References

1. McCann CM, Vyse AJ, Salmon RL, Thomas D, Williams DJ, McGarry JW, et al. Lack of serologic evidence of *Neospora caninum* in humans, England. Emerg Infect Dis. 2008;14:978–80. DOI: 10.3201/eid1406.071128
2. Dubey JP, Lindsay DS. A review of *Neospora caninum* and neosporosis. Vet Parasitol. 1996;67:1–59. DOI: 10.1016/S0304-4017(96)01035-7
3. Lobato J, Silva DA, Mineo TW, Amaral JD, Segundo GR, Costa-Cruz JM, et al. Detection of immunoglobulin G antibodies to *Neospora caninum* in humans: high seropositivity rates in patients who are infected by human immunodeficiency virus or have neurological disorders. Clin Vaccine Immunol. 2006;13:84–9. DOI: 10.1128/CVI.13.1.84-89.2006
4. Tranas J, Heinzen RA, Weiss LM, McAllister MM. Serological evidence of human infection with the protozoan *Neospora caninum.* Clin Diagn Lab Immunol. 1999;6:765–7.
5. Petersen E, Lebech M, Jensen L, Lind P, Rask M, Bagger P, et al. *Neospora caninum* infection and repeated abortions in humans. Emerg Infect Dis. 1999;5:278–80.

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Escherichia coli and *Klebsiella pneumoniae* Carbapenemase in Long-term Care Facility, Illinois, USA

To the Editor: *Escherichia coli* harboring *Klebsiella pneumoniae* carbapenemases (KPCs) are now rarely being reported. Worldwide, KPC-2 has been detected in Israel and the People's Republic of China (*1*,*2*). Within the United States, carbapenem-resistant *E. coli* carrying bla_{KPC} has been isolated in New Jersey (*3*) and Cleveland, Ohio (*4*), and 7 carbapenem-resistant *E. coli* isolates were obtained from 3 different hospitals in Brooklyn, New York (*5*). Urban et al. (*6*) recently reported 9 KPC-2 and KPC-3 carbapenemases in urinary *E. coli* isolates from 7 long-term care facilities. We report such an isolate from a resident of a long-term care facility.

This case involved a 68-year-old female resident of a long-term care facility in Centralia, Illinois, who had multiple chronic medical problems, including cerebral palsy, a seizure disorder, and recurrent urinary tract infections. A urine culture grew $>10^5$ CFU/mL of *E. coli* susceptible to amikacin, gentamicin, tobramycin, piperacillin/tazobactam, trimethoprim/sulfamethoxazole, imipenem, and nitrofurantoin. Tigecycline susceptibility was not determined. Trimethoprim/sulfamethoxazole therapy was initiated. Follow-up urine culture almost 3 weeks later again grew $>10^5$ CFU/mL of *E. coli*, now susceptible to amikacin, gentamicin, tobramycin, nitrofurantoin, and tigecycline. The isolate was resistant to imipenem and meropenem. A modified Hodge test demonstrated production of a carbapenemase (*7*), and the bla_{KPC} gene was detected by PCR at the Centers for Disease Control and Prevention (CDC). The patient was treated with a 10-day course of nitrofurantoin, 100 mg by gastrostomy tube 2× per day. Chart review indicated that contact precautions were instituted only after discovery of the second *E. coli* isolate.

Seventeen days later, a repeat urine culture grew $>10^5$ CFU/mL of *K. pneumoniae* susceptible only to amikacin, gentamicin, tobramycin, and tigecycline. No treatment was given. Follow-up urine culture grew $>10^5$ CFU/mL of *K. pneumoniae* again with a similar resistance pattern. The modified Hodge test result was positive (*7*) and was confirmed as bla_{KPC} positive by PCR at CDC. The resident was transferred to an acute care facility for further evaluation and was treated with amikacin. At completion of therapy, a repeat urine culture was negative for organisms.

Our case, like that of Urban et al. (*6*), involved a urinary isolate from a resident of a long-term care facility. As increasing numbers of resistant gram-negative rods colonize such patients, the patients may acquire a bacterium carrying a KPC plasmid conferring broad-spectrum resistance as described in our patient. These plasmids may then be laterally transferred to other gram-negatives, which may have occurred in this case.

Our case underscores the gravity of the evolutionary process of emergent, multidrug–resistant enterobacteriaceae. Even though *E. coli* strains that harbor carbapenemase genes are not ubiquitous, additional therapeutic interventions are needed to prevent the spread of these bacteria, which are likely to infect increasing numbers of patients.

