

A National Outbreak of *Salmonella* Serotype Tennessee Infections From Contaminated Peanut Butter: A New Food Vehicle for Salmonellosis in the United States

Anandi N. Sheth,^{1,2} Mike Hoekstra,¹ Nehal Patel,¹ Gwen Ewald,¹ Cathy Lord,³ Carmen Clarke,⁴ Elizabeth Villamil,⁵ Katherine Niksich,² Cheryl Bopp,¹ Thai-An Nguyen,¹ Donald Zink,⁶ and Michael Lynch¹

¹Division of Foodborne, Bacterial, and Mycotic Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia; ²Epidemic Intelligence Service and Epidemiology Elective Program, Office of Workforce and Career Development, Centers for Disease Control and Prevention, Atlanta, Georgia; ³Iowa State Hygienic Laboratory, Iowa City, Iowa; ⁴Oklahoma State Department of Health, Oklahoma City, Oklahoma; ⁵New York State Department of Health, Albany, New York; and ⁶US Food and Drug Administration, Center for Food Safety and Applied Nutrition, College Park, Maryland

Background. *Salmonella* serotype Tennessee is a rare cause of the estimated 1 million cases of salmonellosis occurring annually in the United States. In January 2007, we began investigating a nationwide increase in *Salmonella* Tennessee infections.

Methods. We defined a case as *Salmonella* Tennessee infection in a patient whose isolate demonstrated 1 of 3 closely related pulsed-field gel electrophoresis patterns and whose illness began during the period 1 August 2006 through 31 July 2007. We conducted a case-control study in 22 states and performed laboratory testing of foods and environmental samples.

Results. We identified 715 cases in 48 states; 37% of isolates were from urine specimens. Illness was associated with consuming peanut butter more than once a week (matched odds ratio [mOR], 3.5 [95% confidence interval {95% CI}, 1.4–9.9]), consuming Brand X peanut butter (mOR, 12.1 [95% CI, 3.6–66.3]), and consuming Brand Y peanut butter (mOR, 9.1 [95% CI, 1.0–433]). Brands X and Y were produced in 1 plant, which ceased production and recalled products on 14 February 2007. Laboratories isolated outbreak strains of *Salmonella* Tennessee from 34 Brands X and Y peanut butter jars and 2 plant environmental samples.

Conclusions. This large, widespread outbreak of salmonellosis is the first linked to peanut butter in the United States; a nationwide recall resulted in outbreak control. Environmental contamination in the peanut butter plant likely caused this outbreak. This outbreak highlights the risk of salmonellosis from heat-processed foods of nonanimal origin previously felt to be low risk for *Salmonella* contamination.

Approximately 1 million infections from >2500 *Salmonella* serotypes occur annually in the United States [1]. Most occur after ingestion of contaminated foods, either foods of animal origin or raw agricultural products. In recent years, however, *Salmonella* outbreaks

have been associated with a variety of food vehicles, including cereal, orange juice, and ice cream, some previously thought to be at low risk for *Salmonella* contamination [2–6].

Salmonella serotype Tennessee infections are rare, and the sources of most of these infections are unknown. An average of 52 cases of *Salmonella* Tennessee infection were reported to the National *Salmonella* Surveillance System annually during 1995–2004, 0.1% of all *Salmonella* strains [7]. The only previously reported *Salmonella* Tennessee outbreak was due to contaminated powdered milk [8]. *Salmonella* usually causes gastroenteritis, but certain serotypes, including *Salmonella* Tennessee, also infect the urinary tract [9].

Received 1 February 2011; accepted 13 May 2011.

Correspondence: Anandi N. Sheth, MD, Centers for Disease Control and Prevention, 1600 Clifton Rd NE, Mailstop A-38, Atlanta, GA 30333 (asheth@cdc.gov).

Clinical Infectious Diseases 2011;53(4):356–362

© The Author 2011. Published by Oxford University Press on behalf of the Infectious Diseases Society of America. All rights reserved. For Permissions, please e-mail: journals.permissions@oup.com.
1058-4838/2011/534-0006\$14.00
DOI: 10.1093/cid/cir407

In November 2006, United States public health officials noted a widespread increase in the number of *Salmonella* Tennessee isolates with matching pulsed-field gel electrophoresis (PFGE) patterns reported to the Centers for Disease Control and Prevention (CDC) PulseNet, the molecular subtyping network for foodborne disease surveillance. By 1 December, 52 isolates with identical PFGE patterns had been reported from 25 states during the preceding 4 months, compared with 55 isolates reported during the preceding 2 years. We conducted a multistate investigation of this increase to identify the source and recommend control measures. Preliminary findings from this investigation were previously reported [10].

