MAJOR ARTICLE

Listeria monocytogenes Infection from Foods Prepared in a Commercial Establishment: A Case-Control Study of Potential Sources of Sporadic Illness in the United States

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(See the article by Voetsch et al. on pages 513–20 and the editorial commentary by Goulet on pages 529–30)

Background. *Listeria monocytogenes* has been estimated to cause >2500 illnesses and 500 deaths annually in the United States. Efforts to reduce foodborne listeriosis have focused on foods frequently implicated in outbreaks. Potential sources for *L. monocytogenes* infection not associated with outbreaks remain poorly understood.

Methods. The Foodborne Diseases Active Surveillance Network conducts surveillance for culture-confirmed listeriosis at clinical laboratories in 9 states. After excluding outbreak-associated cases, we attempted to enroll eligible case patients with *L. monocytogenes* infection in a case-control study from 2000 through 2003. Control subjects were recruited through health care providers and were matched to case patients by state, age, and immunosuppression status. Data were collected about exposures occurring in the 4 weeks before specimen collection from the case patients.

Results. Of the 249 case patients with *L. monocytogenes* infection, only 12 (5%) had cases that were associated with outbreaks; 6 other patients were ineligible for other reasons. Of 231 eligible case patients, 169 (73%) were enrolled in the study. We classified 28 case patients as having pregnancy-associated cases. We enrolled 376 control subjects. In multivariable analysis, *L. monocytogenes* infection was associated with eating melons at a commercial establishment (odds ratio, 2.6; 95% confidence interval, 1.4–5.0) and eating hummus prepared in a commercial establishment (odds ratio, 5.7; 95% confidence interval, 1.7–19.1).

Conclusions. Most cases of *L. monocytogenes* infection were not associated with outbreaks. Reducing the burden of foodborne listeriosis may require interventions directed at retail environments and at foods, such as melons and hummus, that are not commonly recognized as high risk. Because of the severity of listeriosis, pregnant women and other persons at risk may wish to avoid eating these newly implicated foods.

Listeria monocytogenes infection has been estimated to cause >2500 illnesses annually in the United States [1]. Illness is characterized by febrile gastroenteritis, sepsis, meningitis, or fetal loss [2]. The overall mortality rate is ~17%.[3]. Microbiologic and epidemiologic data demonstrate that food is the source of infection in most cases of *L. moncytogenes* infection [4–13]. Although less common than other foodborne pathogens, *L. monocytogenes* results in a disproportionate share of the foodborne disease burden, accounting for 4% of all hospitalizations and 28% of all deaths from foodborne disease in the United States [1].

Established risk factors for infection include immunosuppression, pregnancy, and extremes of age [11].

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Identifying sources of *L. monocytogenes* infection has been challenging. Evidence that *L. monocytogenes* could be foodborne was first reported in 1981 in an investigation of an outbreak in Nova Scotia, Canada, that implicated cabbage [4]. Subsequent outbreaks have been associated with milk, unpasteurized soft cheeses, turkey frankfurters, and ready-to-eat meats, including pâté and delicatessen turkey [5, 6, 8, 14, 15]. Most *L. monocytogenes* infections, however, occur without a clear connection to an outbreak; these cases are considered to be "sporadic." Three case-control studies of sporadic infection have been published. A 1986–1987 US study implicated frankfurters and chicken, a 1989–1990 Danish study implicated milk and pâté, and a 1988–1990 US study implicated soft cheese, food purchased from delicatessens and, in immunosuppressed patients, chicken [11, 12, 16]. Microbiology surveys have verified that *L. monocytogenes* may be present in the foods identified as high risk in these studies [5, 6, 8, 14, 15, 17, 18]. Only 1 epidemiologic study analyzed potential sources of *L. monocytogenes* infection according to pathogen subtype [13].

