(7). The remaining 1 is from Japan and assigned to ST28 (8). Unlike in previous reports, 80% of the human clinical isolates (16 isolates) characterized in this study were assigned to the ST27 complex. Although previous studies suggested that members of the ST27 complex may have lower potential to cause invasive diseases in swine (7), all the isolates were isolated from blood or cerebrospinal fluid of the patients, suggesting a high degree of invasiveness (Table). Because it is unknown whether the ST27 complex is also dominant among isolates from diseased pigs in Thailand, future surveillance will be necessary to know the situation in pigs. However, our data indicate that the ST27 complex is another clonal group that should be assessed for its importance for human infection. Because mrp, epf, and sly are not appropriate as virulence markers for the ST27 complex members, development of novel virulence markers will be needed for efficient discrimination of S. suis strains virulent for humans.

This study made use of the *Streptococcus suis* Multilocus Sequence Typing website (http://ssuis.mlst.net); this site is hosted at Imperial College and development is funded by the Wellcome Trust. The study was supported by a grant-in-aid from the Zoonoses Control Project of the Ministry of Agriculture, Forestry and Fisheries of Japan and the Endowment Fund for Medical Research, Faculty of Medicine, Chiang Mai University.

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References

- Staats JJ, Feder I, Okwumabua O, Chengappa MM. *Streptococcus suis*: past and present. Vet Res Commun. 1997;21: 381–407.
- Vecht U, Wisselink HJ, van Dijk JE, Smith HE. Virulence of *Streptococcus suis* type 2 strains in newborn germfree pigs depends on phenotype. Infect Immun. 1992;60:550–6.
- Jacobs AAC, Loeffen PLW, van den Berg AJG, Storm PK. Identification, purification, and characterization of a thiol-activated hemolysin (suilysin) of *Streptococcus suis*. Infect Immun. 1994;62:1742–8.
- Smith HE, Damman M, van der Velde J, Wagenaar F, Wisselink HJ, Stockhofe-Zurwieden N, et al. Identification and characterization of the *cps* locus of *Streptococcus suis* serotype 2: the capsule protects against phagocytosis and is an important virulence factor. Infect Immun. 1999;67:1750–6.
- Vecht U, Wisselink HJ, Jellema ML, Smith HE. Identification of two proteins associated with virulence of *Streptococcus suis* type 2. Infect Immun. 1991;59:3156–62.
- Gottschalk M, Lebrun A, Wisselink H, Dubreuil JD, Smith H, Vecht U. Production of virulence-related proteins by Canadian strains of *Streptococcus suis* capsular type 2. Can J Vet Res. 1998;62:75–9.
- King SJ, Leigh JA, Heath PJ, Luque I, Tarradas C, Dowson CG, et al. Development of a multilocus sequence typing scheme for the pig pathogen *Streptococcus suis*: identification of virulent clones and potential capsular serotype exchange. J Clin Microbiol. 2002;40:3671–80.
- Chang B, Wada A, Ikebe T, Ohnishi M, Mita K, Endo M, et al. Characteritics of *Streptococcus suis* isolated from patients in Japan. Jpn J Infect Dis. 2006;59:397–9.
- Ye C, Zhu X, Jing H, Du H, Segura M, Zheng H, et al. *Streptococcus suis* sequence type 7 outbreak, Sichuan, China. Emerg Infect Dis. 2006;12:1203–8.
- Silva LMG, Baums CG, Rehm T, Wisselink HJ, Goethe R, Valentin-Weigand P. Virulence-associated gene profiling of *Streptococcus suis* isolates by PCR. Vet Microbiol. 2006;115:117–27.

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Streptococcus suis Meningitis, United States

To the Editor: Streptococcus suis, commensal and opportunistic pathogens of swine, and prevalent zoonotic agents worldwide, are α-hemolytic gram-positive cocci with 35 different serotypes (1). In humans, S. suis infection has been associated with bacterial meningitis, septic shock, arthritis, pneumonia, endocarditis, endophthalmitis, and spontaneous bacterial peritonitis (2,3). Most at risk are those who handle or eat undercooked pork, e.g., farm workers, butchers, and slaughterhouse workers (4). Most cases have been reported in Europe or Southeast Asia (2,3). Meningitis, first recognized in 1968 in Denmark (1), is the most common clinical manifestation of human infection with S. suis. A case of S. suis meningitis in a pig farmer was reported in the United States (5). Here, we describe another case in a 60-year-old man from San Francisco who had consumed raw pork while traveling in the Philippines.

In June 2003, this man became ill with fever, diaphoresis, headache, nausea, and anorexia. He had just returned from a 7-month vacation in the Philippines. Three days after symptoms onset, his physician prescribed doxycycline. Symptoms continued and he was admitted to a local hospital 5 days later with a fever of 38.9°C, nuchal rigidity, headache, and general malaise.

