

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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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-harboring 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 3% 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 *esp* 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 (*bla*_{CTX-M}^p, *bla*_{SHV}^p and *bla*_{TEM}) 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. Drobní 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. Drobní 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 *bla*_{CTX-M-14} or *bla*_{TEM-19}, whereas *K. pneumoniae* isolates mainly carried *bla*_{CTX-M-15}, *bla*_{SHV-12}, or *bla*_{SHV-102}. To our knowledge, ESBLs in *E. coli* and *K. pneumoniae* have not been reported from Alaska, but in two 10-year perspective reports from Canada (8,9), similar patterns and genotypes are reported 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.†	<i>bla</i> genotype			MLST profile
	CTX-M	SHV	TEM	
<i>E. coli</i>				
12	14	—	1	ST38 (ST2253)‡
11	14	—	—	ST131 (ST10)§
5	—	—	19	ST2967¶¶
3	27	—	—	ST405
1	15	—	—	ST131
1	—	—	1	ST2967¶¶
<i>K. pneumoniae</i>				
4	15	12	1	ND
5	—	12	1	ND
2	—	12	—	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, and 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 ERI, University College, Cork, Ireland. (<http://mlst.ucc.ie/mlst/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 *bla*_{TEM-1} may contain undetected ESBL genes because of the non-ESBL phenotype of TEM-1.

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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 *mecA*_{LGA251}), 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 Antibiotic Susceptibility Testing (EUCAST; www.eucast.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 (Trek 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 (MICs 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 $\leq$ 0.25 mg/L).

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Technical Appendix

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

Antibacterial drug	Resistance frequency		Co-resistance frequency		
	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.

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