infections. Although we did not conduct a case-control study, these findings also support the results of other studies that previously reported the demographic characteristics of patients with pandemic influenza infections and the risk factors for severe or fatal pandemic influenza infections (3,4), especially with respect to obesity (5).

Evaluation of tissues collected during autopsy from patients with a suspected infectious process can provide an etiologic diagnosis that was not available from routine premortem and postmortem testing. Other etiologic agents detected in this study included reportable disease agents (e.g., *Rickettsia rickettsii*, *Legionella pneumophila*, dengue virus), vaccine-preventable diseases (e.g., pneumococcal, meningococcal diseases), and zoonotic agents (*Leptospira* and *Capnocytophaga* spp.). These findings underscore the need for autopsies for diagnosing fatal infectious diseases (6). They also confirm the need for coordinated surveillance programs that identify deaths potentially attributable to infectious causes, including the unexplained deaths program (7) and medical examiner infectious diseases death surveillance program (8). Partnerships of medical examiners with local, state, and federal public health departments are crucial for detecting and monitoring pandemic diseases and for assessing the scope and magnitude of infectious agents that continuously affect human populations (9). These infections often result in sudden or unexplained death; thus, a standardized approach to death investigations is recommended.

Acknowledgments

We thank the state and local public health departments, the state and local public health laboratories, and all the pathologists and medical examiners who submitted specimens to the Infectious Diseases Pathology Branch.

Dianna M. Blau, Amy M. Denison, Julu Bhatnagar, Marlene De Leon-Carnes, Clifton Drew, Christopher Paddock, Wun-Ju Shieh, Sherif R. Zaki, and Infectious Diseases Pathology Branch Working Group

Author affiliation: Centers for Disease Control and Prevention, Atlanta, GA, USA

DOI: http://dx.doi.org/10.3201/eid1711.110429

References


1Infectious Diseases Pathology Branch Working Group members were Patty Adem, Jeanine Bartlett, Brigid Batten, Reema Dedania, Amy Green, Pat Greer, Tara Jones, Lindy Liu, Jeltley Montague, Mitesh Patel, Dominique Rollin, Chalanda Smith, and Libby White.


Address for correspondence: Dianna M. Blau, Centers for Disease Control and Prevention, 1600 Clifton Rd NE, Mailstop G32, Atlanta, GA 30333, USA; email: dblau@cdc.gov

---

**Epidemic Meningococcal Meningitis, Cameroon**

To the Editor: In 2010, the city of Ngaoundéré in Cameroon experienced its first reported epidemic of meningococcal meningitis. Ngaoundéré, with an estimated population of 180,000, is the main city in the Adamaoua region in northern Cameroon. The 2 northernmost regions of Cameroon, North and Far North, are considered to belong to the African meningitis belt (/) and are periodically affected by meningococcal meningitis outbreaks. However, the Adamaoua region had been spared because of its altitude, latitude, and low population density in comparison with the North and Far North regions. Fewer than 10 sporadic cases have been reported in the Adamaoua region every year.
During February–April 2010, a total of 126 cases of meningitis (70 cases/100,000 inhabitants) were reported in the Adamaoua region. Of the 126 cases, 34 were confirmed by identification of *Neisseria meningitidis* serogroup A in cerebrospinal fluid (CSF) samples, 46 cases were apparent meningitis in which the patients had turbid CSF, and 46 were clinical cases diagnosed in an epidemic context. The male:female ratio of the patients was 2.7:1. The mean age of patients was 19 years, and median was 17 years.

CSF specimens from 34 patients were sent to the Centre Pasteur du Cameroun in Garoua for testing. Laboratory procedures included assessing CSF turbidity, Gram staining, searching for soluble capsular antigens by using the Pastorex latex agglutination kit (Bio-Rad, Hercules, CA, USA), and testing by the dipstick rapid diagnostic test for *N. meningitidis* serogroups A, C, W135, and Y (provided by the Centre de Recherche Médicale et Sanitaire, Niamey, Niger). All 34 specimens were positive for serogroup A by agglutination, rapid test, or both. CSF specimens were cultured on blood agar and chocolate agar supplemented with PolyViteX (bioMérieux, Marcy-l’Etoile, France) and incubated at 37°C in an atmosphere of 5% CO₂. Susceptibility to antimicrobial drugs was tested according to the recommendations of the Antibiogram Committee of the French Society for Microbiology (www.sfm.asso.fr). An isolate of *N. meningitidis* was sent to the World Health Organization Collaborating Centre for Reference and Research on Meningococci in Oslo, Norway, for molecular analyses, as described (www.neisseria.org). The result was that the isolate, a *N. meningitidis* serogroup A clone of sequence type 7, was susceptible to β-lactams and chloramphenicol but resistant to trimethoprim/sulfamethoxazole.