The patient described no recent contact with sick persons; past medical

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history was unremarkable. On physical examination, he was somnolent but fully oriented, with no focal findings on neurologic examination and only slight nuchal rigidity. He had a leukocyte count of 21,000/mm³, including 16,400/mm³ neutrophils. Cerebrospinal fluid (CSF) showed leukocyte count of 487/µL with 80% polymorphonuclear cells and 18% lymphocytes, and glucose and protein levels <20 mg/dL and <167 mg/dL, respectively. Gram stain of CSF showed gram-positive cocci in pairs. Empiric therapy (ceftriaxone, vancomycin, and ampicillin) for bacterial meningitis was begun. Computed tomographic scan of the head showed only sinusitis; findings of chest radiograph and transesophageal echocardiogram were negative.

On hospital day 2, blood cultures grew gram-positive cocci in pairs and chains (Figure). The organism was catalase-negative, bile esculin-negative, and pyrrolidonyl aminopeptidasenegative, consistent with *Streptococcus* spp. A latex agglutination test did not detect *Streptococcus pneumoniae* antigen. Antimicrobial susceptibility testing showed that the isolate was sensitive to penicillin (MIC = 0.03), ceftriaxone, and vancomycin but resistant to tetracycline and clindamycin. Antimicrobial therapy was changed to penicillin G, 24 million units intravenously per day.

On hospital day 5, the patient complained of hearing loss in his left ear. Results of nasopharyngeal endoscopy were negative. By hospital day 7, the organism was identified by the API 20 Strep System (bioMerieux, Marcy l'Etoile, France) as S. suis serotype 2. The patient subsequently stated that he was a butcher with a culinary preference for partially cooked pork, which he had eaten in the Philippines until the week prior to onset of symptoms. On hospital day 9, a formal audiology evaluation showed severe bilateral sensorineural high-frequency hearing loss (-70 dB). The patient completed a 10-day course of parenteral antimicrobial drugs and was discharged on continued oral therapy with close followup. Two months after discharge, the patient reported much improved hearing without other sequelae.

Most *S. suis* infections occur in older men and patients who report con-

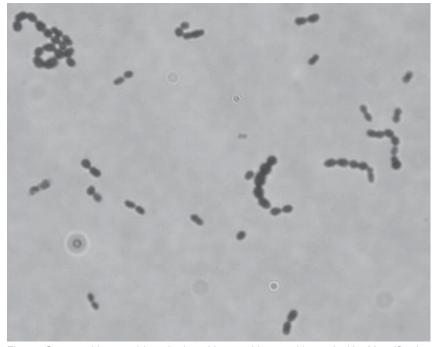


Figure. Gram-positive cocci in pairs in a 60-year-old man with meningitis. Magnification x1,000.

tact with pigs or eating undercooked pork products. Invasion of the bloodstream can occur directly through skin abrasions or the oral or respiratory route (6). Once bloodborne, *S. suis* can cause toxic shock syndrome and sepsis (7). The mechanism by which the organism traverses the bloodbrain barrier to cause meningitis is not known, although bacterial toxins and host inflammatory mediators may play a role (8).

Hearing loss from S. suis meningitis, although not specific for the organism, occurs frequently in half to two thirds of patients and can be irreversible (3,7,9). Administering dexamethasone may ameliorate hearing loss in some cases (10). Penicillin G is the preferred treatment for S. suis infection, although penicillin resistance has emerged in S. suis because of the farm practice of supplementing feeds with antimicrobial drugs. As an alternative therapy, vancomycin may be used (6). Thus, empiric therapy for adult bacterial meningitis (ceftriaxone and vancomycin with or without ampicillin) would likely be sufficient to treat S. suis meningitis. Although the death rate from this disease can be high, varying from 7% in one study (3) to 30% in another (6), infection can be prevented by treating abrasions promptly, wearing gloves when handling pork, adhering to proper hand washing techniques, and sufficiently cooking pork products (3).

S. suis infection may go unrecognized since many laboratories do not routinely speciate α -hemolytic streptococci. However, in the United States, specialized tests such as the API 20 Strep System (API System; La Balme Les Grottes, Montalieu-Vercieu, France) or reference laboratories are readily available for diagnosis of all unidentified streptococci. In severe cases where infection is suspected, physicians may request that laboratories conduct definitive tests to identify the organism. In countries that lack these resources and where undercooked pork is a diet staple, underdiagnosis of *S. suis* infection is likely. Greater understanding of this organism and its disease spectrum would promote earlier diagnosis and prevention of sequelae.