METHODS

Case Finding

We defined an outbreak case as infection with *Salmonella* Tennessee with 1 of 3 closely related CDC PulseNet PFGE patterns (*Xba*I patterns JNXX01.0010, JNXX01.0011, and JNXX01.0026) in a person residing in the United States with illness onset from 1 August 2006 to 31 July 2007 (or, if onset date was unknown, with *Salmonella* Tennessee isolated from 1 August 2006 to 31 July 2007). Initial case finding focused on isolates with pattern JNXX01.0011, for which increased reports to PulseNet were noted. The case definition was expanded to include patterns JNXX01.0026 (after some patients were found to have strains with this pattern and JNXX01.0011 in the same specimen) and JNXX01.0010 (after strains with this pattern were isolated from implicated foods). We requested state public health laboratories to perform PFGE analysis on *Salmonella* Tennessee isolates received during this time period, and we queried the PulseNet database for isolates with matching PFGE patterns. To ascertain cases internationally, alerts were issued through the World Health Organization's International Food Safety Authorities Network, the Global *Salmonella* Surveillance Program, and PulseNet International.

In January 2007, to develop hypotheses regarding possible food vehicles, public health officials in 13 states interviewed 31 patients with an outbreak strain and onset of gastrointestinal symptoms during the preceding month using a standardized survey instrument of ~200 food items. Case exposures were compared with food consumption data from the Foodborne Diseases Active Surveillance Network (FoodNet) population survey, a population-based survey of respondents in 10 FoodNet sites, collected from May through August 2006 [11]. A single interviewer then conducted detailed interviews of 6 patients to obtain additional product information.

Case-Control Study

In February 2007, we conducted a case-control study to identify the food vehicle. For this study, a case was defined as infection

with *Salmonella* Tennessee (pattern JNXX01.0011) in a person ≥ 18 years old with a history of diarrhea and illness onset from 1 August 2006; cases with urine and stool isolates were included. Control participants were adults who did not report diarrhea in the preceding 2 weeks, matched to case patients by geographic location using an online reverse telephone directory. Case patients were asked about consumption of suspected food items identified by the hypothesis-generating interviews and specific brands of foods during the 7 days before diarrhea onset; control participants were asked about the 7 days before the interview. Patients with the most recent illness onset were prioritized for interview.

Further Case Finding and Case Exposure Assessment

To assess exposure to implicated products among patients with illness occurring after the products were recalled, we interviewed patients with illness onset after 14 February 2007. In addition, to determine whether cases of *Salmonella* Tennessee infection reported before the outbreak period were associated with exposure to Brand X or Y peanut butter, we attempted to interview 55 patients infected with an outbreak strain reported from 1 December 2004 to 1 August 2006; these patients were asked about typical food consumption rather than food consumption before illness onset.

Trace-Back and Environmental Investigation

The US Food and Drug Administration (FDA) investigated the manufacturing plant that produced Brands X and Y peanut butter. FDA inspectors reviewed quality control and production records, including consumer complaints, maintenance and installation of equipment, cleaning and sanitizing procedures, raw materials, product inventory, distribution, and in-house testing procedures and results; inspected plant facilities; and collected environmental swabs, raw ingredient samples, and finished peanut butter product for bacterial culture.

Microbiologic Investigation

Patients' *Salmonella* isolates were serotyped and subtyped by means of PFGE at 31 state public health laboratories using standard methods [12, 13]. Samples of leftover Brands X and Y peanut butter collected from patients and grocery stores in February 2007 were cultured for bacteria at state public health laboratories [14]. Environmental and peanut butter samples collected during the FDA's investigation of the manufacturing plant were cultured for bacteria at FDA laboratories, and the FDA performed serotyping and PFGE on food and environmental *Salmonella* isolates.

Statistical Analysis

Statistical analysis was performed using SAS, version 9.1. We estimated matched odds ratios (mORs) and 95% confidence intervals (CIs) using exact conditional logistic regression. Comparisons were performed using χ^2 , Fisher exact, or Wilcoxon rank sum tests where appropriate.

