An International Outbreak of *Salmonella* Infections Caused by Alfalfa Sprouts Grown from Contaminated Seeds

Barbara E. Mahon,* Antti Pönkä, William N. Hall, Kenneth Komatsu, Stephen E. Dietrich, Anja Siitonen, Gary Cage, Peggy S. Hayes, Mary Ann Lambert-Fair, Nancy H. Bean, Patricia M. Griffin, and Laurence Slutsker

Branches of Foodborne and Diarrheal Diseases and of Biostatistics and Information Management, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia; Helsinki City Centre of the Environment and Laboratory of Enteric Pathogens, National Public Health Institute, Helsinki, Finland; Sections of Disease Surveillance and of Virology, Michigan Department of Public Health, Lansing; Sections of Infectious Disease Epidemiology and of Clinical and Reference Microbiology, Arizona Department of Health Services, Phoenix

An outbreak of *Salmonella* serotype *stanley* infections occurred in the United States and Finland in 1995. The outbreak was investigated through case-control studies in Arizona, Michigan, and Finland; by isolate subtyping; and by tracing and culturing of the implicated food. Alfalfa sprout consumption was the only exposure associated with *S. stanley* infections in Arizona (matched odds ratio [MOR] = 11.1; 95% confidence interval [CI], 1.4–513), Michigan (MOR = 5.5; CI, 1.6–23), and Finland (MOR undefined; CI, 4.9–∞). US and Finnish patient isolates were a unique outbreak strain distinct from *S. stanley* isolates not linked to the outbreak. Alfalfa sprouts eaten by patients in 6 US states and Finland were traced to seed shipped by a Dutch shipper. Thus, it was concluded that alfalfa sprouts grown from contaminated seed caused an international outbreak of ⩾242 *S. stanley* infections in ⩾17 US states and Finland. This outbreak illustrates a new mechanism through which contamination of fresh produce can cause large, widely dispersed outbreaks.

Fresh produce is an increasingly important cause of outbreaks of foodborne diseases, including salmonellosis [1–3], shigellosis [4, 5], and others [6]. These outbreaks have usually been linked to fecal contamination of fruits and vegetables during postharvest handling, shipping, or processing in circumstances that permitted bacterial multiplication. We report an outbreak of salmonellosis traced to alfalfa sprouts in which the source was organisms present on seeds used to grow the sprouts. This is the first such outbreak recognized in the United States, and it highlights the need for new measures to assure that sprouts and other fresh produce are safe to eat.

In May 1995, a new outbreak detection algorithm was implemented at the Centers for Disease Control and Prevention (CDC) to analyze routine laboratory-based *Salmonella* surveillance data submitted electronically through the Public Health Laboratory Information System (PHLIS) by state health departments [7]. This algorithm compares the actual number of reports of each *Salmonella* serotype each week with the number expected on the basis of historical data. Early the next month, the algorithm flagged a nationwide increase in reports of *Salmonella* serotype *stanley* isolates. The largest increases were reported in Arizona and Michigan. Federal health officials then queried Salm-Net, a network for communicating information about *Salmonella* among European health officials [8] regarding *S. stanley* outbreaks in European countries. Salm-Net officials reported that an outbreak of *S. stanley* infections had begun in Finland in March, and many of those infected reported eating alfalfa sprouts before they became ill. A fax and electronic mail message was then sent from CDC to state health officials describing the US outbreak and the possible link to alfalfa sprout consumption in Finland.

We report the epidemiologic, environmental, and laboratory investigations that led to the identification of alfalfa sprouts grown from contaminated seed as the vehicle of this international outbreak.

Methods

Epidemiologic studies. Independent studies were done in Arizona, Michigan, and Finland to investigate exposures associated with *S. stanley* infection. Cases of *S. stanley* infections were identified in these sites through routine serotyping of *Salmonella* isolates submitted from clinical laboratories to the laboratories of the Arizona Department of Health Services (ADHS), the Michigan Department of Public Health (MDPH), and Finland's National Public Health Institute.

Arizona. During 16–19 June, a case-control study was conducted using a questionnaire designed to test hypotheses developed during in-depth interviews with 4 patients. A case was defined as an illness in a person who was in Arizona during the 3 days before

Received 3 May 1996; revised 16 October 1996.

Presented in part: 45th annual Epidemic Intelligence Service Conference, 22–26 April 1996, Centers for Disease Control and Prevention, Atlanta.

Reprints or correspondence: Dr. Laurence Slutsker, Foodborne and Diarrheal Diseases Branch, Mailstop A-38, Centers for Disease Control and Prevention, 1600 Clifton Rd., Atlanta, GA 30333.

* Present affiliation: Department of Pediatrics, UMDNJ–Robert Wood Johnson Medical School, New Brunswick, New Jersey.

