

## A foodborne outbreak of gastroenteritis involving *Listeria monocytogenes*

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### SUMMARY

An outbreak of gastroenteritis occurred in Italy among 39 persons who had attended a private supper. All guests were previously healthy, young, non-pregnant adults; 18 (46%) had symptoms, mostly gastrointestinal (78%), with a short incubation period. Four were hospitalized with acute febrile gastroenteritis, two of whom had blood cultures positive for *Listeria monocytogenes*. No other microorganisms were recovered from the hospitalized patients' specimens. Epidemiological investigation identified rice salad as the most likely vehicle of the food-borne outbreak. *L. monocytogenes* was isolated from three leftover foods, the kitchen freezer and blender. Isolates from the patients, the foods and the freezer were indistinguishable: serotype 1/2b, same phage type and multilocus enzyme electrophoretic type. Eight (36%) of 22 guests tested were found to have antibodies against *L. monocytogenes*, compared with none of 11 controls from the general population. This point source outbreak was probably caused by infection with *L. monocytogenes*. Unusual features included the high attack rate among immunocompetent adults and the predominance of gastrointestinal symptoms.

### INTRODUCTION

The role of *Listeria monocytogenes* in foodborne outbreaks has been recognized clearly in the last decade [1–6]. Case-control studies have been employed with success in identifying food vehicles and in certain outbreaks the same strain of *L. monocytogenes*

has been isolated from both infected individuals and from the epidemiologically implicated food [1, 3, 4]. These outbreaks have typically involved several cases presenting over a long period of time and distributed over a large geographic area. Cases have been reported mainly among pregnant women, neonates, the elderly, and persons with underlying conditions (i.e. cancer, immunosuppression, etc.); affected individuals have experienced abortion, stillbirth, neonatal sepsis, meningitis and sepsis. By contrast, we observed and described here an outbreak with predominance of gastrointestinal symptoms among immunocompetent

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adults whose onset of symptoms occurred from 11–60 h after a common exposure and where *L. monocytogenes* was the most likely agent.

### Background

On 14 June 1993, the Local Health Unit (LHU) of the town of San Giorgio di Piano (located near the city of Bologna in Northern Italy) received from the local hospital a report of a suspected foodborne outbreak. Four individuals had been hospitalized in the same day with acute febrile gastroenteritis. All had attended a supper held in a private home on the evening of 12 June together with 35 other participants.

On the day the report was received, a routine epidemiological investigations for a foodborne outbreak was commenced by interviewing all participants at the supper. Rice salad was identified as the most likely vehicle by a comparison of attack rates among consumers and non-consumers (90% vs. 0%). Most foods served at the dinner were still available for laboratory analysis and were found to be heavily contaminated with coliforms but negative for enteropathogens. On 20 June, 8 days after the supper, the local hospital informed the LHU that blood cultures from two of the four hospitalized patients were positive for *L. monocytogenes*, while faeces were negative for the common enteropathogens tested (see Methods section). Given this unusual finding, additional microbiological and epidemiological investigations were conducted to clarify the role of *L. monocytogenes* in the aetiology of the outbreak.

San Giorgio di Piano has a population of 71 587 inhabitants and is located in a rural area approximately 10 km to the northeast of the city of Bologna. Farms for raising sheep, cattle, and pigs are common in the area. No previous cases of listeriosis had been reported in this area since 1990, when listeriosis was included among conditions subject to statutory notification.

## MATERIALS AND METHODS

### Epidemiologic investigation

All 39 attenders at the supper were interviewed using a standardized questionnaire on type and quantity of each food consumed, presence and time of onset of symptoms, and antibiotic treatment received. An outbreak-associated case was defined as an illness occurring in a person attending the supper on 12 June

and who within 3 days developed fever ( $\geq 37.5$  °C) plus one or more of the following symptoms: diarrhoea (three or more loose stools per day), nausea, vomiting, or arthromyalgias.

To assess the local rate of infection of *L. monocytogenes*, a blood sample for detection of anti-*L. monocytogenes* antibodies was obtained from 22 of the 39 attenders (4 non-cases and 18 cases) and from 11 controls who were healthy adult volunteers from the LHU personnel who resided in the same area, 15–30 days after the supper. Stool specimens for enteropathogen culture were also obtained approximately 30 days after the supper from all attenders and from 14 healthy adult LHU personnel (including the 11 above).

### Environmental and laboratory investigation

During the initial investigation, information on ingredients and methods of food preparation were obtained by interviewing the woman who had prepared the meal and at whose home the supper had taken place. All but one of the courses were prepared in her kitchen from industrially or locally produced ingredients.

