Previously reported babesiosis cases in children have been mostly acquired by blood transfusion (10). The patient had no history of transfusions with blood products and had never traveled outside his home town before disease onset. Although he and his parents did not recall any tick bites, he was at high risk for exposure to ticks because he often played with his dog, which frequently went outdoors in a tick-infested forested area. The dog may have transmitted a Babesia sp.–infected tick to the patient. However, ticks from the dog were not available for identification and testing.

The patient in our study was presumed to be healthy and immunocompetent, which indicates that Babesia species can cause infections even in healthy persons. Babesiosis should be considered in the differential diagnosis of patients with a history of tick exposure and prolonged and irregular fever. Blood smear evaluation for intraerythrocytic parasites should be considered.

The patient was treated with azithromycin and atovaquone and the parasites were cleared within 1 month. This combined treatment was well tolerated and effective, and it can be recommended as an alternative treatment to the commonly used therapy of quinine and clindamycin (1).

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References

1. Vannier E, Krause PJ. Human babesiosis. N Engl J Med. 2012;366:2397–407. http://dx.doi.org/10.1056/NEJMra1202018
2. Herwaldt BL, Cacció S, Gherlinzoni F, Aspöck H, Slemenda SB, Piccaluga P, et al. Molecular characterization of a non–Babesia divergens organism causing zoonotic babesiosis in Europe. Emerg Infect Dis. 2003;9:942–8. http://dx.doi.org/10.3201/eid0908.020748
3. Häselbarth K, Tenter AM, Brade V, Krieger G, Hunfeld KP. First case of human babesiosis in Germany: clinical presentation and molecular characterisation of the pathogen. Int J Med Microbiol. 2007;297:197–204. http://dx.doi.org/10.1016/j.ijmm.2007.01.002
4. Su GG, Zhao NF, Ye XX. A case report of babesiosis [in Chinese]. Chinese Journal of Zoonoses. 2002;18:112.
5. Yao LN, Wei R, Zeng CY, Li ZH, Zhang X, Lei Y, et al. Pathogen identification and clinical diagnosis for one case infected with Babesia [in Chinese]. Zhongguo Ji Sheng Chong Xue Yu Ji Sheng Bing Bing Za Zhi. 2012;30:118–21.
6. Zhou X, Li SG, Chen SB, Wang JZ, Xu B, Zhou HJ, et al. Co-infections with Babesia microti and Plasmodium parasites along the China–Myanmar border. Infect Dis Poverty. 2013;2:24. http://dx.doi.org/10.1186/2049-9957-2-24
7. Qi C, Zhou D, Liu JZ, Cheng ZQ, Zhang L, Wang L, et al. Detection of Babesia divergens using molecular methods in anemic patients in Shandong Province, China. Parasitol Res. 2011;109:241–5. http://dx.doi.org/10.1007/s00436-011-2382-8
8. Armstrong PM, Katavolos L, Caporale DA, Smith RP, Spilman A, Telford SR III. Diversity of Babesia infecting deer ticks (Ixodes dammini), Am J Trop Med Hyg. 1998;58:739–42.
9. Hunfeld KP, Lambert A, Kampen H, Albert S, Epe C, Brade V, et al. Seroreivalence of Babesia infections in humans exposed to ticks in midwestern Germany. J Clin Microbiol. 2002;40:2431–6. http://dx.doi.org/10.1128/JCM.40.7.2431-2436.2002
10. Fox LM, Wingeter S, Ahmed A, Arnold AP, Chou J, Rhein L, et al. Neonatal babesiosis: case report and review of the literature. Pediatr Infect Dis J. 2006;25:169–73. http://dx.doi.org/10.1097/01.inf.0000195438.09628.b0

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Extended-Spectrum β-Lactamases in Escherichia coli and Klebsiella pneumoniae in Gulls, Alaska, USA

To the Editor: Resistance to β-lactam antibacterial drugs has spread rapidly, particularly through the CTX-M β-lactamase enzymes (CTX-M) (1). Although CTX-Ms are geographically widely distributed, reports of extended-spectrum β-lactamase (ESBL) dissemination are few from remote regions. In 2008, we reported phenotypic resistance traits in Escherichia coli isolates in 8.2% of wild birds sampled in the Arctic (2). We sampled approximately 260 wild birds, mainly gulls and geese, but found no ESBL-harbouring isolates (J. Bonnedahl et al., unpub. data). Here we report results of our 2010 study at Barrow, Alaska, USA, a follow up to our 2005 study in which we found vancomycin-resistant enterococci (VRE) with clear traits of human origin in glaucous gulls (3). Our findings show a remarkable change, not in VRE dissemination, which is fairly unchanged, but in the emergence of ESBLs and general resistance of E. coli isolates.

