A University Outbreak of Gastroenteritis Due to a Small Round-Structured Virus: Application of Molecular Diagnostics to Identify the Etiologic Agent and Patterns of Transmission


An epidemiologic investigation of a gastroenteritis outbreak in December 1994 indicated that salad consumption during lunch was linked with illness on 2 days (5 December: odds ratio [OR] = 3.1, 95% confidence interval [CI] = 2.0–5.0; 6 December: OR = 3.1, 95% CI = 1.9–4.9). Single stool or vomitus specimens from ill students and staff (case-patients) were examined for bacterial and viral pathogens. Small round-structured viruses (SRSVs) were detected by electron microscopy in stool specimens from 9 of 19 case-patients and in vomitus specimens from 3 of 5 case-patients. By reverse transcription–polymerase chain reaction (RT-PCR), the SRSVs were shown to be a G-2/P2-B type strain. The nucleotide sequences of RT-PCR products from vomitus and stool specimens of ill students were identical to stool specimens from the ill salad chef. These findings suggest that a single SRSV strain was the etiologic agent in the outbreak that was possibly transmitted to students through consumption of contaminated salad. Epidemiologic investigation in conjunction with molecular diagnostics may enable early identification of sources of infection and improve outbreak control.

Small round-structured viruses (SRSVs) are antigenically related 30-nm members of the Caliciviridae family [1, 2]. Norwalk virus (NV), the prototype strain, was discovered in fecal specimens collected during an outbreak of gastroenteritis in Norwalk, Ohio, in 1968 [3, 4]. Subsequent studies have shown that SRSVs are the major cause of outbreaks of nonbacterial gastroenteritis, and transmission of SRSVs by contaminated food and water has been documented [5–9]. While gastroenteritis outbreaks associated with SRSVs are common, the incidence of disease and its importance as a cause of sporadic illness has not been extensively studied, in part, because of difficulties in making an accurate and rapid diagnosis.

In the past, investigations of foodborne outbreaks of gastroenteritis associated with SRSVs relied on classic epidemiologic methods to demonstrate the association of illness with food consumption [10, 11]. Diagnostic methods included the examination of fecal specimens by electron microscopy and the measurement of antibody response, neither of which could distinguish between related strains [12]. Consequently, it was difficult to link illness in a food handler to an outbreak strain or to determine if patients had become ill from an SRSV originating from a common source. The recent development of novel molecular methods to detect and differentiate SRSVs has allowed the diagnosis of gastroenteritis with increased sensitivity and specificity and provided techniques to determine if one or multiple strains of the virus are responsible for an outbreak [13–17].

On 6 December 1994, the University Health Service (UHS) of a Massachusetts university notified the state Department of Public Health that a number of students had presented in the preceding 24 h for treatment of acute gastroenteritis. The abrupt onset and frequent vomiting among patients suggested an etiology consistent with either a preformed toxin (e.g., Bacillus cereus, Staphylococcus aureus) or an SRSV. Since these agents may originate from different sources and have distinct modes of transmission, we began an investigation to identify the etiologic agent and determine its mode of transmission so that appropriate control measures could be implemented.

Materials and Methods

Background. The university, located in Cambridge, Massachusetts, has an undergraduate population of 6400. Of these, 1660
are freshmen and live primarily in 14 residence halls. During the academic year, students receive both inpatient and outpatient care from the UHS, which maintains computerized records on all admissions. On 6 December, we began our investigation by surveying staff and students hospitalized for gastroenteritis. We reviewed UHS records to determine the number of cases and assessed the presenting signs and symptoms and the severity of illness. Since the majority of cases occurred among freshmen and the only common exposure easily identified among ill students was eating at the freshman dining hall, the facility was closed after dinner on 6 December, pending further investigation.

Epidemiologic investigation. On 8 December, we distributed questionnaires to residents of student dormitories to determine demographic information, illness characteristics, and food consumption histories over 3 days (4--6 December). We restricted our investigation to persons who had eaten or worked in the dining hall during this period. We defined a case of gastroenteritis as an illness consisting of at least 1 episode of vomiting or diarrhea (3 liquid stools within a 24-h period) after 2 December. We maintained surveillance for additional cases among students and staff through the student advisor network, the UHS emergency room, and the UHS outpatient clinic.

