

in Mexico (6) and the United States (7). In the United States, several pregnant women died, and the hospitalization rate for pregnant women was 4× higher than for the general population (8). Despite a fairly high birth rate on Reunion Island (19 births/1,000 population), our small series does not support these findings.

During the epidemic (July 20–September 20, 2009), acute respiratory infections, including presumed cases of pandemic (H1N1) 2009, accounted for 20.6% of the total case load of physicians on the island. The attack rate was ≈12.9% among the 810,000 inhabitants, and 8 deaths among persons with confirmed infection were reported. Therefore, the minimal overall death rate was ≈7.5 per million population and the case-fatality rate, 1 per 10,000 population.

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Crimean-Congo Hemorrhagic Fever Virus, Northeastern Greece

To the Editor: Crimean-Congo hemorrhagic fever virus (CCHFV) causes a disease in humans that is characterized by fever and hemorrhagic manifestations, with death rates up to 30%. Humans are infected through tick bites or contact with the viremic blood of patients or livestock. CCHFV belongs to the genus *Nairovirus* (family *Bunyaviridae*), which contains 7 serogroups: CCHFV, Dugbe virus, Hughes virus, Sakhalin virus, Dera Ghazi Khan virus, Qalyub virus, and Thiafora virus.

A CCHFV strain, AP92, was isolated from *Rhipicephalus bursa* ticks collected in 1975 from goats in Vergina, a village in northern Greece (1). Seroprevalence among Vergina residents was 6.1% (2). During 1981–1988, the seroprevalence among 3,388 persons in Greece was 1.1% (range 0%–9.6%) (3). The first Crimean-Congo hemorrhagic fever case in Greece was reported in 2008, when a woman died in Komotini in northeastern Greece (4). The causative strain (Rodopi) differs from strain AP92 (5).

To determine the prevalence of CCHFV antibodies in the human population of northeastern Greece, serum samples were collected prospectively during November 2008–April 2009 from 1,178 residents of Drama, Kavala, Xanthi, Rodopi, and Evros prefectures. A predefined number of participants were enrolled in the study on the basis of prefecture population. Participants were selected randomly among persons who were referred to health care settings for blood testing, regardless of reason for testing, and regardless of CCHFV risk factors. Oral consent was given by all participants. A questionnaire was completed concerning age, sex, occupation, place of residence, history of tick bite, symp-

toms after the bite, contact with animals, and any other situation related with increased risk for tick bite. All age groups were included (range 0–97 years, mean \pm SE age 53.2 ± 0.63).

Serum samples were tested for CCHFV immunoglobulin (Ig) G by ELISA (Vektor-Best, Koltsovo, Novosibirsk, Russia). The data were analyzed by using Stata Special Edition 9 (StataCorp LP, College Station, TX, USA). Multivariate logistic regression modeling was adopted to identify potential risk factors for acquisition of CCHFV infection. Odds ratios (ORs) with 95% confidence intervals (CIs) were obtained. *p* values <0.05 were considered significant.

In total, 37 (3.14%) of 1,178 persons were positive for CCHFV by IgG. The mean \pm SE age of seropositive and seronegative persons was 68.7 ± 2.54 years (range 0–87 years) and 55.6 ± 0.79 years (range 0–97 years). The female:male ratio was 1.6 among tested persons and 0.6 among seropositive persons. Seroprevalence differed among prefectures: Rodopi, where the fatal Crimean-Congo hemorrhagic fever case was observed, and Evros had the highest values (4.95% and 4.49%), Drama and Xanthi the lowest (1.34%

and 1.09%), and no IgG-positive person was found in Kavala. The distribution of regions where IgG-positive persons were found is presented in the Figure. Seropositive persons lived in rural areas at an altitude of 10m to 270 m; however, this factor was not significant ($p = 0.358$).

Crude analysis showed that age, sex, prefecture, occupation, contact with goats and sheep, slaughtering, and history of tick bite were significantly associated with seropositivity. Multivariate analysis showed that the following variables were significant risk factors for acquisition of CCHFV infection: age (OR 1.05, 95% CI 1.02–1.08; $p = 0.002$), residence in Rodopi prefecture (with Drama prefecture as reference category) (OR 6.55, 95% CI 1.36–31.60; $p = 0.019$), contact with goats (OR 3.40, 95% CI 1.22–9.43; $p = 0.019$), history of slaughtering (OR 2.53, 95% CI 1.01–6.45; $p = 0.048$), and history of tick bite (OR 2.51, 95% CI 1.03–6.15; $p = 0.044$).

When we compared our results with those of Antoniadis et al. (3), marked differences were seen: seroprevalence in Rodopi, Evros, Xanthi, and Drama was 0.5%, 0%, 1.2%,

and 0%, respectively, compared with 4.95%, 4.49%, 1.09%, and 1.34% in the present study, which suggests that during the past 20 years CCHFV was introduced in some areas in Greece or increased its circulation in others. Climatic and environmental changes and infested livestock movements (legal or illegal) in a habitat suitable for ticks might play a role in the current situation (6).

Further studies are necessary to estimate the seroprevalence in the whole country and detect disease-endemic foci of the disease. In addition, surveys for CCHFV in Ixodid ticks are necessary to enable the construction of risk maps and risk assessment analysis.