We collected 150 fecal samples from a population of adult gulls residing close to a landfill site. For a description of general resistance levels (4,5), susceptibility of 1 randomly selected E. coli isolate per sample (137 isolated from 150 samples) was tested to a set of 10 antibacterial agents. Nearly half (48%) of the 137 E. coli isolates were resistant to at least 1 of the drugs tested. Resistance to 1 or 2 antimicrobial agents was found in 32% and 13% of the tested isolates, respectively, and resistance to ≥3 was found in 7% of isolates (online Technical Appendix Table, wwwnc.cdc.gov/EID/article/20/5/13-0325-Techapp1.pdf).
We analyzed samples for presence of VRE (3). Seven (4.7%) *E. faecium* isolates were found, all of which harbored both the *vanA* and the *exp* genes (found in isolates of the CC17 lineage) (3). No other VRE were found.

To investigate the presence of ESBL-producing bacteria, we conducted a selective screen as described (6). ESBL-producing bacteria were found (*E. coli* and *K. pneumoniae*), and ESBL genes (*blaCTX-M*, *blaSHV*, and *blaTEM*) in ESBL-positive isolates were analyzed (6). We found 33 *E. coli* and 35 *K. pneumoniae* ESBL-producing isolates in 55 samples (12 samples had >1 unique isolate), a total of 37% of ESBL-harboring samples (Table).

We performed multi-locus sequence typing (MLST) on ESBL-producing *E. coli* isolates (4). Isolates were of described sequence types (STs) (ST131 [12 isolates], ST38 [10], ST405 [3], and ST10 [1]), and of previously undescribed STs (designated ST2253 [1 isolate] and ST2967 [6 isolates]) (Table).

In our 2005 study in Barrow, general resistance was relatively low, and no ESBL was found; surprisingly, however, 2 VRE isolates of a human clonal lineage were found (3; M. Drobiñi et al., unpub. data). Since then, resistance dissemination, particularly that of ESBLs, has exploded globally (1). In 2010, we found a high level of general resistance; 48% of randomly selected *E. coli* isolates displayed resistance toward ≥1 antibacterial drugs. This level is similar to the level we found in gulls in France in 2008, an area with high current and historical clinical antibacterial drug use and where birds have close contact with human activities (4).

We screened samples for VRE and ESBL-producing bacteria. The prevalence of VRE decreased from 6% in 2005 to 4.7% in the current study (3), indicating a slow decline or stability in VRE. ESBL, on the other hand, was not found in the 2005 study (M. Drobiñi et al., unpub. data) but emerged in 37% of samples carrying *E. coli* and/or *K. pneumoniae* harboring ESBLs. In the study from France, only 9.4% of birds carried ESBLs (4), although a study of gulls in Portugal during 2007–2008 reported an ESBL carriage of 32% (7), more similar to results of our current study but in contrast also because they investigated gulls from a highly populated area.

*E. coli* isolates mainly carried *blaCTX-M-14* or *blaTEM-19* whereas *K. pneumoniae* isolates mainly carried *blaCTX-M-15*, *blaSHV-12*, or *blaSHV-102*. To our knowledge, ESBLs in *E. coli* and *K. pneumoniae* in clinical isolates (mainly from samples of persons with urinary tract infections and urosepsis). Our MLST of *E. coli* indicated 4 known STs; ST10, ST38, ST131, and ST405, all very common in the material from Canada (8), and major STs responsible for CTX-M dissemination worldwide (1). Two novel STs were found; several isolates were designated to 1 of them. We conclude that the relatively limited variation in clonal variants (STs) and ESBL genotypes is a consequence of recent introduction from connecting areas, such as Canada, possibly directly by bird migration or human activities, of a few resistant clones, followed by a local clonal expansion. This conclusion is supported by our 2005 study showing no ESBLs and by studies showing where different clones might have been introduced continuously for long periods, such as our study in France (4), which display a much larger diversity.

The dissemination of ESBLs to Barrow is part of this global pattern, and it is safe to say that humans and wildlife share resistant *E. coli* flora. When areas such as remote parts of Alaska are affected, global coverage is imminent.