Samples of food still available when the investigation was initiated were collected and sent to the local microbiological laboratory (Presidio Multizonale di Prevenzione) located in Bologna, where cultures for the following enteric pathogens were performed: *E. coli*, *Salmonella* spp., *Bacillus cereus*, and *Staphylococcus aureus*. Foods remaining after microbiological assessments were stored at +4 °C in the same laboratory; none of the rice salad remained after the initial microbiological examination.

During the additional investigation, some basic ingredients of dishes served at the supper were also collected, as were environmental samples from various kitchen surfaces. In the 2 weeks following the supper, the same type and brand of ingredients used for the supper were retrieved from the food-store and from the farm where the original ingredients had been purchased.

### Laboratory methods

#### *Bacteriological investigations*

Stool specimens from the four hospitalized individuals were plated on MacConkey and Salmonella–Shigella (SS) agar, incubated at 37 °C for 18 h, and examined

for the presence of salmonella, shigella, and other enteropathogens by standard methods [7]. For *Salmonella* spp. a portion of each sample was enriched in sodium selenite medium, incubated for 18 h at 37 °C, and then plated on SS and held at 37 °C. All specimens were also plated on cefsulodin–Irgasan–novobiocin (CIN) (Difco, Detroit) agar for the isolation of *Yersinia enterocolitica*, and stored at room temperature for 48 h [7]. Blood-free agar (CCDA) plates, incubated at 42 °C for 48 h in a microaerophilic environment, were used to test for the presence of *Campylobacter* spp. [8].

Blood cultures on all hospitalized individuals were performed using a commercial kit: Bactec NR-730 (Becton-Dickinson). Food and stool specimens were cultured for *L. monocytogenes* according to the methodology described by Lovett [9] with a modification consisting of a second phase of selective enrichment [10]. Isolates were biochemically confirmed as *L. monocytogenes* and subjected to mouse lethality testing using the procedure of Lovett [9]. Serotyping was performed using the method described by Seeliger and Hohne [11]. Quantitative determination of *L. monocytogenes* was conducted using the most probable number (MPN) technique [12]. Environmental samples from surfaces were taken using sterile gauze saturated with Ringer solution (Oxoid, England) and transferred to the laboratory in enrichment broth (LEB, Biolife).

The detection of anti-*L. monocytogenes* antibodies was performed by the laboratory of the local hospital using an *in vitro* agglutination test with antigens O and H type 1 and 4b provided in a commercial kit (Behring).

#### Characterization of *L. monocytogenes* strains

All isolates of *L. monocytogenes* were sent to the Division of Bacterial and Mycotic Disease at the US Centers for Disease Control, where multilocus enzyme electrophoresis was performed according to the methodology of Selander [13] and Bibb [14]. The same strains were sent to the Centre Collaborateur de l'OMS pour la Listériose d'Origine Alimentaire, Institut Pasteur, where phage typing was carried out using the methods described by Rocourt [15].

#### Statistical analysis

All data collected from interviews were entered and analysed using EPI INFO version 5.01b. Non-para-

metric (Kruskal–Wallis) tests were used to compare distributions of quantitative variables. Attack rates, relative risks (R.R.) and 95% confidence limits were calculated for each of the foods served at the supper. Proportions were compared by the chi-square test or Fisher's exact test.

## RESULTS

### Epidemiological and clinical findings

Of the 39 supper attendees, 18 (46%) reported fever and one or more additional symptoms in the 3 days following the supper, meeting the case definition for the investigation; 16 consulted a physician and 13 underwent antibiotic treatment for an average duration of 5 days. None of the non-hospitalized individuals had undergone any microbiological test at the onset of symptoms or before treatment. The median age of attendees was 28 years, and all were previously healthy. Cases ranged in age from 17–54 years; their median age was higher than that of non-cases (36 vs. 22 years;  $P < 0.001$ ). Thirty-nine percent of all attendees were female; none was pregnant. None of the cases had a history of cancer, immunosuppression, or any other underlying condition.

Symptoms of cases are shown in Table 1; most had febrile gastroenteritis characterized by diarrhoea (up to 20 loose stools per day) (78%), nausea (78%) and vomiting (44%). Other common symptoms were arthromyalgia (78%), headache (78%) and sore throat (72%). In four cases only flu-like symptoms were reported with arthromyalgias, sore throat and headache, but no diarrhoea or vomiting. Among the latter, mean body temperature was significantly lower than that of those with gastrointestinal symptoms (38.0 °C vs. 39.5 °C,  $P < 0.01$ ), and clinical features were generally milder. The cook and her husband experienced flu-like symptoms, but their two daughters, who had eaten at the supper, remained healthy (none of the family members reported diarrhoea or flu-like illness in the week before the supper). Seven guests reported symptoms but did not meet the case definition. The interval between the supper and the onset of symptoms for cases ranged from 11–60 h. As shown in the epidemic curve (Fig. 1), gastrointestinal illness had an earlier onset than the influenza-like syndrome, the median incubation times being 18 and 43 h, respectively ( $P = 0.06$ ).