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Figure. Five prefectures in northeastern Greece (inset), showing locations of persons who were immunoglobulin G-positive for Crimean-Congo hemorrhagic fever virus (solid circles), 2008–2009. Size of circle indicates number of persons with positive test results in each location.

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Class D OXA-48 Carbapenemase in Multidrug-Resistant Enterobacteria, Senegal

To the Editor: Class D OXA β -lactamases are characterized as penicillinases that can hydrolyze oxacillin and cloxacillin and are poorly inhibited by clavulanic acid and EDTA. OXA-48 is one of the few members of this family to possess notable carbapenem-hydrolyzing activity (1). First described in 2004 in Turkey, OXA-48 has recently started to spread in Europe and the Middle East (2). We report the recent emergence of the plasmid-encoded *bla*_{OXA-48} gene in multidrug-re-

sistant *Enterobacteriaceae* recovered from patients in Dakar, Senegal, in hospitals and in the community.

From November 2008 through October 2009, 11 *Enterobacteriaceae* isolates (8 *Klebsiella pneumoniae*, 1 *Escherichia coli*, 1 *Enterobacter cloacae*, and 1 *Enterobacter sakazakii*) with reduced susceptibility to imipenem were identified at the Institut Pasteur (Dakar, Senegal). Antibacterial drug susceptibility was determined by the disk diffusion method and interpreted according to the European Committee on Antimicrobial Susceptibility Testing guidelines (www.eucast.org). Nine isolates were resistant to expanded-spectrum cephalosporins and also to other antibacterial drug classes.

The isolates were recovered from 6 patients with urinary tract infections, 4 patients with surgical infections, and 1 patient with omphalitis. Nine infections were hospital acquired (Le Dantec and Principal Hospitals). Because the patients died before antibacterial drug susceptibility testing could be completed, all 5 patients with surgical infections or omphalitis received only empirical therapy with amoxicillin/clavulanate. One patient with a nosocomial urinary tract infection caused by a co-trimoxazole-susceptible strain was successfully treated with this antibacterial agent. The antibacterial drug regimens of the remaining 4 patients were not known, and they were lost to follow-up. We determined the MICs of imipenem, meropenem, and ertapenem by using the Etest method (AB Biodisk, Solna, Sweden), which showed that 9 isolates were susceptible to imipenem and meropenem but either intermediately susceptible or resistant to ertapenem (Table). The 2 imipenem-nonsusceptible isolates were susceptible or intermediately susceptible to meropenem, and both were resistant to ertapenem.

We used previously described PCRs (1,3–7) to screen for carbapenem-hydrolyzing β -lactamase genes

(*bla*_{VIM}, *bla*_{IMP}, *bla*_{KPC}, and *bla*_{OXA-48}), as well as plasmid-encoded *bla*_{CTX-M}, *bla*_{AmpC}, *bla*_{OXA-1}, and *bla*_{TEM} β -lactamase genes; the *aac(6)-Ib* aminoglycoside resistance gene; the quinolone resistance genes *qnrA, B, S*; the tetracycline resistance genes *tetA, B, D*; and class 1 integron. The *bla*_{OXA-48}, *bla*_{CTX-M}, *bla*_{AmpC}, and *aac(6)-Ib* genes and the variable region of class 1 integron were then characterized by direct DNA sequencing of the PCR products. *bla*_{OXA-48} was present in all 11 isolates. *bla*_{VIM}, *bla*_{IMP}, and *bla*_{KPC} were not detected. The *qnr* genes were present in 7 isolates resistant to ciprofloxacin. The *aac(6)-Ib-cr* variant was present in 7 isolates resistant to gentamicin, tobramycin, and ciprofloxacin.

The 9 isolates resistant to expanded-spectrum cephalosporins all harbored the *bla*_{CTX-M-15} gene. The *E. coli* isolate also harbored the plasmid-encoded *bla*_{AmpC} gene ACT-1; *bla*_{CTX-M-15}, *bla*_{OXA-1}, *bla*_{TEM}, and *aac(6)-Ib-cr* were associated in 6 isolates. Long-range PCRs showed that these latter 4 genes were located in the same “multidrug resistance region,” as described in Senegal (6). Positive conjugation experiments with sodium azide-resistant *E. coli* J53 showed through PCR results, plasmid DNA extraction, and antibiogram patterns of the obtained transconjugants that *bla*_{OXA-48} was located on a 70-kb self-conjugative plasmid.

The genetic environment of *bla*_{OXA-48} was then investigated by PCR with primers specific for insertion sequence IS1999 and for the 5' part of *bla*_{OXA-48} (1). *bla*_{OXA-48} was found to be part of a *Tn1999* composite transposon composed of 2 copies of the insertion sequence IS1999, as reported (2). Further sequencing of the IS1999 located upstream of *bla*_{OXA-48} showed that it was consistently truncated by the insertion sequence IS1R, as initially described in Turkey and more recently in Lebanon and Egypt (2,8).

*Xba*I pulsed-field gel electrophoresis was then used to study the genetic relatedness of the 8 *K. pneumoniae*