Hospital-Acquired Listeriosis Outbreak Caused by Contaminated Diced Celery—Texas, 2010

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Background. Listeria monocytogenes causes often-fatal infections affecting mainly immunocompromised persons. Sources of hospital-acquired listeriosis outbreaks can be difficult to identify. We investigated a listeriosis outbreak spanning 7 months and involving 5 hospitals.

Methods. Outbreak-related cases were identified by pulsed-field gel electrophoresis (PFGE) and confirmed by multiple-locus variable-number tandem-repeat analysis (MLVA). We conducted patient interviews, medical records reviews, and hospital food source evaluations. Food and environmental specimens were collected at a hospital (hospital A) where 6 patients had been admitted before listeriosis onset; these specimens were tested by culture, polymerase chain reaction (PCR), and PFGE. We collected and tested food and environmental samples at the implicated processing facility.

Results. Ten outbreak-related patients were immunocompromised by ≥1 underlying conditions or treatments; 5 died. All patients had been admitted to or visited an acute-care hospital during their possible incubation periods. The outbreak strain of L. monocytogenes was isolated from chicken salad and its diced celery ingredient at hospital A, and in 19 of >200 swabs of multiple surfaces and in 8 of 11 diced celery products at the processing plant. PCR testing detected Listeria in only 3 of 10 environmental and food samples from which it was isolated by culturing. The facility was closed, products were recalled, and the outbreak ended.

Conclusions. Contaminated diced celery caused a baffling, lengthy outbreak of hospital-acquired listeriosis. PCR testing often failed to detect the pathogen, suggesting its reliability should be further evaluated. Listeriosis risk should be considered in fresh produce selections for immunocompromised patients.

Keywords. listeriosis; disease outbreak; hospital infections; food contamination; immunocompromised patient.

Listeria monocytogenes, the gram-positive bacterial agent of listeriosis, is ubiquitous [1] and usually transmitted to humans in food. Because of its reservoirs and ability to grow under refrigeration, it is not infrequently found during routine testing of deli meats and poultry, hot dogs, soft cheeses, and unpasteurized milk. Food vehicles identified in outbreaks include milk, cheese, butter, hot dogs, ready-to-eat meat and poultry, and several vegetable or grain salads [2]. Invasive listeriosis occurs among pregnant women, newborns, older adults, and those with immunocompromising conditions or receiving immunosuppressive therapy. The incubation period of invasive listeriosis is 3–70 days; sepsis, meningitis, meningoencephalitis, and fetal loss are common signs. Although the infectious dose of L. monocytogenes is unknown, it is believed to be lower among severely immunocompromised persons than those with healthier immune systems [2]. Case-fatality rates average 20%–30% [1]. Healthy persons exposed to L. monocytogenes have lower risk of developing invasive listeriosis, but might develop self-limited gastrointestinal listeriosis accompanied by fever. Neither clinical nor public health laboratories test stool specimens for L. monocytogenes. In the United States, approximately 800 cases of listeriosis are reported each year, with an average of 44 cases in Texas annually [3–7].
Outbreaks provide opportunities for determining vehicles of infection. However, because of the long incubation period for listeriosis and difficulty of obtaining reliable food histories, as well as its relatively high morbidity and mortality, listeriosis outbreaks can be even more difficult to investigate than those involving other pathogens. A large proportion of hospitalized patients are at high risk for listeriosis, which might account for the number of reported hospital-acquired outbreaks [8–17]. These outbreaks ranged in size from 2–23 cases each, had an average case fatality rate of ≥50%, and often lasted months or years [8–17].

In February 2010, the Texas Department of State Health Services (DSHS) laboratory identified a cluster of 4 listeriosis cases with specimens having the same or very similar pulsed-field gel electrophoresis (PFGE) patterns collected from mid-January through mid-February. Six additional cluster-associated cases were identified during the following 6 months. We collaborated with local health departments, the Centers for Disease Control and Prevention (CDC), and the Food and Drug Administration (FDA) to identify the source of infections and to prevent additional cases.