Four cases, two males and two females, were hospitalized within 24 h of the supper. Their ages

Table 1. Frequency distribution of symptoms among participants at the supper

Symptoms	Gastrointestinal (n = 14)	Flu-like (n = 4)	Total (n = 18)
Diarrhoea*	14 (100)†	0 (0)	14 (78)
Fever	14 (100)	4 (100)	18 (100)
Mean temperature °C	39.5 —	38.0 —	39.2 —
Nausea	12 (86)	2 (50)	14 (78)
Headache	11 (79)	3 (75)	14 (78)
Sore throat	10 (72)	3 (75)	13 (72)
Arthromyalgias	10 (71)	4 (100)	14 (78)
Vomiting	8 (57)	0 (0)	8 (44)
Abdominal pain	8 (57)	0 (0)	8 (44)
Swollen lymphnodes	2 (14)	2 (50)	4 (22)
Median onset in hours	18 —	39 —	18 —

\* From 3–20 loose stools/day (median = 3).

† Percentages are given in parentheses.

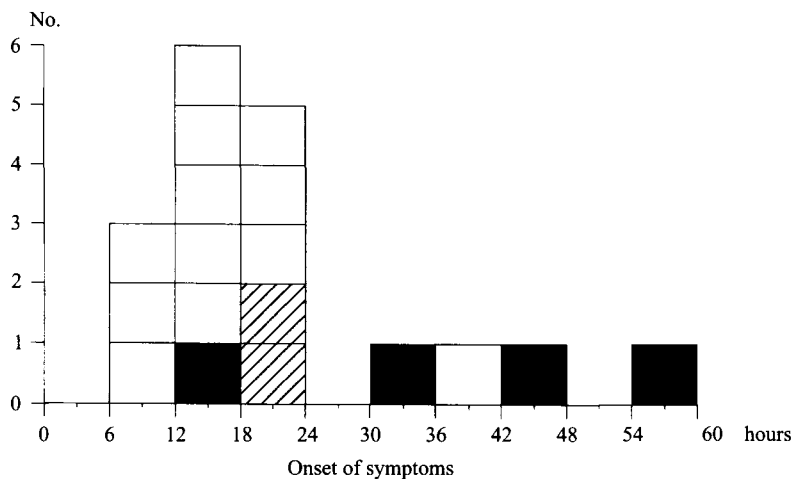


Fig. 1. Epidemic curve. □, Gastrointestinal symptoms; ▨, bacteraemic patients; ■, flu-like symptoms.

ranged from 17–27 years. Upon hospital admission, all had high fever (average temperature was 39.6 °C), diarrhoea, nausea, and abdominal pain; they also complained of arthromyalgias, headache, and sore throat. Stool cultures were negative for salmonella, shigella, and other common enteric pathogens. During hospitalization, no patient's stool was cultured for *L. monocytogenes*. Blood samples were collected and cultured on the day of hospital admission from all four patients. In two patients, *L. monocytogenes* was isolated from blood cultures. Patients were discharged from the hospital after 3–14 days, following treatment with trimethoprim-sulphamethoxazole (two patients) or with ampicillin (two patients). Strains from the two blood specimens were both serotype 1/2b, electrophoretic type (ET) 16, and phage type 1967:10:43.

Of the 22 attenders tested serologically for *L. monocytogenes*, 7/18 (39%) cases and 1/4 (25%)

non-cases had antibodies (dilution 1:200) against the somatic antigen type 1, while none had antibodies against type 4b antigens (both O and H). Among the 4 hospitalized individuals, 3, including the 2 bacteraemic patients, were positive. The highest titre (1600) was seen in the woman who had prepared the food. All sera from the 11 controls were negative. The prevalence of seropositivity among supper attenders was significantly higher than amongst controls (0/11 vs 8/22;  $P = 0.03$ ). Stool samples collected 1 month after the supper from all 39 guests and from the 14 controls, were negative for *L. monocytogenes*.