In 1989, after a case of listeriosis was linked to turkey frankfurters, the US Department of Agriculture implemented a "zero-tolerance" policy for *L. monocytogenes* in ready-to-eat meat and poultry [19–21]. In 1992, the US Centers for Disease Control and Prevention (CDC) disseminated guidelines to reduce listeriosis in high-risk populations [22]. Throughout the late 1980s and 1990s, food manufacturers instituted aggressive new hygiene policies in their plants [23]. These combined efforts appear to have contributed to decreases in the incidence of human *L. monocytogenes* infection from 1989–1993 [24]. Listeriosis incidence, however, did not decrease as markedly during 1996–2003, and large multistate outbreaks of *L. monocytogenes* infection continued to occur [15, 25]. In response to these concerns, the CDC launched a multicenter case-control study of sporadic listeriosis, and the US Food and Drug Administration and US Department of Agriculture conducted a Listeria Risk Assessment and revised the National Listeria Action Plan [26]. The primary purpose of this case-control study was to determine whether foods associated with *L. monocytogenes* infection have changed since widespread implementation of industry and government interventions and to determine whether subtyping of isolates could assist in identifying potential sources not previously targeted for interventions.

METHODS

Initiated in 1996 as a part of the CDC's Emerging Infections Program, the Foodborne Diseases Active Surveillance Network (FoodNet) is a collaboration between the CDC, the US Department of Agriculture, the US Food and Drug Administration, and participating state health departments that conducts surveillance and epidemiologic studies [27]. At the time of this study, FoodNet conducted active surveillance for culture-confirmed cases of *L. monocytogenes* infection in >600 clinical diagnostic laboratories located in a patient catchment area (mean population, 37.7 million) that included all or part of 9 states: California, Colorado, Connecticut, Georgia, Maryland, Minnesota, New York, Oregon, and Tennessee. For surveillance, a case is defined as isolation of *L. monocytogenes* from a normally sterile site, including from blood or CSF samples, or from placenta or the products of conception. A mother-infant pair was counted as a single case. During the period 2000–2003, FoodNet sites attempted to enroll all patients with culture-confirmed cases of *L. monocytogenes* infection in the case-control study. Sites began this study at different times during 2000; the Colorado site enrolled patients for 29 consecutive months, and the other 8 sites enrolled patients for 36 consecutive months. Patients with cases of culture-confirmed *L. monocytogenes* infection were excluded from the study if the patient did not report illness, lived outside the catchment area, did not speak either English or Spanish, could not be reached after ⩾15 telephone attempts and within 1 month after specimen collection, or acquired infection as part of a recognized outbreak. For this study, we defined an outbreak as the occurrence of ⩾2 ill persons with culture-confirmed cases of *L. monocytogenes* infection in which a public health investigation clearly identified a common source of infection.

Patients were classified as being immunosuppressed if the person reported active malignancy, HIV infection or AIDS, a history of solid-organ or bone marrow transplantation, end-stage renal disease, diabetes, receipt of radiation therapy, or treatment with an immunosuppressing medication (e.g., oral steroids, cyclosporine, or cancer chemotherapy). Patients were classified as having pregnancy-associated cases if illness occurred in a pregnant woman or an infant <31 days old; for all pregnancy-associated cases, including those in which only the infant was clinically ill, the mother was considered to be the case patient for recording the history of exposures.

After obtaining verbal informed consent, trained FoodNet staff conducted standardized telephone interviews with all eligible patients. If the patient was <12 years old or was not well enough to answer questions, then a surrogate—the person most familiar with the patient's dietary habits—was interviewed.

We attempted to recruit 4 control subjects for each case patient through physicians who treated the case patient. For pregnancy-associated cases, control subjects were matched within 1 month of the estimated date of delivery. For all other cases, control subjects were matched by age group (<6, 6–19, 20–59, and ⩾60 years old) and by primary immunosuppressing condition. If 4 control subjects could not be obtained from a physician's practice, study team members recruited persons from a predetermined multispecialty clinic, health maintenance organization, or medical center in the FoodNet catchment area.

Control subjects were ineligible if they did not speak English or Spanish.

