

## **Ceftazidime-resistant *Salmonella enterica*, Morocco.**

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## Lessons from a Special Service for Public Health, Brazil

**To the Editor:** Many thanks for your interesting and informative special section on infectious diseases in the Amazon Region (1). Your readers should also be interested in a little known, but extremely successful, sustainable health program that had its start in the Amazon.

In 1942, the governments of Brazil and the United States agreed to establish a special service for public health (called the Serviço Especial de Saúde Pública). The purpose of this program was to improve health conditions in key areas in the Amazon, expedite the collection and export of native rubber, and counteract the growing influence of Nazi Germany in Latin America (2). The program spread to the Vale do Rio Doce, where there were resources of iron ore, mica, and optical quartz, which were important for the war effort. Although the program eventually moved to all states of Brazil, the Amazon program remained an important activity for ≈50 years before it was integrated into the Brazilian Ministry of Health (3).

The program in the Amazon focused primarily on infectious disease. It comprised programs of immunization, provision of small sustainable water systems, development of privy programs (sewer systems in the larger centers of population), malaria control, improvement of residences and living conditions for Chagas disease control, epidemiologic intelligence, and extensive training for auxiliary and professional personnel.

The effects of this program are shown by the increase in life expectancy for all age groups, with an increase of >10 years for those childhood age groups for whom infectious disease control would have the greatest effect from 1939–1941 to 1950–1951 (4).

This program contains many lessons for the planners of health and disease control projects in tropical, low-income countries.

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## Ceftazidime-Resistant *Salmonella enterica*, Morocco

**To the Editor:** Nontyphoidal salmonellosis (NTS) is a major food-borne illness worldwide. Extended-spectrum cephalosporins (ESCs) are currently preferred drugs for treatment of children with NTS. However, resistance to ESCs has emerged worldwide and has become a serious public health problem. This resistance is caused by

production of various class A extended-spectrum  $\beta$ -lactamases (ESBLs) and class C cephalosporinases in *Salmonella enterica* (1).

National surveillance systems, ideally based on integration of data for animals, food, and humans, are needed to develop strategies for containing antimicrobial drug resistance. Such systems are primarily based on a network of public or private clinical laboratories that refer *Salmonella* isolates to public health laboratories for identification. However, this laboratory-based surveillance system in developing countries is hampered by cost constraints and poor access to quality health facilities, resulting in a low rate of isolation of bacterial pathogens from patients having mild infections. These constraints account for the lack of data and underestimation of the number of NTS cases in many countries, including Morocco.

According to the World Health Organization Global Salm Surv database ([www.who.int/salmsurv/activities/en](http://www.who.int/salmsurv/activities/en)), the Moroccan National Institute of Hygiene reported only 210 human non-Typhi isolates and 999 animal non-Gallinarum isolates during 1999–2003. Antimicrobial drug resistance data are extremely rare. We report the presence of nontyphoidal *Salmonella* isolates resistant to ESCs during an outbreak of food poisoning and in food products in Morocco.

In March 2008, an *S. enterica* serotype Typhimurium strain was isolated from stool samples of 45 persons who had attended a wedding ceremony in Errachidia. Clinical symptoms were diarrhea, vomiting, and stomach cramps, beginning 24–72 hours after these persons had eaten a tagine prepared with poorly cooked broiler chickens. Five patients were hospitalized for 3 days, but no deaths were recorded. *S. enterica* serotype Typhimurium was isolated from leftovers of a broiler carcass stored in a refrigerator.

PulseNet (<http://pulsenetinternational.org/pulsenet/pulsenet.asp>)

standard pulsed-field gel electrophoresis (PFGE) of *Xba*I-digested chromosomal DNA showed that human and poultry isolates had identical profiles. Antimicrobial drug susceptibility was determined by the disk diffusion method and E-tests, as described (2). Isolates were resistant to penicillins and ceftazidime but were susceptible to other antimicrobial drug classes tested. A positive double-disk synergy test result suggested that these strains produced an ESBL. Isolates showed higher levels of resistance to ceftazidime (MIC 128 mg/L) than to ceftriaxone (MIC 8 mg/L).

For identification of the ESBL gene, we conducted PCR amplifications of *bla*<sub>TEM</sub>, *bla*<sub>SHV</sub>, and *bla*<sub>CTX-M</sub> group genes, as described (3). Only the SHV amplicon was obtained, and DNA sequencing showed this amplicon to be 100% identical to *bla*<sub>SHV-12</sub>. Resistance to ESCs and the *bla*<sub>SHV-12</sub> gene were transferred into *Escherichia coli* by conjugation. An ≈60-kb plasmid was isolated from *E. coli* transconjugants and the parental strain. PCR-based replicon typing analysis identified replicon IncII (4). Although the broilers had been reared locally, no environmental investigation was conducted.

In November 2007, an *S. enterica* serotype Newport strain was isolated from a pastry made with locally produced eggs during a food survey conducted in southern Morocco. The isolate was resistant to penicillins, ceftioxin (MIC 128 mg/L), ceftriaxone (MIC 64 mg/L), ceftazidime (MIC 128 mg/L), streptomycin, sulfonamides, chloramphenicol, and tetracycline. We identified the *bla*<sub>CMY-2</sub> gene carried by a 210-kb nonconjugative plasmid of replicon IncA/C. These CMY-2-producing isolates are also known as *Salmonella* Newport multidrug-resistant (MDR)-AmpC. *Xba*I-PFGE showed a profile similar to the New8a profile described in 2003 in France during a small outbreak linked to consumption of imported horse meat (2).