The Journal of Infectious Diseases 1997; 175:876–82

0022–1899/97/7504–0020$01.00

onset of illness in which a clinical specimen yielded *S. stanley*. Two controls per case were matched by age (the match range increased with age from ±1 year for cases ≤5 years old to ±20 years for cases >60 years old) and neighborhood by systematically adding to or subtracting from the case-patient's telephone number until controls of appropriate ages were obtained. Controls were eligible if they had not had symptoms of gastroenteritis since 25 April. The questionnaire included items about medical history and 24 specific food and environmental exposures during the 3 days before illness onset (case-patients) or during the most recent 3 days that were the same days of the week as the days of interest for the matched case (controls).

Michigan. A case-control study was conducted 12–19 June using a questionnaire with questions regarding 42 specific food and environmental exposures. The study design and questionnaire were not discussed with the Arizona investigators. A case was defined as the first illness in a household in which an isolate submitted to the MDPH laboratory after 1 May was identified as *S. stanley*. Two controls per case were identified by asking case-patients to name 2 well-persons of about their age who lived in their neighborhood. Case-patients were interviewed about exposures during the 7 days before the onset of illness; matched controls were interviewed about exposure during the same 7 days. The first 30 cases were included in the case-control study.

Finland. A case-control study was initiated in May 1995. A case-patient was defined as a person whose address was in Helsinki or Espoo (the capital region), who had domestically acquired illness, and from whom a *Salmonella* isolate submitted to the National Public Health Institute in 1995 was identified as *S. stanley*. Case-patients were mailed a questionnaire with questions regarding 10 specific food exposures in the 5 days before illness onset. One control per case also completed the questionnaire; controls were well family members of case-patients, when available, and well public health inspectors otherwise.

US surveillance. After consumption of alfalfa sprouts was implicated by the studies in Arizona and Michigan, CDC investigators asked health officials in other states in which *S. stanley* infections were reported in 1995 to interview persons about whether they had eaten alfalfa sprouts in the 5 days before illness onset and to send up to 5 1995 isolates to the CDC for subtyping with information on the subject's history of eating alfalfa sprouts.

Statistical methods. Maximum likelihood estimates of matched odds ratios with exact 95% confidence intervals are reported as measures of association.

Laboratory methods. *S. stanley* isolates submitted to the CDC were analyzed by pulsed-field gel electrophoresis (PFGE) and by antimicrobial resistance testing. Isolates were analyzed from the outbreaks in Arizona, Michigan, and Finland, from cases occurring in other states at the time of the outbreak, and, for comparison, from the CDC reference collection of *S. stanley* isolates (obtained in 1984–1995). Chromosomal DNA was prepared in agarose plugs and restricted with *Xba*I as previously described [9], except that the cells were grown for 16 h in trypticase soy broth. Restricted DNA fragments were separated by electrophoresis (CHEF DR II system; Bio-Rad Laboratories, Richmond, CA) [9]. Isolates with PFGE patterns differing from the outbreak strain by no more than 1 band were considered to be the outbreak strain. PFGE was done by similar methods in Arizona (Clinical and Reference Microbiology Laboratory, ADHS), Michigan (Molecular Biology Section, Virology Division, MDPH), and Finland (Laboratory of Enteric Pathogens, National Public Health Institute) on additional outbreak-related isolates not submitted to the CDC.

Antimicrobial resistance testing was by the disk diffusion technique with zone size criteria standardized for Enterobacteriaceae [10]. A standard panel of antimicrobial agents was used and included chloramphenicol, trimethoprim-sulfamethoxazole, tetracycline, ampicillin, sulfisoxazole, streptomycin, ciprofloxacin, nalidixic acid, gentamicin, ceftriaxone, amoxicillin-clavulanic acid, and kanamycin.

Implicated alfalfa seed and sprouts grown from that seed were cultured for salmonellae. Sprouts were grown from seed by twice-daily spraying with sterile tap water. Sampling was done in triplicate. In addition to the methods described [11], seeds and sprouts were blended with 0.1% Tween 80 and preenriched in universal buffered peptone.

Trace-back methods. State health departments conducted interviews to determine the source(s) of sprouts eaten by case-patients. Case-patients identified retail outlets (restaurants, markets) and dates of purchase. Retail outlets identified shippers and growers and delivery dates. Growers identified seed suppliers, lot numbers of alfalfa seed, and dates of sprouting. The origin of implicated seed was determined by reviewing seed supplier invoices and delivery records. A successful trace back was defined as one in which a patient ate sprouts from only one retail outlet during the 5 days before illness onset, so that the grower and seed supplier(s) could be identified. A patient's trace-back result was defined as ''definite'' when seed from a single supplier or lot was clearly documented to be the only seed from which the sprouts eaten by that patient could have been grown. The result was defined as ''probable'' when a single supplier or lot was very likely but could not be definitively documented and as ''possible'' when it could not be determined which of two or more possible seed suppliers or lots was most likely.

