AN OUTBREAK OF FEBRILE GASTROENTERITIS ASSOCIATED WITH CORN CONTAMINATED BY *LISTERIA MONOCYTOGENES*

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ABSTRACT

Background On May 21, 1997, numerous cases of febrile gastrointestinal illness were reported among the students and staff of two primary schools in northern Italy, all of whom had eaten at cafeterias served by the same caterer.

Methods We interviewed people who ate at the cafeterias about symptoms and foods consumed on May 20. There were no samples of foods left at the cafeterias, but we tested routine samples taken on May 20 by the caterer and environmental specimens at the catering plant. The hospitalized patients were tested for common enteropathogens and toxins.

Results Of the 2189 persons interviewed (82 percent of those exposed), 1566 (72 percent) reported symptoms; of these, 292 (19 percent) were hospitalized. Among samples obtained from hospitalized patients, all but two of the stool specimens and all blood specimens were negative for common enteropathogens. *Listeria monocytogenes* was isolated from one blood specimen and from 123 of the 141 stool specimens. Consumption of a cold salad of corn and tuna was associated with the development of symptoms (relative risk, 6.19; 95 percent confidence interval, 4.81 to 7.98; $P<0.001$). *L. monocytogenes* was isolated from the caterer's sample of the salad and from environmental specimens collected from the catering plant. All listeria isolates were serotype 4b and were found to be identical on DNA analysis. Experimental contamination of sterile samples of the implicated foods showed that *L. monocytogenes* grew on corn when kept for at least 10 hours at 25°C.

Conclusions Food-borne infection with *L. monocytogenes* can cause febrile illness with gastroenteritis in immunocompetent persons. (N Engl J Med 2000;342:1236-41.)

LISTERIA monocytogenes, a gram-positive bacterium that can grow at low temperatures, causes several illnesses in humans and animals. In immunocompromised persons, food contaminated with *L. monocytogenes* may cause severe invasive disease.[1-4] Few cases of listeriosis have been reported in previously healthy persons, and these cases were attributed to exposure to high infective doses.[5-7] Although listeriosis is known to be transmitted through food, only recently has it begun to be considered a gastrointestinal disease. Unlike other food-borne infections, which produce intestinal symptoms, the clinical manifestation of infection with *L. monocytogenes* is usually described as an illness of the central nervous system, as sepsis, or as a flulike disease; it affects mainly immunocompromised persons and has a limited effect on the general population. Some of the episodes reported[8,9] have involved gastroenteric symptoms such as diarrhea, nausea, vomiting, and abdominal cramps, often accompanied by fever. Ingestion of listeria was the suspected cause of two small outbreaks of gastroenteric illness.[10,11] However, only recently has it been demonstrated that food-borne listeriosis can present as a gastrointestinal illness with fever.[12-14]

On May 21, 1997, the local health units of two adjacent towns (Moncalieri and Giaveno) in northern Italy received an unusually high number of reports of febrile illness and gastroenteric disease among children (who were 6 to 10 years of age) and adult staff members at local primary schools. On the same day, a local health unit in nearby Turin received reports of similar cases among students who had eaten at the cafeteria of the University of Turin. The cafeterias of the primary schools and the university were served by the same caterer. The outbreak received wide national press coverage, and in response to public concern, the primary schools were closed for the summer holidays two weeks early. To identify the source and cause of the illness, we conducted epidemiologic and microbiologic investigations.

METHODS

Background

Moncalieri and Giaveno have populations of approximately 55,000 and 6000, respectively. Both towns are located within 30 km of Turin (population, 1.2 million), which is the principal city of the Piedmont region in northern Italy.

Separate local health authorities are responsible for the surveillance and prevention of communicable diseases in the three towns.