For case patients, the questionnaire covered illness history. For both case patients and control subjects, the questionnaire covered medical history and medicines used, foods and drinks consumed, animals contacted, and places visited in the 4 weeks before specimen collection for the case patient. Food and drink questions involved >100 potential exposures and asked about the frequency with which items were consumed during the 4-week exposure period and where the item was prepared or consumed.

Isolates were obtained from clinical laboratories and forwarded to the CDC via state public health laboratories. All isolates were confirmed as *L. monocytogenes* with use of AccuProbe (GenProbe). Serotyping was done using the method of Seeliger and Hohne [28]. PFGE subtyping was performed using the *Asc*I restriction endonuclease, in accordance with the PulseNet standardized protocol [29].

We included all eligible, interviewed case patients in the analysis. We accounted for the matched study design by creating strata for the primary matching factors (e.g., age group, state, and medical condition). Medical condition was classified as immunosuppressed, pregnant, or not immunosuppressed. In all analyses, we controlled for the matching factors, for whether the subject was enrolled as part of a matched set, and for whether a surrogate respondent was used to report the food exposure history [30, 31]. We introduced individual exposure variables sequentially into an unconditional logistic regression model to determine adjusted, univariate ORs. Candidate multivariable models included variables known to be associated with *L. monocytogenes* infection based on prior studies and variables from the univariate analysis that were associated with risk of *L. monocytogenes* infection (OR, >1). We excluded from the models those variables that had ORs <1 in univariate analysis. We employed automated forward selection to derive a final multivariable model. We defined statistical significance as a P value of <.05. We performed subset analysis among patients with the most common *L. monocytogenes* serotypes, among patients with unique PFGE patterns, and among case patients not associated with pregnancy. Population-attributable fractions were calculated for the primary multivariable analysis using the method of Bruzzi to evaluate the relative importance of each exposure [32]. Data was managed using EpiInfo software, version 6.04b (CDC), and was analyzed using SAS software, version 9.0 (SAS Institute).

RESULTS

Study population. There were 249 reported cases of *L. monocytogenes* infection during the study period. Twelve cases were associated with 2 multistate outbreaks that were traced to turkey delicatessen meat in 2000 (8 cases; serotype 1/2a) and 2003 (4 cases; serotype 4b). Of 231 eligible case patients, 169 (73%) were enrolled in the study (figure 1). Enrolled case patients were similar to eligible, nonenrolled case patients with respect to age, sex, hospitalization, and proportion of isolates obtained from blood or CSF samples. Among enrolled case patients, the most common racial and ethnic categories were non-Hispanic white (75%), non-Hispanic black (10%), and Hispanic (10%). Twenty-eight (17%) of the enrolled case patients were classified as having pregnancy-associated cases. The median age of pregnancy-associated case patients was 28 years (range, 16–40 years); the median age of all other case patients was 71 years (range, 1–100 years). Among the 141 enrolled case patients whose cases were not associated with pregnancy, 108 (77%) were immunosuppressed.

We enrolled 376 control subjects. Control subjects were demographically similar to enrolled case patients with respect to age, sex, and a variety of other socioeconomic factors, including education, rural residence, health insurance, and self-reported household income. Control subjects were more likely to be non-Hispanic black than were enrolled case patients (19% vs. 10%; $P = .005$). The proportion of individuals classified as pregnancy-associated or immunosuppressed was similar among control subjects and enrolled case patients.

