

showing phenotypic characteristics typical of *S. mexicana* and *S. schenckii* (2). In contrast, type reference strain *S. brasiliensis* CBS 120339 was included in the test, and it was able to assimilate only dextrose.

A presumptive identification based on phenotypic characteristics allowed us to classify this fungus as *S. mexicana*, although this species has an atypical morphologic profile. The diameter of colonies grown at 30°C and 37°C are smaller than those proposed by Marimon and collaborators but much closer to those of *S. schenckii* (2). These differences could be attributable to the intraspecific variation of this single isolate.

Genomic DNA was obtained from the yeast phase of *S. mexicana* MUM 11.02, and the partial sequencing of the nuclear calmodulin gene was based on the amplicon generated by PCR reaction by using CL1 and CL2A primers (2,3). Sequencing was performed at Fundação Oswaldo Cruz, Rio de Janeiro, Brazil. A BLAST analysis (www.ncbi.nlm.nih.gov/BLAST) comparing the sequence of the calmodulin gene with sequences AM398382, AM398393, AM117444, AM116899, and AM116908 in the GenBank database confirmed the identity of this isolate as *S. mexicana*. The MUM 11.02 isolate showed 99% similarity with the sequences of *S. mexicana* (i.e., GenBank accession no. AM398393) with high bootstrap support values (Figure, panel B). The calmodulin sequence of MUM 11.02 was deposited in GenBank as JF970258.

In vitro susceptibility tests with fluconazole, itraconazole, and terbinafine were performed by the microdilution method (9) and revealed MICs of 128 µg/mL, 32 µg/mL, and 0.5–1.0 µg/mL, respectively, which corresponds to the findings of Marimon et al. (1) for *S. mexicana*. Thus, *S. mexicana* is an emerging cause of human sporotrichosis.

Financial support for this work was provided by Fundação Carlos Chagas Filho de Amparo à Pesquisa do Estado do Rio de Janeiro (Grant Proc. E-26/111.619/2008). R.M.Z.O. is in part supported by Conselho Nacional de Desenvolvimento Científico e Tecnológico 350338/2000-0.

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DOI: <http://dx.doi.org/10.3201/eid1710.110737>

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Swinepox Virus Outbreak, Brazil, 2011

To the Editor: *Swinepox virus* (SWPV), which replicates only in swine, belongs to the *Suipoxvirus* genus of the *Poxviridae* family. It is the etiologic agent of a skin disease of pigs, characterized by generalized pustular lesions and associated with high rates of illness (occasionally >80%). It occurs mainly on farms with poor management and housing conditions and affects primarily pigs <3 months of age; adult pigs show milder signs. The disease is mechanically transmitted by pig lice or through direct animal contact (1). *Vaccinia virus* (VACV; *Orthopoxvirus* genus) also causes a similar pustular disease in pigs that is difficult to distinguish clinically from SWPV infections. VACV infections were common during smallpox vaccination

campaigns, when VACV was transmitted to domestic animals from lesions of vaccinees (1,2).

Swinepox disease has a worldwide distribution, and 4 outbreaks of similar infections were reported in pig herds in Brazil during 1976–2001 (3). Nevertheless, the etiologic agents of these outbreaks have never been identified through molecular techniques. Specific virus identification in such infections is particularly relevant in Brazil, considering the persistence of VACV in nature in this country, causing frequent outbreaks of pustular skin disease in dairy cattle (4–6). Therefore, distinguishing between SWPV and VACV infections during outbreaks of pustular disease in pigs is essential for evaluating whether VACV infection might have spread to pigs and whether SWPV could be detected in Brazil.

We describe the molecular identification of SWPV as the etiologic agent of an outbreak of pustular disorder in pig herds. In November 2010 and January 2011, ~850 of 3,460 animals on 3 pig farms in Holambra, São Paulo, Brazil, had generalized pustular lesions on the body, fever (38.0°C–39.7°C) and mild weight loss. Lesions evolved from macules or papules to umbilicated lesions with pustular content, followed by crusting (online Appendix Figure, www.cdc.gov/EID/content/17/10/11-0549-appF.htm). Secondary dermatitis was also noticed. Healing occurred after 3–4 weeks, but the disease started subsequently in previously healthy animals. Although the first clinical signs of disease started in the nursery units (pigs 40–50 days old), nearly 70% of the sick pigs were at the finishing units (pigs 127–134 days old), where elevated animal density and deficient sanitation conditions were observed. These findings may account for the high attack rate (nearly 50%) in finisher pigs, although overall illness was moderate (nearly 25%) when animals from all units

were analyzed together. No deaths were associated with the outbreak, in concordance with the low death rates reported for SWPV infections (<5%) (1). The affected farms belonged to the same owner, who reported frequent movement of animals between the farms.

