The IS\textit{Ecp1} insertion sequence has been described as a flanking region of several \( \text{bla}_{\text{CTX-M}} \) genes and has been implicated in the expression and mobilization of the genes (5). A recent study by Lartigue et al. showed that a CTX-M-2 progenitor in \textit{K. ascorbata} could be mobilized and transferred to a conjugative \textit{E. coli} plasmid by the \textit{ISEcp1B} element; enhanced mobilization was observed in the presence of ceftazidime, cefotaxime, and pipera
cillin (10).

This Salmonella isolate’s resistance to cepfime and ceftquinome, fourth-generation cephalosporins, is troubling. Cefquinome is not approved for use in the United States but has been used in Europe for treating food animals since 1994. ESBLs, including CTX-M enzymes, are more common in Europe than in the United States (1). Further studies are warranted to clarify the extent to which the use of ceftquinome has contributed to high CTX-M prevalence in Europe.

In conclusion, we report a domestically acquired CTX-M-producing Salmonella isolate in the United States. Because third-generation cephalosporins are important for treating invasive Salmonella infections, continued monitoring of ESBL-producing bacteria is important.

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Yersinia pseudotuberculosis

\( 0:1 \) Traced to Raw Carrots, Finland

To the Editor: Illness caused by \textit{Yersinia pseudotuberculosis} is mainly characterized by fever and acute abdominal pain due to mesenteric lymphadenitis that mimics appendicitis. Secondary manifestations include erythema nodosum and reactive arthritis (1). Outbreaks have been reported in the Northern Hemisphere, including Canada (2,3), Japan (4), and Russia (5). Several community outbreaks have also been reported in Finland since 1982 (1,6–9). Only in a few of the outbreaks has the vector or source of the infection been identified. Recently, fresh produce, such as iceberg lettuce (7) and carrots (9), has been implicated by epidemiologic investigations as a source of infection, but mechanisms of contamination of fresh produce have remained unknown.

On April 8, 2004, the National Public Health Institute of Finland was informed of several cases of gastroen-
teritis in schoolchildren in 1 municipality in northern Finland. On April 13, 2004, stool samples from symptomatic schoolchildren confirmed Y. pseudotuberculosis infections. At the same time, an increase occurred in Y. pseudotuberculosis cases reported to the National Infectious Disease Register (NIDR) from other parts of the country. We conducted epidemiologic, microbiologic, trace-back, and environmental investigations to determine the source of the outbreak and the origin of contamination.

In the school outbreak, a survey concerning symptoms of gastrointestinal illness was conducted among all schoolchildren (7–18 years of age) and personnel (N = 900) of the 7 schools in the municipality. A case was defined as a laboratory-confirmed Y. pseudotuberculosis infection in a child or staff member who attended a school that received lunches from the school central kitchen, or as abdominal pain and fever, or erythema nodosum with illness onset from March 8 through March 28, 2004. Of respondents to the survey, 53 met the case definition. Among these respondents, Y. pseudotuberculosis was isolated from stool samples of 5 persons.

A case–control study was conducted to identify the source of infection; self-administered questionnaires asked about consumption of items on school menus from March 8 through March 26, 2004. For each of the 53 case-patients identified in the survey, 4 controls were selected from the same class; 39 cases and 107 controls were included in the analysis. Univariate analysis showed that a vegetable mixture of carrots and white cabbage served on March 8 and a mixture of cucumber and white cabbage served on March 25 were associated with illness. Multivariate analysis showed that only the carrot–white cabbage mixture was associated with illness.

We also conducted a case–control study by mailing questionnaires to 37 persons with microbiologically confirmed Y. pseudotuberculosis infections reported to the NIDR from March 15 through May 7, 2004. These cases were from other parts of the country and were not associated with the school outbreak. For each case, 4 controls matched by age, sex, and municipality were randomly selected from the national population registry. Risk of illness increased with increased frequency of eating fresh carrots.

Carrots served in the school were traced back to the farm level. Samples checked included grated carrots, which were available from the school kitchen. The kitchen had received all vegetables from 1 fresh-food processing plant. Samples were taken from the carrot-peeling line, carrot-peeling leftovers, grated carrots, and other vegetable-processing lines at the plant. Carrots originated from only 2 farms, which were inspected, and samples were obtained for bacteriologic examination. Small mammals at the farms were caught in carrot fields and investigated microbiologically to identify the reservoir of Y. pseudotuberculosis. This bacterium was isolated from 1 environmental sample from the carrot-peeling line in the fresh-food processing plant, from spoiled carrots, from fluid draining from spoiled carrots, and from a pooled sample of common shrew (Sorex araneus) intestines from 1 farm.