Outcomes and serotype distribution. Among the 28 pregnancy-associated case patients, 18 (64%) were admitted to the hospital for *L. monocytogenes*–associated illness, with hospital-

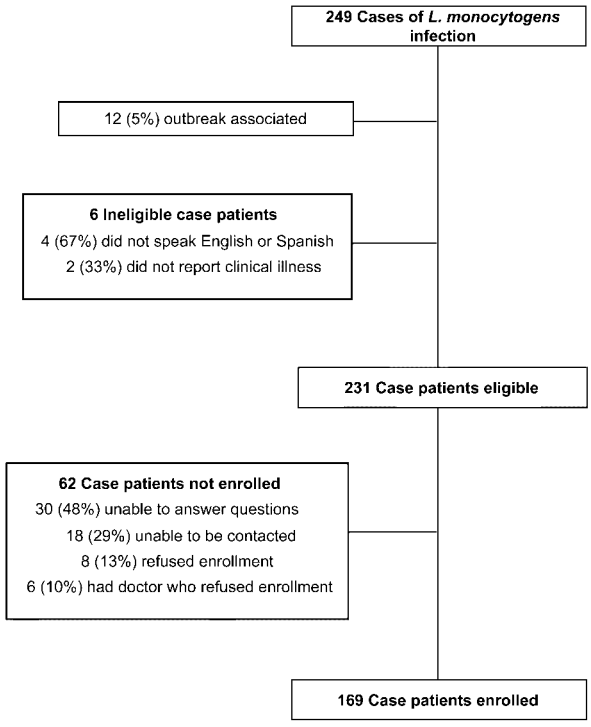

Figure 1. Study eligibility and enrollment of patients with cases of *Listeria monocytogenes* in the surveillance area during the period 2000–2003.

ization lasting a median of 8 nights (range, 2–9 nights). Outcome of pregnancy was not known for 3 case patients. Seven (28%) of the patients with pregnancy-associated cases experienced a spontaneous abortion or fetal demise, with fetal loss occurring at a median of 18 weeks gestation (range, 16–35 weeks). Among 18 live births, 15 infants were hospitalized after delivery for a median of 10 nights (range, 1–54 nights).

Among the 141 enrolled case patients not associated with pregnancy, 131 (94%) were admitted to the hospital for a median of 8 nights (range, 1–46 nights). Of these 141 case patients, 22 (16%) died.

Isolates for serotyping were available from 162 (96%) of 169 enrolled case patients (table 1). Serotype 4b was the most common serotype in case patients without immunosuppression or associated with pregnancy. Serotype 1/2a was the most common serotype among case patients with immunosuppression. Among 7 patients with known HIV infection, 3 had serotype 1/2a, 2 had serotype 4b, and 2 did not have isolates available for testing.

Isolates for PFGE subtyping with *Asc*I were available for 159 (94%) of the enrolled case patients. Of the 159 isolates, 93 (58%) had unique patterns. The 66 isolates with a pattern shared by at least 1 other isolate were clustered as follows: there were 8 clusters containing 2 isolates each, 5 clusters with 3 isolates each, 4 clusters with 4 isolates each, 1 cluster with 5 isolates, and 2 clusters with 7 isolates each.

Case-control study. In bivariate analysis, 11 exposures were associated with an increased likelihood of *L. monocytogenes* infection: having preexisting liver disease, eating hummus, eating hummus prepared in a commercial establishment, eating Mexican-style cheese that was purchased from a delicatessen counter, having a soft cheese (such as brie, camembert, or queso fresco) in the refrigerator, buying any soft cheese, eating melons at a commercial establishment, eating watermelon at a commercial establishment, eating "ice milk," eating leftovers heated on a stove, and living on a cattle farm (table 2). Five exposures were included in the final multivariable model, and 3 of these exposures had strong statistical associations with infection: eating hummus prepared in a commercial establishment, eating melons at a commercial establishment, and living on a cattle farm (table 3). When we restricted our analysis to the 93 isolates with unique PFGE patterns, there were no appreciable changes in the measures of association for the 5 exposures that had been included in the final multivariable model.

In a multivariable model restricted to serotype 1/2a–related cases, 3 exposures had strong statistical associations with infection: eating hummus prepared in a commercial establishment, eating melons at a commercial establishment, and eating ice milk. In a multivariable model restricted to serotype 4b–related cases, 2 exposures had strong statistical associations with infection: eating hummus and eating mussels.

Table 1. Distribution of *Listeria monocytogenes* serotypes among patients with non–outbreak-associated cases of *L. monocytogenes* infection, stratified by level of immunosuppression, 2000–2003.

