strains was associated with increased isolate diversification (8), which suggests a selective restriction of the dominant circulating strain (2). In France, the W135/ST11 strain was rare after 2005; no cases were culture confirmed in 2010, and the 2 cases that were confirmed in 2011 showed the FetA2-19 marker. However, isolates from Africa during 2000–2011 frequently showed the FetA1 marker; in sub-Saharan Africa, the decline of W135/ST11 isolates was also associated with isolates showing diversified FetA markers (M.-K. Taha, unpub. data). The reemergence in 2012 of W135/ST11 strains that had the FetA1-1 marker suggests an antigenic shift that may have involved membrane proteins other than FetA or other surface structures, such as the lipooligosasccharide. Such antigenic shifts were associated with increased incidence of serogroup C and serogroup Y meningococcal disease in the United States (9). Antigenic shift could be a marker of changes in virulence and transmission of meningococcal isolates. Extensive molecular typing of meningococcal isolates is more likely to detect antigenic shifts and escape variants that may undergo clonal expansion and therefore should be employed in outbreak investigations. Enhanced surveillance was setup in France to identify imported W135 cases.

Our findings indicate that travelers to the meningitis belt of sub-Saharan Africa may be at risk for infection with *N. meningitidis* of serogroup W135. A vaccination campaign using the meningococcal A conjugate vaccine is ongoing in this region (10), but a conjugate bivalent vaccine that includes W135 should also be considered. Vaccination of travelers to this region with quadrivalent meningococcal vaccine should be recommended.

Information regarding the patients and their contacts were provided by the clinicians, the French Institute for Public Health Surveillance (wwwinvs.sante.fr), and the Regional Health Agencies of Pays-deLoire, Rhône-Alpes and Ile-de-France. In Côte d'Ivoire, the work was supported by the Agence Médecine Préventive and the mobile laboratory; patient information was provided by clinicians at health districts and the National Institute of Public Health.

Muhamed-Kheir Taha, Adèle Kacou-N'Douba, Eva Hong, Ala Eddine Deghmane, Dario Giorgini, Sophia Lurette Okpo, Tatiana Kangah, and Mireille Dosso

Author affiliations: Institut Pasteur, Paris, France (M.-K. Taha, E. Hong, A.E. Deghmane, D. Giorgini); and Institut Pasteur, Abidjan, Côte d'Ivoire (A. Kacou-N'Douba, S.L. Okpo, T. Kangah, M. Dosso)

DOI: http://dx.doi.org/10.3201/eid1906.120515

References

- Parent du Châtelet I, Traore Y, Gessner BD, Antignac A, Naccro B, Njanpop-Lafourcade BM, et al. Bacterial meningitis in Burkina Faso: surveillance using field-based polymerase chain reaction testing. Clin Infect Dis. 2005;40:17–25. http://dx.doi.org/10.1086/426436
- Traoré Y, Njanpop-Lafourcade BM, Adjogble KL, Lourd M, Yaro S, Nacro B, et al. The rise and fall of epidemic Neisseria meningitidis serogroup W135 meningitis in Burkina Faso, 2002–2005. Clin Infect Dis. 2006;43:817–22. http:// dx.doi.org/10.1086/507339
- Meningitis in Chad. Niger and Nigeria: 2009 epidemic season. Wkly Epidemiol Rec. 2010;85:47–63.
- Collard JM, Maman Z, Yacouba H, Djibo S, Nicolas P, Jusot JF, et al. Increase in *Neisseria meningitidis* serogroup W135, Niger, 2010. Emerg Infect Dis. 2010;16:1496–8. http://dx.doi. org/10.3201/eid1609.100510
- World Health Organization. Meningococcal disease: situation in the African meningitis belt. 2012 Mar 23 [cited 2013 Mar 18]. http://www.who.int/csr/ don/2012 03 23/en/index.html
- Vienne P, Ducos-Galand M, Guiyoule A, Pires R, Giorgini D, Taha MK, et al. The role of particular strains of *Neisseria* meningitidis in meningococcal arthritis, pericarditis, and pneumonia. Clin Infect Dis. 2003;37:1639–42. http://dx.doi. org/10.1086/379719