This epidemic occurred in an area where the mean annual rainfall for the past 30 years was 1,460 mm (Agency for Aerial Navigation Safety in Africa and Madagascar, unpub. data). This value should exclude Ngaoundéré from the African meningitis belt, for which the southern limit of annual rainfall was classically considered to be the 1,100-mm isohyet (Figure).

This epidemic at the border of the African meningitis belt raises the question of the belt limitation and its potential expansion southward. These topics should be addressed through active and standardized surveillance in countries such as Cameroon, which are not entirely included in the belt (2,3).

This meningitis epidemic has 2 other noteworthy characteristics. First, 80 (63%) of 126 suspected cases had a lumbar puncture performed at the Ngaoundéré Regional Hospital or at the Norwegian hospital. With the help of the laboratory, an increasing number of cases of meningitis in Cameroon are confirmed cases (4). Second, the etiologic agent was serogroup A meningococcus, a serogroup that had not been identified in north Cameroon since 2006 (5) but that had been isolated previously (6) and in south Cameroon (7).

Acknowledgments
We thank the provinces’ authorities and the health districts’ staff for their collaboration and Pascal Boisier for assistance in preparing the manuscript.

This work was supported by the French Ministry of Foreign Affairs.
Foodborne-associated Shigella sonnei, India, 2009 and 2010

To the Editor: Infection with Shigella spp. is a major cause of foodborne diseases, which have increased considerably during the past decades, but only a small fraction of cases are reported (1). S. dysenteriae and S. flexneri are the predominant species in the tropics; clinically, S. dysenteriae serotype 1 is associated with severe disease, large outbreaks, or epidemics. S. sonnei occurs more frequently in industrialized than in developing countries and causes milder illness than S. dysenteriae and S. flexneri. However, occasional foodborne outbreaks by antimicrobial drug–resistant S. sonnei have been reported from the United States, Japan, and European countries, mostly among children (2–5). During recent years, in Thailand, Vietnam, and Sri Lanka, the predominant species has shifted from S. flexneri to S. sonnei, a phenomenon possibly linked with country’s level of development (6,7). As a result, S. sonnei outbreaks are also being reported from developing countries (8). In India, the scenario differed somewhat. Devastating outbreaks of dysentery by multidrug-resistant S. dysenteriae type 1, with high case-fatality rates, affected major parts of the country during 1984–1985 (9). After a gap of 18 years, during 2002–2003, S. dysenteriae type 1 with an altered antimicrobial drug resistance pattern (100% fluoroquinolone resistance) reemerged, causing several dysentery outbreaks in West Bengal (10). Although S. flexneri was the major species, since 2005, S. dysenteriae type 1 has not been isolated (9).

We report 2 foodborne outbreaks of S. sonnei in India, 1 each from Kerala (southern part) in February 2009 and Maharashtra (western part) in February 2010, which support extension of S. sonnei into India. The outbreak isolates were characterized by antimicrobial drug resistance and plasmid and pulsed-field gel electrophoresis profiles.

On February 1, 2009, >300 persons (age range 2–70 years) attended a marriage party at Thiruvananthapuram, Kerala, where they were served local food made of rice, lentils, milk, and water. Within 12 hours after eating, ≈60% of persons had onset of acute diarrhea, vomiting, and abdominal pain and were admitted to local village or district hospitals or the nearest government general hospital for treatment. Illness was more severe in children; because of clinical severity, 10 children (<10 years of age) were admitted to a referral hospital for children in Thiruvananthapuram. One child (7 years of age) was moved to the pediatric intensive care unit because of altered sensorium and drowsiness and was treated with intravenous ceftriaxone and metronidazole. Others were treated with oral cefotaxime until recovery and were discharged. Fecal samples from 15 patients were processed at the local public health laboratory for enteric pathogens; 9 (60%) of 15 samples yielded S. sonnei as the sole pathogen. On microscopic examination, 12 (80%) samples had erythrocytes.

The second outbreak occurred on February 11, 2010, at Kolhapur, Maharashtra, among day laborers and their family members who had eaten in 1 madrasa (religious place). Approximately 150 persons reported diarrhea, vomiting, abdominal pain, and fever. They were admitted to the Government Medical College, Kolhapur, and treated with intravenous fluid (lactated Ringer’s solution), oral rehydration solution, intravenous ceftriaxone, and metronidazole. All patients were discharged after complete recovery. S. sonnei was