Scabs from 7 animals were used for DNA extraction (4), followed by PCR detection of poxvirus DNA (7). We used primers designed to anneal to gene regions conserved in different poxviruses: FP-A2L, 5'-TAGTTTCAGAACAAGGATATG-3' and RP-A2L, 5'-TTCCCATATTAATTGATTACT-3' directed the

amplification of a 482-bp fragment of the virus late transcription factor-3 (www.poxvirus.org); primer sets for the DNA polymerase gene (543-bp fragment) and DNA topoisomerase gene (344-bp fragment) were previously described (7). Amplicons were directly sequenced as described (4,5). Consensus primers that specifically detect the full-length hemagglutinin gene of Eurasian-African orthopoxviruses were used to investigate VACV in the samples (4).

The nucleotide sequences obtained for the fragments of the DNA polymerase, DNA topoisomerase, and virus late transcription factor-3

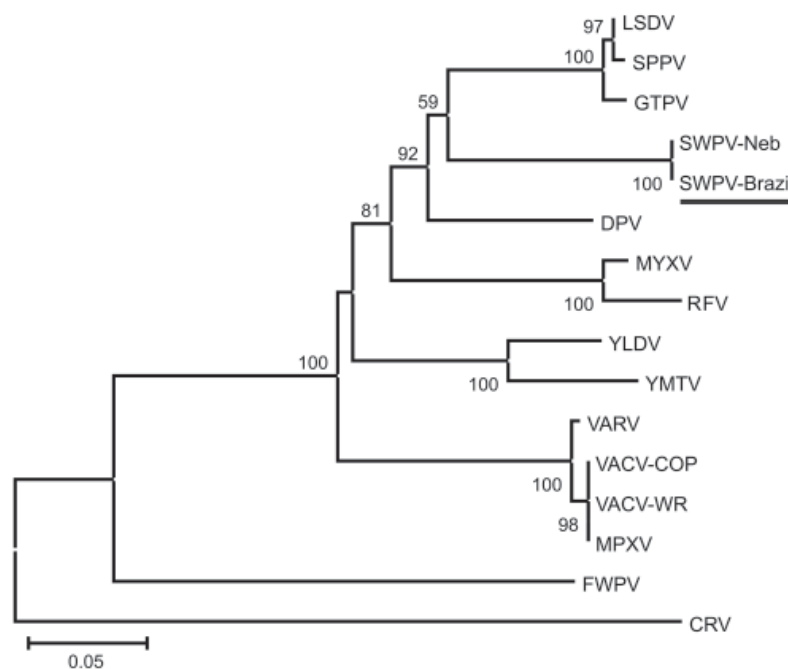


Figure. Phylogenetic tree based on the predicted amino acid sequences of fragments of the DNA polymerase, DNA topoisomerase, and viral late transcription factor-3 of the clinical isolate (GenBank accession nos. JF770341, JF770342, and JF770343) and 15 poxviruses. Sequences were aligned by ClustalX version 1.81 (www.clustal.org), and the concatenated alignments were used for phylogeny inference (MEGA4; www.megasoftware.net) opting for the neighbor-joining method and Poisson correction. We computed 1,500 replicates for bootstrap support. Values >50% are shown. The tree is drawn to scale, with branch lengths in the same units as those of the evolutionary distances used to infer the phylogenetic tree. Virus species and GenBank accession numbers: LSDV (lumpy skin disease virus; AF409137), SSPV (sheeppox virus; AY077834), GTPV (goatpox virus; AY077834), SWPV-Neb (swinepox virus Nebraska strain; NC_003389), DPV (deerpox virus; AY689437), MYXV (myxoma virus; NC_001132), RFV (rabbit fibroma virus; NC_001266), YLDV (Yaba-like disease virus; NC_002642), YMTV (Yaba monkey tumor virus; NC_002642), VARV (variola virus; NC_002642), VACV-COP (vaccinia virus Copenhagen strain; M35027), VACV-WR (vaccinia virus WR strain; NC_006998), MPXV (monkeypox virus; DQ011154), FWPV (fowlpox virus; NC_002188), CRV (crocodilepox virus; NC_008030). Virus isolated in this study is underlined.

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 assess VACV spread and host range in Brazil.

This work was supported by grants from the Conselho Nacional de Desenvolvimento Científico e Tecnológico, Ministério da Agricultura, Pecuária e Abastecimento, Fundação de Amparo à Pesquisa do Estado do Rio de Janeiro, and Instituto Nacional de Pesquisa Translacional na Amazônia to C.R.D.; M.L.G.M. received a fellowship from the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior.

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DOI: <http://dx.doi.org/10.3201/eid1710.110549>

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Plasmodium vivax Seroprevalence in Bred Cynomolgus Monkeys, China¹

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

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