On 8 December, we surveyed students eating at the freshman dining hall to determine risk factors for gastroenteritis and compared cases (n = 188) with students who did not have gastroenteritis (controls, n = 175). Since controls were underrepresented in surveys on 8 December, we performed an additional survey at the freshman dining hall on 10 December to permit a case-control analysis. We analyzed a subset of data from controls in the dormitory survey and controls in the dining hall survey for comparability of responses for selected food items and found no significant differences. We compared attack rates of gastroenteritis for case-patients and controls by using a $\chi^2$ test of significance for nonstratified data and calculated odds ratios (OR) using 95% confidence intervals (CI) [18].

We interviewed the dining hall food service and administrative staff (n = 19) to obtain information on food handling procedures, raw food sources, preparation and storage of food items, and absenteeism and symptoms among those who were ill.

Environmental investigation. Potential sources of contamination of water and food were investigated by staff of the university Office of Environmental Health and Safety and the city Department of Sanitation. Water and sewage systems supplying the freshman dormitories and dining hall were inspected, and water samples were obtained for bacteriologic analysis. All available specimens of food, ice, and water and swabs of kitchen surfaces and utensils were collected on 6 and 7 December, shortly after closure of the dining hall, and sent to Morrell Associates (Marshfield, MA) for bacteriologic culture. Samples demonstrating growth of enteric pathogens were further analyzed at the US Food and Drug Administration (Washington, DC) microbiologic laboratories to identify S. aureus enterotoxin using an EIA (Tecra Diagnostics, Roseville, Australia). Assays for toxins of B. cereus and Clostridium perfringens were not done. We reviewed kitchen facilities and procedures used in the processing and handling of cooked and uncooked food and collected information on specific vendors, time of food delivery, and inspection of foods upon delivery.

Laboratory investigation. Rectal swabs were obtained from 75 patients (collected 6--9 December) and cultured for Campylobacter, Salmonella, Shigella, Vibrio, and Yersinia species at the UHS laboratory (n = 60) and the state health department laboratory (n = 15). Specimens of vomitus and stool, collected from ill students and food service workers within 48 h of the onset of illness, were kept at 4°C and transported to the Centers for Disease Control and Prevention for viral diagnosis [19]. Electron microscopy (EM) was done using standard methods [20].

Since investigation of past outbreaks has shown that analysis of a subset of patient specimens does not alter the conclusions regarding either the causative agent or the genotype classification, our molecular analysis included RNA extracted from 9 stool and 5 vomitus specimens. SRSVs were detected by reverse transcription–polymerase chain reaction (RT-PCR) and further characterized by Southern hybridization of oligonucleotide probes [15]. The amount of RT-PCR product from vomitus specimens was increased by additional purification of the RNA using Oligo(dT)30 before sequencing (Takara Biomedicals, Kyoto, Japan). To determine relatedness of strains, amplification products from 6 patients were sequenced: 1 ill food handler (stool) and 5 ill students (2 stool, 3 vomitus). The RT-PCR products were sequenced along both strands, and a consensus sequence was generated.

A stool specimen containing human astrovirus or human parechovirus A rotavirus was used as the negative control for RT-PCR and Southern hybridization. We used a specimen containing the prototype NV strain from a volunteer as the positive control. The coded specimens, including controls, were processed for RNA extraction and RT-PCR in separate groups of 10 specimens or less. All assays were repeated for confirmation.

Acute- and convalescent-phase sera from ill students and staff were tested for IgG antibody to NV and Toronto virus (TV) using baculovirus-expressed NV and TV capsid protein immunon assays [2, 21, 22]. NV is representative of an antigenic type G1, and TV is representative of a G2 type SRSV strain.

Results

UHS case investigation. A review of UHS hospital records indicated that the epidemic began abruptly on 6 December, peaked on 7 December, and continued at a low level during the following week (figure 1B). Of note, most patients were freshmen or student advisors living in freshman dormitories.