METHODS

Identification of Outbreak and Initial Case Investigations

Clinical isolates of *L. monocytogenes* from Texas patients were sent to the DSHS laboratory for PFGE subtyping. Outbreak cases of listeriosis were identified by similarity of PFGE patterns. PFGE was performed using *ApaI* and *ApaI* enzymes, according to standardized procedures for PulseNet, the national foodborne pathogen molecular subtyping network [18, 19]. The DSHS laboratory submitted PFGE data electronically to PulseNet’s central database, where the patterns were compared with patterns for clinical isolates submitted from other states, and food and environmental isolates from specimens collected by state and federal regulatory partners. Outbreak-associated isolates were sent to the CDC for serotyping and multiple-locus variable-number tandem-repeat analysis (MLVA). MLVA was performed using a previously described protocol, with 1 additional locus in the typing scheme [20, 21]. We defined an outbreak-related case as a laboratory-confirmed infection of *L. monocytogenes* with an isolate having PulseNet *ApaI* pattern GX6A16.0074, GX6A16.0096, GX6A16.1270, or GX6A16.1299, and *ApaI* pattern GX6A12.0174 or GX6A12.1573 from a specimen collected during 1 January–1 September 2010. Any combination of 1 of the 4 *ApaI* patterns and 1 of the 2 *ApaI* patterns was defined as the outbreak strain of *L. monocytogenes*. Relatedness of cases to the outbreak was confirmed using MLVA.

All outbreak-related patients alive at the time of case confirmation were interviewed to determine possible exposures to *L. monocytogenes*. Surrogate interviews with family members were conducted for patients unable to respond themselves. Interviews focused on food histories, using a standardized questionnaire including specific listeriosis-associated foods and open-ended questions, and medical histories. Medical records were reviewed for food and medical histories.

Investigations at Acute Care Hospitals

Sources for all food items served at the acute care hospitals where outbreak case patients had been admitted or visited were obtained from hospital staff. Invoices from the single common distributor to all hospitals were obtained for 2 months preceding the specimen collection date for each case. Our evaluations focused on raw and cooked poultry, because consumption of these food items was reported by many cases and because *L. monocytogenes* has been detected in similar items. The kitchen of one hospital (hospital A), where 6 outbreak-related patients had been admitted prior to developing listeriosis, was inspected by local and state health department sanitarians and epidemiologists in mid-May 2010. Environmental and food samples collected at the hospital (Supplementary Data) were tested at the DSHS laboratory by simultaneous polymerase chain reaction (PCR)–based BAX (DuPont) and culture methodologies suitable for detecting *Listeria* species, and *L. monocytogenes* in particular [22, 23]. Isolates were PFGE subtyped and sent to CDC for serotyping and MLVA.

The last case patient was identified in August 2010. A food history was obtained from her using the structured questionnaire. Because this case patient had been admitted to hospital A prior to listeriosis onset, the kitchen was visited 2 more times and additional samples were collected there (Supplementary Data).

Investigation of Produce Processing Facility

The produce processing facility implicated by the epidemiologic and environmental investigations was visited on 11 October 2010 by DSHS sanitarians. Sanitarians collected 10 unopened, vacuum-sealed final product samples for testing at the DSHS laboratory, and inspected the facility to determine possible sources and mechanisms of ongoing contamination. The FDA also investigated the facility 2 days later and collected >200 environmental and 19 unopened finished product samples and tested them by PCR-based and culture methodologies for *L. monocytogenes*. All *L. monocytogenes* isolates were subtyped by PFGE and MLVA, and serotyped. We also obtained the produce processing facility’s records for private laboratory Association of Analytical Communities–Enzyme Linked Fluorescent Assay (AOAC–ELFA) antibody-based rapid testing of environmental and food samples.

Linkage of Outbreak-Related Patients to Produce Processing Facility

Product tracebacks were conducted from each hospital with outbreak-related patients. Trace-forwards were conducted by
using invoices provided by the produce facility. We linked the implicated product to cases through ≥1 distributors from the produce facility to the hospitals where these patients had visited or been admitted.