	No. (%) of patients			
		With cases not associated with pregnancy		
Serotype	With pregnancy-associated cases (n = 28)	Without IS (n = 33)	With IS (n = 108)	All patients (n = 169)
1/2a	7 (25)	9 (27)	44 (41)	60 (36)
1/2b	4 (14)	6 (18)	28 (26)	38 (23)
1/2c	0 (0)	0 (0)	1 (1)	1 (1)
3a	0 (0)	0 (0)	1 (1)	1 (1)
4b	16 (57)	16 (49)	29 (27)	61 (36)
Not typable	1 (4)	0 (0)	0 (0)	1 (1)
No isolate	0 (0)	2 (6)	5 (5)	7 (4)

NOTE. IS, immunosuppression.

DISCUSSION

This is the first study of potential sources of *L. monocytogenes* infection conducted since the implementation of large-scale pathogen-reduction measures and dissemination of dietary guidelines to the public. We found that most cases of *L. monocytogenes* infection were not part of a recognized outbreak (i.e., they were sporadic) and that previously unrecognized foods, including melons eaten at a commercial establishment or hummus prepared in a commercial establishment, might be potential sources for sporadic listeriosis. These findings suggest that retail environments play a role in the contamination of foods and/or amplification of *L. monocytogenes.* Outbreaks of *L. monocytogenes* infection have been clearly linked to contamination at the time of food production or processing, but links to specific retail environments have been harder to demonstrate [33, 34]. This study also suggests that interventions directed at retail environments may be an important way to reduce sporadic disease, which represents the greatest burden of *L. monocytogenes* infection.

Melons are a well-established risk factor for bacterial foodborne infections and continue to be the vehicle of infection in many large bacterial gastroenteritis outbreaks in the United States [35, 36]. Although we did not ask about how the melons were prepared, it is likely that melons eaten at a commercial establishment (e.g., a restaurant) were sliced and probably refrigerated in the commercial establishment. *L. monocytogenes* may be present on the exterior of melons and preslicing could allow *L. monocytogenes* to multiply [37]. Food microbiology studies indicate that *L. monocytogenes* can contaminate melons and that refrigeration slows, but does not inhibit, the growth of *L. monocytogenes* [38].

Hummus, a food usually prepared from pureed chickpeas, has been found to be contaminated with *L. monocytogenes* in

Table 2. Bivariate analysis of risk factors for *Listeria monocytogenes* illness among patients with non–outbreak-associated cases of *L. monocytogenes* infection, 2000–2003, including subgroup analysis of serotype 1/2a and 4b cases.