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All the persons in whom illness was reported had eaten meals provided by a local catering company, which has a permanent staff of 18 employees and prepares about 8000 meals a day. The caterer's food handlers have taken a course on safe food preparation, and the manager ensures the quality of the food by following the recommendations of the Fédération Européenne de la Restoration Collective. Specifically, routine samples of prepared food are collected every day before distribution and stored at 4°C until microbiologic tests are performed.

Epidemiologic Investigation

The caterer provided us with a list of foods served at the cafeterias of the primary schools and the university on the day before the onset of symptoms. Because the common exposure of the persons who became ill was evident, we designed the epidemiologic investigation as a cohort study. To identify the students and staff of the primary schools who ate meals at the cafeteria on May 20, we contacted the administrative offices of the schools. For the university, we were able to trace only some of the students who ate in the cafeteria, by counting the payment slips. Overall, 2930 persons were estimated to have been exposed to the implicated meals (2658 in the primary schools and 272 at the university).

During the three weeks that followed the outbreak, we attempted to trace the exposed persons and to interview them by telephone using a standard questionnaire, which included questions on the specific food items eaten on May 20 and on the presence, time of onset, and type of symptoms that occurred within one week after ingestion of the meal; fever was defined as a body temperature of at least 38°C, and diarrhea was defined as at least three loose stools within 24 hours. There was also a question about hospital admission. For patients who had been hospitalized (in a total of nine hospitals), we reviewed clinical records to obtain information on the clinical characteristics of the illness and the results of microbiologic tests conducted on stool samples. We then matched this information to that obtained from the questionnaire. The food handlers at the catering firm were also interviewed about the onset of symptoms and about food-preparation procedures and were asked to provide stool samples.

Microbiologic Investigation

Blood and Stool Samples

We reviewed the results of the stool cultures performed by the nine hospitals. For some patients, the hospitals had also performed blood cultures, depending on the practice of the hospital and the clinical presentation of the patient. The hospitals initially tested the stool samples for salmonella, shigella, clostridium, yersinia, campylobacter, rotavirus, astrovirus, and adenovirus; after testing, one of the hospitals froze the specimens for future analysis.

After a strain of listeria was isolated from one patient's blood, all available stool specimens (a total of 141, which included the specimens that had been frozen and the specimens that were still available from patients) were tested for listeria. Culture for *L. monocytogenes* was performed on a selective agar base (Oxford formulation, Oxoid, Basingstoke, United Kingdom). For confirmation, bacterial isolates were sent to the Istituto Superiore di Sanità (the national institute of health of Italy).

Food

Because there was no leftover food at the cafeterias from the meals served on May 20, we obtained the food samples that the caterer had taken on that day for routine testing. The samples were of pasta with olive oil, a salad made with canned tuna and canned corn, and julienned carrots; all had been stored at 4°C. We also collected samples from sealed cans of corn and tuna that belonged to the same batches as those served at the meals. The canned foods were tested for sterility, and (given the possibility of corn toxicity caused by mycotoxins produced by molds) the corn was also tested for the presence of vomitoxin, zearalenone, and fumonisin, with the use of commercial enzyme-linked immunosorbent assay kits (Tenca, R and D Diagnostics–Biotechnology, Trieste, Italy; and Ridascreen, R-Biopharm, Darmstadt, Germany). All foods were analyzed for major food-borne pathogens, including *L. monocytogenes*.[15] A colony count of *L. monocytogenes* was performed in a petri dish with selective medium.[16]

Environmental Samples

At the catering plant, moistened swabs were used to wipe the work surfaces, the utensils, the sinks in the area where the salad of corn and tuna had been prepared and the vegetables had been washed, and the floor drains in the room where the tools had been washed. Swabs were examined for salmonella, *Yersinia enterocolitica*, *Escherichia coli*, *Clostridium perfringens*, and *L. monocytogenes*.