RESULTS

Outbreak and Patient Characteristics

Ten patients (5 female) meeting the outbreak case definition were identified. The Ascl and Apal PFGE patterns of the case patients’ isolates were the same or very similar, and all had the same MLVA patterns and serotypes. Median case-patient age was 80 years (range, 56–93 years). Nine patients resided in central Texas, and 1 resided in south Texas. No case-patient isolates with the same or similar PFGE patterns were reported from other states. Nine patients had been hospitalized for 5–23 days, and 1 visited 3 hospitals during the typical incubation period for listeriosis; all either remained in a hospital (n = 5) or had a new hospital admission (n = 5) for listeriosis (Table 1). Five patients died within 3 months of the collection date for the specimen that yielded L. monocytogenes. For 3 deceased patients, listeriosis was identified on death certificates as an immediate or contributing cause. For 2 deceased patients, listeriosis might have contributed to death. We were unable to determine precise listeriosis onset dates for the majority of case patients because they had underlying conditions or received treatments that caused gastrointestinal symptoms, which are not used to define invasive listeriosis. In addition, fever was not reported for 4 patients when listeriosis was diagnosed, and 1 patient had fever associated with treatments for underlying conditions. We therefore used collection dates for L. monocytogenes clinical specimens as surrogates for onset dates. Specimens (9 blood and 1 pleural fluid) for the 10 patients were collected during 11 January–14 August 2010. All 10 patients had ≥1 immunocompromising conditions or were receiving corticosteroid or acid-reducing treatments that could have increased their susceptibility to invasive listeriosis [12, 24].

Table 1. Demographic, Medical, and Epidemiologic Characteristics Among Patients in an Outbreak of Hospital-Acquired Listeriosis (Texas, 2010)

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (y)/Sex</th>
<th>Underlying Conditions</th>
<th>Immunotherapy/Acid-Reducer Use</th>
<th>Hospital</th>
<th>Signs and Symptoms on Admission</th>
<th>Specimen Source</th>
<th>Outcome (Death Certificate Diagnosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>66/M</td>
<td>AID, CA, CVD, DII, PD</td>
<td>Chronic steroid, acid-reducer use</td>
<td>A</td>
<td>Unresponsiveness, seizures, fever</td>
<td>Blood</td>
<td>Died (glioblastoma, listeriosis contributing)</td>
</tr>
<tr>
<td>2</td>
<td>86/M</td>
<td>CA, CVD</td>
<td>Chronic steroid, acid-reducer use</td>
<td>B</td>
<td>Dyspnea, pleural effusion</td>
<td>Pleural fluid</td>
<td>Died (pneumonia, renal failure)</td>
</tr>
<tr>
<td>3</td>
<td>79/M</td>
<td>CA, CVD, DII, HepC, RD</td>
<td>None</td>
<td>C</td>
<td>Dyspnea</td>
<td>Blood</td>
<td>Recovered</td>
</tr>
<tr>
<td>4</td>
<td>93/M</td>
<td>CA, CVD, PD</td>
<td>Chronic acid-reducer use</td>
<td>A</td>
<td>Fever, hypotension, altered mental status</td>
<td>Blood</td>
<td>Recovered</td>
</tr>
<tr>
<td>5</td>
<td>74/F</td>
<td>PD</td>
<td>Chronic steroid use</td>
<td>D</td>
<td>Fever, diarrhea</td>
<td>Blood</td>
<td>Recovered</td>
</tr>
<tr>
<td>6</td>
<td>81/F</td>
<td>CVD, Hip, PD</td>
<td>Chronic steroid use</td>
<td>A</td>
<td>Unresponsiveness, hypotension</td>
<td>Blood</td>
<td>Died (hypoxemia, listeriosis contributing)</td>
</tr>
<tr>
<td>7</td>
<td>88/F</td>
<td>CVD</td>
<td>Chronic steroid use</td>
<td>A</td>
<td>Episodes of syncope</td>
<td>Blood</td>
<td>Died (chronic anemia)</td>
</tr>
<tr>
<td>8</td>
<td>56/F</td>
<td>AID, CVD, HepC, PD, RD</td>
<td>Chronic steroid, acid-reducer use</td>
<td>E</td>
<td>Upper chest pain</td>
<td>Blood</td>
<td>Recovered</td>
</tr>
<tr>
<td>9</td>
<td>81/M</td>
<td>AID, BD, CA, CVD</td>
<td>None</td>
<td>A</td>
<td>Gastrintestinal bleeding</td>
<td>Blood</td>
<td>Died (listeriosis)</td>
</tr>
<tr>
<td>10</td>
<td>61/F</td>
<td>CA, CVD</td>
<td>Chronic steroid, acid-reducer use</td>
<td>A</td>
<td>Fever, diarrhea</td>
<td>Blood</td>
<td>Recovered</td>
</tr>
</tbody>
</table>

Abbreviations: AID, autoimmune disease; BD, blood dyscrasia; CA, cancer; CVD, cardiovascular disease; DII, type II diabetes; DOC, date of collection for Listeria monocytogenes; HepC, chronic hepatitis C infection; Hip, recent hip fracture; PD, pulmonary disease; RD, renal disease.
Determination of Hospital-Acquired Nature of Outbreak

Interviews of patients and surrogates and reviews of patient medical records did not identify any common medication, procedure, or other possible exposure. In the majority of patient medical records, only a static menu based on type of diet (e.g., unrestricted, diabetic, renal) was available for review. None of the patients were placed on a diet that restricted foods commonly associated with listeriosis. Several hospitals purged menus upon patient discharge.

