qnrS1, in nontyphoidal salmonellae in the United Kingdom. These data are in contrast to those of recent studies in the United States and France, which show low incidences of *qnrS* genes in larger strain collections (*9*,*10*). The *qnr* phenotype is in contrast to resistance mediated by mutations in the topoisomerase genes whereby 1 mutation confers low-level resistance to fluoroquinolones and full resistance to nalidixic acid. Our previous study demonstrated that *qnrS1* was sufficient to cause decreased susceptibility to ciprofloxacin in the absence of mutations in *gyrA* (*1*). In this study, a *qnr* gene was sufficient to increase the ciprofloxacin MIC to 0.38–0.75 μg/mL. In addition, a *qnr* gene contributed to high-level ciprofloxacin resistance in 10 isolates, thereby potentially jeopardizing first-line treatment of vulnerable patient groups with ciprofloxacin.

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Saksenaea vasiformis Infection, French Guiana

To the Editor: The Zygomycetes are a class of filamentous fungi that are ubiquitous in the environment. Most of the species known to cause human or animal infections belong to a few genera within the order Mucorales. *Saksenaea vasiformis*, isolated from soil in India and described by Saksena in 1953, was reported to cause human infection for the first time by Ajello et al. (*1*). We report a case of a cutaneous lesion caused by *S. vasiformis* in French Guiana.

A nonimmunocompromised 47-year-old woman with a long history of non–type 1 diabetes mellitus, who had lived in French Guiana for many years, was admitted to Cayenne Hospital on November 18, 2005, with a cutaneous lesion of the abdominal wall and a fever that had lasted for 5 days before she was hospitalized. A skin biopsy specimen was obtained, and the first surgical debridement was performed on day 4 of hospitalization. A diagnosis of zygomycosis was made after direct examination and histopathologic examination of the tissue samples. Treatment was initiated on day 8, beginning with liposomal amphotericin B and itraconazole for 10 days, followed by liposomal amphotericin B alone for 12 days. Persistence of necrotic tissues at the infection site required additional surgical debridement on day 10. Histopathologic examination of the resected tissues showed damaged hyphae of zygomycetes. Resolution of clinical signs was excellent. Additional biopsy specimens taken by the end of treatment on day 21 were negative for fungi by direct examination and culture. Finally, a cicatrix was formed.

Histologic examination of the initial excised tissues showed a localized periumbilical cutaneous lesion of

14 cm × 13 cm. The skin was covered by a 1-mm layer of necrosis. The necrosis extended into all the abdominal adipose tissue at the rectus abdominis muscle and linea alba. Microscopy examination showed extensive superficial mycotic proliferation, with wide and irregular ribbonlike nonseptate hyphae and right-angle branching. These hyphae extended toward the hypodermic fat tissues and were associated with a break in the cell membrane of adipocytes and with crystals inside the adipocytes. These lesions were associated with massive nonsuppurative vascular thrombosis.

Culture of tissues samples on Sabouraud-chloramphenicol-gentamicin agar after 4 days at 30°C and 37°C grew a white aerial mold, which covered the entire surface of the agar. Examination by microscopy showed nonseptate sterile hyphae typical of a zygomycete. The fungal isolate was sent to the National Reference Center for Mycology and Antifungals at the Institut Pasteur, Paris. Subcultures on different media including malt extract agar and potato dextrose agar grew sterile mycelia. The isolates were then cultured in nutritionally deficient medium consisting of sterile distilled water supplemented with 0.05% filter-sterilized yeast extract (Difco, Becton, Dickinson and Company, Sparks, MD, USA) solution for 7 days at 37°C (*2*). Typical flask-shaped sporangia enabled identification of *S. vasiformis* (Figure). Sporulation also occurred on Czapek agar after 7 days' incubation at 37°C.

Molecular identification based on PCR amplification and sequencing of rDNA internal transcribed spacer (ITS) regions was also performed. Briefly, mycelia were grown in liquid Roswell Park Memorial Institute medium, and DNA was extracted as previously described (*3*). Ribosomal DNA, including the complete ITS1–5.8S-ITS2 region, was amplified with the fungal universal primer pairs V9D/LS266 (*4*) and ITS1/ITS4 (*5*), and both strands of PCR products were sequenced. The sequence has been deposited in GenBank (accession no. EU182902). Sequence alignment with the only *S. vasiformis* ITS sequence available in the GenBank database showed 82% similarity over 530 bp. This low degree of sequence homology is probably reflective of the need for further phylogenetic study of this genus.

Sporulation on Czapek agar enabled preparation of a sporangiospore suspension used for antifungal susceptibility testing. Sporangiospore suspension was counted microscopically and adjusted to the required density. MICs, determined by the EUCAST reference microdilution method (*6*), after 48 h of incubation were >8, 2, >8, 0.5, and >8 μg/mL for amphotericin B, itraconazole, voriconazole, posaconazole, and caspofungin, respectively. The MIC of 0.5 μg/mL for posaconazole suggests the potential clinical utility of this agent.

S. vasiformis has been isolated from soil samples in different parts of the world (*7*). This fungus has been rarely responsible for human infections. A recent review (*8*), which did not include infrequently cited articles (*9*,*10*), found only 30 human cases. This scarcity may occur because the diagnosis is often based on histologic features and *S. vasiformis* does not sporulate in routine mycology media.