Identification of the Listeria Strain

Listeria isolates were biochemically confirmed (10300 API Listeria system, BioMérieux Italia, Florence, Italy) and assessed for pathogenicity in immunocompromised mice.[17] For phage typing and serotyping, *L. monocytogenes* strains were sent to the World Health Organization Collaborating Center in Paris and to the Swiss National Reference Center in Lausanne. The isolates from the environment, food, and hospitalized patients were further characterized by DNA macrorestriction analysis, with the use of field-inversion gel electrophoresis,[18] contour-clamped homogeneous field electrophoresis, and analyses of randomly amplified polymorphic DNA, as described.[19]

Experimental Contamination of Food

To determine the conditions required for the growth of listeria, we performed experimental contamination of the specific foods that were implicated in the epidemiologic investigation (tuna and corn), using SCOTT A strain (kindly provided by M.P. Doyle, Center for Food Safety and Quality Enhancement, University of Georgia, Griffin) as the control. Specifically, sterile specimens of tuna and corn were separately inoculated with the *L. monocytogenes* isolates obtained from patients and from the environmental swabs (approximately 10 microorganisms per gram of product). The experimentally contaminated foods were incubated at 8°, 15°, and 25°C for 48 hours; these temperatures corresponded, respectively, to the storage temperature recommended for perishables, the temperature recognized to be hazardous for refrigerated foods,[20] and the environmental temperature in the area on May 20, as reported by the National Meteorological Service. At preset intervals (0, 5, 10, and 24 hours), aliquots of the contaminated food samples were taken for counting of listeria by the surface-spread method (culture medium, tryptone soy agar and 0.6 percent yeast extract [TSAYE, Oxoid]).

Statistical Analysis

The data collected were entered into an ad hoc data base and analyzed with the use of Epi Info (version 6.04b) software. The statistical significance of differences between proportions was assessed by means of a chi-square test, and food-specific relative risks were computed; 95 percent confidence intervals were calculated. All reported P values are two-sided.

RESULTS

Epidemiologic Investigation

Of the 2930 children and adults who were exposed to the implicated foods, 2217 (76 percent) were traced and interviewed: 2053 primary-school students, 136 primary-school staff members, and 28 university students. Interviews were conducted with 82 percent of the primary-school students and staff members who were exposed. Because most of the

university students could not be traced or declined to be interviewed, we succeeded in interviewing only 10 percent of them and thus decided to exclude cases in university students from further investigation and analysis. No other cases outside this outbreak were reported in the same period.

Of the 2189 primary-school students and staff members interviewed, 1566 reported one or more symptoms (93 adults and 1473 students), for an attack rate of 72 percent. The mean age of symptomatic persons was 8.4 years for children and 42.2 years for school staff. The most frequently reported symptoms were headache (88 percent of adults and 86 percent of children), abdominal pain (72 percent in both groups), and fever (68 percent of adults and 86 percent of children) (Table 1). Fever and vomiting were reported significantly more frequently in children than in adults (relative risk of fever, 1.3; 95 percent confidence interval, 1.1 to 1.5; and relative risk of vomiting, 1.8; 95 percent confidence interval, 1.2 to 2.7). Diarrhea and pain in the joints and muscles were significantly more frequent among adults (Table 1). The median amount of time that elapsed between the consumption of the meal and the onset of symptoms was 24 hours (range, 6 to 51), with no significant difference between children and adults.

A total of 292 persons (19 percent of those reporting symptoms) were admitted to the hospital (median duration of hospital stay, three days); all were children. None had a diagnosis of sepsis, and there were no deaths. The frequency distribution of symptoms among hospitalized patients did not differ significantly from that among nonhospitalized patients. According to the medical records of the hospitalized patients, diarrhea and fever lasted a median of three days (range, one to seven). None of the hospitalized patients had positive results for blood in loose stools.

