symptom onset, increased during the symptomatic period (as shown by higher titers on day 10), peaked by day 30 (2 weeks after recovery), then declined slowly over several years. *Zaire ebolavirus* IgG remained detectable, often at high levels, >11 years after the infection.

These long-lasting IgG antibody responses found in 20 survivors of 3 different *Zaire ebolavirus* outbreaks rule out the hypothesis that low Ebola virus (and Marburg virus) seroprevalence rates found in epidemic regions of Africa are due to rapid loss of specific IgG. Whether this immunity is sufficient to protect from recurrent infection remains undetermined. These findings show that IgG ELISA is suitable for epidemiologic and epizootiologic investigations of Ebola and that *Zaire ebolavirus* IgG is an excellent indicator of *Zaire ebolavirus* circulation in humans.

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**References**

1. Georges AJ, Leroy EM, Renault AA, Tevi Benissan C, Ndoz MB, Minet TR, et al. *Ebola* hemorrhagic fever outbreaks in Gabon, 1994–1997: epidemiologic and health control issues. *J Infect Dis*. 1999;179(Suppl 1):S65–75. doi: 10.1086/514290

2. Becquart P, Becquart C, Gasquet C. *Varicella zoster* virus transmission events and rapid decline of central African wildlife. *Science*. 2004;303:387–90. doi: 10.1126/science.1092528

3. Walsh PD, Abernethy KA, Bermejo M, Beyers R, De Wachter P, Ella Akou M, et al. *Catastrophic ape decline in western equatorial Africa*. *Nature*. 2003;422:611–4. doi: 10.1038/nature01566

4. Bermejo M, Rodriguez-Tijeiro JD, Illera G, Barroso A, Vila C, Walsh PD. *Ebola* outbreak—killed 5000 gorillas. *Science*. 2006;314:1564. doi: 10.1126/science.1133105

5. Rowe AK, Bertolli J, Khan AS, Makunu R, Muyembe-Tamfum JJ, Bressler D, et al. Clinical, virologic, and immunologic follow-up of convalescent *Ebola* hemorrhagic fever patients and their household contacts, Kikwit, Democratic Republic of the Congo. *J Infect Dis*. 1999;179(Suppl 1):S28–35. doi: 10.1086/354138

6. Ksiazek TG, Rollin PE, Williams AJ, Bressler DS, Martin ML, Swanepoel R, et al. Clinical virology of *Ebola* hemorrhagic fever (EHF): virus, virus antigen, and IgG and IgM antibody findings among EHF patients in Kikwit, Democratic Republic of the Congo, 1995. *J Infect Dis*. 1999;179(Suppl 1):S177–87. doi: 10.1086/514321

7. Ksiazek TG, West CP, Rollin PE, Jahrling PB, Peters CJ. ELISA for the detection of antibodies to *Ebola* viruses. *J Infect Dis*. 1999;179(Suppl 1):S192–8. doi: 10.1086/514313

8. Heffernan RT, Pambo B, Hatchett RJ, Leman PA, Swanepoel R, Ryder RW. Low seroprevalence of IgG antibodies to *Ebola* virus in an epidemic zone: Ogooué-Lindé region, northeastern Gabon, 1997. *J Infect Dis*. 2000;191:964–8. doi: 10.1086/3052994

9. Bausch DG, Borchert M, Grein T, Roth C, Swaminathan S, Lihande ML, et al. Risk factors for Marburg hemorrhagic fever, Democratic Republic of the Congo. *Emerg Infect Dis*. 2003;9:1531–7.

10. Borchert M, Mulangu S, Swanepoel R, Tshomba A, Afounde A, Kulidiri A, et al. Clinical virology of *Ebola* hemorrhagic fever (EHF): virus, virus antigen, and IgG and IgM antibody findings among EHF patients in Kikwit, Democratic Republic of the Congo, 1995. *J Infect Dis*. 1999;179(Suppl 1):S28–35. doi: 10.1086/514321

11. Rowe AK, Bertolli J, Khan AS, Makunu R, Muyembe-Tamfum JJ, Bressler D, et al. Clinical, virologic, and immunologic follow-up of convalescent *Ebola* hemorrhagic fever patients and their household contacts, Kikwit, Democratic Republic of the Congo. *J Infect Dis*. 1999;179(Suppl 1):S28–35. doi: 10.1086/354138

Varicella zoster virus infection is an anergic, gram-positive, diphtheroid bacterium that was described by Hall et al. in 2003. Biochemical testing, electrophoretic analysis of whole-cell proteins, and phylogenetic analysis of 16S rRNA gene sequences showed that *V. cambriense* is related to but distinct from *Actinomyces* spp and related taxa, including the genera *Actinobaculum*, *Arcanobacterium*, and *Mobiluncus*.