Case-control study. On 8 December, we surveyed 363 students. Of these, 188 (52%) had gastrointestinal symptoms that met the case definition and 175 (48%) did not (controls). The students surveyed appeared similar to those attending the UHS for treatment of gastroenteritis with respect to sex, age, and residence location; slightly more students surveyed were female (52%). Among the ill students, 99 (53%) reported onset of illness on 6 December, and 69 (37%) reported onset on 7 December (figure 1A). For the students surveyed, the illness peaked 1 day before the peak for cases seen at the UHS, a delay that may reflect the time from onset of illness to the seeking of treatment. The median age of both cases and controls...
was 18 years (range, 16–28). Ninety-nine percent of cases and controls were freshmen; the rest were graduate student advisors. At the 14 freshmen dormitories, attack rates were 7%–17%. We found no significant clustering of cases within dormitories ($P = .74$, Walter’s test for aggregation) [23].

The symptoms most commonly reported among 188 cases were nausea (95%), vomiting (84%), headache (78%), chills (73%), cramps (72%), fever (67%), diarrhea (62%), muscle ache (56%), and cough (22%). The mean number of bowel movements was 6 per day, and the mean duration of illness was 1.5 days (range, 1–7). Ten of 19 food service workers surveyed reported an illness similar to that of the students. Four of the ill staff were general service workers, 3 were cooks, and 1 each was a cleaner, supervisor, and a salad chef. Three workers developed illness on 3 December (a cook, general service worker, and supervisor); the salad preparer became ill on 4 December. A second employee who prepared salad on 2 and 3 December did not develop gastroenteritis.

The survey results were examined to identify meals and foods that might be linked with illness (table 1). We identified
had no evidence of contamination with enteric pathogens. Specimens retained after closure of the kitchen, 35 items were
dining hall sites showed no bacterial growth. Among the food
gastroenteritis. We further analyzed individual salad bar foods but found no
Swabs from dishes and utensils and ice samples from different
additional exposures that were significantly associated with
(OR = 1.9; CI, 1.3-2.9), and lunch on 6 December (OR = 3.1;
CI, 1.9-4.9) were significantly associated with illness (table 2).
December (OR = 3.1; CI, 2.0-5.0), dinner on 5 December
the consumption of items from the salad bars for lunch on 5
of diners (cases) who were already ill.
0.3; CI, 0.2-0.7), perhaps representing the absence of a number
individual foods may have posed an increased risk for gastroen­
teritis; 85% of
of controls (OR = 1.7; CI, 1.0-2.9). A protective effect was
associated with consumption of dinner on 6 December (OR =
consumer 4-6 December 1995.

<table>
<thead>
<tr>
<th>Date</th>
<th>Meal</th>
<th>% (n) who ate meal (n = 188)</th>
<th>Controls (n = 175)</th>
<th>OR (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>12/4/94</td>
<td>Brunch</td>
<td>75 (140)</td>
<td>78 (137)</td>
<td>0.8 (0.5-1.3)</td>
</tr>
<tr>
<td>12/4/94</td>
<td>Dinner</td>
<td>90 (170)</td>
<td>87 (153)</td>
<td>1.4 (0.7-2.6)</td>
</tr>
<tr>
<td>12/5/94</td>
<td>Lunch</td>
<td>85 (166)</td>
<td>77 (135)</td>
<td>1.7 (1.0-2.9)</td>
</tr>
<tr>
<td>12/5/94</td>
<td>Dinner</td>
<td>90 (170)</td>
<td>95 (166)</td>
<td>0.5 (0.2-1.2)</td>
</tr>
<tr>
<td>12/6/94</td>
<td>Lunch</td>
<td>83 (156)</td>
<td>78 (136)</td>
<td>1.4 (0.8-2.4)</td>
</tr>
<tr>
<td>12/6/94</td>
<td>Dinner</td>
<td>83 (156)</td>
<td>93 (163)</td>
<td>0.3 (0.2-0.7)</td>
</tr>
</tbody>
</table>

* Odds ratio (OR) calculated for unmatched case-control analysis; CI, confidence interval.

one meal—lunch on 5 December—associated with gastroen­
teritis; 85% of ill students ate this lunch compared with 77%
of controls (OR = 1.7; CI, 1.0–2.9). A protective effect was
associated with consumption of dinner on 6 December (OR =
0.3; CI, 0.2–0.7), perhaps representing the absence of a number
of diners (cases) who were already ill.