Results

Clinical and epidemiologic studies. Illness peaked during May and June in the United States as a whole as well as in Arizona, Michigan, and Finland (figure 1).

Arizona. The ADHS received 22 reports of *S. stanley* infections in 1995 through 16 June; all occurred in April and May. By comparison, 1 *S. stanley* infection each year was reported during 1993 and 1994. Clinical and demographic information is summarized in table 1. Nineteen case-control sets were interviewed (1 case occurred outside Arizona, and 2 case-patients could not be reached). Eating alfalfa sprouts was significantly associated with illness; 39% of case-patients who could recall, reported eating alfalfa sprouts in the 3 days of interest, compared with 8% of controls (table 2). Neither gender nor any other exposure was associated with illness.

Michigan. The MDPH received 51 *S. stanley* isolates during May, June, and July 1995, a marked increase from the 2 and 6 infections reported during the same 3 months in 1993 and 1994. Forty-eight of the 51 case-patients were interviewed (2 were the second case in a household, 1 re-

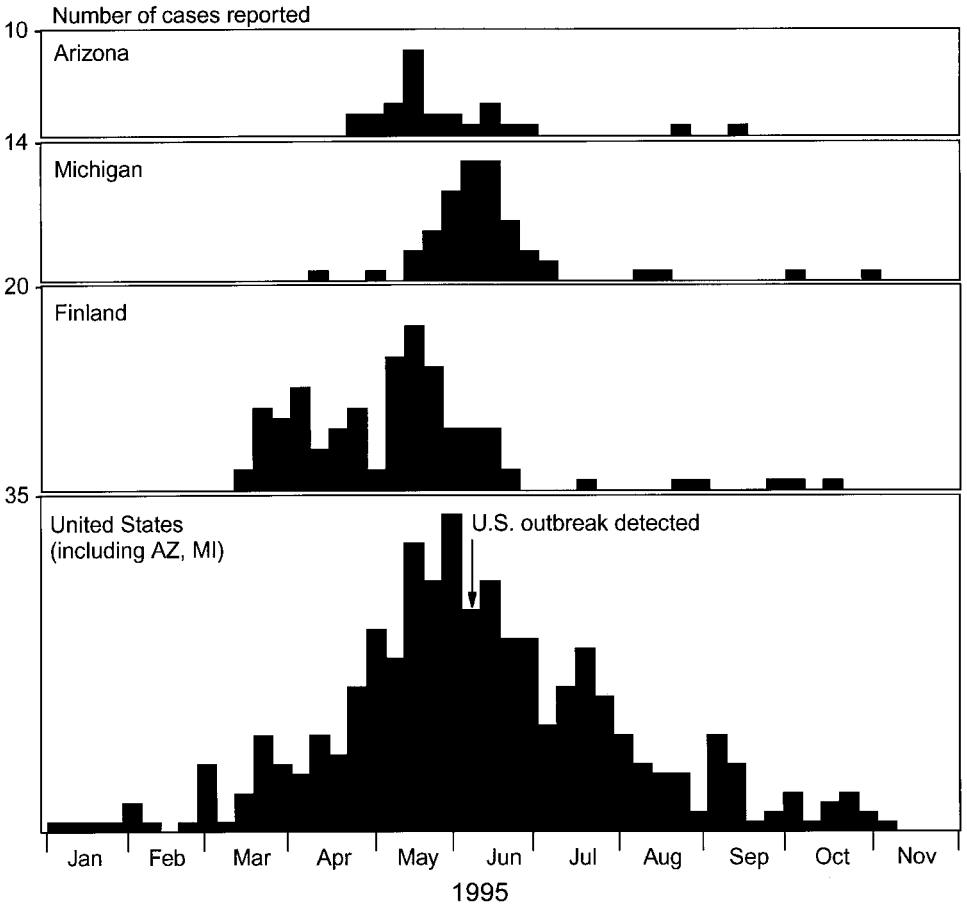

Figure 1. Week of illness onset (Arizona, Michigan, United States) or stool sampling (Finland) of patients with *Salmonella stanley* infections, 1 January through 30 November 1995.

fused). The median age difference between cases and matched controls was 2 years (range, 0–22). Twenty-nine case-control sets were interviewed (1 refused). As in Arizona, eating alfalfa sprouts was the only exposure significantly associated with illness; 41% of case-patients and 10% of controls reported exposure (table 2).

Finland. From March through November 1995, 114 domestically acquired *S. stanley* infections were reported in Finland; in 1993 and 1994, none were reported. Demographic data for the cases were similar to that in Arizona and Michigan (table 1). Questionnaires were mailed to the 36 case-patients who lived in the study area; 25 (69%) returned the questionnaire. The median age difference between cases and matched controls was 14 years (range, 1–46). Eating alfalfa sprouts during the 5 days of interest was the only exposure significantly associated with illness; 100% of case-patients and 20% of controls reported this exposure (table 2).