Characteristic	Control subjects	Case patients								
		All	OR (95% CI)	*P*	With serotype 1/2a	OR (95% CI)	*P*	With serotype 4b	OR (95% CI)	*P*
Pre-existing liver disease	16/374 (4)	21/167 (13)	3.12 (1.34–7.26)	.01	9/59 (15)	3.91 (1.13–13.5)	.03	7/60 (12)	3.92 (1.19–12.89)	.02
Receipt of acid reducing medication	84/362 (23)	53/161 (33)	1.41 (0.84–2.34)	.19	18/54 (33)	1.13 (0.51–2.49)	.76	18/59 (31)	1.6 (0.74–3.44)	.23
Eating hummus										
Any hummus	15/373 (4)	13/167 (8)	2.66 (1.08–6.55)	.03	4/59 (7)	2.82 (0.72–11.03)	.14	7/60 (12)	3.92 (1.19–12.88)	.02
Hummus prepared in a commercial establishment	6/372 (2)	11/166 (7)	5.86 (1.83–18.81)	<.01	4/59 (7)	8.92 (1.86–42.75)	.01	5/59 (9)	5.12 (1.08–24.24)	.04
Eating hot dogs										
Any hot dogs	201/369 (55)	76/167 (46)	0.57 (0.36–0.89)	.01	29/58 (50)	0.71 (0.36–1.4)	.32	26/61 (43)	0.43 (0.22–0.84)	.01
Undercooked hot dogs	6/367 (2)	5/166 (3)	1.09 (0.25–4.74)	.91	1/57 (2)	1.01 (0.08–12.23)	.99	4/61 (7)	2.05 (0.37–11.51)	.41
Eating food purchased from a delicatessen counter										
Any ready-to-eat food	41/372 (11)	24/166 (15)	1.3 (0.68–2.5)	.42	11/58 (19)	1.67 (0.66–4.18)	.28	4/60 (7)	0.47 (0.13–1.71)	.25
Any meat	225/376 (60)	91/169 (54)	0.59 (0.37–0.94)	.03	33/60 (55)	0.59 (0.29–1.2)	.15	34/61 (56)	0.55 (0.28–1.11)	.1
Turkey breast	102/370 (28)	36/159 (23)	0.63 (0.37–1.07)	.09	12/56 (21)	0.64 (0.28–1.48)	.3	12/58 (21)	0.47 (0.2–1.09)	.08
Eating pâté	9/376 (2)	7/166 (4)	1.28 (0.38–4.24)	.69	2/59 (3)	0.8 (0.12–5.14)	.81	4/61 (7)	1.98 (0.4–9.9)	.41
Eating brie cheese	18/370 (5)	14/167 (8)	1.68 (0.7–3.99)	.24	5/58 (9)	1.26 (0.3–5.25)	.75	5/61 (8)	2.72 (0.75–9.87)	.13
Eating camembert cheese	5/371 (1)	7/166 (4)	2.83 (0.71–11.21)	.14	2/57 (4)	1.15 (0.15–9.1)	.89	2/61 (3)	1.77 (0.2–15.51)	.61
Eating Mexican-style cheese										
Any	22/371 (6)	13/166 (8)	1.48 (0.63–3.48)	.37	5/57 (9)	2.4 (0.63–9.14)	.2	4/61 (7)	0.97 (0.28–3.44)	.97
Purchased from a delicatessen counter	3/371 (1)	4/166 (2)	5.67 (1.01–31.88)	.05	3/57 (5)	37.67 (3.51–404.68)	<.01	1/61 (2)	3.4 (0.3–38.99)	.33
Having a soft cheese in the refrigerator[a]	27/373 (7)	29/165 (18)	2.39 (1.17–4.87)	.02	13/58 (22)	3.35 (1.22–9.18)	.02	12/60 (20)	4.02 (1.44–11.24)	.01
Buying any soft cheese[a]	32/374 (9)	30/165 (18)	2.03 (1.03–4.02)	.04	13/58 (22)	2.84 (1.06–7.62)	.04	12/60 (20)	2.88 (1.08–7.7)	.04
Eating mussels	17/375 (5)	13/168 (8)	2.13 (0.88–5.18)	.09	4/59 (7)	2.29 (0.63–8.33)	.21	6/61 (10)	3.83 (1.11–13.2)	.03
Eating any smoked fish	37/371 (10)	17/169 (10)	1.19 (0.59–2.4)	.63	5/60 (8)	0.57 (0.17–1.92)	.36	10/61 (16)	2.61 (1.06–6.43)	.04
Eating smoked salmon	34/375 (9)	16/169 (10)	1.2 (0.58–2.48)	.62	5/60 (8)	0.65 (0.19–2.26)	.5	10/61 (16)	2.96 (1.19–7.35)	.02
Eating melons										
Any	245/376 (65)	99/169 (59)	0.86 (0.55–1.35)	.51	35/60 (58)	1.08 (0.54–2.17)	.82	35/61 (57)	0.81 (0.42–1.55)	.52
At a commercial establishment	44/376 (12)	29/169 (17)	2.54 (1.39–4.65)	<.01	11/60 (18)	3.36 (1.36–8.32)	.01	10/61 (16)	2.31 (0.93–5.73)	.07
Watermelons										
Any	164/373 (44)	66/166 (40)	0.97 (0.63–1.5)	.9	24/58 (41)	1.3 (0.66–2.58)	.44	23/60 (38)	0.9 (0.47–1.72)	.07
At a commercial establishment	22/365 (6)	13/165 (8)	2.27 (1.02–5.03)	.04	6/57 (11)	3.48 (1.14–10.6)	.03	5/60 (8)	2.26 (0.68–7.44)	.18
Eating carrots at a commercial establishment	24/371 (7)	13/166 (8)	2.0 (0.88–4.54)	.10	7/58 (12)	8.25 (2.65–25.63)	<.001	4/60 (7)	1.53 (0.41–5.7)	.53
Eating ice milk	5/374 (1)	6/169 (4)	4.14 (1.08–15.91)	.04	3/60 (5)	10.3 (1.75–60.71)	.01	2/61 (3)	2.69 (0.34–21.01)	.35
Eating sorbet	57/373 (15)	36/166 (22)	1.69 (0.98–2.94)	.06	16/59 (27)	2.72 (1.23–6.04)	.01	9/61 (15)	0.95 (0.39–2.32)	.92
Eating leftovers heated on the stove	52/373 (14)	33/165 (20)	1.89 (1.06–3.38)	.03	13/59 (22)	1.88 (0.79–4.46)	.15	14/61 (23)	2.52 (1.12–5.68)	.03
Living on a farm with direct contact with animal feces	2/375 (1)	3/169 (2)	4.25 (0.5–36.31)	.19	1/60 (2)	2.41 (0.07–84.05)	.63	2/61 (3)	12.06 (1.2–120.69)	.03
Living on a cattle farm	1/376 (0)	3/169 (2)	11.87 (1.12–126.14)	.04	1/60 (2)	34.91 (0.82–1495.06)	.06	1/61 (2)	11.67 (0.59–228.98)	.11
Visiting a petting zoo with a pig present	1/375 (0)	1/166 (1)	6.12 (0.34–109.17)	.22	1/59 (2)	32.16 (1.67–620.55)	.02	0/61 (0)	0.00	…