We then analyzed food items served at meals on 5 and
6 December, since we were concerned that consumption of
individual foods may have posed an increased risk for gastroen­
teritis (table 2). Despite the 187 menu items surveyed, only
the consumption of items from the salad bars for lunch on 5
December (OR = 3.1; CI, 2.0–5.0), dinner on 5 December
(OR = 1.9; CI, 1.3–2.9), and lunch on 6 December (OR = 3.1;
CI, 1.9–4.9) were significantly associated with illness (table 2).
We further analyzed individual salad bar foods but found no
additional exposures that were significantly associated with
gastroenteritis.

Environmental investigation. Samples from water supply
lines serving the student dormitories and freshman dining hall
had no evidence of contamination with enteric pathogens. Swabs from dishes and utensils and ice samples from different
dining hall sites showed no bacterial growth. Among the food
specimens retained after closure of the kitchen, 35 items were
tested for bacterial pathogens. Of these, 3 grew S. aureus:
chopped egg, 430 S. aureus/g (normal, <10); sliced cucumber,
290 S. aureus/g; and rigatoni noodles, 44,000 S. aureus/g. De­
spite these results, no S. aureus toxin was detected in any of
the samples in which the bacterium was isolated.

During our review of kitchen procedures, we interviewed 2
salad chefs: 1 worked 5 days per week and reported having
 gastroenteritis; the other worked 2 days each week (Friday and
Saturday) and remained well. Among 8 additional food han­
dlers interviewed, 6 reported nausea, vomiting, or both at work
during 4–9 December. Most raw vegetables for the salad bar
were prepared in a local factory and delivered vacuum-packed
to all university dining facilities. Selected items, including
tomatoes and cucumbers, were processed daily in each dining
facility kitchen. Salad chefs, wearing plastic gloves, unpacked
the prepared ingredients and mixed salad items according to
the menu of the day. Leftover foods, including salads, were
stored in a refrigerator for inspection by the production man­
ger before they were served in subsequent meals.

Laboratory investigation. All rectal swab specimens cul­
tured from ill persons were negative for Shigella, Salmonella,
Vibrio, and Campylobacter species, except those from 2 food
handlers in which Yersinia frederiksenii (part of the commensal
flora in humans) was isolated. SRSVs were identified by EM
in 3 of 5 vomitus and in 9 of 19 fecal specimens (table 3).

Six of 9 fecal specimens and 3 of 5 vomitus specimens were
characterized by Southern hybridization as belonging to the
P2-B phylogenetic group, which includes the Snow Mountain
agent. Of note, all 3 vomitus specimens positive for SRSVs
by EM had SRSV detected by RT-PCR. Sequence analysis of
the 81-bp RT-PCR product from the stool of an
food handler showed it was identical to RT-PCR products of both stool
and vomitus from ill students, suggesting a common source of
infection. SRSV was detected in 2 more patients by RT-PCR
than by EM (8); only 1 patient had a stool positive by EM
that was negative by RT-PCR, indicating a positive correlation
between EM and RT-PCR studies.

Among 12 paired sera tested for antibody to NV, 3 demon­
strated a titer rise of 4-4-fold using the recombinant NV EIA,
was already ill, contaminated the salad during preparation. The salad chef became ill before others) or that the salad chef, who worked in the salad bar, was the vehicle for virus transmission (and the vomitus suggests either that food contaminated and then served to the strain identified in both stool and vomitus of the salad chef was identical (in an 81-bp region) to the strain identified in other students in the outbreak, demonstrating a common infectious source and a plausible causal link to the salad chef. This observation suggests either that food contaminated and then served in the salad bar was the vehicle for virus transmission (and the salad chef became ill before others) or that the salad chef, who was already ill, contaminated the salad during preparation.