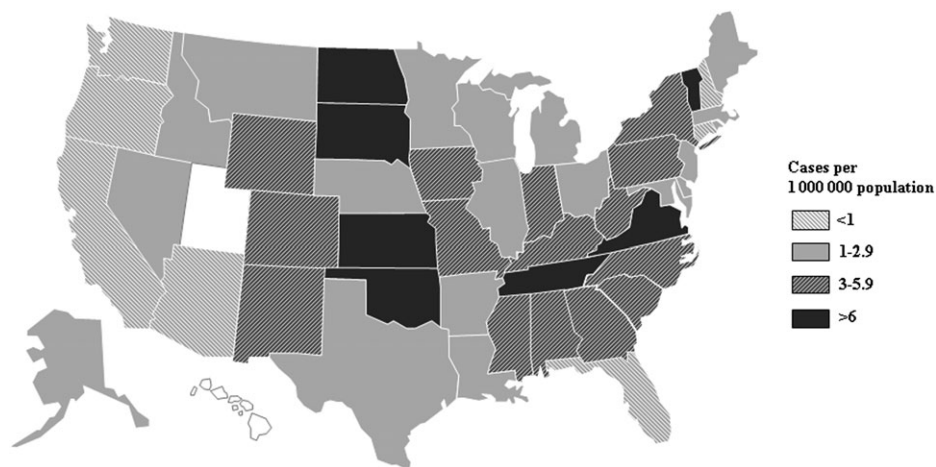


Figure 1. Number of reported cases of *Salmonella* Tennessee infection with outbreak-associated pulsed-field gel electrophoresis pattern per 1 000 000 population, by state—1 August 2006 through 31 July 2007 (N = 715).

RESULTS

Case Finding and Descriptive Epidemiology

A total of 715 cases were identified in 48 states (Figure 1). Patients had a median age of 53 years (range, 2 months to 95 years); 519 of 708 (73%) were female. Among 707 *Salmonella* isolates where specimen site was available, 421 (60%) were from stool specimens, 264 (37%) were from urine specimens, and 22 (3%) were from other or multiple specimen sources. The median age of patients with urine isolates was 62 years (range, 2 to 94 years), compared with 48 years (range, 2 months to

95 years) for patients with stool isolates. Ninety-four percent (247/263) of patients with urine isolates were female, compared with 62% (257/416) of patients with stool isolates (Figure 2).

Symptom onset dates (known for 546 patients) ranged from 1 August 2006 to 24 July 2007 (Figure 3). Clinical data (known for 442 patients) indicated that 118 of 415 (28%) visited an emergency department for their illness, 321 of 411 (78%) received antibiotics, 93 of 423 (22%) were hospitalized, and none died from *Salmonella* infection. Symptoms of infection included diarrhea (303/427, 71%), abdominal cramps (263/412, 64%), nausea (216/406, 54%), dysuria (183/398, 46%), fever (165/399,

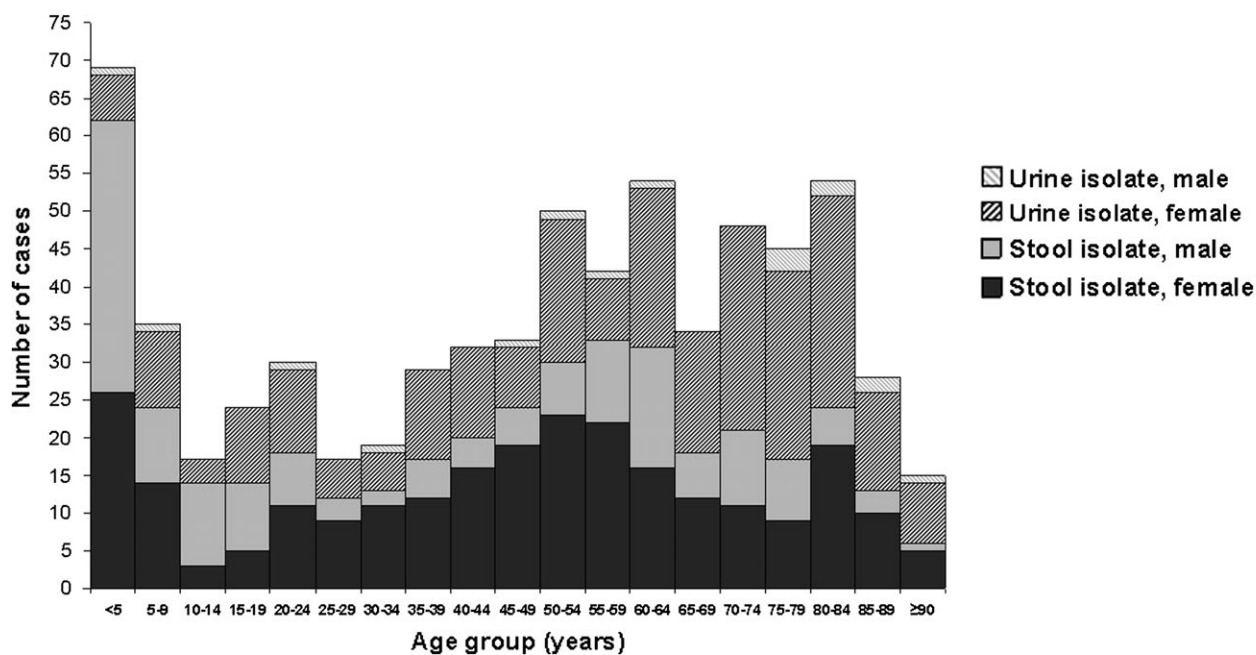


Figure 2. Age distribution of individuals with *Salmonella* Tennessee infection with outbreak-associated pulsed-field gel electrophoresis pattern by sex and isolate site (N = 675).