All United States. From March through July 1995, 349 *S. stanley* isolates were reported to the CDC (figure 1). By comparison, during the same 5 months in 1993 and 1994, 60 and 92 *S. stanley* infections were reported, respectively. An excess of *S. stanley* infections in 1995 through 30 November was reported among young adults compared with the age distribution of persons infected with all other *Salmonella* serotypes (figure 2). Among the *S. stanley* isolates, 60% were from females compared with 51% for all other *Salmonella* serotypes. In 16 states other than Arizona and Michigan, 95 persons were interviewed; 32 (34%) in 11 states said they had eaten alfalfa sprouts in the 5 days before illness onset, 46 (48%) said they had not, and 17 (18%) were not sure.

Laboratory results. At the CDC, *S. stanley* isolates collected during the outbreak period from Arizona ($n = 22$), Michigan ($n = 3$), Finland ($n = 4$), and 16 other states ($n = 75$) were compared with 38 *S. stanley* isolates from the CDC reference collection. The same PFGE pattern (figure 3) and unique antibiogram (resistance to trimethoprim-sulfamethoxazole, tetracycline, sulfisoxazole, streptomycin, and kanamycin) were found in 20 of the 22 Arizona isolates and in all isolates from Michigan and Finland. One Arizona isolate that did not match the outbreak strain antibiogram differed only by lacking kanamycin resistance; it had the outbreak PFGE pattern. In contrast, the 38 reference isolates had 25 different PFGE patterns and 7 different antibiograms. Only 1 reference isolate had the outbreak antibiogram and PFGE pattern; it was collected in May 1995, during the outbreak period. Among the 75 isolates submitted by states other than Arizona and Michigan, 56 (75%)

Table 1. Demographic characteristics and symptoms reported by persons with *Salmonella stanley* infections in Arizona, Michigan, and Finland, 1995.

Characteristic	Arizona (n = 19)*	Michigan (n = 48)†	Finland (n = 114)‡
Median age, years (range)	25 (<1–81)	29 (2–79)	29 (0–93)
% female	58	60	64
Reported signs and symptoms, %			
Diarrhea	100	100	80§
Abdominal cramps	100	85	68§
Subjective fever	84	87	80§
Nausea	74	55	68§
Bloody stool	37	47	20§
Vomiting	32	36	28§
Hospitalized	26	33	20§
Median duration, days (range)	7 (3–49)	8 (4–21)	10 (0–30)§

* Includes only patients enrolled in case-control study.

† Includes all patients with isolates submitted to Michigan Department of Public Health laboratory from 1 May to 31 July who had first case in their household and who agreed to be interviewed.

‡ Includes all patients with domestically acquired isolates reported to National Public Health Institute from 1 March to 31 October 1995.

§ Includes 25 patients in case-control study only.

from 15 states were identical to the outbreak strain. These 56 isolates were examined either by PFGE only (n = 5), antibiogram only (n = 45), or both methods (n = 6).

Results at other laboratories supported the CDC findings. In Arizona, the 22 isolates had the same PFGE results as at CDC. In Michigan, all 46 outbreak-related isolates and 4 of 5 Arizona isolates had identical *Xba*I PFGE patterns (n = 47) or had minor one-band differences (n = 3). In Finland, all 6 isolates tested from Finnish patients and 4 of 4 US outbreak isolates had identical *Xba*I PFGE patterns.

Isolates from 53 patients were submitted to the CDC by 8 states (Arizona, California, Illinois, Indiana, Michigan, Ohio, Oklahoma, and Virginia) with information on whether the patient had eaten alfalfa sprouts in the 5 days before illness onset. Among the 47 patients whose isolates were the outbreak strain, 20 (43%) reported eating alfalfa sprouts, whereas, of the 6 patients whose isolates were not the outbreak strain, none reported eating alfalfa sprouts.

Less than 5 kg of ⩾20,000 kg of alfalfa seed implicated in the US trace-back investigation was available for culture. A sample of these seeds and sprouts grown from them did not yield *S. stanley*.

Trace-back investigation. Trace backs were successful for 50 patients in 6 states (Arizona, Colorado, Illinois, Michigan, Ohio, and Pennsylvania). These patients had eaten alfalfa sprouts grown by ⩾9 growers. All growers had obtained alfalfa seed from a single US supplier whose market share is estimated to be 60%–70%. All of the patients had either definitely (n = 48) or possibly (n = 2) eaten alfalfa sprouts grown from seed from this supplier. This supplier bought the seed from other sources; 96% of the US patients had either definitely (n = 27), probably (n = 19), or possibly (n = 2) eaten sprouts grown from seed that the US supplier bought from a shipper in the Netherlands. The Netherlands shipper supplied 46% of the alfalfa seed that the US supplier delivered to US growers in March and April, when most implicated seed was delivered. The Netherlands shipper also shipped the alfalfa seed, via another supplier, that was grown in Finland and eaten by the Finnish patients. The Netherlands shipper reportedly mixed the seed sent to the United States from lots bought in Italy, Hungary, or Pakistan, but this information could not be confirmed, and the ultimate origin and harvest date of the seed was not determined. Inspection of the seed shipper's warehouse by Netherlands health authorities ~6 months after the implicated seed was shipped did not reveal any specific problems.