Although we could not identify a specific breach in hygiene practices, salad preparation and storage was confined to a single room of the kitchen, and the salad was prepared by the same employee each Sunday through Thursday. Contamination of salad bar foods by this food handler appears to be consistent with her onset of symptoms on Sunday, 4 December, her continued illness while at work on 5 December, and clustering of cases in the outbreak on 6 December. Common salad items served in other university cafeterias did not cause outbreaks. The food-specific attack rates suggest that consumption of food in the salad bar was strongly associated with illness. On the basis of these data, however, we cannot determine the relative contribution to illness of each meal since persons consuming salad bar items at one meal may be likely to consume salad bar items at other meals.

Since vomiting was a major feature of the gastroenteritis in this outbreak, our differential diagnosis included infection caused by bacterial toxins and SRSVs. The detection of SRSVs with an identical nucleotide sequence in vomitus and stool specimens from ill students and food service workers and the finding of probe-positive vomitus specimens by Southern hybridization suggests that viruses and not food toxins were the causative agents of this outbreak. This finding extends those of volunteer studies in which 4 of 5 vomitus specimens were positive for SRSV antigen by RIA and 1 specimen had SRSV particles visualized by immune EM [24]. Application of molecular diagnostics to vomitus specimens will better define the usefulness of vomitus for diagnosis in future outbreaks. Identification of SRSV particles in vomitus lends further support to the view that exposure to vomitus (and virus aerosolized from vomitus) may pose a risk for illness in susceptible persons [25].

During this investigation, we considered alternative hypotheses and exposures that could have resulted in the outbreak. For example, we were concerned about food contamination at the factory before packaging into sealed containers; however, this scenario seemed unlikely because gastroenteritis rates in groups eating at other campus facilities supplied by the factory were not increased. Second, fecal contamination of water used during food preparation appears unlikely, since the university water supply was bacteriologically pure [26]. Third, although other food service employees who were ill on 3 December could have contaminated food prior to serving, these workers stated they had little or no contact with the salad bar or other foods.

The epidemiologic and environmental studies in this investigation helped rule out other exposures, such as outside group activities and contaminated water. The early hypothesis that a food served at the dining hall was the likely source of infection led to closure of the dining facility and the abrupt termination of epidemic gastroenteritis among the students. The appearance of secondary cases among students suggests that person-to-person transmission may have occurred in the dormitory setting. However, rapid control of SRSV-associated outbreaks

### Table 3. Laboratory results from clinical specimens from students and staff with gastroenteritis.

<table>
<thead>
<tr>
<th>Specimen type, patient no.</th>
<th>Electron microscopy</th>
<th>Polymerase chain reaction</th>
<th>Southern hybridization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stool</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Negative</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>2</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>3</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>4</td>
<td>Positive</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>5</td>
<td>Negative</td>
<td>Negative</td>
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</tr>
<tr>
<td>6</td>
<td>Positive</td>
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<td>NT</td>
</tr>
<tr>
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<td>Positive</td>
<td>NT</td>
<td>NT</td>
</tr>
<tr>
<td>8</td>
<td>Positive</td>
<td>NT</td>
<td>NT</td>
</tr>
<tr>
<td>9</td>
<td>Negative</td>
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<tr>
<td>10</td>
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<td>NT</td>
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<tr>
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<tr>
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</tr>
<tr>
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<td>Negative</td>
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</tr>
<tr>
<td>Total (no. positive/no. tested)</td>
<td>9/19</td>
<td>3/9</td>
<td>6/9</td>
</tr>
<tr>
<td>Vomitus</td>
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</tr>
<tr>
<td>20</td>
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</tr>
<tr>
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</tr>
<tr>
<td>24</td>
<td>Positive</td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>Total (no. positive/no. tested)</td>
<td>3/5</td>
<td>3/5</td>
<td>3/5</td>
</tr>
</tbody>
</table>

**NOTE.** NT, specimen not tested by this method.

and 4 of 12 patients showed seroconversion using the recombinant TV EIA.