Analysis of the food consumed (Table 2) confirmed that rice salad was the most likely vehicle: the attack rate was 90% for those who ate rice salad and 0% for those who did not ( $P < 0.001$ ). The protective role played by the pizza (R.R. = 0.3) could be explained by the fact that those who ate pizza were less likely to

Table 2. Attack rates among consumers and non-consumers and Relative Risks (R.R.) by food

	Those who ate		Those who did not eat		R.R.	95% C.I.
	ill/total	(%)	ill/total	(%)		
Rice salad	18/20	90	0/19	0	n.c.*	—
Fruit tart	14/25	56	4/14	29	2.0	[0.8–4.8]
Onion relish	7/10	70	11/29	38	1.9	[1.0–3.4]
Salmon canapé	9/15	60	9/24	38	1.6	[0.8–3.1]
Cheese canapé	7/12	58	11/27	41	1.4	[0.7–2.8]
Cream puff	11/23	48	7/16	44	1.1	[0.5–2.2]
Spinach pie	3/6	50	15/33	46	1.1	[0.5–2.7]
Vol-au-vent	8/19	42	10/20	50	0.8	[0.4–1.7]
Pizza	9/30	30	9/9	100	0.3	[0.2–0.5]

\* Not computable;  $P < 0.001$ .

Table 3. Microbiological analysis of food samples

Food	Total bacterial count c.f.u./g	Coliforms c.f.u./g	<i>E. coli</i> c.f.u./g	<i>Listeria monocytogenes</i> c.f.u./g*
Fresh eggs	—	—	—	neg/25 g
Onion relish	$1.1 \times 10^{10}$	$1.0 \times 10^7$	neg	—
Mayonnaise, tuna fish	$1.8 \times 10^4$	$4.0 \times 10^2$	neg	—
Cream puff	$6.0 \times 10^7$	$2.5 \times 10^6$	neg	—
Shrimp vol-au-vent	$3.0 \times 10^4$	$1.5 \times 10^3$	neg	$2.1 \times 10^3$
Mayonnaise vol-au-vent	$2.0 \times 10^4$	neg	neg	neg/25 g
Cream cheese canapé	$6.0 \times 10^7$	$5.0 \times 10^5$	$5 \times 10^2$	$4.6 \times 10^5$
Fresh fruit tart	$5.6 \times 10^9$	$8.0 \times 10^6$	$4 \times 10^3$	0.93
Pizza	$1.5 \times 10^3$	$8.0 \times 10^2$	neg	—
Salmon canapé	$4.0 \times 10^5$	$1.1 \times 10^4$	neg	—
Rice salad	$5.6 \times 10^9$	$1.5 \times 10^6$	$1 \times 10^3$	—
Swiss cheese	$8.0 \times 10^2$	neg	neg	—
Frozen peas†	$5.0 \times 10^4$	neg	neg	neg/25 g
Stuffed olives†	$2.7 \times 10^6$	$2.5 \times 10^6$	neg	neg/25 g
Raw onions†	—	—	—	neg/25 g
Raw onions‡	—	—	—	neg/25 g
Cream cheese‡	$2.0 \times 10^2$	neg	neg	neg/25 g

\* Determined by the three-tube most probable number method.

† Foods sampled on the second visit of the kitchen.

‡ Foods obtained from the supermarket and the farmer.

eat rice salad than those who did not (9/30 vs. 9/9;  $P < 0.001$ ). No association was observed between symptoms and the consumption of other foods, even after adjustment for the quantity of food consumed.

The ingredients in the rice salad were: boiled rice, swiss cheese, pickled vegetables, hard-boiled eggs, and frozen vegetables (peas, carrots, etc.). All of the dishes, excluding the spinach pie brought by one of the guests, were prepared by the host in the 4–5 h preceding the supper and placed in a refrigerator, with the exception of the rice salad and the fresh fruit tart,

which were prepared 24 h in advance, and which, because of their large volume, remained outside the refrigerator at room temperature. The average daily temperature in this area during June is 27–28 °C.

#### Laboratory findings in foods and environmental samples

Many foods, including rice salad, were found to be heavily contaminated with coliforms, reaching a value of  $10^7$  c.f.u./g (Table 3). All foods were negative on

culture for *Salmonella* spp., *Bacillus cereus*, and *Staphylococcus aureus*. When the initial laboratory investigation was conducted, only these enteric pathogens were sought, as routinely done when a foodborne outbreak is suspected. No rice salad remained in the laboratory for culturing for *L. monocytogenes*. However, *L. monocytogenes* was isolated from 3 of the five other foods left over from the supper and still available in the laboratory (shrimp vol-au-vent, cheese canapé, and fresh fruit tart) and from the blender and the freezer in the home of the cook. *L. monocytogenes* was not found either in foods taken from the kitchen which were used as ingredients at the supper or in foods retrieved from the local food-store and farm. All *L. monocytogenes* isolates were pathogenic when tested in the mouse model.