Although its natural habitat remains unknown, *V. cambriense* has been isolated from intratruterine devices and human vagina and abscess specimens. Commercial systems, such as analytical profile index (API) Rapid ID 32 Strep and Coryne kits (bioMérieux, Marcy l’Etoile, France), used in differentiation of novel bacteria provide biochemical profiles useful for identification of *V. cambriense* (I). However, the absence of data on this organism in manufacturers’ databases has hampered recognition of *V. cambriense* in routine clinical laboratories. We report 4 cases of *V. cambriense* infection and show that this bacterium is a potential pathogen in skin and soft tissue infections.

In 2006, 4 isolates of gram-positive curved bacilli that grew on Columbia agar with 5% horse blood under anaerobic conditions were referred to 2 regional hospitals in Hong Kong to our laboratory for identification. These isolates originated from the abscess specimens of 4 patients.

Patient 1 was a 45-year-old woman with a right ovarian chocolate cyst and endometriosis who had undergone laparotomy, right salpingo-oophorectomy, and lysis of adhesions in 2001. Since then, she had a recurrent abscess over the umbilical scar that was treated conservatively. Culture of pus from
the umbilical scar grew an unidentified gram-positive bacillus (M124). Histologic analysis of umbilical tissue showed acute suppurative inflammatory cells and microabscess formation. The patient refused follow-up and no antimicrobial drug treatment was given.

Patient 2 was a 25-year-old man who had a history of excision of multiple sebaceous cysts in the groin, and buttock pain and swelling for 4 days. He had no previous trauma and was afebrile. An abscess was incised and drained. Culture of tissue obtained grew an unidentified gram-positive bacillus (M397) and a Peptostreptococcus spp. The infection later subsided without antimicrobial drug treatment.

Patient 3 was a 34-year-old man with a lump in the left groin that had been present for 1 year. He was hospitalized with erythema and increased swelling of the lesion. An abscess was diagnosed, incised, and drained. Gram staining of pus showed numerous leukocytes and gram-positive bacilli. Culture yielded an unidentified gram-positive bacillus (M380). He was treated with ampicillin and cloxacillin, and the wound healed.

Patient 4 was a 55-year-old woman with an abscess on her back. She had a 3-year history of seronegative rheumatoid arthritis but was not receiving immunosuppressant therapy. The abscess was drained, and gram staining of pus showed numerous leukocytes and gram-positive cocci and bacilli. Culture yielded Peptostreptococcus spp. and an unidentified gram-positive bacillus (M398). The abscess healed without antimicrobial drug treatment.

The 4 unidentified gram-positive bacilli from these patients were initially characterized by using the API Rapid ID 32 Strep and Coryne kits. Doubtful identifications at various confidence levels were obtained, including Corynebacterium diphtheriae var. mitis or var. belfanti, Gardnerella vaginalis, Streptococcus mitis, S. oralis, Gemella morbillorum, and Aerococcus urinae. Sequencing of full length 16S rRNA genes suggested that the isolates were *V. cambriense*; GenBank BLAST (http://blast.ncbi.nlm.nih.gov/Blast.cgi) results showed *V. cambriense* type strain CCUG 44998T sequence as the best match (identities 98%–99%) (3).

Biochemical reactions of the strains were similar to those of CCUG 44998T (1). All 4 isolates grew poorly in air and 5% CO₂, were catalase negative, did not hydrolyse esculin or gelatin, or reduce nitrates. The 4 isolates did not ferment most of the carbo-

| Characteristic | Varibaculum cambriense type strain CCUG 44998T | Isolate |
|---------------|-----------------------------------------------|---------|
| Hospital       | B                                             | B       | A       | A       |
| Patient sex/age, y | F/45                                         | M/25    | M/34    | F/55    |
| Date referred/isolated | 2006 May                                     | 2006 Dec| 2006 Dec|
| API test code  | Rapid ID32 Strep                              | 00100010100 | 00102010100 | 02122001100 | 0002021100 |
| Coryne         | 1010321                                       | 1410321 | 1010721 | 1010321 |
| Acid production from glucose | +                                                | +       | +       | +       |
| Lactose        | –                                             | –       | –       | –       |
| Maltose        | V                                             | – (+)   | – (+)   | +       |
| Melibiose      | –                                             | –       | –       | –       |
| Pullulan       | –                                             | –       | –       | –       |
| Raffinose      | –                                             | –       | –       | –       |
| Ribose         | V                                             | +       | +       | +       | – (+) |
| Sucrose        | V                                             | +       | +       | +       |
| Trehalose      | V                                             | –       | –       | +       |
| MBDG           | –                                             | –       | –       | –       |
| Hydrolysis of hippurate | V                                              | +       | +       | –       |
| Production of | α-galactosidase                               | –       | –       | –       | +       | – |
| β-galactosidase| V                                             | –       | – (+)   | – (+)   | +       |
| α-glucosidase  | +                                             | – (+)   | – (+)   | – (+)   |
| Alkaline phosphatase | –                                           | –       | –       | –       |
| APPA           | V                                             | –       | +       | +       |
| GenBank accession no. † | FJ169866                                    | FJ169867 | FJ169868 | FJ169869 |
| Identity to type strain CCUG 44998T, % | 99                                             | 98      | 99      | 99      |