- Mayer LW, Reeves MW, Al-Hamdan N, Sacchi CT, Taha MK, Ajello GW, et al. Outbreak of W135 meningococcal disease in 2000: not emergence of a new W135 strain but clonal expansion within the electophoretic type-37 complex. J Infect Dis. 2002;185:1596–605. http://dx.doi. org/10.1086/340414
- Taha MK, Giorgini D, Ducos-Galand M, Alonso JM. Continuing diversification of *Neisseria meningitidis* W135 as a primary cause of meningococcal disease after emergence of the serogroup in 2000. J Clin Microbiol. 2004;42:4158–63. http://dx.doi.org/10.1128/JCM.42.9.4158-4163.2004
- Harrison LH, Jolley KA, Shutt KA, Marsh JW, O'Leary M, Sanza LT, et al.; Maryland Emerging Infections Program. Antigenic shift and increased incidence of meningococcal disease. J Infect Dis. 2006;193:1266– 74. http://dx.doi.org/10.1086/501371
- Marc LaForce F, Ravenscroft N, Djingarey M, Viviani S. Epidemic meningitis due to Group A *Neisseria meningitidis* in the African meningitis belt: a persistent problem with an imminent solution. Vaccine. 2009;27(Suppl 2):B13–9. http://dx.doi. org/10.1016/j.vaccine.2009.04.062

Address for correspondence: Muhamed-Kheir Taha, Invasive Bacterial Infections Unit, Institut Pasteur, 28 rue de Dr Roux, 75015 Paris, France; email: mktaha@pasteur.fr

Clostridium difficile Infection Associated with Pig Farms

To the Editor: Clostridium difficile of PCR ribotype 078 causes enteric disease in humans and pigs (1,2); a recent pan-European study revealed that this type was the third most frequently found type of C. difficile (1). The finding of identical C. difficile PCR ribotype 078 isolates in piglets with diarrhea and in humans with C. difficile infection (CDI) led to the suggestion that interspecies transmission might occur (3,4). Because C. difficile can be detected in the immediate environment of pig farms, we investigated intestinal colonization with C. difficile in pigs and in pig farmers, their relatives, and their employees in the Netherlands.

Persons living on 32 pig farms were enrolled as part of a longitudinal intervention study of several zoonotic agents. Pig farmers were partly recruited through the Dutch Farmer's Association or by veterinarians, who informed potential participants about the aims of the study. Inclusion criteria for participants were that they should work and/or live on the farm; the farms were either closed farms or multipliers (farms at which piglets are bred and then sold to other farms, where they are raised until ready for slaughter). The number of persons willing to submit a fecal sample per farm ranged from 1 to 10 (mean 4, median 5). Veterinarians who normally provided veterinary services to each farm collected fresh fecal samples from the floors of 10 animal wards per farm. No a priori knowledge of C. difficile colonization status of the pigs on the farms was available. Fecal samples from humans and from animal wards were cultured for the presence of toxinogenic C. difficile by using previously described methods (1,3,4).

Of the 128 persons who enrolled in the study, 48 had daily contact with pigs, 22 had weekly contact with pigs, and 36 had contact with pigs varying from monthly to less than yearly; no contact information was available for 22 participants. A total of 12 (25%) of 48 persons who had daily contact with pigs had fecal samples positive for C. difficile colonization; for persons who had weekly contact with pigs, 3 (14%) of 22 had positive samples. Daily to weekly contact with pigs versus monthly to less than yearly contact was significantly associated with an intestinal presence of C. difficile (p = 0.003). C. difficile was also found in fecal samples from 3 persons for whom no contact information was available. The C. difficile carriage rate among those with daily to weekly contact with pigs (15/70, 21%) was higher than the carriage rate of <5% reported for nonhospitalized adults with CDI (5).

A total of 18 *C. difficile*—positive human samples were detected at 16 of 32 pig farms investigated. At 2 of these farms, only 1 person submitted a sample, but at the other 14 farms, the number of participants ranged from 2 to 9 (mean 4, median 3). *C. difficile* was found in pig manure at all farms; 10%–80% of the wards were positive per farm.