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| Table. Characterization of Escherichia coli and Klebsiella pneumoniae ESBL-producing isolates, Barrow, Alaska, USA* |
|---------------------------------------------------------------|
| **Isolates, no.†**            | **bla genotype** | **MLST profile** |
|                               | **CTX-M** | **SHV** | **TEM** |                                |
| **E. coli**                   |           |       |       |                                   |
| 12                             | 14       | –     | 1      | ST38 (ST2253);‡                  |
| 11                             | 14       | –     | –      | ST131 (ST10)$§                   |
| 5                              | –        | –     | 19     | ST2967¶†                        |
| 3                              | 27       | –     | –      | ST405†                           |
| 1                              | 15       | –     | –      | ST131§                           |
| 1                              | –        | 1     | –      | ST2967¶†                        |
| **K. pneumoniae**             |           |       |       |                                   |
| 4                              | 15       | 12    | 1      | ND                               |
| 5                              | –        | 12    | 1      | ND                               |
| 2                              | –        | 12    | 1      | ND                               |
| 2                              | –        | 102   | 19     | ND                               |
| 8                              | –        | 102   | –      | ND                               |
| 1                              | –        | –     | 19     | ND                               |
| 4                              | 15       | 1     | 1      | ND                               |
| 1                              | –        | 2     | –      | ND                               |

*ESBL, extended-spectrum β-lactamase; MLST, multilocus sequence type; ST, sequence type; ND, no data.
†*E. coli* comprised 33 isolates from 32 samples. *K. pneumoniae* comprised 35 isolates from 35 samples. 12 samples were both *E. coli* and *K. pneumoniae* ESBL-harboring isolates but did not display horizontal transfer resulting from deviating resistance genotypes.
‡One of the isolates harbored a novel MLST allele, giving the novel ST2253 (deposited in the *E. coli* MLST database at the ERL, University College, Cork, Ireland. (http://mlst.ucc.ie/mlst/dbs/Ecoli/)).
§One of the isolates had ST10; the remaining 10 had ST131.
¶Isolates harbored a novel MLST allele, rendering the novel ST2967 (deposited in the MLST database). The single isolate with only *blaTEM*† may contain undetected ESBL genes because of the non-ESBL phenotype of TEM-1.
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References

1. Naseer U, Sundsfjord A. The CTX-M conundrum: dissemination of plasmids and Escherichia coli clones. Microb Drug Resist. 2011;17:83–97. http://dx.doi.org/10.1089/mdr.2010.0132
2. Sjölund M, Bonnedahl J, Hernandez J, Bengtsson S, Cederbrant G, Finlass J, et al. Dissemination of multidrug-resistant bacteria into the Arctic. Emerg Infect Dis. 2008;14:70–2. http://dx.doi.org/10.3201/eid1401.070704
3. Drobni M, Bonnedahl J, Hernandez J, Haemig P, Olsen B, Vancomycin-resistant enterococci, Point Barrow, Alaska, USA. Emerg Infect Dis. 2009;15:838–9. http://dx.doi.org/10.3201/eid1505.081219
4. Bonnedahl J, Drobni M, Gauthier-Clerc M, Hernandez J, Granholm S, Kaysier Y, et al. Dissemination of Escherichia coli with CTX-M type ESBL between humans and yellow-legged gulls in the south of France. PLoS ONE. 2009;4:e5958. http://dx.doi.org/10.1371/journal.pone.0005958
5. Gordon DM. Geographical structure and host specificity in bacteria and the implications for tracing the source of coliform contamination. Microbiology. 2001;147:1079–85.
6. Bonnedahl J, Drobni P, Johansson A, Hernandez J, Melhus Å, Stedt J, et al. Characterization, and comparison, of human clinical and black-headed gull (Larus ridibundus) extended-spectrum β-lactamase-producing bacterial isolates from Kalmar, on the southeast coast of Sweden. J Antimicrob Chemother. 2010;65:1939–44. http://dx.doi.org/10.1093/jac/dkq222
7. Simões RR1, Poirel L, Da Costa PM, Nordmann P. Seagulls and beaches as reservoirs for multidrug-resistant Escherichia coli. Emerg Infect Dis. 2010;16:110–2. http://dx.doi.org/10.3201/eid1601.090896
8. Piraino G, van der Bij AK, Gregson DB, Pitout JD. Molecular epidemiology over an 11-year period (2000 to 2010) of extended-spectrum β-lactamase-producing Escherichia coli causing bacteremia in a centralized Canadian region. J Clin Microbiol. 2012;50:294–9. http://dx.doi.org/10.1128/JCM.06025-11
9. Peirano G, Sang JH, Pitondo-Silva A, Laupland KB, Pitout JD. Molecular epidemiology of extended-spectrum-β-lactamase-producing Klebsiella pneumoniae over a 10 year period in Calgary, Canada. J Antimicrob Chemother. 2012;67:1114–20. http://dx.doi.org/10.1093/jac/dks026

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Staphylococcus aureus Carrying mecC Gene in Animals and Urban Wastewater, Spain

To the Editor: A new methicillin resistance mechanism gene, a divergent mecA homologue named mecC (formerly mecA1GA251), was recently described in Staphylococcus aureus (1). Methicillin-resistant S. aureus (MRSA) isolates carrying mecC have been recovered from humans, ruminants, pets, and other animals such as rats, seals, and guinea pigs (1–3). It has been suggested that mecC-carrying MRSA isolates might not be detected by using MRSA selective media (4). For mecC-carrying S. aureus isolates, cefoxitin MICs of 4–64 mg/L have been demonstrated (1–2,4), values that would normally include susceptible isolates, according to the epidemiologic cutoff value established by the European Committee on Antimicrobial Susceptibility Testing (EUCAST; www.eucaost.org). mecC-carrying S. aureus isolates have been classified as heteroresistant (5), and MICs can be affected by the drug-susceptibility testing method used (1,5).