The food-specific attack rates for the two schools are shown in Table 2. Consumption of corn-and-tuna salad at the school cafeterias was associated with a significantly increased risk of disease (relative risk, 6.19; 95 percent confidence interval, 4.81 to 7.98; $P<0.001$). On May 20, the food-processing plant had prepared 2750 portions of corn-and-tuna salad and 200 portions of corn salad (served only at the university), using 57 cans of corn packed in water and 18 cans of tuna packed in olive oil. According to the caterer, the cans of corn and tuna had been opened in the early morning, and the contents had been left to drain on separate trays. The corn and tuna were then mixed without the addition of any dressing or spices. Individual portions of corn-and-tuna salad (approximately 80 to 100 g each) were

TABLE 1. SYMPTOMS REPORTED IN ADULTS AND CHILDREN AFTER EXPOSURE.

SYMPTOM	ADULTS (N=93)	CHILDREN (N=1473)
	no. (%)	
Body temperature		
≥38°C	53 (57.0)	1124 (76.3)*
<38°C	2 (2.2)	47 (3.2)
Fever (unspecified)	8 (8.6)	94 (6.4)
Headache	82 (88.2)	1267 (86.0)
Abdominal pain	67 (72.0)	1061 (72.0)
Nausea	56 (60.2)	791 (53.7)
Vomiting	18 (19.4)	586 (39.8)*
Diarrhea (3 times/day)	49 (52.7)	586 (39.8)*
Joint pain	42 (45.2)	348 (23.6)*
Sore throat	11 (11.8)	190 (12.9)
Muscular pain	47 (50.5)	300 (20.4)*
Sleepiness	58 (62.4)	934 (63.4)

*The percentages of adults and children differed significantly ($P<0.001$).

TABLE 2. FOOD-SPECIFIC ATTACK RATES AND RELATIVE RISKS AMONG PRIMARY-SCHOOL STUDENTS AND STAFF.

FOOD	PERSONS WHO ATE THE FOOD		PERSONS WHO DID NOT EAT THE FOOD		RR (95% CI)*	P VALUE
	ATTACK RATE	NO. ILL/TOTAL NO.	ATTACK RATE	NO. ILL/TOTAL NO.		
	%		%			
Pasta with olive oil	71.4	1041/1458	71.8	525/731	0.99 (0.94–1.05)	0.84
Parmesan cheese	71.9	1025/1426	70.9	541/763	1.01 (0.96–1.07)	0.63
Salad of corn and tuna	83.9	1514/1805	13.5	52/384	6.19 (4.81–7.98)	<0.001
Carrots	71.1	994/1399	72.4	572/790	0.98 (0.93–1.04)	0.50
Medlars	71.3	978/1372	72.0	588/817	0.99 (0.94–1.05)	0.73
Bread	72.2	1135/1571	69.7	431/618	1.04 (0.98–1.10)	0.24
Tap water	72.6	954/1314	69.9	612/875	1.04 (0.98–1.10)	0.13

*RR denotes relative risk, and CI confidence interval.

placed in small plastic trays and covered with plastic film. The trays were placed in polystyrene boxes, transported to the schools, and served for lunch. The preparation of the corn salad served at the university cafeteria followed the procedures used for the corn-and-tuna salad, but the corn salad was prepared later in the day and served at dinnertime. None of the food handlers at the plant reported symptoms.

Microbiologic Investigation

All but 2 of the 292 stool samples from the hospitalized patients and all of the blood samples were found to be negative for common enteropathogens; *Salmonella arizonae* and *S. muenchen* were each isolated from 1 stool sample. One of the 40 blood cultures performed was positive for *L. monocytogenes.* Of the 141 stool samples subsequently tested for *L. monocytogenes,* 123 (87 percent) were found to be positive. Of the 12 cultures of stool samples from food handlers, 1 was positive for *L. monocytogenes.* Of the 45 environmental specimens collected at the catering plant, 3 were positive for *L. monocytogenes* (1 from the sink drain where vegetables were handled, 1 from the sink drains where utensils were washed, and 1 from the work surface where meals were prepared).