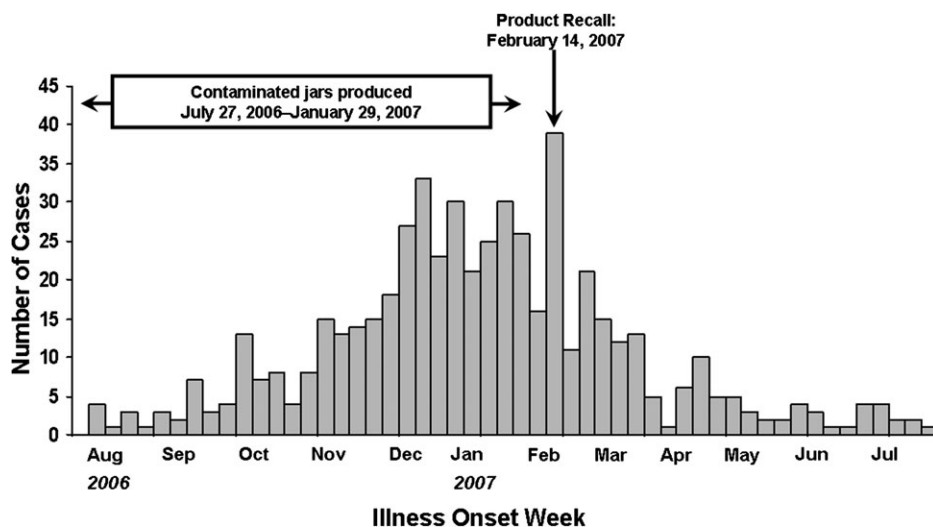


Figure 3. Number of cases of *Salmonella* Tennessee infection with outbreak-associated pulsed-field gel electrophoresis pattern for which date of symptom onset was available, by week of symptom onset—United States, 1 August 2006 through 31 July 2007 (N = 546).

41%), vomiting (126/431, 29%), and bloody diarrhea (63/402, 16%). Among patients with urine isolates, 87% (134/154) reported dysuria and 36% (55/152) reported diarrhea. Among patients with stool isolates, 18% (42/233) reported dysuria and 91% (241/266) reported diarrhea. Diarrhea and urinary symptom onset dates were available for 24 patients with urine isolates who reported both symptoms. The median time from diarrhea onset until urinary symptom onset was 12.5 days (range, 3–59 days).

Case Investigations

An initial investigation indicated that cases were not clustered geographically. Routine patient interviews conducted by local officials did not reveal common food exposures. Interviews of 31 patients using a standard food-consumption survey instrument indicated that, of the 200 exposures listed in the survey, consumption of turkey and peanut butter among patients (48% and 85%, respectively) were higher than would be expected from the FoodNet population survey (Table S1, available online). Five of 6 patients who underwent detailed, directed interviews had eaten peanut butter in the week before illness onset, and 4 patients named Brand X as at least 1 of the brands they ate before illness onset.

Case-Control Study

The median ages for the 65 patients and 124 control participants enrolled in the case-control study were 53 (range, 18 to 81 years) and 58 (range, 18 to 98 years) years, respectively ($P = .44$). Fifty-three of 65 (82%) patients and 70 of 119 (59%) control participants were female ($P = .003$). Eighty-one percent of patients and 65% of control participants reported eating peanut butter (mOR, 1.9 [95% CI, 0.8–5.2]). Patients were more likely than control participants to have eaten peanut butter more than once a week (66% vs 40%; mOR, 3.5 [95% CI, 1.4–9.9]), to have eaten Brand X peanut butter (61% vs 13%; mOR, 12.1 [95% CI, 3.6–63.3]), and to have eaten Brand Y peanut butter (8% vs 1%; mOR, 9.1 [95% CI, 1.0–433]). Neither consumption of other peanut butter brands nor consumption of turkey products was associated with illness (Table 1). Controlling for age and sex had negligible effects on the magnitudes of these associations. The population attributable fractions were 56% and 7% for Brands X and Y, respectively.