These observations led us to retrospectively investigate the presence of mecC gene in a set of 361 mecA-negative S. aureus isolates collected during 2009–2012 (Table), independently of their susceptibility to cefoxitin. Isolates were recovered from healthy carriers in livestock (n = 39), from wild animals (n = 254), and from wastewater (effluents) from an urban sewage plant (n = 68). Specific amplification of the mecC gene was performed as described (6). The mecC-carrying S. aureus isolates were tested by broth microdilution using Microtiter EUST plates (Prog Diagnostic Systems, East Grinstead, UK) for susceptibility to benzylpenicillin, cefoxitin, chloramphenicol, ciprofloxacin, clindamycin, erythromycin, florfenicol, fusidic acid, gentamicin, kanamycin, linezolid, mupirocin, rifampin, sulfamethoxazole, streptomycin, quinupristin-dalfopristin, tetracycline, thiamulin, trimethoprim, and vancomycin. Additionally, susceptibility to oxacillin was determined by using microScan Gram Positive Combo panel 37 (Siemens, Erlangen, Germany). MICs were interpreted according to EUCAST epidemiologic cutoff values.

mecC was detected in a total of 4 isolates from wild boar (n = 1), fallow deer (n = 2), and urban wastewater (n = 1); these isolates represent 1% of the 361 tested isolates. The 3 isolates recovered from animals were susceptible to all antimicrobial drugs tested other than β-lactams and to oxacillin (MIC 0.5–1 mg/L) but were resistant to penicillin (MICs 0.5–2 mg/L). Two of the isolates were resistant to cefoxitin (MICs 8 and 16 mg/L) and the third was susceptible (MIC 4 mg/L). The wastewater isolate was resistant to penicillin (MIC 2 mg/L) and erythromycin (MIC 16 mg/L) and susceptible to all other antimicrobial drugs tested, including cefoxitin (MIC 4 mg/L) and oxacillin (MIC ≤0.25 mg/L).

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Extended-Spectrum $\beta$-Lactamases in *Escherichia coli* and *Klebsiella pneumoniae* in Gulls, Alaska, USA

Technical Appendix

Technical Appendix Table. Resistance profile of randomly selected *Escherichia coli* isolates*

| Antibacterial drug                        | No. samples† | % of total | No. antibacterial drugs‡ | No. samples† | % Total |
|------------------------------------------|--------------|------------|--------------------------|--------------|---------|
| Ampicillin                               | 62           | 45         | 0                        | 71           | 52      |
| Tetracycline                             | 10           | 7.3        | 1                        | 44           | 32      |
| Cefadroxil                               | 8            | 5.8        | 2                        | 18           | 13      |
| Trimethoprim/sulfamethoxazole            | 6            | 4.4        | 3                        | 1            | 0.7     |
| Streptomycin                             | 5            | 3.6        | 4                        | 1            | 0.7     |
| Nalidixic acid                           | 4            | 2.9        | 5                        | 1            | 0.7     |
| Chloramphenicol                          | 1            | 0.7        | 6                        | 1            | 0.7     |
| Tigecycline                              | 0            | 0          | >1                       | 66           | 48      |
| Nitrofurantoin                           | 0            | 0          |                          |              |         |
| Mecillinam                               | 0            | 0          |                          |              |         |

*Resistance was determined by antibacterial disk diffusion in accordance with recommendations from The European Committee on Antimicrobial Susceptibility Testing (EUCAST) (www.eucast.org). For antibacterial drugs lacking defined breakpoints for *E. coli* (tetracycline and streptomycin), the normalized resistance interpretation method (1) used by EUCAST, was implemented to define a local breakpoint.

†Total number of randomly selected *E. coli* was 137, isolated from 150 viable samples.

‡Denotes number of simultaneous antibacterial resistance phenotypes in each isolate.

Reference

1. Kronvall G, Kahlmeter G, Myhre E, Galas MF. A new method for normalized interpretation of antimicrobial resistance from disk test results for comparative purposes. Clin Microbiol Infect. 2003;9:120–32. PubMed [http://dx.doi.org/10.1046/j.1469-0691.2003.00546.x](http://dx.doi.org/10.1046/j.1469-0691.2003.00546.x)