Discussion

We conclude that alfalfa sprouts grown from contaminated seed were the vehicle of an international outbreak of *S. stanley* infections. The following lines of evidence support this conclusion.

First, three independently designed and conducted case-control studies in Arizona, Michigan, and Finland found that consumption of alfalfa sprouts was the only exposure associated

Table 2. Study design and association of reported consumption of alfalfa sprouts with *Salmonella stanley* infection in matched case-control studies in Arizona, Michigan, and Finland, 1995.

Location	Cases	Controls/case	Patient exposed (%)	Control exposed (%)	MOR*	Confidence bounds
Arizona	19	2	7/18 (39)	3/38 (8)	11.1	1.4–513†
Michigan	29	2	12/29 (41)	6/58 (10)	5.5	1.6–23†
Finland	25	1	25/25 (100)	5/25 (20)	Undefined	4.9‡

NOTE. Case-patients were asked about exposures during 3 days before illness onset in Arizona, 7 days before onset in Michigan, and 5 days before onset in Finland.

* Exact 95% confidence interval for maximum likelihood estimate of matched odds ratio (MOR).

‡ Exact lower 95% confidence bound for maximum likelihood estimate of MOR.

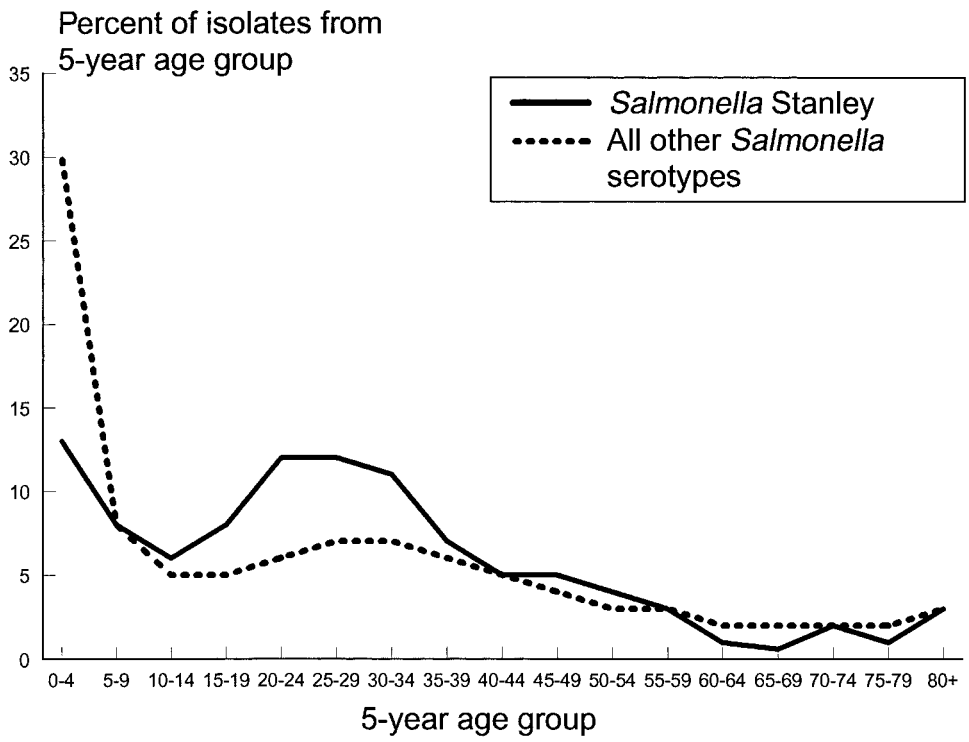

Figure 2. Percentage of isolates of *Salmonella stanley* and of all other *Salmonella* serotypes reported to CDC 1 January through 30 November 1995 within each 5-year age group.

with illness. Second, in 16 other states in which persons with *S. stanley* infections were interviewed, 34% reported eating alfalfa sprouts in the 5 days before illness onset; in contrast, in the most recent National Health and Nutrition Examination Survey (NHANES III), <0.5% of Americans reported eating alfalfa sprouts on a random day (unpublished data, CDC). Since patients were interviewed weeks or months after their illnesses, and since alfalfa sprouts are often served inconspicuously in salads or sandwiches, many more were probably exposed to alfalfa sprouts than could definitely recall eating them. Before the study results were made public, the Arizona case-patients were reinterviewed to ask whether they might have eaten alfalfa sprouts during the 3 days before onset, 75% said they definitely, probably, or possibly had (unpublished data, CDC). The relative excess of *S. stanley* isolates from persons 20 to 40 years old and from women, compared with *Salmonella* isolates as a whole, likely reflects the demographics of persons who eat alfalfa sprouts. Third, *S. stanley* isolates from American and Finnish patients linked epidemiologically to the outbreak had a unique PFGE pattern and antibiogram not present in other *S. stanley* isolates. Finally, the alfalfa sprouts eaten by American and Finnish case-patients were produced by many different growers but were grown from seed from a single common source—a seed shipper in the Netherlands—indicating that the seed was contaminated before it was shipped.