Product Investigation and Further Case Finding

Epidemiologic data suggesting Brands X and Y peanut butter as the possible sources of the outbreak were provided to FDA officials on 13 February 2007. The following day, the FDA issued

Table 1. Selected Food Exposures From Case-Control Study

| Exposure | Case patients (n = 65) | Control participants (n = 124) | mOR (95% CI) |
|---------------------------------------|------------------------|--------------------------------|-----------------|
| Peanut butter | 50/62 (81) | 80/124 (65) | 1.9 (.8–5.2) |
| Peanut butter more than once per week | 39/59 (66) | 49/124 (40) | 3.5 (1.4–9.9) |
| Brand X peanut butter | 37/61 (61) | 15/119 (13) | 12.1 (3.6–63.3) |
| Brand Y peanut butter | 5/61 (8) | 1/119 (1) | 9.1 (1.0–433) |
| Turkey | 15/61 (25) | 18/123 (15) | 2.3 (.9–6.3) |

NOTE. Data are proportion (%) of participants. CI, confidence interval; mOR, matched odds ratio.

a consumer health alert indicating not to eat Brand X or Y peanut butter, both of which were manufactured in a single facility operated by Company A. Company A voluntarily recalled the products, destroyed existing products in their possession, and halted production pending further investigation. Peanut butter from this plant had been distributed to 50 states and exported to 70 countries. Although several cases of salmonellosis associated with Brand X peanut butter consumption were investigated, no laboratory-confirmed outbreak cases were reported from other countries.

New case reports decreased substantially after the 14 February peanut butter recall but did not return to preoutbreak levels until months later (Figure 3). Among 685 patients with known illness onset or specimen collection dates, 212 (31%) had illness or specimen onset dates that occurred ≥ 1 week after the recall. Compared with those reporting illness before the recall, patients who reported illness after the recall were more likely to be older (median age, 59 vs 49 years; $P < .001$) and to have urine *Salmonella* Tennessee isolates (52% vs. 30%; $P < .001$). Among 64 patients with illness after the recall who provided food consumption histories, 79% ate peanut butter during the week before illness onset, 55% ate Brand X or Y peanut butter, and 19% had Brand X or Y peanut butter jars in their homes at the time of the interview. Of these patients, 95% were aware of a peanut butter recall, 81% correctly identified that the recall involved Brand X, and 51% correctly identified that it involved Brand Y.

On 9 March 2007, Company A extended its recall to include all Brands X and Y peanut butter produced at the implicated plant purchased since October 2004. Among 12 of 55 patients with cases occurring before the outbreak period that could be contacted, 11 (91%) reported eating peanut butter in a typical week; 8 (73%) of these ate peanut butter more than once per week, and 5 (60%) ate Brand X.

FDA investigation of plant operations was limited, because the plant had halted operations 1 day before. Company A did not report malfunctions of the peanut roaster, but the actual roasting temperature of peanuts during normal operations could not be measured. Some roasted peanuts were held in uncovered bins. Company A did not report maintenance or repair issues of production equipment. No trends in employee illnesses were noted. Review of finished product and environmental testing performed by Company A from 2005 and 2006 noted no evidence of contamination. However, records showed that *Salmonella* had been isolated from peanut butter jars in the plant in October 2004; these jars had previously been destroyed, and molecular typing was not available [15].

Microbiologic Investigation

Salmonella Tennessee with a PFGE pattern matching 1 of the outbreak strains (JNXX01.0011) was first isolated from leftover peanut butter from a patient by the Iowa State Public Health

Laboratory on 22 February 2007; eventually, an outbreak strain was isolated from 34 of 298 opened and unopened Brand X and Y peanut butter jars with production dates from 27 July 2006 through 29 January 2007. An additional outbreak-associated strain (JNXX01.0010) was isolated from some of these peanut butter samples (Figure S1, available online). A Brand X peanut butter jar obtained from a store in the Philippines also yielded 1 of the outbreak strains (JNXX01.0011) (C. Carlos, MD, written communication, April 2007).

The FDA isolated *Salmonella* Tennessee with a PFGE pattern matching 1 of the outbreak strains (JNXX01.0011) from 2 plant environmental samples that were collected from the peanut roasting room (Figure S1, available online).

Public Health Response

In response to this outbreak, Company A installed new equipment at the manufacturing plant, made repairs to the roof, cleaned and sanitized the plant, ensured separation of raw ingredients from post-peanut-roasting areas that handle processed product, created a new Hazard Analysis and Critical Control Points Plan, and implemented a revised environmental testing program for *Salmonella* [16]. The plant reopened in September 2007; since then, *Salmonella* Tennessee infections have not increased. One of the outbreak strains (JNXX01.0010) has not been reported to CDC PulseNet since 2007, and only 14 isolates matching the other 2 outbreak strains (JNXX01.0011 and JNXX01.0026) have been reported from 1 January 2009 until 30 April 2010.