NOTE. Data are proportion (%) of patients, unless otherwise indicated. Only selected risk factors are presented (those that were statistically significant in univariate analysis and those presumed to be associated with a high risk of illness on the basis of prior evidence).

[a] For example, brie, camembert, and queso fresco.

Table 3. Multivariable analysis of risk factors for *Listeria monocytogenes* illness among patients with non–outbreak-associated cases of *L. monocytogenes* infection, 2000–2003.

Characteristic, by serotype	OR (95% CI)	*P*	Population-attributable fraction (%)
All serotypes			
Pre-existing liver disease	2.89 (1.14–7.33)	.07	8.2
Eating hummus prepared in a commercial establishment	5.74 (1.72–19.13)	<.01	5.5
Eating Mexican-style cheese purchased from a delicatessen counter	5.03 (0.83–30.56)	.06	1.9
Eating melons at a commercial establishment	2.63 (1.39–4.96)	.01	10.6
Living on a cattle farm	13.75 (1.2–157.74)	.02	1.6
Serotype 1/2a			
Pre-existing liver disease	3.01 (0.81–11.16)	.06	10.2
Eating hummus prepared in a commercial establishment	9.23 (1.79–47.68)	<.01	6.0
Eating melons at a commercial establishment	2.59 (0.97–6.93)	.02	11.3
Eating ice milk	7.04 (0.97–51.36)	<.01	4.3
Eating sorbet	1.99 (0.85–4.66)	.11	13.5
Serotype 4b			
Eating hummus	3.19 (0.98–10.33)	.02	8.0
Eating mussels	2.98 (0.81–11)	.01	6.5
Eating smoked salmon	2.27 (0.87–5.92)	.09	9.2

US Food and Drug Administration inspections [39]. A recent food microbiology survey reported isolating 2 *L. monocytogenes* strains from hummus that were identical to strains implicated in *L. monocytogenes* outbreaks [40]. Hummus prepared in a commercial establishment may be contaminated during preparation or storage and exposed to prolonged storage times before serving, permitting *L. monocytogenes* growth.