This is the first *Salmonella* outbreak in the United States linked to alfalfa sprouts grown from contaminated seeds. However, alfalfa sprouts are a highly plausible vehicle of *Salmonella* transmission. A 1994 *Salmonella bovismorbificans* outbreak in Finland and Sweden was traced to Australian alfalfa seed [12], and a 1988 outbreak of *Salmonella saint paul* infections in Europe was linked to mung bean sprouts [13, 14]. A small 1990 cluster of *Salmonella anatum* infections in the United States was suspected to be linked to one grower's alfalfa sprouts, but the source of contamination was not determined (unpublished data, CDC). Although it is not known how the seeds implicated in this investigation became contaminated, alfalfa seeds are a raw agricultural product that may come in contact with salmonellae from the feces of birds, rodents, or other animals during growth, harvest, processing, storage, or shipping; *S. stanley* has been isolated from a wide variety of animal species. Salmonellae have been recovered from alfalfa seeds [15] and from alfalfa [12] and bean [16] sprouts. *Salmonella* organisms can survive for months under the dry conditions used for alfalfa seed storage [17]. The presence of even a few salmonellae on seeds may be hazardous, because the sprouting process is a highly effective enrichment step. The numbers of *S. stanley* [18] and other salmonellae [19] increased 3–5 orders of magnitude per gram during sprouting of experimentally inoculated seed. The same phenomenon has been observed for *Bacillus cereus,* which caused an outbreak of food poisoning linked to home-sprouted vegetable sprouts [20, 21].

It is not surprising that cultures of implicated alfalfa seeds and sprouts grown from them did not yield *S. stanley*. Because of their relatively short shelf-life, implicated sprouts were not available for culture. Contamination was probably not uniform

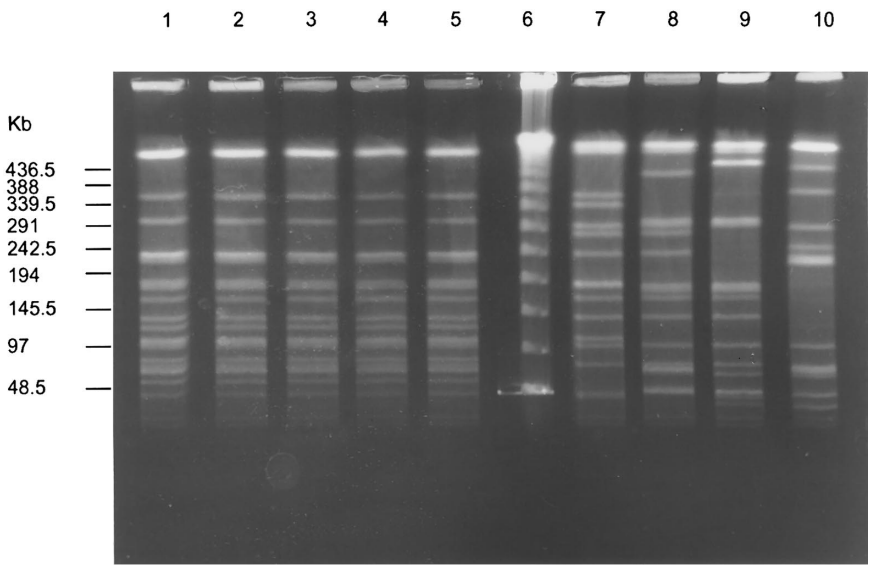

Figure 3. Pulsed-field gel electrophoresis patterns of *Salmonella stanley* isolates. Lanes 1–5, outbreak isolates (1 and 2, Arizona; 3, Michigan; 4, Finland; 5, Ohio). Lanes 7–10, sporadic isolates not linked to outbreak (7, Arizona; 8, Missouri; 9, Finland; 10, Virginia). Lane 6, λ ladder molecular weight marker.

in the lots of seed from which the implicated sprouts were grown, and, since a single bag of <0.03% of that seed was available for culture, we were unlikely to recover *S. stanley*.