DISCUSSION

This is the first reported outbreak caused by peanut butter consumption in the United States. The outbreak was widespread and prolonged, with 715 cases reported in 48 states during a period of 1 year. Considering underascertainment of cases through laboratory-based surveillance, estimating 29 outbreak cases for each culture-confirmed case [1], there may have been $>20\,000$ persons ill from this outbreak. The source of the outbreak was traced to peanut butter produced at a single facility and distributed under 2 brands, although because of higher population exposure, 1 brand of peanut butter accounted for the majority of outbreak-related illnesses. One peanut butter-associated outbreak was previously reported, a smaller outbreak of *Salmonella* Mbandaka infections occurring in Australia in 1996 [17–19]. Other peanut-associated outbreaks include *Salmonella* Agona infections associated with a peanut-flavored snack produced in Israel [20, 21] and *Salmonella* Stanley and Newport infections associated with Asian-style peanuts [22]. In 2008–2009, contaminated peanut butter and peanut butter-containing products from a different manufacturer resulted in a large multistate outbreak of *Salmonella* Typhimurium infections. In this *Salmonella* Typhimurium

outbreak, *Salmonella* Tennessee with a PFGE pattern matching the outbreak described in this paper was also isolated from an unopened container of the implicated brand of peanut butter, but no concomitant increase in human *Salmonella* Tennessee infections was seen [23].

Although peanut butter may have previously been considered at low risk for *Salmonella* contamination, these outbreaks indicate that this is not the case. It is unclear how peanut butter became contaminated in this outbreak, although several factors may have contributed. Peanuts could have become contaminated with salmonellae during growth, harvest, or storage. Salmonellae can enter food-processing plants by various mechanisms, such as through raw agricultural products, water, animals, humans, or other surfaces. The organisms are able to survive high temperatures in high-fat, low water activity environments [24]. Peanut butter provides such an environment, and *Salmonella* has been shown to survive for at least 6 months in peanut butter [25]. Although peanuts are typically roasted at $>150^{\circ}\text{C}$ and peanut butter is heated to $>70^{\circ}\text{C}$ during grinding and milling, such heating might not reliably eliminate salmonellae from the finished product if heating is heterogeneous or if contamination occurs after heating [26, 27].

Investigation of cases that occurred prior to August 2006 suggests that contamination may have begun even before the apparent outbreak period. The high proportion of cases since December 2004 with Brand X peanut butter consumption, isolation of the outbreak strain from environmental samples from the plant, and the report of *Salmonella* found in peanut butter tested in 2004, all suggest that the outbreak could have been related to prolonged environmental contamination at the manufacturing plant.

Case counts decreased the week after the recall, and identification of the outbreak vehicle likely prevented hundreds of illnesses. However, case counts did not return to baseline levels until months later. Regulatory actions and the scope of the recall were expansive, and the outbreak received intensive media attention [28]; however, some people continued to consume the implicated product. Interviews of individuals who became ill after the recall indicate that a high proportion of them were aware of the recall, suggesting that they did not understand that the recall applied to a product in their possession or did not appreciate the risk associated with consuming a recalled product. The typical shelf life of peanut butter is 12–18 months, so implicated brands may have remained in consumer homes for months after they were no longer available in stores. This experience suggests that product recalls related to outbreak investigations, although often necessary and effective public health measures, are imperfect tools for controlling the safety of a food.

The frequency of urinary tract infections (UTIs) due to *Salmonella* was unusually high in this outbreak and may have

contributed to its prolonged nature. *Salmonella* UTIs are thought to most commonly arise after gastrointestinal infection. A report of a 1998 outbreak of *Salmonella* Havana described urinary isolates in 33% of patients; gastrointestinal symptoms were not described [29]. A review from Austria reported that 10% of patients with symptomatic *Salmonella* UTIs had gastrointestinal symptoms and 50% had *Salmonella* isolated from the stool without gastrointestinal symptoms [30]. In this outbreak, more than one-third of patients with UTIs reported diarrhea associated with their illness. Although the incubation period of *Salmonella* gastroenteritis has been well described, the incubation period for UTIs after gastrointestinal infection is not known. The time from diarrhea onset until urinary symptom onset in this outbreak ranged from 3 to 59 days, suggesting that *Salmonella* shedding may have lasted for weeks after gastrointestinal infection in these patients.

The high rate of UTIs in this outbreak suggests that organisms that cause urinary infections may be widely distributed through contaminated food. A foodborne reservoir for certain strains of uropathogenic *Escherichia coli* has been suggested, but its distribution through a specific food vehicle has not been clearly demonstrated [31]. A review of urinary *Salmonella* isolates reported to the CDC between 1995 and 1999 showed that urinary isolates represented 4% of all *Salmonella* isolates, an increase from previous years [9]. *Salmonella* Tennessee had the highest urine-to-stool isolate ratio among reported serotypes, suggesting that currently unrecognized serotype-specific factors may either increase uropathogenicity or decrease enteropathogenicity, resulting in overrepresentation of these serotypes isolated from urine.