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References

1. Navon-Venezia S, Chmelnitsky I, Leavitt A, Schwaber M, Schwartz D, Carmeli Y. Plasmid-mediated imipenem-hydrolyzing enzyme KPC-2 among multiple carbapenem-resistant *Escherichia coli* clones in Israel. Antimicrob Agents Chemother. 2006;50:3098–101. DOI: 10.1128/AAC.00438-06
2. Cai JC, Zhou HW, Zhang R, Chen GX. Emergence of *Serratia marcescens, Klebsiella pneumoniae*, and *Escherichia coli* isolates possessing the plasmid-mediated carbapenem-hydrolyzing β-lactamase KPC-2 in intensive care units of Chinese hospital. Antimicrob Agents Chemother. 2008;52:2014–8. DOI: 10.1128/AAC.01539-07

3. Hong T, Moland ES, Abdalhamid B, Hanson ND, Wang J, Sloan C, et al. *E. coli* producing KPC-3 carapenem hydrolyzing enzyme. In: Program and abstracts of the 43rd Interscience Conference on Antimicrobial Agents and Chemotherapy (Chicago). Washington: American Society for Microbiology; 2003. p. 75. Abstract C1-265.
4. Deshpande LM, Rhomberg PR, Sader HS, Jones RN. Emergence of serine carbapenemases (KPC and SME) among clinical strains of *Enterobacteriaceae* isolated in the United States Medical Centers: Report from the MYSTIC Program (1999–2005). Diagn Microbiol Infect Dis. 2006;56:367–72. DOI: 10.1016/j.diagmicrobio.2006.07.004
5. Bratu S, Brooks S, Burney S, Kochar S, Gupta J, Landman D, et al. Detection and spread of *Escherichia coli* possessing the plasmid-borne carbapenemase KPC-2 in Brooklyn, New York. Clin Infect Dis. 2007;44:972–5. DOI: 10.1086/512370
6. Urban C, Bradford PA, Tuckman M, Segal-Maurer S, Wehbeh W, Grenner L, et al. Carbapenem-resistant *Escherichia coli* harboring *Klebsiella pneumoniae* carbapenemase β-lactamases associated with long-term care facilities. Clin Infect Dis. 2008;46:e127–30. DOI: 10.1086/588048
7. Anderson KF, Lonsway DR, Rasheed JK, Biddle J, Jensen B, McDougal LK, et al. Evaluation of methods to identify the *Klebsiella pneumoniae* carbapenemase in *Enterobacteriaceae.* J Clin Microbiol. 2007;45:2723–5. DOI: 10.1128/JCM.00015-07

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Bedbugs and Healthcare-associated Dermatitis, France

To the Editor: Bedbugs (*Cimex lectularius*) are hematophagous insects. Adults are 4–6 mm long, flattened, oval and wingless, and brown to brownish–red (Figure, panel A) (*1*). They may feed in the wild on birds or bats (*2*), but they are mainly associated with human dwellings and can be found on furniture and clothing (*3*). Because bedbugs are nocturnal and feed painlessly only in the dark, while humans sleep, initial bedbug proliferation usually goes unnoticed until several weeks later when the patient discovers a pruritic cutaneous eruption of unknown origin (*4*). Decades ago, bedbugs were frequently found worldwide, but reports of cases in industrialized countries have progressively declined, probably the result of improved living conditions (*3*). They nonetheless remain a pest in less-developed countries and in the wild (*5*). The past 10 years have seen the revival of this insect in industrialized countries (*3*,*6*,*7*). Increasing reports describe isolated cases or bedbugs spreading throughout a single building (*8*). We report an outbreak of healthcare-associated dermatitis caused by bedbugs in a hospital nursing home in Cannes, French Riviera.

In July 2007, Mrs. Q arrived, with her bed and mattress, for admission to a single room in a hospital nursing home. This facility has 112 rooms located on 2 floors, each having A and B wings. Mrs. Q's first lesions, diagnosed as insect bites, appeared in October 2007. Concomitantly, Mrs. T, a long-term resident of the room across the hall (1.5 m away), developed similar lesions. Examination of Mrs. Q's room led to the discovery of an aggregation of 200 *C. lectularius* bedbugs beneath her mattress. In Mrs. T's room, 15 bedbugs were identified (Figure 1, panel B). Suspected insect excreta were also found in another nearby room. A private company conducted a nonspecific pest-control intervention in these 3 rooms.

In November 2007, another 2 residents in rooms located 3 and 6 m away from Mrs. Q's had insect-bite dermatitis: 15 bedbugs were found in each room. Over a 3-week period, the nursing home staff performed the second pest-control intervention in these 2 infested rooms and also treated 10 adjacent rooms. They disassembled furniture and applied insecticides to furniture, room corners (imiprothrine and cypermethrine), and clothing (esdepallethrine and piperonyl butoxide).

No additional skin lesions occurred during the next 4 months, and no new resident was admitted. In March 2008, a new long-term resident developed similar bedbug-dermatitis lesions (Figure, panel C); 12 *C. lectularius* bedbugs were found in his room (33 m from Mrs. Q's room, same floor, wing B). This time, a specialized private company conducted the pest-control intervention over a 2-month period in the 56 rooms on the second floor (wings A and B); they treated furniture and clothing and placed silicone sealer around doors and floorboards to obstruct potential pest refuges. All furniture was removed, disassembled, and washed. When no bedbugs or eggs were found, bendiocarb was applied preventively; otherwise, curative *d-trans*-tetramethrin was applied (*3*). No further infestation has been observed.

Three pest-control interventions were required to eliminate these infestations. The first was not specific for bedbugs, and the second was not sufficiently extensive. Only specific and extensive insecticide application achieved elimination. The temporal–spatial distribution of dermatitis in this facility suggests 2 types of transmission: during the first 2 waves, spontaneous movement of the bedbugs is the most likely hypothesis because