The specimens taken from unused sealed cans of corn and tuna still stored at the plant were found to be sterile. The tests for mycotoxins were negative. The laboratory sample of corn-and-tuna salad yielded a high bacterial load of *L. monocytogenes* (more than 10^6 colony-forming units [CFU] per gram), but none of the other pathogens or mycotoxins for which we tested were detected. Other microbial tests were conducted; the mesophilic aerobic plate count, Enterobacteriaceae count, and sulfite-reducing clostridium count yielded bacterial loads of more than 10^6 CFU per gram, 6×10^3 CFU per gram, and less than 100 CFU per gram, respectively.

The strains isolated from hospitalized patients and from food handlers, the strain isolated from the food, and the strains isolated from environmental specimens all belonged to serogroup type 4b, which is not phage typable. All strains were positive in tests for pathogenicity in immunodeficient mice. On comparison of DNA, the strains from humans, food, and the environment were indistinguishable (Fig. 1): the restriction enzymes *Sma*I, *Sal*I, *Apa*I, *Asc*I, *Not*I, and random amplified polymorphic DNA yielded identical DNA profiles.

The inoculation experiment showed that the corn kernels supported the growth of *L. monocytogenes* if kept at 25°C. After 10 hours at this temperature, the bacterial load was remarkably high (more than 10^6 CFU per gram). At the other temperatures tested (8°C and 15°C), no growth of the outbreak strain was observed over a period of seven days. The growth curve for listeria in experimentally contaminated corn (pH, 6.3; sodium chloride, 1.5 percent) correlated well with the growth curve predicted with the use of modeling software (Pathogen Modeling Program, version 5.1, U.S. Department of Agriculture, Philadelphia).[21]

DISCUSSION

We investigated a large outbreak of noninvasive gastroenteritis and fever caused by *L. monocytogenes.* The etiologic role of *L. monocytogenes* was demonstrated by both microbiologic and epidemiologic findings. Although not all the clinical specimens were

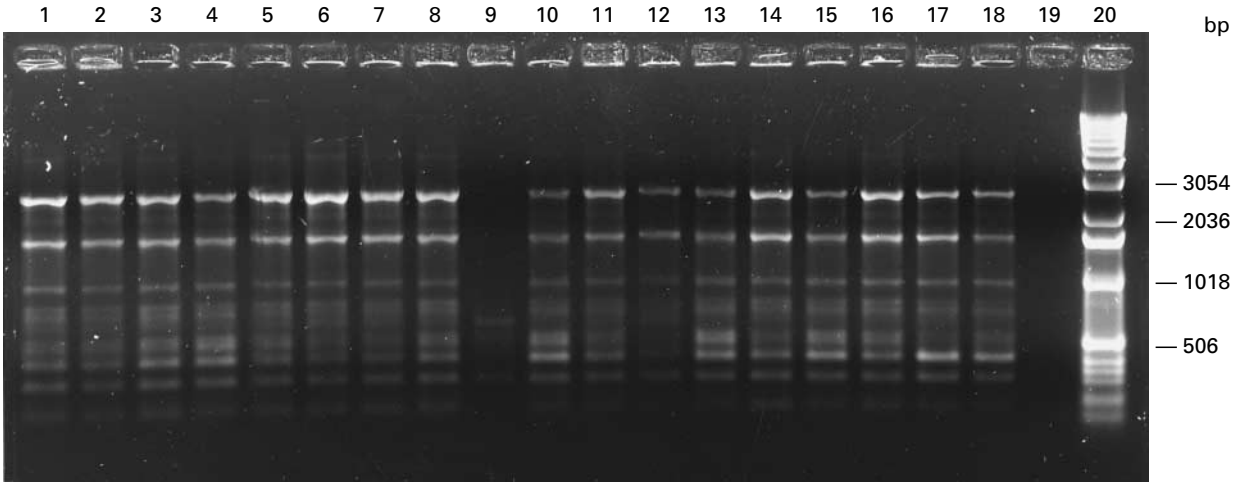

Figure 1. Profiles of Random Amplified Polymorphic DNA of *Listeria monocytogenes* Strains with Primer OPM-01.