This outbreak reveals the potential for widespread illness from a broadly distributed contaminated product with a long shelf life. Such outbreaks are often diffuse and evolve slowly, with illnesses occurring over months to years. *Salmonella* surveillance with serotyping and molecular subtyping is essential in detecting such outbreaks. This outbreak also shows that processed foods can become contaminated even when the production process includes a heat treatment step, underscoring the need for effective controls in food processing plants to ensure adequate heat treatment and to prevent contamination after heat treatment. Further investigations of *Salmonella* outbreaks and processing plants associated with these outbreaks will guide recommendations for preventive controls.

Supplementary Data

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

Acknowledgments

Additional contributions: The *Salmonella* Tennessee Outbreak Investigation Team included public health officials at local and state health departments and public health laboratories in the United States, including Catina James (Alabama), Karen Martinek (Alaska), Shoana Anderson (Arizona), Linda Gladden (Arkansas), Jennifer Schneider (California), Nicole Comstock (Colorado), Wendy Bamberg (Colorado), Quyen Phan (Connecticut), Susan Shore (Delaware), Mike Friedman (Florida), Carrie Shuler (Georgia), Leslie Tengelsen (Idaho), Ingrid Trevino (Illinois), Lynae Granzow (Indiana), Sarah Brend (Iowa), Cheryl Banez-Ocfemia (Kansas), Peggy Ellis (Kentucky), Annu Thomas (Louisiana), Anthony Yartel (Maine), Sonhi Kim (Maryland), Emily Harvey (Massachusetts), Katie Sheline (Michigan), Sally Bidol (Michigan), Stacy Holzbauer (Minnesota), Sheryl Hand (Mississippi), JoAnn Rudroff (Missouri), Bonnie Barnard (Montana), Dennis Leschinsky (Nebraska), Rick Sowadsky (Nevada), Beth Daly (New Hampshire), Michelle Malavet (New Jersey), Christina Ewers (New Mexico), Geraldine Johnson (New York), Scott Nowicki (Ohio), David Bergmire-Sweat (North Carolina), Megan Davis (North Carolina), Julie Goplin (North Dakota), Bill Keene (Oregon), Nancy Rea (Pennsylvania), Leanne Chiaverini (Rhode Island), Julie Schlegel (South Carolina), Linda Schaefer (South Dakota), Samir Hanna (Tennessee), Lesley Bullion (Texas), Mary Spayne (Vermont), Seth Levine (Virginia), Kathryn MacDonald (Washington), Suzanne Wilson (West Virginia), Rachel Klos (Wisconsin), Kelly Weidenbach (Wyoming), Samir Sodha (CDC), and Manoj Menon (CDC).