By early May 2010, a total of 7 cases in 4 hospitals were identified. Examination of patient histories revealed that all had visited or been admitted to an acute care hospital within 70 days (the maximum incubation period) before listeriosis specimen collection (Figure 1). Our working hypothesis was that all patients had acquired listeriosis from a food item distributed to multiple hospitals.

We determined that 5 hospitals (hospitals A–E) with outbreak-related patients used the same distributor for the majority of food items, including meat, poultry, prepared, and frozen foods. The additional 2 hospitals visited by the case patient who had not been inpatient during the likely incubation period for listeriosis had no distributors in common with all hospitals A–E. Certain hospitals purchased dairy, produce, and baked goods from this distributor, and others purchased these items elsewhere. We identified poultry items, both raw and cooked, from the same manufacturer or branded with the common distributor’s name. Although no patients were restricted from eating these items, no mechanism existed for determining which patients had actually consumed them. The last case patient reported frequent consumption of chicken salad. Precooked, diced chicken used in the preparation of chicken salad was distributed to multiple hospitals in the cluster.

Investigation of Multicase Hospital

We focused our investigation on hospital A, where by early May, 4 patients had been hospitalized before listeriosis onset. Analyses of environmental and food samples collected during the first visit to hospital A, in mid-May 2010, identified no obvious source of contamination; *L. monocytogenes* was not detected in any environmental or food specimens. During the second visit to hospital A, after 2 additional outbreak-related cases including case patient 10 were reported from there, the outbreak strain of *L. monocytogenes* was detected in chicken salad prepared in the hospital kitchen, but not in a previously unopened packaged of diced chicken of the type used to prepare the salad nor in any other food items or environmental specimens.

Based on the positive findings of *L. monocytogenes* in the chicken salad, we visited hospital A a third time (Supplementary Data) and sampled ingredients used to prepare a fresh batch of chicken salad and the finished salad. *Listeria monocytogenes* of the outbreak strain was detected only in the freshly

![Figure 1](https://academic.oup.com/cid/article-abstract/56/1/20/417020)
made chicken salad and the diced celery used in its preparation.

Investigation and Closure of Produce Processing Facility

DSHS inspectors noted structural defects, including cracks in floors and walls, inadequate produce handling and cleaning techniques, and absent operating plans in the implicated produce processing facility. *Listeria monocytogenes* of the outbreak strain was detected in 7 of 10 bags of diced celery finished product they collected. Culture plates had only a few colonies of each of *L. monocytogenes*. Nineteen of the environmental specimens and 1 diced celery finished product sample collected by FDA inspectors yielded the outbreak strain of *L. monocytogenes*. Positive environmental swabs were collected from floors, surfaces, and equipment in every room of the facility. Of note, all of the negative food samples were whole or hand-cut, not diced, products. Diced celery was the only machine-cut product present in the facility when sampling was done.

The company refused DSHS’ request to voluntarily close, and DSHS issued an Emergency Order of Closure and Recall on 20 October 2010, for all products with production dates from 1 January 2010 forward. Four additional DSHS environmental samplings before the end of 2010 in the closed facility indicated the continued presence of the outbreak strain of *L. monocytogenes*. The company closed permanently in February 2011, and no additional cases have been detected.

Summary of Laboratory Findings

PFGE results for clinical, environmental, and food samples are summarized in Table 2. The 4 *ApaI* PFGE patterns (GX6A16.0074, GX6A16.0096, GX6A16.1270, and GX6A16.1299) differed from each other by 2–6 bands, and the 2 *ApaI* PFGE patterns (GX6A16.1573 and GX6A16.0174) differed from each other by 4 bands. All clinical, environmental, and food isolates were identified as *L. monocytogenes* serotype 1/2a with indistinguishable MLVA patterns. Of note, only 3 of 10 DSHS food specimens that grew *L. monocytogenes* were presumptively positive using PCR-based methodology on initial testing. The 7 specimens that were initially negative by PCR but yielded isolates by culturing were positive when tested by PCR methodology.