The strains were isolated from sweet corn (lane 1), a sink drain (lane 2), a floor drain (lane 3), blood (lane 4), and stool (lanes 5 through 8 and 10 through 18). Lanes 9 and 19 show DNA from negative controls. Lane 20 shows a 1-kb DNA ladder (GIBCO BRL, Madison, Wis.).

tested for listeria, the only pathogen detected in the clinical specimens and in the epidemiologically involved food was listeria serogroup 4b, which was not phage typable. The three tests for molecular subtyping that were used to analyze the isolated strains showed that all the cases of noninvasive disease and the one case of invasive disease (in which the blood was positive for listeria) were caused by the same clone. Because no other microbiologic or toxic causative agent was identified and because, as far as we were capable of determining, the strains isolated from food, the environment, and hospitalized patients were identical, all the cases in the outbreak can be attributed to the contamination of sterile canned corn kernels with listeria.

The experimental contamination showed that sterile canned corn kernels sustained the growth of bacteria from clinical isolates until a high load was reached after 10 hours at room temperature. Therefore, given the high ambient temperature, cross-contamination of the corn-and-tuna salad from other untreated foods through use of the same utensils could easily have occurred if the salad had been prepared and left out at room temperature many hours before it was served, if the time taken to prepare the salad was longer than usual, or both. It is possible that cross-contamination occurred, because listeria is able to persist in specific areas (usually cold, damp areas in food-processing plants) by adhering to surfaces and forming biofilms that are resistant to biocidal treatment.[22,23] Furthermore, the likelihood of cross-contamination may be increased by food handlers' perception that the risk associated with sterile foods is low. In our study, the role of the infected food handler could not be determined, because of the absence of reported symptoms, but we believe that his infection was due to the fact that he had eaten the implicated food and that he was not the source of the infection.

Our investigation confirms that *L. monocytogenes* requires only a brief incubation period (24 hours) to cause a disease with gastrointestinal symptoms and fever. Salamina et al.[11] and Dalton et al.[12] reported shorter incubation periods (18 hours and 20 hours, respectively), whereas Miettinen et al.[14] reported a longer period (28 hours). However, differences in the duration of incubation and in the proportion of cases that are invasive may depend on the specific dose or strain, or they may reflect some unknown individual variation in susceptibility to the microorganism.[24] Nonetheless, the consistency of symptoms reported in this outbreak, together with the high attack rate, suggests that the characteristics of the specific strain have an important role in the clinical picture.

Our results emphasize that food-borne listeriosis in immunocompetent persons is not uncommon and that listeriosis should be considered in the etiologic diagnosis of fever and gastrointestinal disease. To increase the likelihood that listeria that is present will be detected, cold enrichment (a technique to enhance the growth of psychrophilic bacteria selectively) can be used during the processing of stool samples. In many parts of the world, the official sources of information probably underestimate the true burden of disease attributable to listeria. For example, in the United States, only 1000 to 2000 cases are reported each year,[25] and in Italy, an average of 32 cases per year (0.6 case per 1 million inhabitants) were reported from 1991 to 1997.[26]

The outbreak that we studied had a serious effect in terms of health costs, as a result of the high number of hospital admissions and the large number of assays carried out. The outbreak also had an important effect in terms of public concern, which was enhanced by the young age of most of the patients. Nonetheless, this burden could have been avoided. Containing the risks associated with the contamination of foods with listeria or other alimentary pathogens is of crucial importance for safety in food preparation, especially for food prepared in large catering operations and for ready-to-eat foods. Food-processing plants have tackled the problem by adopting the Hazard Analysis and Critical Control Point scheme.[27]

The present investigation confirms that procedures aimed at containing contamination with listeria can prevent not only cases of invasive diseases in immunocompromised patients but also large outbreaks of gastrointestinal febrile illness in immunocompetent persons.

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