolgus Monkeys, China[1]

To the Editor: Having worked with numerous species of research nonhuman primates over the past 26 years, I have a keen interest in related occupational health and safety. In this regard, I was quite interested in the recent report by Li et al. (*1*) and have some comments and questions relative to this article.

The occurrence of *Plasmodium* spp. infection in feral primates, feral source captive primates, or primates

[1]Li et al. have declined to respond to this letter.

bred outdoors in malaria-endemic areas is not uncommon. However, with the exception of *P. knowlesi*, it is my understanding that malarial organisms found in cynomolgus monkeys do not pose a major zoonotic concern (although this can always change). Furthermore, it is my understanding that *P. vivax* does not infect macaques, including cynomolgus monkeys.

Other malarial parasites of cynomolgus monkeys, apart from *P. knowlesi*, may include *P. cynomologi*, *P. inui*, *P. fieldi*, and *P. coatneyi*. A recent publication reported that in wild-source cynomolgus monkeys in Malaysia, >90% of the animals tested were positive for ≥1 *Plasmodium* species. Furthermore, >80% of samples from these animals were positive by specific PCR for ≥1 of these organisms (*2*).

Using PCR for *Plasmodium* spp. identification, I have tested newly imported research cynomolgus monkeys from various breeding centers in China. I can confirm that some animals have subclinical malarial infections.

Except for the report by Li et al. (*1*), I am unaware of other reports of *P. vivax* in cynomolgus monkeys. It would be interesting to confirm the presence of this organism by using PCR primers specific for *Plasmodium* spp. My questions to the authors relate to the test method used in their study. Was an ELISA for detecting *P. vivax* antibodies the only diagnostic method used to identify this parasite? It may be useful to re-address the specificity of this test in differentiating various *Plasmodium* spp. Until these issues are clearly addressed, their reported results are not reliable.

David B. Elmore

Author affiliation: DBE Veterinary Consulting, San Diego, California, USA

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Address for correspondence: David B. Elmore, DBE Veterinary Consulting, 1785 Guizot St, San Diego, CA 92107, USA; email: dbevetcon@aol.com

Dengue Virus Serotype 4, Roraima State, Brazil

To the Editor: Temporão et al. recently reported the detection and characterization of dengue virus serotype 4 (DENV-4) in Boa Vista, Roraima, Brazil (*1*). To date, 4 subtypes of DENV-4 have been recognized: genotype I, which comprises Asian strains (e.g., Thailand-1978-U18441); genotype II, which has been detected since the early 1980s in the Americas (e.g., Brazil-1982-U18425); genotype III, which comprises recently emerged Thai strains (GenBank accession no. AY618989); and genotype IV, which comprises sylvatic strains (GenBank accession no. EF457906) (*2*).

Temporão et al. conducted phylogenetic analysis of envelope gene sequences and concluded that 3 samples of DENV from Roraima in 2010 were DENV-4, genotype I (*1*). Unfortunately, the authors mistakenly labeled Asian strains (Thailand-1978 and -1985) as genotype II, and American strains (e.g., Brazil-1982) as genotype I. Those DENV-4 strains isolated in Roraima in 2010 in fact belong to genotype II (*2*,*3*). We had previously analyzed 2 samples isolated from Roraima in 2010 by using C/prM nucleotide sequencing and maximum-likelihood phylogenetic reconstruction. Our results, presented at the XXI National Meeting of Brazilian Society for Virology in October 2010, show that both isolates are indeed genotype II (*3*). Nucleotide sequences are available in GenBank under accession nos. HQ822125 and HQ822126.

Temporão et al. also concluded that because only genotype II (reported as genotype I) was identified in their samples, "[it] excludes the possibility that Asian genotypes previously circulated in Brazil." Beyond its obviously flawed logic, we believe that this statement lacks scientific support; DENV-4 genotype I, closely related to Chinese and Philippine strains, has in fact been shown to occur in the city of Manaus, ≈800 km south of Boa Vista, as reported in 2 recent articles (*4*,*5*). Circulation of DENV-4 genotype I in northern Brazil, probably related to increasingly intense trade with Asian countries, may be sporadic and geographically limited as yet (*5*), but ignoring this evidence can hardly be helpful for dengue surveillance and control.

Pablo Oscar Amézaga Acosta, Rodrigo Melo Maito, Fabiana Granja, Joel da Silva Cordeiro, Thalita Siqueira, Mayara Nunes Cardoso, André de Lima Corado, Raphaela Honorato Barletta-Naveca, and Felipe Gomes Naveca

Author affiliations: Universidade Federal de Roraima, Boa Vista, Brazil (P.A. Amézaga Acosta, F. Granja, J. da Silva Cordeiro, T. Siqueira, M. Nunes Cardoso, A. de Lima Corado); Laboratório Central de Roraima, Boa Vista (R. Melo Maito); Instituto Nacional de Pesquisas da Amazônia, Manaus, Brazil (R.H. Barletta-Naveca); and Instituto Leônidas e Maria Deane, Fiocruz, Brazil (F. Gomes Naveca)