After implication in the outbreak, the produce processing facility provided private laboratory results for AOAC-ELFA antibody-based rapid test procedures, without culturing, of green pepper and lettuce finished product samples collected in January and September 2010, respectively. Both samples were collected before the facility was implicated in the outbreak and were presumptively positive for *L. monocytogenes*. We are not aware of any corrective actions taken by the facility after it received these results. Three of 32 environmental swabs collected in late October 2010 after the facility had closed were also presumptively positive for *L. monocytogenes*.

Summary of Epidemiology Findings

Nine outbreak-related cases were linked to the produce processing facility because those patients had been admitted to or visited hospitals that had received the facility’s diced celery during the listeriosis incubation period. No link was documented for 1 case, though the hospital where this patient had been admitted received items other than diced celery from the produce processing facility.

### Table 2: Clinical, Environmental, and Food Specimen Pulsed-Field Gel Electrophoresis Results, Hospital-Acquired Listeriosis Outbreak (Texas, 2010)

<table>
<thead>
<tr>
<th>PFGE-AscI Pattern</th>
<th>PFGE-Apal Pattern</th>
<th>Isolate Date(s)</th>
<th>Submitter</th>
<th>Location</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>GX6A16.0096</td>
<td>GX6A12.0174</td>
<td>11 Jan–2 June 2010</td>
<td>Hospital</td>
<td>Hospitals A, D, E</td>
<td>7 clinical isolates, cases 1, 3, 4, 5, 7, 8, 9</td>
</tr>
<tr>
<td>GX6A16.0096</td>
<td>GX6A12.0174</td>
<td>11 Oct 2010</td>
<td>DSHS</td>
<td>Produce facility</td>
<td>6 diced celery finished product</td>
</tr>
<tr>
<td>GX6A16.0096</td>
<td>GX6A12.0174</td>
<td>14 Oct 2010</td>
<td>FDA</td>
<td>Produce facility</td>
<td>3 environmental swabs</td>
</tr>
<tr>
<td>GX6A16.1270</td>
<td>GX6A12.0174</td>
<td>21 Jan 2010</td>
<td>Hospital</td>
<td>Hospital B</td>
<td>1 clinical isolate, case 2</td>
</tr>
<tr>
<td>GX6A16.1299</td>
<td>GX6A16.1573</td>
<td>21 April 2010</td>
<td>Hospital</td>
<td>Hospital A</td>
<td>1 clinical isolate, case 6</td>
</tr>
<tr>
<td>GX6A16.1299</td>
<td>GX6A16.1573</td>
<td>5 Oct 2010</td>
<td>DSHS</td>
<td>Hospital A</td>
<td>1 chicken salad</td>
</tr>
<tr>
<td>GX6A16.1299</td>
<td>GX6A16.1573</td>
<td>13 Dec 2010</td>
<td>DSHS</td>
<td>Produce facility</td>
<td>3 environmental swabs</td>
</tr>
<tr>
<td>GX6A16.0074</td>
<td>GX6A12.0174</td>
<td>14 Aug 2010</td>
<td>Hospital</td>
<td>Hospital A</td>
<td>1 clinical isolate, case 10</td>
</tr>
<tr>
<td>GX6A16.0074</td>
<td>GX6A12.0174</td>
<td>21 Sept 2010</td>
<td>DSHS</td>
<td>Hospital A</td>
<td>1 chicken salad</td>
</tr>
<tr>
<td>GX6A16.0074</td>
<td>GX6A12.0174</td>
<td>11 Oct 2010</td>
<td>DSHS</td>
<td>Produce facility</td>
<td>1 diced celery</td>
</tr>
<tr>
<td>GX6A16.0074</td>
<td>GX6A12.0174</td>
<td>14 Oct 2010</td>
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<td>Produce facility</td>
<td>3 environmental swabs</td>
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<td>GX6A16.0074</td>
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<tr>
<td>GX6A16.0074</td>
<td>GX6A12.1573</td>
<td>14 Oct 2010</td>
<td>FDA</td>
<td>Produce facility</td>
<td>1 environmental swab</td>
</tr>
</tbody>
</table>

Abbreviations: DSHS, Texas Department of State Health Services; FDA, Food and Drug Administration.
produce processing facility. One patient reported having eaten chicken salad sandwiches at hospital A and another reported eating various sandwiches at hospital C; both hospitals received the implicated diced celery. All other patients might have eaten diced celery–containing sandwiches because these were on their menus, might have been ordered, or were not restricted from their menu.