With this outbreak, a new mechanism is added to the growing list of causes of large, widely dispersed, produce-related outbreaks. Previous outbreaks have been linked to cut cantaloupes inoculated by salmonellae on the skin [1, 2], to tomatoes that may have been contaminated with salmonellae when rinsed in contaminated water taken up at the stem scar [2, 3], and to shigellae on shredded lettuce [4]. In those outbreaks, the natural barriers of the produce vehicle were breached by cutting or shredding, allowing bacterial access to the interior of the produce, where rapid growth could occur. In contrast, in this outbreak, the alfalfa sprouts were intact; it is likely that rapid growth of *Salmonella* organisms was fostered by the sprouting process itself. This outbreak suggests that even a few salmonellae present on seed used for sprouting can lead to an extensive outbreak.

In the present outbreak, as in other large produce-related outbreaks [2], cases were widely distributed geographically, and few household clusters were recognized. This pattern likely reflects the broad distribution of a product, in this case alfalfa seed, with low-level, nonuniform contamination. We identified ⩾242 outbreak-related cases (Arizona, 21; Michigan, 51; and Finland, 114, in which the outbreak strain was identified or that were linked epidemiologically; and other US states, 56 with the outbreak strain) from 17 US states and Finland. The actual number of cases was probably 5000–24,000, based on rates of underreporting defined in other *Salmonella* outbreaks [22]. The outbreak was probably even more extensive than we were able to document; *S. stanley* isolates from Canada collected during the outbreak period were indistinguishable from the outbreak strain (unpublished data, CDC); however, there was no information available about alfalfa sprout consumption by Canadian patients.

Preventing similar outbreaks in the future will depend on new measures to reliably decontaminate seeds before sprouting. Recent studies of surface decontamination of alfalfa seed experimentally inoculated with *S. stanley* showed that soaking seeds in chlorine bleach at concentrations of 2000 ppm hypochlorite or in 57–60°C water at the time of germination can greatly reduce bacterial populations without decreasing germination [18]. In a survey of alfalfa sprout growers regarding production practices at the time of the outbreak, 50% reported they used a chlorine soak to decontaminate seeds, but they typically used solutions much more dilute than 2000 ppm (unpublished data, CDC). Further efforts are needed to define acceptable effective decontamination procedures that can be used by sprout growers. The importance of rapid evaluation and implementation of these control measures is highlighted by the fact that, since this investigation, another US *Salmonella* outbreak has been linked to alfalfa sprouts [23].

This outbreak was detected because of routine serotyping of *Salmonella* isolates by state public health laboratories, timely electronic reporting of isolates through PHLIS to CDC, application of a new outbreak detection algorithm to *Salmonella* surveillance data, and national and international communication among public health workers. It illustrates the value of newly developed electronic surveillance and outbreak detection methods in identifying widely dispersed outbreaks that may otherwise be missed and in allowing timely public health action in response to surveillance information. The assistance of SalmNet, the European public health communication network for information about salmonellae, was instrumental. Widely dispersed foodborne outbreaks such as this one are likely to pose an increasing challenge to public health, as food is increasingly

centrally produced and widely distributed [24]. The surveillance and communication tools used in investigating this outbreak will be essential to meet this challenge.

Acknowledgments

We gratefully acknowledge the assistance of Ian Fisher, Salm-Net; Bruce Hoar; Lawrence Sands, Clare Kioski, Karen Cowgill, Brock Marlin, and Donald Reese, ADHS and Maricopa, Pima, and Coconino county health departments, Arizona; Barbara Robinson-Dunn, Linda Reese, Susan Shiflett, and the staff of the Microbiology Section, MDPH, Andrew Al-Shab, Jaime V. Altamirano, Robert M. Barrie, Marcia K. Brooks, Garold A. Goza, James C. Hospedales, James B. Kent, Norman B. Keon, Cynthia M. Kramer, Mark A. Miller, Mary Grace Stobierski, Judith A. Weber, and Jeffrey J. Zoellner, Disease Control Division, MDPH, Gerald Wojtala and the field staff of the Food Division, Michigan Department of Agriculture, and the communicable disease control programs of the local Michigan health departments; Liisa Immonen, Tarja Heiskanen, and Ritvaleena Puohiniemi, Laboratory of Enteric Pathogens, and Sirpa Passoja, Automatic Data Processing Office, National Public Health Institute, Finland; Benson Werner, California Department of Health Services; Pamela Shillam, Colorado Department of Health; Carl Langkop and Constance Austin, Department of Public Health, and Keith Pigg, Division of Food, Drugs and Dairies, Illinois; Alan Oglesby, Indiana State Department of Health; Rebecca Olson, New Mexico Department of Health; Ellen Peterson Salehi, Ohio Department of Health; Tim Graves, Oklahoma State Department of Health; Marshall Deasy, Pennsylvania Department of Health; Suzanne Jenkins and Mary Jean Lin, Virginia State Health Department; John Spika and Wendy Johnson, Laboratory Centre for Disease Control, Canada; Martien Borgdorff, Department of Infectious Diseases Epidemiology, Netherlands; Cynthia Ogden, National Center for Health Statistics; Cincinnati office, Food and Drug Administration; Kathleen Maloney, Biostatistics and Information Management Branch, and Lewis Graves, Joy Wells, Timothy Barrett, Bala Swaminathan, and Robert Tauxe, Foodborne and Diarrheal Diseases Branch, CDC; and the International Sprout Growers' Association and others in the alfalfa sprout industry.