ESC-resistant *Salmonella* isolates have been reported in Morocco (5). This report described a serotype Typhimurium clone that produced TEM-3 that was isolated from 10 children hospitalized in Casablanca in 1994. Few studies have been conducted on ESC-resistant *S. enterica* in northern Africa, and most have reported hospital-acquired infections (1). Our study identified ESC-resistant *Salmonella* strains in the human food chain and in poultry for human consumption in Morocco. *Salmonella* isolates resistant to ESCs were not identified in food during 2002–2005 at the Institut Pasteur de Casablanca (104 *Salmonella* isolates from 11,516 food samples) (6). Emergence in poultry and humans of an MDR serotype Keurmassar strain that produced SHV-12 was reported in Senegal in 2001 (7).

Although CMY-2 was originally identified in a serotype Senftenberg isolate from a child in Algeria (8), we report the *Salmonella* Newport MDR-AmpC strain in Africa. *Salmonella* Newport MDR-AmpC isolates were reported in 1998 in the United States (9), where they quickly spread to cattle and humans. Recent potential spread of this strain into poultry in the United States was suggested by Varma et al. (10). Because of the risk for spreading, an efficient national antimicrobial drug resistance monitoring system for foodborne pathogens in Morocco is required to prevent dissemination of bacterial strains resistant to first-line antimicrobial drugs in humans.

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## Group B Streptococcus Meningitis in Child with Cochlear Implant

**To the Editor:** *Streptococcus agalactiae*, designated group B streptococcus (GBS), is a major cause of infections in neonates and young infants (1). Invasive GBS disease in children beyond infancy is uncommon, occurring mainly as bacteremia without a focus; meningitis caused by GBS is rarely reported (2). Cochlear implant recipients have been documented as having a higher rate of postimplantation bacterial meningitis than a cohort of the same age in the general US population (3). However, no cochlear implant recipient described has been reported to be infected with GBS. We report a case of GBS meningitis in a 6-year-old boy with a cochlear implant.

The patient was hospitalized in 2007 with a 1-day history of fever, headache, and vomiting. His medi-

cal history indicated congenital bilateral deafness diagnosed at 1 month of age and consistent with Patterson syndrome (i.e., unusual facies, deafness, bronzed hyperpigmentation of the skin, cutis laxa, mental retardation, and bony deformities) (4). At 4 years of age, he received a right-ear cochlear implant with good functional result. Preoperative high-resolution computed tomography of the temporal bones showed bilateral inner ear malformations of both the cochlear and vestibular labyrinth, conditions consistent with bilateral Mondini deformity (5). Mastoids and middle ears were well aerated. No evidence of cerebrospinal fluid leak appeared during physical examination or imaging. He received a dose of 23-valent pneumococcal polysaccharide vaccine.

At the time of hospital admission, he was somnolent but could be aroused and was cooperative. He had nuchal rigidity, dysmorphic facies, and oligodactyly. Fundi, skin, and ears were unremarkable on examination. Lumbar puncture showed a total protein level of 204 mg/dL, a glucose level of 1.6 mmol/L (blood glucose 3 mmol/L), and 4,800 leukocytes/mm<sup>3</sup> with 88% neutrophils; no bacteria were seen on the Gram stain. Blood count was remarkable for leukocytosis of 30,000/mm<sup>3</sup> and neutrophil predominance.

The patient received treatment with dexamethasone, vancomycin, and ceftriaxone; after treatment, his condition improved rapidly. Blood culture was sterile, but GBS grew in the cerebrospinal fluid culture (the isolate being resistant only to tetracycline). Therapy was continued with ampicillin for 3 weeks. Repeated testing of his hearing and speech perception with the cochlear implant showed no deterioration.

GBS plays a major role in early- and late-onset infections in neonates and young infants (1). Infections in older children and adults have been described, especially in elderly patients

or those suffering from chronic conditions such as diabetes mellitus, malignancy, or HIV infection (6). A review of medical records of patients with GBS infections over a 7-year period at a children's hospital in Memphis, Tennessee, USA, showed that, among 18 patients >3 months of age (13% of all GBS infections in the study), bacteremia was most commonly reported; 3 cases of ventriculo-peritoneal shunt infections were recorded, but no cases of meningitis without foreign devices were found (2). GBS meningitis in children beyond infancy is rare; only a few cases have been reported (7).

Cochlear implantation is the standard treatment for children and adults affected by severe and severe-to-profound sensorineural hearing loss. The implant is a neural stimulator with an electrode array surgically placed near the auditory nerve fibers in the scala tympani of the cochlea. Pediatric cochlear implant recipients were found to be at higher risk for developing bacterial meningitis than children in the general US population (3). Increased risk was evident in the perioperative period but extended to >2 years postimplantation (8). Most meningitis cases were associated with an implant with a positioner, a silastic wedge inserted next to the implanted electrode in the cochlea to position the electrode closer to the cochlear nerve endings and thus facilitate electrical signal transmission. Most of those infections were caused by *Streptococcus pneumoniae*, and none by GBS (3,8). In our patient, the implant did not include a positioner. The timing of meningitis was consistent with the timing indicated in previous reports, but the infecting organism was unique.

Inner ear malformations themselves are associated with increased risk for meningitis (9). The patient reported here had bilateral inner ear malformations; therefore, estimating the relative role of the deformity compared with the cochlear implant's role in the pathogenesis of meningitis in