**DISCUSSION**

We traced the source of an outbreak of 10 cases (5 deaths) of hospital-acquired listeriosis to chicken salad initially, and then to diced celery used in the salad. For some hospital-acquired listeriosis outbreaks, no specific food or other vehicle was implicated [9, 11, 14]. The most commonly reported vehicle was cold sandwiches [8, 10, 12, 15, 16]. Although investigations have implicated specific sandwich types, including chicken salad, none have identified the specific ingredient containing *L. monocytogenes* [8, 15, 16]. One of the difficulties of hospital-acquired foodborne illness outbreak investigations is that foods consumed by patients are frequently not reported in medical records. A survey of New York City hospitals found that 57% of hospitals do not maintain a record of foods ordered by patients [25]. Furthermore, there are no uniform guidelines regarding serving ready-to-eat produce to immunocompromised hospitalized patients. We are conducting a study of hospital food safety practices in Texas hospitals to provide the evidence needed to develop such guidelines.

We were unable to determine the original source of *L. monocytogenes* in the produce facility. The celery used by the facility was grown in another state, came from multiple companies and farms, and was distributed nationally. It is possible that the outbreak source was nationally distributed celery with no cases detected in other states, or a different produce item had been contaminated during processing. Most of the other products processed using this dicer, including potatoes and onions, would have been cooked prior to consumption.

The initial negative PCR-based tests and the low colony-count plates at the DSHS laboratory suggest low-level contamination in celery and chicken salad because the organism required culturing in order to form enough colonies to yield the positive PCR results from isolates. These findings suggest that rapid testing alone might not be reliable for ensuring product safety. Testing of food products often involves only rapid testing which, unlike culture, might not detect low levels of contamination. Therefore, reevaluation of the effectiveness of PCR rapid testing of food products is needed.

There were likely many more susceptible patients who ate the celery than the 10 reported listeriosis outbreak cases. The small number of cases over the period of 7 months suggests intermittent, low-level contamination. Hospital A was a transplant hospital and had a high proportion of critically ill persons whose immunocompromised status likely facilitated this outbreak. Given the wide distribution of the product, there were likely many more undetected cases in the community, and possibly hospitals, because illness resolved before care was sought or before a diagnosis of invasive listeriosis was made, or death occurred without listeriosis diagnosis. Healthy individuals who consumed the contaminated product would not likely have developed invasive listeriosis and would not have been tested for *Listeria* gastroenteritis. Fresh produce has been increasingly implicated as a cause of foodborne illness outbreaks from different pathogens, including *L. monocytogenes* [28, 29]. A recent listeriosis outbreak was linked to cantaloupe [30]. Outbreaks caused by other vehicles (eg, meat items) have not similarly increased in numbers [28]. Increased consumption of fresh fruits and vegetables and increased use of processing steps (eg, washing, dicing, and peeling) that can introduce or spread a contaminant likely contribute to the increase in produce-related outbreaks [28]. The consumption of produce items present in mixed-ingredient foods is often not recalled by patients; this likely contributed to the increase [31]. Foodborne illnesses are usually self-limited in healthy individuals. However, *L. monocytogenes*, *Salmonella* species, Shiga toxin–producing *Escherichia coli*, and other pathogens can cause serious outcomes, including death, among immunocompromised persons, especially those who are hospitalized. This outbreak highlights the particular vulnerability of hospitalized and immunocompromised patients to invasive *L. monocytogenes* infections. We concur with other investigators who have called for minimizing exposure to ready-to-eat produce and other high-risk foods in order to reduce morbidity and mortality among these vulnerable persons [10, 12–16, 32].

**Supplementary Data**

Supplementary materials are available at Clinical Infectious Diseases online (http://www.oxfordjournals.org/our_journals/cid/). Supplementary materials consist of data provided by the author that are published to benefit the reader. The posted materials are not copyedited. The contents of all
supplementary data are the sole responsibility of the authors. Questions or messages regarding errors should be addressed to the author.

Notes
Acknowledgments. The authors acknowledge the molecular biology contribution of Aaron P. Benfield, PhD, at the DSHS laboratory in conducting PFGE analyses of the isolates obtained in this outbreak.

Disclaimer. The findings and conclusions in this report are those of the author(s) and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

Potential conflicts of interest. All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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