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

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

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

References

1. Scallan E, Hoekstra RM, Angulo FJ, et al. Foodborne illness acquired in the United States—major pathogens. *Emerg Infect Dis* 2011; 17:7–15.
2. Centers for Disease Control and Prevention. Multistate outbreak of *Salmonella* serotype Agona infections linked to toasted oats cereal—United States, April–May, 1998. *MMWR* 1998; 47:462–4.
3. Jain S, Bidol S, Austin J, et al. Multistate outbreak of *Salmonella* Typhimurium and Saintpaul infections associated with unpasteurized orange juice—United States, 2005. *Clin Infect Dis* 2009; 48:1065–71.
4. O’Ryan M. An outbreak of *Salmonella* infection from ice cream. *NEJM* 1996; 335:824–5.
5. Lynch M, Painter J, Woodruff R, Braden C. Surveillance for foodborne-disease outbreaks—United States, 1998–2002. *MMWR* 2006; 55:1–42.
6. Centers for Disease Control and Prevention. Surveillance for foodborne disease outbreaks—United States, 2006. *MMWR* 2009; 58:609–15.
7. Centers for Disease Control and Prevention. *Salmonella* surveillance summary, 2004. <http://www.cdc.gov/ncidod/dbmd/phlisdata/salmonella.htm>. Published 2006. Accessed 15 April 2007.
8. Centers for Disease Control and Prevention. *Salmonella* serotype Tennessee in powdered milk products and infant formula—Canada and United States, 1993. *MMWR* 1993; 42:516–7.
9. Sivapalasingam S, Hoekstra RM, McQuiston JR, Fields PI, Tauxe RV. *Salmonella* bacteriuria: an increasing entity in elderly women in the United States. *Epidemiol Infect* 2004; 132:897–902.
10. Centers for Disease Control and Prevention. Multistate outbreak of *Salmonella* serotype Tennessee infections associated with peanut butter—United States, 2006–2007. *MMWR* 2007; 56:521–4.
11. Centers for Disease Control and Prevention. Foodborne Diseases Active Surveillance Network (FoodNet): population survey atlas of exposures, 2002. <http://www.cdc.gov/foodnet/surveys/pop/2002/2002atlas.pdf>. Published 2004. Accessed 15 January 2007.
12. Nataro JP, Bopp CA, Fields PI, Kaper JB, Strockbine NA. *Escherichia*, *Shigella*, and *Salmonella*. In Murray PR, Baron EJ, Jorgensen JH, Landry JL, Pfaller MA eds: *Manual of clinical microbiology*, 9th ed. Washington, DC: ASM Press, 2007.
13. Ribot EM, Fair MA, Gautom R, et al. Standardization of pulsed-field gel electrophoresis protocols for the subtyping of *Escherichia coli* O157:H7, *Salmonella*, and *Shigella* for PulseNet. *Foodborne Pathog Dis* 2006; 3:59–67.
14. Andrews W, Hammack T. *Bacteriological analytical manual*, 8th edition, revision A, 1998. <http://www.fda.gov/Food/ScienceResearch/LaboratoryMethods/BacteriologicalAnalyticalManualBAM/default.htm>. Accessed 3 December 2009.
15. Food and Drug Administration. Establishment Inspection Report: February 14–March 2, 2007. <http://www.fda.gov/downloads/AboutFDA/CentersOffices/ORA/UCM133011.pdf>. Published 2007. Accessed 23 April 2010.
16. Food and Drug Administration. Establishment inspection report: August 20–23, 2007. <http://www.fda.gov/downloads/AboutFDA/CentersOffices/ORA/UCM133012.pdf>. Published 2007. Accessed 23 April 2010.
17. Scheil W, Cameron S, Dalton C, Murray C, Wilson D. A South Australian *Salmonella* Mbandaka outbreak investigation using a database select controls. *Aust N Z J Public Health* 1998; 22:536–9.
18. Ng S, Rouch G, Dedman R, et al. Human salmonellosis and peanut butter. *Commun Dis Intell* 1996; 20:326.
19. Oliver G. The *Salmonella* Mbandaka outbreak—an Australian overview. *Commun Dis Intell* 1996; 20:326.
20. Killalea D, Ward LR, Roberts D, et al. International epidemiological and microbiological study of outbreak of *Salmonella* Agona infection from a ready to eat savoury snack—I: England and Wales and the United States. *BMJ* 1996; 313:1105–7.
21. Shohat T, Green MS, Merom D, et al. International epidemiological and microbiological study of outbreak of *Salmonella* Agona infection from a ready to eat savoury snack—II: Israel. *BMJ* 1996; 313:1107–9.
22. Kirk MD, Little CL, Lern M, et al. An outbreak due to peanuts in their shell caused by *Salmonella enterica* serotypes Stanley and Newport—sharing molecular information to solve international outbreaks. *Epidemiol Infect* 2004; 132:571–7.
23. Centers for Disease Control and Prevention. Multistate outbreak of *Salmonella* infections associated with peanut butter and peanut butter-containing products—United States, 2008–2009. *MMWR* 2009; 58:85–90.
24. Mattick KL, Jorgensen F, Legan JD, Lappin-Scott HM, Humphrey TJ. Habituation of *Salmonella* spp. at reduced water activity and its effect on heat tolerance. *Appl Environ Microbiol* 2000; 66:4921–5.
25. Burnett SL, Gehm ER, Weissinger WR, Beuchat LR. Survival of *Salmonella* in peanut butter and peanut butter spread. *J Appl Microbiol* 2000; 89:472–7.
26. Shachar D, Yaron S. Heat tolerance of *Salmonella enterica* serovars Agona, Enteritidis, and Typhimurium in peanut butter. *J Food Prot* 2006; 69:2687–91.
27. Ma L, Zhang G, Gerner-Smidt P, Mantripragada V, Ezeoke I, Doyle M. Thermal inactivation of *Salmonella* in peanut butter. *J Food Prot* 2009; 72:1596–601.
28. Funk J. Peanut butter recalled over *Salmonella*. 2007. The Washington Post. <http://www.washingtonpost.com/wp-dyn/content/article/2007/02/15/AR2007021500597.html>. Accessed 23 April 2010.
29. Vincent C, Boerlin P, Daignault D, et al. Food reservoir for *Escherichia coli* causing urinary tract infections. *Emerg Infect Dis* 2010; 16:88–95.
30. Backer HD, Mohle-Boetani JC, Werner SB, Abbott SL, Farrar J, Vugia DJ. High incidence of extra-intestinal infections in a *Salmonella* Havana outbreak associated with alfalfa sprouts. *Public Health Rep* 2000; 115:339–45.
31. Allerberger FJ, Dierich MP, Ebner A, et al. Urinary tract infection caused by nontyphoidal *Salmonella*: report of 30 cases. *Urol Int* 1992; 48:395–400.