Strains from the three foods and from freezer specimens were similar to strains isolated from blood of hospitalized patients: serotype 1/2b, electrophoretic type (ET) 16, and phage type 1967:10:43; while the strain from the blender showed a different pattern: serotype 1/2a and phage type 575.

## DISCUSSION

Although *L. monocytogenes* is ubiquitously distributed, the incidence of recognized listeriosis in the community is low [16]. Moreover, despite the observation that food has often been the vehicle of transmission, gastroenteritis is an infrequent presentation. Nonetheless, gastrointestinal symptoms before the onset of meningitis or other invasive diseases due to *L. monocytogenes* have been reported in both sporadic [17–19] and outbreak-associated cases [20–24].

Syndromes associated with recognized adult listeriosis are in most cases represented only by severe clinical manifestations such as infections of the central nervous system, sepsis, or focal infections [16]. The potential for *L. monocytogenes* to cause gastroenteritis alone has been considered previously, but mild disease due to *L. monocytogenes* has been difficult to document [22, 25, 26]. Riedo and colleagues reported on an investigation initiated after two pregnant women developed sepsis due to *L. monocytogenes* several weeks after a catered party; mild gastrointestinal and musculoskeletal symptoms were identified retrospectively in several other partygoers [25].

The present outbreak presents several unusual features, the most important being the recovery of *L.*

*monocytogenes* and the predominance of gastrointestinal symptoms experienced by immunocompetent adults attending the dinner. Although infection by *L. monocytogenes* was confirmed microbiologically only for two hospitalized cases, the following findings support the hypothesis that *L. monocytogenes* played an aetiological role in the outbreak of gastroenteric illnesses:

(1) *L. monocytogenes* was isolated from blood specimens of two of the outbreak-associated cases affected by diarrhoea and fever, symptoms presented by the majority of the cases; (2) identical strains of *L. monocytogenes* were isolated from patients and from several foods served at the supper; (3) several cases with diarrhoea had high titres of anti-listerial antibodies against the same serotype of the outbreak-associated strain, and the rate of seroprevalence of antibodies against the microorganism was significantly higher among the supper attenders than among controls; (4) no other common enteric bacterial pathogens were revealed by laboratory assessments; and (5) the short incubation period observed is not consistent with gastrointestinal viral infections but is similar to that observed in a few sporadic cases where relatively clear links were established between the onset of invasive disease and consumption of listeria-contaminated food [19, 28, 29].

The attack rate in this outbreak was high (46%) and 4 of the 18 cases were hospitalized; this may have been due to virulence of the causative strain or a very high infecting dose, as suggested by results of quantitative cultures for *L. monocytogenes* in the available foods and by the inappropriate storage conditions of the rice salad.

The foods implicated in previously reported major foodborne outbreaks of listeriosis were commercially produced and widely distributed, such as soft cheese in Europe [4] and Los Angeles [3], pâté in the United Kingdom [5], and pork tongue in jelly in France [6]. Consequently, efforts to reduce listerial contamination of ready-to-eat foods became a focus of food regulatory policy internationally. In this outbreak, we were unable to identify the original source of *L. monocytogenes* from which contamination of the rice salad might have occurred. However, the same strain of *L. monocytogenes* was found in several foods left over from the supper, with no ingredient in common, as well as on one environmental surface, indicating that extensive cross-contamination may have occurred. Recent investigations suggest that cross-contamination of foods at retail facilities, such as

delicatessen counters in the US [30] or 'charcuteries' in France [6], contributes to the spread of infection. In the present case, *L. monocytogenes* could have entered the home in any number of foods, with time and temperature abuse leading to high level contamination of the rice salad.

Subtyping was useful in this outbreak investigation. Serotyping alone is only of minimal help in epidemiological studies, since the majority of human listeriosis is caused by three serotypes of *L. monocytogenes* (4b, 1/2a, and 1/2b) [16]; in fact, one of the strains found on the surface of the kitchen, although having the same serotype was completely different from the other strains in terms of phage and electrophoretic type. Though *L. monocytogenes* is frequently recovered from the environment and from a variety of foods [16], identification of the same phage type and enzyme type in patients and in foods served at the supper was unlikely to have occurred by chance.

Prevention of listeriosis has focused on efforts to eliminate contamination during food processing and to educate consumers at increased risk due to immunosuppression or pregnancy. However, this outbreak reinforces the importance of proper food handling practices in reducing the risk of foodborne disease, particularly when large food quantities are prepared in non-commercial settings. Finally, given the atypical clinical presentation of *L. monocytogenes* observed in this outbreak, culture for this pathogen should be considered during investigations of foodborne outbreaks.

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