References

1. Centers for Disease Control. Multistate outbreak of *Salmonella poona* infections: United States and Canada, 1991. MMWR **1991**;40:549–51.
2. Hedberg CW, MacDonald KL, Osterholm MT. Changing epidemiology of food-borne disease: a Minnesota perspective. Clin Infect Dis **1994**; 18:671–82.
3. Zhuang RY, Beuchat LR, Angulo FJ. Fate of *Salmonella montevideo* on and in raw tomatoes as affected by temperature and treatment with chlorine. Appl Environ Microbiol **1995**;61:2127–31.
4. Davis H, Taylor JP, Perdue JN, et al. A shigellosis outbreak traced to commercially distributed shredded lettuce. Am J Epidemiol **1988**;128: 1312–21.
5. Martin DL, Gustafson TL, Pelosi JW, Suarez L, Pierce GV. Contaminated produce—a common source for two outbreaks of *Shigella* gastroenteritis. Am J Epidemiol **1986**;124:299–305.
6. Rosenblum LS, Mirkin IR, Allen DT, Safford S, Hadler SC. A multifocal outbreak of hepatitis A traced to commercially distributed lettuce. Am J Public Health **1990**;80:1075–9.
7. Martin SM, Bean NH. Data management issues for emerging diseases and new tools for managing surveillance and laboratory data. Emerging Infect Dis **1995**;1:124–8.
8. Fisher IST, Rowe B, Bartlett CLR, Gill ON. ''Salm-Net''—laboratory based surveillance of human salmonella infections in Europe. PHLS Microbiol Dig **1994**;11:181–2.
9. Barrett TJ, Lior H, Green JH, et al. Laboratory investigation of a multistate food-borne outbreak of *Escherichia coli* O157:H7 by using pulsed-field gel electrophoresis and phage typing. J Clin Microbiol **1991**;32: 3013–7.
10. National Committee for Clinical Laboratory Standards. Performance standard for antimicrobial disk susceptibility test: approved standard, PSM-7. Villanova, PA: NCCLS, **1993**.
11. Food and Drug Administration. In: Bacteriological analytic manual. 7th ed. Arlington, VA: Association of Official Analytical Chemists International, **1992**:51–60.
12. Ponka A, Andersson Y, Siitonen A, et al. *Salmonella* in alfalfa sprouts. Lancet **1995**;345:462–3.
13. Andersson Y, de Jong B. *Salmonella* associated with bean sprouts. In: Proceedings of the World Association of Veterinary Food Hygienists, 10th International Symposium, **1989**:319–22.
14. O'Mahony M, Cowden J, Smyth B, et al. An outbreak of *Salmonella saintpaul* infection associated with bean sprouts. Epidemiol Infect **1990**;104: 229–35.
15. Andrews WH, Wilson CR, Poelma PL, Romero A, Mislivec PB. Bacteriological survey of sixty health foods. Appl Environ Microbiology **1979**; 37:559–66.
16. Jerngklinchan J, Saitanu K. The occurrence of salmonellae in bean sprouts in Thailand. Southeast Asian J Trop Med Public Health **1993**;24: 114–8.
17. Mitscherlich E, Marth EH. Microbial survival in the environment. Berlin: Springer-Verlag, **1984**.
18. Jaquette CB, Beuchat LR, Mahon BE. Efficacy of chlorine and heat treatment in killing *Salmonella stanley* inoculated onto alfalfa seeds and growth and survival of the pathogen during sprouting and storage. Appl Environ Microbiol **1996**:62:2212–5.
19. Andrews WH, Mislivec PB, Wilson CR, et al. Microbial hazards associated with bean sprouting. J Assoc Off Anal Chem **1982**;65:241–8.
20. Portnoy BL, Goepfert JM, Harmon SM. An outbreak of *Bacillus cereus* food poisoning resulting from contaminated vegetable sprouts. Am J Epidemiol **1976**;103:589–94.
21. Harmon SM, Kautter DA, Solomon HM. *Bacillus cereus* contamination of seeds and vegetable sprouts grown in a home sprouting kit. J Food Protection **1987**;50:62–5.
22. Chalker RB, Blaser MJ. A review of human salmonellosis. III Magnitude of *Salmonella* infection in the United States. Rev Infect Dis **1988**;10: 111–24.
23. Oregon Health Division. Salmonellosis outbreak traced to alfalfa sprouts—Oregon and BC. Communicable Disease Summary. Albany, OR: OHD, **1996**;45.
24. Tauxe RV. *Salmonella:* a postmodern pathogen. J Food Protection **1991**; 54:563–8.