Corresponding *C. difficile* PCR ribotypes were cultured from samples from pigs and humans; type 078 was found in humans and pigs on 15 farms and type 045 in a farmer and his pigs on 1 farm. Multilocus variable number tandem repeat analysis (MLVA) and antimicrobial drug susceptibility testing (E-test) were performed on human isolates from 15 farms and 1 porcine isolate per farm. One human isolate could not be typed because the isolate was lost during laboratory activities.

MLVA results showed that, at 2 farms, the human and porcine isolates were not genetically related, whereas at the other 13 farms, human and porcine isolates were genetically related, including 100% identical MLVA results for type 078 human and porcine isolates at 3 farms. Isolates were considered genetically related when the summed tandem repeat differences were <10 (3,4,6).

Antimicrobial drug susceptibility testing demonstrated similar susceptibility levels among isolates. For human and porcine isolates from 9 of 15 farms, MIC variability of <1 μg/L was found for imipenem, cotrimoxazole, erythromycin, clindamycin, tetracycline, and moxifloxacin. For the remaining 6 farms, drug susceptibility patterns for human isolates differed from pig isolates for 1 drug only: for 1 farm, MICs of erythromycin were 256 μg/L for human isolates and 0.38 μg/L for pig isolates; for 3 farms, MICs of erythromycin were 256 µg/L for pig isolates and 0.25 µg/L for human isolates; and for 2 farms, MICs of imipenem were 32 µg/L for pig isolates and 1.5 or $2 \mu g/L$ for human isolates.

In summary, the high *C. difficile* carriage rate among persons who had direct contact with pigs and the fact that these *C. difficile* isolates were genotypically and phenotypically similar to the pig isolates from the same farms indicates that transmission occurs either by direct contact or through the environment. Prospective studies are needed to determine the relationship between *C. difficile* carriage and development of CDI in this population.

This research was financially supported by ZonMW, the Netherlands organization for health research and development. None of the authors have any conflict of interest related to this article. This research is approved by the Animal Experiments Committee of Utrecht University and by the Medical Ethical Committee of Utrecht University.

Elisabeth C. Keessen, Céline Harmanus, Wietske Dohmen, Ed J. Kuijper, and Len J.A. Lipman

Author affiliations: Utrecht University, Utrecht, the Netherlands (E.C. Keessen, W. Dohmen, L.J.A. Lipman); and University Medical Center, Leiden, the Netherlands (C. Harmanus, E.J. Kuijper)

DOI: http://dx.doi.org/10.3201/eid1906.121645

References

- Bauer MP, Notermans DW, van Benthem BH, Brazier JS, Wilcox MH, Rupnik M, et al. Clostridium difficile infection in Europe: a hospital-based survey. Lancet. 2011;377:63–73. http://dx.doi. org/10.1016/S0140-6736(10)61266-4
- Songer JG, Anderson MA. Clostridium difficile: an important pathogen of food animals. Anaerobe. 2006;12:1–4. http://dx.doi.org/10.1016/j.anaerobe. 2005.09.001
- Debast SB, van Leengoed LA, Goorhuis A, Harmanus C, Kuijper EJ, Bergwerff AA. Clostridium difficile PCR ribotype 078 toxinotype V found in diarrhoeal pigs identical to isolates from affected humans. Environ Microbiol. 2009;11: 505–11. http://dx.doi.org/10.1111/j.1462-2920.2008.01790.x

- Goorhuis A, Bakker D, Corver J, Debast SB, Harmanus C, Notermans DW, et al. Emergence of *Clostridium difficile* infection due to a new hypervirulent strain, polymerase chain reaction ribotype 078. Clin Infect Dis. 2008;47:1162–70. http:// dx.doi.org/10.1086/592257
- Loo VG, Bourgault AM, Poirier L, Lamothe F, Michaud S, Turgeon N, et al. Host and pathogen factors for *Clostridium difficile* infection and colonization. N Engl J Med. 2011;365:1693–703. http://dx.doi. org/10.1056/NEJMoa1012413
- Marsh JW, O'Leary MM, Shutt KA, Pasculle AW, Johnson S, Gerding DN, et al. Multilocus variable-number tandem-repeat analysis for investigation of Clostridium difficile transmission in hospitals. J Clin Microbiol. 2006;44:2558–66. http://dx.doi.org/10.1128/JCM.02364-05

Address for correspondence: Elisabeth C. Keessen, PO Box 80175, 3508 TD, Utrecht, The Netherlands; email: E.C.Keessen@uu.nl

Prolonged Incubation Period for Cryptococcus gattii Infection in Cat, Alaska, USA

To the Editor: We report a case of *Cryptococcus gattii* infection in a 12-year-old neutered male cat in Alaska. The cat traveled to Anchorage, Alaska (61°N) from San Diego County, California, with its owner in August 2003. Although *C. gattii* has not been detected in Alaska (where only extremely limited sampling has occurred) or above 49°N (i.e., Vancouver Island, British Columbia), the case suggests that the incubation period for *C. gattii* could be >8 years.