Due to zygomycetes' lack of susceptibility to most of the antifungal agents, identification of a zygomycete as the etiologic agent of an infection is essential for rapid and accurate management of the disease. Rare Zygomycetes species such as *S. vasiformis* or *Apophysomyces elegans* should be suspected when a nonsporulating zygomycete is isolated from an infected lesion. When this acute infection is suspected after examination of tissue by microscopy, the fungi should be cultured in specific culture media to induce sporulation or they should be identified by molecular tools.

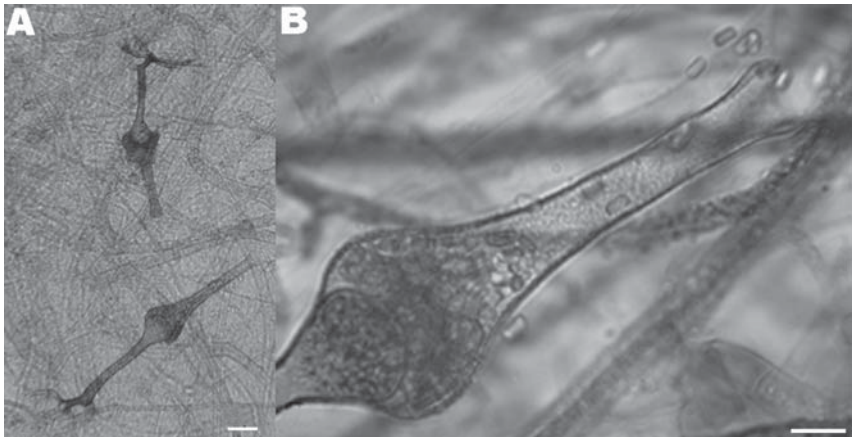

Figure. Microscopic characteristics of the isolate of *Saksenaea vasiformis* cultured on Czapek agar. A) Typical flask-shaped sporangia (scale bar = 25 μm) containing B) smooth-walled, rectangular sporangiospores (scale bar = 10 μm).

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Letters

Letters commenting on recent articles as well as letters reporting cases, outbreaks, or original research are welcome. Letters commenting on articles should contain no more than 300 words and 5 references; they are more likely to be published if submitted within 4 weeks of the original article's publication. Letters reporting cases, outbreaks, or original research should contain no more than 800 words and 10 references. They may have one Figure or Table and should not be divided into sections. All letters should contain material not previously published and include a word count.

Q Fever in Young Children, Ghana

To the Editor: Recently, experts identified Q fever, caused by the small, gram-negative bacterium *Coxiella burnetii*, as an important underdiagnosed childhood disease (*1*). Studies on Q fever in children <5 years of age are scarce, especially with respect to sub-Saharan Africa. The only available study from Niger reports a seroprevalence of 9.6% (*2*). Throughout Africa, prevalence of Q fever in adults shows considerable variability and is highest in countries with prominent stockbreeding (*3*).

Clinical manifestations of Q fever in children are similar to those of malaria (*1*,*4*). In malaria-endemic areas, most fevers are attributed to *Plasmodium falciparum* infection and presumptively treated with expensive combination therapies (*5*). In this context, other neglected fever-causing pathogens need to be given appropriate consideration.

We studied the prevalence of Q fever antibodies in 219 randomly selected children living in 9 rural villages of the Ashanti region, Ghana. Plasma was obtained by venous puncture from 2-year-old children after they had participated in a malaria control study and had been clinically monitored for 21 months. Clinical, parasitologic, socioeconomic, and Global Positioning System information was recorded as described elsewhere (*6*,*7*). In addition, 158 healthy adult volunteers from the same area were included. Plasma was stored at –20°C until microimmunofluorescence assays (IFA) (*Coxiella burnetii* I+II, Vircell SL Microbiologists, Granada, Spain) were performed according to manufacturer's instructions. To identify all children with Q fever titers, we regarded the following as positive fluorescence reactions to plasma dilutions: ≥1:64 for phase II immunoglobulin (Ig) G and ≥1:24 for phase II IgM with sensitivity (specificity) of 97.2% (100%) and 100% (56.3%), respectively. IgM testing was only performed on IgG-positive children. Positive and negative controls were run on each IFA slide. Relative risks (RR) for characteristics of children were calculated by χ^2 test; $p<0.05$ was considered significant. Informed consent was obtained from all participants or their parents. The study protocol was approved by the committee on human research and publication, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana.

Positive *C. burnetii* phase II IgG responses were observed in 37 (16.9%) of 219 children and 14 (8.9%) of 158 adults (Figure, panels A and B). In comparison to adults, more children had IgG titers ≥64 (Figure, panels C and D). On the day of the serosurvey 71 (32.4%) of 219 children had fever (measured body temperature >38°C or reported fever within the previous 48 hours). Test outcome did not appear to be influenced by *P. falciparum* infection, since 4 of 37 IgG-positive children (23 of 182 IgG-negative children) had clinical malaria, 11/37 (62/182) had asymptomatic parasitemia, and 6/37 (38/182) had fever without parasitemia, and there were no significant differences between groups. The frequency of prior malaria episodes also did not influence antibody response. Three aparasitemic children had positive phase II IgM titers (24, 96, and 1,536; phase II IgG 64, 64, and 4,096, respectively). The child with the high IgM and IgG titers was clinically ill with nonsevere *C. burnetii* pneumonia. This child was among 10 (27%) of 37 phase II IgG-positive children with detectable anti–*C. burnetii* phase I antibodies. Of all sociodemographic characteristics under consideration, only maternal illiteracy was associated with positive phase II IgG testing (RR 2.1, 95% confidence interval 1.0–4.2, $p<0.05$).

A considerable proportion of Ghanaian children had anti–*C. burnetii* antibodies, which indicates that Q fever might be a common event in