Vancomycin-Resistant Enterococci, Point Barrow, Alaska, USA

To the Editor: An increasing number of bacterial infections are now difficult or impossible to treat (1) because of the misuse of antimicrobial drugs and the epidemic spread of bacterial resistance to these drugs (2). The most alarming reports are of methicillin-resistant Staphylococcus aureus, extended-spectrum β-lactamase producers, and vancomycin-resistant enterococci (VRE). Although knowledge about dissemination mechanisms is poor, the spread of resistance clearly is not restricted to hospitals but occurs also in the community and in the natural environment (3,4). Since the 1990s, the epidemiology in the United States has shifted so that most VRE are Enterococcus faecium. Recent studies indicate clonal spread of the E. faecium CC17 lineage in clinical isolates, exhibiting high-level ampicillin and fluoroquinolone resistance and harboring an enterococcal surface protein–coding esp gene (5,6).

During a polar research expedition to the Beringia region in 2005, we collected fecal samples from birds at sites with no or low human population. The aim was to investigate the current status of resistance dissemination into remote areas of the world. The study site in Alaska was located on the tundra halfway between the city of Barrow and Point Barrow, the northernmost point of the United States (71°23′20″N, 156°28′45″W). Fecal samples from glaucous gulls (Larus hyperboreus) were enriched (18 h at 37°C) in brain–heart infusion broth (Becton Dickinson, Franklin Lakes, NJ, USA) supplemented with aztreonam and vancomycin (10 mg/L and 4 mg/L, respectively; ICN Biomedicals Inc., Aurora, OH, USA), followed by spreading on chromID VRE plates (bioMérieux, Marcy l’Étoile, France) and incubated for 48 h at 37°C. Typical colonies were isolated and species identified by biochemical testing, including the Phoenix Automated Microbiology System (Becton Dickinson). MIC was determined for vancomycin, teicoplanin, ampicillin, and ciprofloxacin by using Etest strips (AB Biodisk, Solna, Sweden), and the presence of vanA, vanB, and esp genes was established by PCR with previously described primers (7,8) (esp primers esp11 and esp12).

Cultures showed 2 isolates of E. faecium; MICs for vancomycin and teicoplanin were >256 and 96 μg/mL, respectively, for both isolates. Genotyping determined that they harbored vanA. Isolates exhibited high-level ampicillin and ciprofloxacin resistance; MICs were >256 and >32 μg/mL, respectively for both isolates. They also harbored the esp gene. Isolates came from 2 of 33 sampled glaucous gulls, a species confined to the Arctic regions, that have limited southbound migration during the nonbreeding season.

Clinical isolates of VRE were first found in the late 1980s. In the United States, vancomycin was widely used in human medicine, and outbreaks occurred in hospitals rather than in the community; the opposite was, and is, true in Europe. Because of massive use of glycopeptide antimicrobial drugs, i.e., avoparcin, as growth promoters in domestic animal production until the mid-1990s, VRE can be found in hospitals and the community (9).

Our findings show that bacteria resistant to antimicrobial drugs, or resistance genes, already have spread to one of the most remote areas of North America, Point Barrow, Alaska. This spread suggests that few (if any) places on earth may be protected against the spread of such resistance, and the dispersal mechanisms are far more efficient than previously thought. Our data also place the isolates as part of the clinically spread clonal E. faecium CC17 lineage, characterized by high-level ampicillin and quinolone resistance and harboring the esp gene, thus strongly supporting a human origin. Possible dispersal mechanisms to remote areas include stepwise horizontal transfer between migratory and nonmigratory bird species and anthropogenic transport.

The increasing evolution and spread of antimicrobial drug–resistant bacteria and resistance genes seriously threaten public health and could escalate to catastrophic proportions (1). Bacteria and drug resistance are easily transferred between humans and animals and consequently between the environment and clinical settings. Much remains to be learned about the effect of human-associated changes of natural ecosystems on the total effect of resistance. Therefore, our finding of VRE at Point Barrow is important to recognize. Decisive action is needed to establish efficient monitoring programs that include not only surveillance and control of clinical bacterial resistance but also environmental levels of resistance.

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References

1. Cohen ML. Epidemiology of drug resistance: implications for a post-antimicrobial era. Science. 1992;257:1050–5. DOI: 10.1126/science.257.5073.1050
Use of Templates to Identify Source of Norovirus Outbreak

To the Editor: On November 22, 2006, an infection control nurse notified the Marion County (Oregon) Health Department about acute gastroenteritis among persons who had attended a reception at a medical facility on November 16, 2006. With a holiday weekend only hours away, the county health department asked the state health department to join the outbreak investigation.

After interviewing the caterer, organizers, and several attendees, we modified a questionnaire template to reflect potential exposures. Using this questionnaire, we conducted a retrospective cohort study by telephone among reception attendees identified from a ticket list. We defined a case of acute gastroenteritis as reported vomiting or diarrhea (>3 loose stools within a 24-hour period) within 18–72 hours of the event.

Sanitarians inspected the facility and the caterer’s kitchen. We traced implicated oysters (the source of the outbreak) through distribution records; screened stool specimens for norovirus by RT-PCR; tested oysters from the implicated lot for norovirus by qRT-PCR; entered data into a custom outbreak database template; calculated relative risks (RRs) and 95% confidence intervals (CIs) using Epi Info (www.cdc.gov/epiinfo); and assessed the significance of the association between acute gastroenteritis and consumption of implicated oysters by the chi-square or Fisher exact test.

Approximately 200 persons attended the reception. We called all households on the reception ticket list with identifiable phone numbers and reached a convenience sample of 66 attendees from 50 households. We determined that 10 had cases of acute gastroenteritis, 53 had no symptoms, and 3 (who were excluded from the analysis) had minor symptoms. The median incubation period was 36 hours (range 31–63 hours). None of the 10 attendees with acute gastroenteritis sought medical attention; stool specimens from 2 of them tested positive for norovirus (1 positive for genogroup II and 1 positive for both I and II).

Illness was associated with consumption of raw oysters on the half shell (RR 11.8; 95% CI 2.8–50; p = 0.0001), which was reported by 8 of the 10 attendees with acute gastroenteritis. No other foods were associated with illness. No significant breaches in food-handling procedures were identified. The only food handler who reported illness had eaten several oysters at the event and became ill 36 hours later.

The oysters had been individually quick frozen on the half shell and packed loosely in cartons after being harvested in South Korea by growers approved by the US Food and Drug Administration. For the reception, a single 6-kg box of oysters was thawed and served raw. The box was from a shipment of 2,200 boxes legally imported in October 2006. Boxes from the same shipment had been distributed to 5 states. Oysters from 4 other cartons were consumed (some cooked) at 2 other Oregon locations. Public health officials in other states were notified and asked to report any related illnesses; none were identified.

Noroviruses (genogroups I and II) were detected in oysters from an intact carton of the implicated lot. Sequencing was not attempted. The implicated lot was voluntarily recalled by the national distributor; most of the lot was embargoed or recalled before the oysters were consumed.

Oysters are a recurrent source of outbreaks and sporadic cases of norovirus infection, vibriosis, and other infections (1) because they are frequently eaten raw or undercooked (2). Microbial monitoring of oyster harvest

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