**Discussion**

We used classic epidemiologic methods to trace this large outbreak of viral gastroenteritis among university freshmen to food served at a salad bar on 5 and 6 December 1994. Patients' symptoms were compatible with infection with SRSV and included vomiting, diarrhea, and abdominal cramping. Using molecular methods, we concluded that the SRSV strain detected in a fecal specimen of the salad chef was identical (in an 81-bp region) to the strain identified in both stool and vomitus of students in the outbreak, demonstrating a common infectious source and a plausible causal link to the salad chef. This observation suggests either that food contaminated and then served in the salad bar was the vehicle for virus transmission (and the salad chef became ill before others) or that the salad chef, who was already ill, contaminated the salad during preparation.
may be most challenging in settings in which close contact occurs between susceptible persons and case-patients, even when exposure to virus in food or water is prevented [27]. Application of molecular diagnostic techniques for SRSVs in ongoing outbreaks may pinpoint previously unknown sources of infection, allowing their elimination and preventing additional cases of gastroenteritis by interrupting transmission of SRSVs.

Recent advances in molecular virology have enabled investigators to delineate the phylogenetic relationship of SRSVs using RT-PCR, Southern hybridization, and nucleotide sequencing [15]. At the present time, ~39 SRSV strains have been classified into 4 phylogenetic genogroups on the basis of sequence analysis of a segment of the RNA polymerase gene [28]. The 4 genogroups are P1-A (prototype, NV), P1-B (no prototype), P2-A (prototype, TV), and P2-B (prototype, Snow Mountain virus). Because the virus sequenced in this outbreak was not identical in the RNA polymerase gene region sequenced to other viruses by our laboratory in previous outbreaks, it represents a unique SRSV strain.

The low rates of seroconversion we observed using highly sensitive assays to detect antibody against NV and TV and the fact that the virus in this outbreak was classified into a separate genogroup (P2-B) from NV or TV further suggest that the virus is antigenically distinct from either of these reference strains. In addition, data from our laboratory has shown 90%-100% seroconversion rates using the recombinant TV EIA for patients involved in outbreaks caused by SRSVs in the P2A genogroup [22].

Our results may be better understood in the context of limitations encountered during the study. Although the risk of illness associated with consumption of salad appears high and the salad chef developed gastroenteritis, food contamination by other ill kitchen workers may have occurred during handling or processing of items not identified during our interviews. Since the number of contact hours the salad chef had with food served in the salad bar greatly exceeded that of any other employee and the salad preparation room was separated from the rest of the kitchen, contamination by food handlers other than the salad chef appears unlikely. Because information collected in food histories may be subject to substantial recall bias, we limited the number of meals (i.e., 9 during the 3-day period) included in the survey to reduce the impact of recall bias in our analysis. The inclusion of controls on 8 and 10 December in the case-control analysis did not appear to introduce additional recall bias, since their recall histories appeared comparable to controls surveyed initially in the dormitory.

In the past, outbreak investigations of foodborne viral gastroenteritis have implicated consumption of salad, shellfish, and other prepared foods. This investigation is consistent with prior studies indicating that food handlers may transmit SRSV through contamination of food during preparation. As in previous oyster-related outbreaks of SRSV gastroenteritis, the present investigation demonstrates that infecting strains of virus identified in stool specimens can link cases occurring in a temporally related cluster [13, 14]. We may now track the transmission of SRSVs using our ability to identify etiologic agents from patients and food sources alike. Furthermore, the use of vomitus as a source for diagnostic material may enable identification of etiologies in future outbreaks that previously would have remained undiagnosed. Application of molecular techniques to identify and characterize SRSVs in conjunction with epidemiologic investigation may improve the diagnosis of SRSVs and suggest novel control measures for outbreaks in which illness propagates over time or the spread of illness resists routine interventions.

Acknowledgments

We thank John O’Connor for editorial assistance, Barbara Skane and Joseph Peppe for evaluation of bacteriologic specimens, John Fontana for virologic specimen processing, Emily Harvey, Richard Knowlton, Silvia Kreindel, and Ann Miller for data processing, and Michael Berry for university staff coordination.

References