*API, analytical profile index; V, variable; MBDG, methyl-β-D-glucopyranoside; APPA, alanyl-phenylalanyl-proline arylamidase. Test results were obtained by using the API Rapid ID32 Strep kit (bioMérieux, Marcy l’Étoile, France). Results in parentheses were obtained by using the API Coryne kit (bioMérieux). †For 16S rRNA gene sequence.
hydrates in the 2 API kits, except for ribose, maltose, glucose, and sucrose. Discrepancies in results between the 2 test kits were seen with ribose in 1 isolate (M398) and maltose in 2 isolates (M124 and M397). One isolate (M380) did not hydrolyze hippurate but produced acid from trehalose and xylose. This isolate was also α-galactosidase positive, a result different from that of the type strain. All 4 isolates were α-glucosidase positive and 3 were alan- yl-phenyl-alanyl-proline arylamidase positive. Some of the biochemical reactions for the 4 isolates, including all tests for delineating *V. cambriense* from other catalase-negative *Actinomyces* spp. (1), are summarized in the Table.

We report the isolation of *V. cambriense* from 4 patients with purulent skin and soft tissue infections. Our findings contribute to understanding of the clinical and pathogenic potential of this anaerobic bacterium. Gram-positive diphtheroid organisms from wound specimens are occasionally considered to be skin commensal organisms. Clinical microbiologists should be aware of this organism and the current inadequacy of commercial systems for its identification. We have shown that 16S rRNA gene sequencing is a useful alternative to gas–liquid chromatographic analyses of cell wall fatty acids or metabolic products for identification of anaerobic gram-positive bacilli.

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**Outbreaks of Hemotrophic Mycoplasma Infections in China**

To the Editor: Infections caused by hemotrophic mycoplasmas (formerly called eperythrozoonoses) in animals and humans have been emerging in the People’s Republic of China in recent years. To date, 6 hemotrophic *Mycoplasma* spp. have been identified in rodents and mammals (1). *M. suis* from pigs, *M. wenyonii* from cattle, and *M. ovis* from sheep have been confirmed; the human pathogen, which is most frequently observed in China, has not been genetically identified (2). However, the zoonotic potential of the bacteria is evident because the disease is more prevalent in farmers and veterinary doctors, who have frequent close contact with domestic animals, than in other persons (2). Vertical transmission from mother to fetus has also been confirmed (2). In animals, especially in piglets, the disease is characterized by febrile acute anemia, jaundice, and eventual death resulting from concurrent infection with other microbes (3–6). Infected humans may be asymptomatic or have various clinical signs, including acute fever, anemia, and severe hemolytic jaundice, especially in infected neonates. Pregnant women and newborns were reported to be more vulnerable to the disease than others and to show more severe clinical signs after infection (2).

We conducted an epidemiologic investigation of hemotrophic mycoplasma infections in China by reviewing all reported cases and outbreaks for 1994–2007. Clinical cases for >6 animal species (including pigs, cows, goats, horses, foxes, chickens, and humans) were reported during the period (Table). The number of reported cases varied from year to year. Human infections were confirmed by clinical and laboratory methods (2). We investigated blood samples of >600 pigs with previous diagnoses of mycoplasma infection accompanied by clinical signs of fever and jaundice. Slides were made and stained in Giemsa-staining solution. We used light microscopy to look for the presence of *M. suis* on the erythrocyte surface. We also used fluorescence microscopy to look for the microbes by mixing a drop of infected blood with acridine orange solution (0.1 mg/mL). The microbes bound to red blood cells were examined with a confocal microscope. Positive cases were further confirmed by PCR using primers of the small subunit RNA gene sequences. All samples were PCR positive, but PCR sensitivity is higher than sensitivity of acridine orange staining, which is higher than sensitivity of Geimsa staining.

Hemotrophic mycoplasma infection is still a neglected zoonotic disease, which poses a threat to public health and the animal industry, especially in China (2,7). The prevalence of the dis-