Human and environmental isolates obtained were serotype O:1, subtype O:1b. Pulsed-field gel electrophoresis (PFGE) profiles of isolates from schoolchildren, fluid of spoiled carrots at the infected farm, and shrew intestines were indistinguishable. All 22 isolates from NIDR cases belonged to 2 PFGE genotypes. One genotype had a PFGE profile that was indistinguishable from the profile of the school outbreak isolates and the other genotype differed from these isolates by only 1 fragment.

Our study provides microbiologic and epidemiologic evidence that the school outbreak was caused by carrots contaminated at the production farm. We isolated a Y. pseudotuberculosis subtype from human patients that was indistinguishable from isolates from the implicated source and a potential animal reservoir. Although the association between shrews and carrots is uncertain, shrews may have been picked up with carrots by harvesting machinery and ended up dead in wooden storage frames with the carrots. If carrots become contaminated, long storage at cold temperatures favors growth of Y. pseudotuberculosis and may result in human infections. Further studies are needed to determine the mechanism of contamination and other natural reservoirs. After the outbreak, the Finnish Food Safety Authority recommended controlling contamination at the farm level by removing spoiled carrots and paying attention to any subsequent spoilage during handling procedures.

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Antibodies against \textit{Rickettsia} spp. in Hunters, Germany

To the Editor: A number of emerging \textit{Rickettsia} species have been recently described \cite{1}. One of these, \textit{R. helvetica}, was first isolated in Switzerland in 1979 and was implicated in perimyocarditis and nonspecific febrile disease in humans \cite{2,3}. PCR showed its prevalence in 1,187 \textit{Ixodes ricinus} ticks in southern Germany to be 8.9\% \cite{4}. This finding raises the question whether autochthonous transmission of rickettsiae to humans may occur in Germany. To help answer this question, we conducted a cross-sectional study of the presence of antibodies against \textit{Rickettsia} spp. in a population in Germany presumably exposed to ticks.

On February 4–5, 2006, we used convenience sampling to enroll 286 hunters at a national hunting fair in Dortmund, Germany. All study participants gave written, informed consent. The Ethics Committee of the Charité approved the study.

Every participant completed a standardized questionnaire. Serum samples were collected from all hunters and analyzed by immunofluorescence assay for 9 \textit{Rickettsia} species (\textit{R. conorii}, \textit{R. slovaca}, \textit{R. helvetica}, \textit{R. massiliae}, \textit{R. mongoliitmonae}, \textit{R. raesiens}, \textit{R. aeschlimannii}, \textit{R. felis}, and \textit{R. typhi}) as described previously \cite{5}. Odds ratios (ORs) and 95\% confidence intervals (CIs) were calculated by using SPSS software version 14 (SPSS, Inc., Chicago, IL, USA). We considered p<0.05 to be significant.

Of the 286 hunters, 252 (88.1\%) were male; median age was 46 years (range 17–79 years). Positive antibody titers (immunoglobulin [Ig] G, IgM, or both) against any \textit{Rickettsia} spp. were found for 26 (9.1\%) hunters (95\% CI 6.2–13.0). Antibodies against different \textit{Rickettsia} spp. were found for 18 hunters; species-specific antibodies against \textit{R. helvetica} were found for 2 hunters and against \textit{R. aeschlimannii} for 6 (Table). Seropositive and seronegative hunters did not differ significantly with respect to sex, age, and total years of hunting. Neither hunting nor traveling in a foreign country within the past 5 years was significantly associated with seropositivity. Neither of the 2 hunters against \textit{R. helvetica}–specific antibody titers had traveled outside Germany in the 5 years before the study, but 3 of the 6 hunters with specific titers against \textit{R. aeschlimannii} had traveled and hunted in countries with unknown endemicity for \textit{R. aeschlimannii} (Russia, Romania, Namibia). A total of 212 (74.1\%) hunters had received at least 1 tick bite in the year before the study; median was 4 tick bites/year. Living in the southern parts of Germany (below 50°N) was significantly related to seropositivity (OR 4.1, 95\% CI 1.3–12.3, p = 0.02). Although the 26 persons with positive serologic results for \textit{Rickettsia} spp. reported arthralgia with higher frequency than did seronegative persons (50\% vs. 37\%, respectively), their reports of arthralgia and of other clinical signs did not differ significantly: temperature >38.5°C (8\% vs. 2\%), enlarged lymph nodes (12\% vs. 9\%). No seropositive hunter reported having had an eschar.

This study provides data for Germany on the seroprevalence of \textit{Rickettsia} spp. in persons highly exposed to ticks. Our results suggest that \textit{Rickettsia} spp. are endemic to southern Germany and may cause autochthonous infections. Although most seropositive hunters exhibited reactivity to

\begin{thebibliography}{9}


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