Although infrequently identified in food microbiologic surveys, serotype 4b is the most common *L. monocytogenes* serotype among patients in the United States, and some have speculated that it may have a greater propensity to cause disease when present on contaminated food [13, 41, 42]. In our study, potential sources for serotype 4b included hummus, mussels, and smoked salmon. Serotype 1/2a, the other common serotype in our study, has previously been linked in outbreaks to turkey frankfurters and turkey delicatessen meat [19, 34]. The association between serotype 1/2a and eating hummus was statistically significant in multivariable analyses, suggesting that this association may be particularly important.

Our study is subject to several limitations. Information bias is possible given the long exposure period for dietary histories, the use of surrogate respondents, and the limited recall among patients who were severely ill and elderly. The use of a 4-week exposure period may make it difficult to demonstrate an association between illness and commonly eaten foods, resulting in case patients and control subjects being equally likely to be exposed to commonly eaten, high-risk foods. Earlier *L. monocytogenes* case-control studies used a methodology similar to that used in this study and enrolled fewer patients; these studies did find an association with commonly eaten foods, such as undercooked hot dogs and foods purchased from delicatessen counters, suggesting that the absence of an association in our study may not be due to bias [11, 12].

Selection bias is also an important limitation. Some otherwise eligible case patients were not enrolled in the study, because they were dead or were too ill to answer questions. Patients who had died or were too ill to answer questions may represent a population with unique host dynamics or food exposures. PFGE analysis suggests that an undetected, widespread outbreak was unlikely to have skewed our findings (i.e., that this was, in fact, a study of sporadic illness). This is further supported by our subset analysis of isolates with unique PFGE patterns. Recruitment of control subjects for a study of *L. monocytogenes* infection is challenging, because no biologic or epidemiologic tool exists to measure and adjust for susceptibility to listeriosis. We, therefore, had to rely on recruitment from physician practices, a method that may introduce bias. Similarities in demographic data and medical conditions between case patients and control subjects suggest that a substantial difference between these 2 populations is unlikely. To assess the impact of information and selection bias, we conducted a sensitivity analysis that confirmed the findings of our study; methods and results for this analysis are available as a separate appendix on the FoodNet Web site [43].

The US Food and Drug Administration risk assessment identified turkey delicatessen meat as one of the most important sources of *L. monocytogenes* infection, but our study found no such association [44]. We excluded 12 outbreak-associated cases from our study. These 12 cases were part of 2 multistate outbreaks caused by *L. monocytogenes*–contaminated turkey deli-

catessen meat, demonstrating that turkey delicatessen meat remains an important source of human *L. monocytogenes* outbreaks. Soft cheeses and cheeses made from nonpasteurized milk are also well-established risk factors for *L. monocytogenes* infection [45, 46]. Although several cheese exposures were associated with *L. monocytogenes* infection in our univariate analysis, none remained so in the multivariable analysis. It is possible that the absence of a strong association in this study between *L. monocytogenes* and several foods targeted for regulatory intervention indicates that government and industry efforts have reduced the risk associated with these foods.

Although uncommon, *L. monocytogenes* remains a pathogen of great public health concern, because it exists throughout the environment, readily contaminates food, and causes high morbidity and mortality in vulnerable populations. Consumers at high-risk for *L. monocytogenes,* such as pregnant women, may wish to avoid eating melons and hummus prepared in a commercial establishment, as well as other foods known to be associated with a high risk for listeriosis, such as soft cheeses and unheated delicatessen meats [47]. Public health officials and physicians should remain aware that the range of potentially hazardous foods remains large and should participate in new public health initiatives to collect detailed food histories for all cases of *L. monocytogenes* infection and conduct PFGE for all *L. monocytogenes* isolates [48]. Interventions directed at retail environments may be the next most-promising approach for controlling this pathogen.

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