In September 2010, the cat was brought to a veterinary clinic in Anchorage because of facial pruritis and excoriation. The cat did not respond to treatment with methylprednisolone acetate and was referred to a veterinary dermatologist in October. At that time, alopecia, thick scaling, and excoriations were observed on ear margins, sides of the head, and between the eyes. The hair coat was sparse, and

there was minimal scaling near the tail and lower legs. All foot pads were excessively cross-hatched and scaly. Cytologic analysis of the skin on the head and pinna showed neutrophils and cocci overgrowth. Skin scrapings were negative for mites. Cytologic analysis of the ears did not identify yeast bodies, parasites, or bacteria. A long-acting antimicrobial drug for treatment of skin infections (cefovecin) was given.

Biopsy specimens were obtained from the head, ears, and paws, and analysis of these samples supported a diagnosis of mural folliculitis and mild plasma cell pododermatitis. Chest radiograph findings and results of routine blood analysis were not unusual. Test results for feline leukemia virus and feline immunodeficiency virus were negative. Prednisolone (1.8 mg/kg/d) was given; the cat showed a good response and eventual resolution of scaling. Hair grew back but the steroid dose could not be reduced to <2.5 mg/d because periodic increases were needed when symptoms flared.

In November 2011, the cat was brought back to the veterinary clinic because of worsening of the skin condition even though the owner had increased the prednisolone dose to 7.5 mg/d during the previous 3 weeks. The cat had also started shaking its head frequently and had a unilateral right nasal discharge. Major nasal discharge had not been a symptom previously reported by the owner. Cytologic analysis of the discharge showed large yeast bodies consistent with a *Cryptococcus* sp. interspersed among neutrophils, cocci, and rods.

The cat was treated with fluconazole, and prednisolone was slowly decreased to minimal doses to control the mural folliculitis. After consultation with the Alaska Office of the State Veterinarian and Division of Public Health, a nasal swab specimen was sent to the Centers for Disease Control and Prevention (CDC) (Atlanta, GA, USA) for confirmation and molecular typing. Since 2009, Alaska has participated in the CDC-led Pacific Northwest *C. gattii* working group, which has been interested in enhanced surveillance for *C. gattii* (1,2). CDC identified the isolate as *C. gattii* molecular type VGIII; this type is commonly reported in the southern United States, particularly California (3).

Given what is known about the potential for dispersal of C. gattii (4), the owner was extensively interviewed about travel history of the cat and household members, and any travelers to or visitors from California who may have brought items into the house. The cat was rescued as a stray at ≈ 1 year of age in California, and after traveling to Anchorage had lived as an indoor/outdoor cat without further travel. The family had not transported or received organic materials from California, except for an elongated (≈45 cm) seedpod. All plants and potting soil had been bought locally from national chain vendors. In May 2012, fifteen environmental samples, including soil from the yard, commercial potting soil, and a planter made from the seedpod brought from California, were taken from the home of the cat; all showed negative results for *C. gattii* when tested at CDC.

In humans, the average incubation period for C. gattii infection is 6 weeks-13 months (5-7). Therefore, case-patients are usually asked to recall potential exposures during the 13 months before symptom onset (5). Although most reported cases of C. gattii infection appear to be primary infections, infrequent reports of C. gattii infections in immunocompetent persons have described symptoms occurring several years after likely exposure, which suggests that C. gattii may have a greater capacity to remain dormant than believed (8–10). Incubation periods are not well described for animals but are generally reported as 2-11 months (R. Wohrle, pers. comm.). For either animal or human case-patients living in disease-endemic areas, precise incubation periods are likely incalculable because potential exposure to fungi is ongoing.