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References

- Staats JJ, Feder I, Okwumabua O, Chengappa MM. *Streptococcus suis*: past and present. Vet Res Commun. 1997;21:381– 407.
- Kopic J, Paradzik MT, Pandak N. Streptococcus suis infection as a cause of severe illness: 2 cases from Croatia. Scand J Infect Dis. 2002;34:683–4.
- Arends JP, Zanen HC. Meningitis caused by *Streptococcus suis* in humans. Rev Infect Dis. 1988;10:131–7.
- Dupas D, Vignon M, Geraut C. Streptococcus suis meningitis: a severe noncompensated occupational disease. J Occup Med. 1992;34:1102–5.
- Willenburg KS, Sentochnik DE, Zakods RN. Human *Streptococcus suis* meningitis in the United States. N Engl J Med. 2006;354:1325.
- Vilaichone RK, Vilaichone W, Nunthapisud P, Wilde H. *Streptococcus suis* infection in Thailand. J Med Assoc Thai. 2002;85(Suppl 1):S109–17.
- Tang J, Wang C, Feng Y, Yang W, Song H, Chen Z, et al. Streptococcal toxic shock syndrome caused by *Streptococcus suis* serotype 2. PLoS Med. 2006;3:e151.
- Vadeboncoeur N, Segura M, Al-Numani D, Vanier G, Gottschalk M. Pro-inflammatory cytokine and chemokine release by human brain microvascular endothelial cells stimulated by *Streptococcus suis* serotype 2. FEMS Immunol Med Microbiol. 2003;35:49–58.
- Kay R, Cheng AF, Tse CY. Streptococcus suis infection in Hong Kong. QJM. 1995;88:39–47.
- de Gans J, van de Beek D. Dexamethasone in adults with bacterial meningitis. N Engl J Med. 2002;347:1549–56.

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Parvoviruses in Blood Donors and Transplant Patients, Italy

To the Editor: Parvoviruses (PARV) 4 and 5 are 2 genotypes of a novel human parvovirus, with 92% nucleotide identity, identified in the plasma sample of a patient screened for acute HIV infection and in samples of manufactured plasma pools (1,2). Recently, PARV4 and PARV5 were identified in blood samples from 3 of 26 cadavers from the United Kingdom, all of whom were positive for hepatitis C virus RNA and had a history of intravenous drug use (3). PARV4/5 were also found in bone marrow (BM) and lymphoid tissues from 17 of 24 HIVpositive cadavers from Scotland (4) and in BM aspirates from 16 of 35 Italian patients with AIDS (5). Little or no information is available about the epidemiology and clinical correlates of infection with these novel viruses. To provide insights into their pathogenic potential in vivo, we assessed the frequency of PARV4/5 viremia in healthy patients, transplant patients, and those with suspected viral disease.

We performed a retrospective molecular study for the presence of PARV4/5 sequences in 4 groups of 417 Italian HIV-negative persons. Group 1 consisted of 100 blood donors recruited from the Transfusion Centre of Modena (northern Italy); group 2, 84 patients with hematologic diseases showing clinical signs of viral etiology but negative results for the most common viruses (herpesviruses, adenovirus, hepatitis virus, and coxsackie virus). For both of these groups, DNA was extracted for analysis from serum specimens and peripheral blood mononuclear cells (PBMCs). Groups 3 and 4 comprised recipients of kidney and allogeneic BM/peripheral blood stem cell (PBSC) transplants. for which DNA was extracted from serum specimens collected at 6 and 12 months, respectively, after transplantation. The nested PCR method was used to amplify a shared sequence of PARV4 and its variant PARV5 and was specific for the open reading frame 1. First step PCR was performed as previously described (2) with a sensitivity of 1-10 copies, on 1 µg PBMC DNA and on one fifth of DNA extracted from 0.25 mL of serum. Primers for second round PCR were PV4NS1Fn2 (5'-GTTGATGGYCCT-GTGGTTAG-3') and PV4NS1Rn2 (5'-CCTTTCATATTCAGTTCCT-GTTCAC-3'). All positive results were confirmed by direct sequencing.

We found 3 positive case-patients, including 2 renal transplant recipients and 1 patient with a suspected viral disease; none of the blood donors tested positive on single-round PCR. On nested PCR, 1 blood donor had positive results; the positivity rate did not increase in the other groups (Table). In the first 2 groups, PARV4/5 sequences were detected only in the serum samples, not in the PBMCs collected at the same time. These sequences suggest that PBMCs are not a major site of viral replication. Similar to B19 infection, which is rarely reactivated in the setting of BM/PBSC transplantation (6,7), none of the BM/PBSC transplant patients were PARV4/5 positive. The detection of PARV4/5 sequences in the serum collected at 12 months after transplantation was not associated with the occurrence of any symptoms in the 2 renal recipients. Of note, the available serum samples collected from both recipients before transplantation, and at 6 and 24 months after