

# A large hotel outbreak of Norwalk-like virus gastroenteritis among three groups of guests and hotel employees in Virginia

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(Accepted 1 March 2002)

## SUMMARY

A large outbreak of acute gastroenteritis occurred among three different groups of guests and the employees of a Virginia hotel within a 2-week period in November 2000. At least 76 of the hotel's guests and 40 hotel employees had acute gastroenteritis during this period. All tested ill persons were infected with the same strain of Norwalk-like virus, as shown by cloning and sequencing of virus detected in stool specimens from the three guest groups and the employees. Epidemiologic investigation suggested food as the probable source for the guests. Most of the employees, including those sick, did not eat in the hotel, suggesting that environmental contamination and person-to-person transmission could have contributed to the outbreak. The disease continued to spread in the hotel, passing from one guest group to another, by food, environmental contamination, and/or by person-to-person transmission through infected employees and guests. The study describes procedures implemented to control the outbreak and makes recommendations for future outbreak control.

## INTRODUCTION

Human caliciviruses (HuCVs) are an important cause of acute gastroenteritis. The characterization of the viral genome of many HuCVs in the past 10 years has led to the classification of this group into two genera in the *Caliciviridae*, 'Norwalk-like viruses' (NLVs) and 'Sapporo-like viruses' (SLVs)[1]. NLVs are the major cause of non-bacterial outbreaks of acute gastroenteritis in children and adults. SLVs mainly infect children. Large outbreaks of NLV-associated gastroenteritis commonly occur in schools, childcare centers, nursing homes, hospitals, restaurants, cruise ships, and military facilities. The organism is transmitted by the faecal-oral route via contaminated food and water, environmental contamination, or by person-to-person transmission [2–18]. HuCVs are highly contagious and usually cause large outbreaks

with high attack rates. Three peaks of gastroenteritis cases were reported to the Peninsula Health Department (PHD) between 3 November 2000 and 9 November 2000. Each outbreak involved a different group of guests staying at a resort hotel. The first group (group A) consisted of 110 people attending a business conference. The second group (group B) consisted of 95 physicians and their families. The third group (group C) consisted of 350 retired persons. This paper describes the investigation of the outbreak of acute gastroenteritis among three groups of guests and employees in a large hotel that occurred over a 2-week period.

## METHODS

### Epidemiologic investigation

A cohort study was conducted with groups A and B using a questionnaire composed of demographic data, characteristics of illness, foods eaten at the hotel, and

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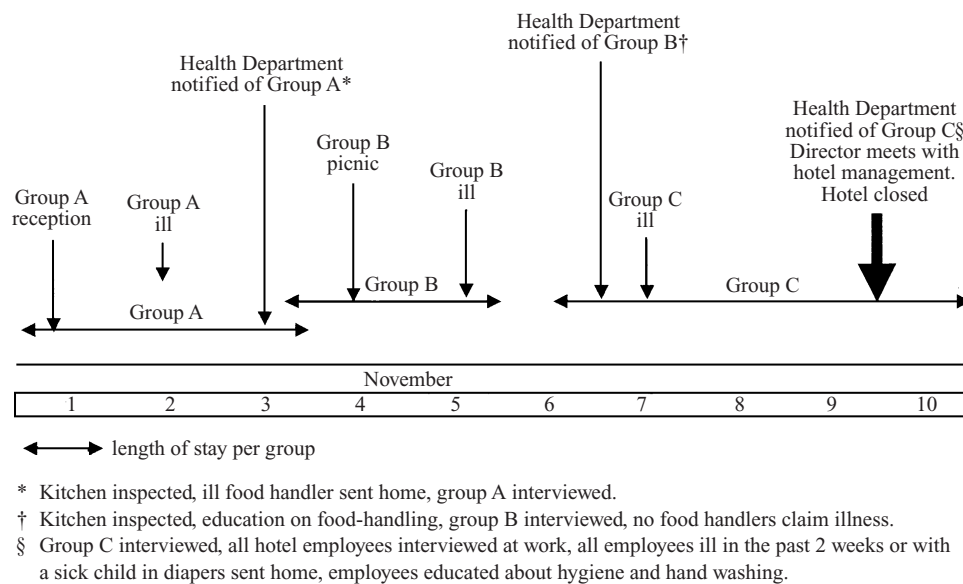


Fig 1. Time line

events attended. A case study was performed on group C and the hotel staff using a questionnaire administered by the PHD. A case was defined as vomiting or diarrhoea in a hotel attendee or staff, which occurred from 27 October to 14 November.

### Statistical analysis

Relative risks and confidence intervals were calculated using Epi Info 2000 software (Epi. Info, CDC, Atlanta, GA). Yates corrected Chi-square or Fisher exact test for statistical significance were performed using Epi Info 2000.

### Environmental investigation

After each outbreak was reported, the environmental health division of the PHD inspected the kitchen and obtained information about foods served, methods of food preparation, food sources, and food temperatures. Food handlers were asked about current illness and knowledge of food safety. Prior to the outbreak, hotel policy required that ill food handlers be excluded from food preparation or service until free of symptoms for 24 h. Any remaining foods served to the three groups were obtained for laboratory investigation. A time line of the investigation is shown in Fig. 1.

### Laboratory investigation

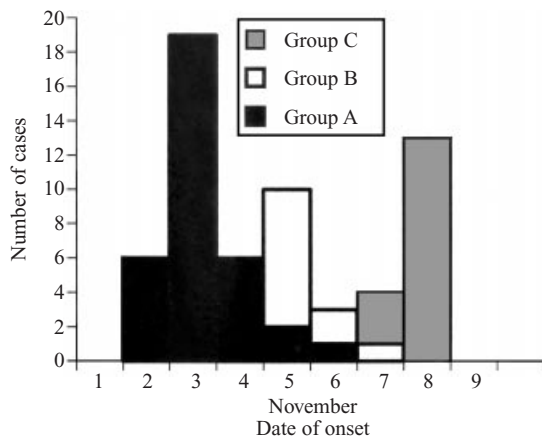
Stool specimens were collected from ill attendees of each group and two ill hotel employees. Stool

specimens were tested for HuCVs by reverse transcriptase – polymerase chain reaction (RT-PCR) at the Division of Consolidated Laboratory Services (DCLS) in Richmond and the Center for Pediatric Research (CPR), Eastern Virginia Medical School in Norfolk, and by an enzyme immune assay (EIA) at the CPR [19, 20]. This EIA utilizes hyperimmune sera obtained from animals cross-immunized with recombinant capsid antigens of nine strains of HuCVs. The stool specimens also were tested for Salmonella, Shigella, *Escherichia coli* O157: H7, Campylobacter, and Vibrio at DCLS. Stool specimens positive for HuCVs by RT-PCR were further characterized by cloning and sequencing using standard protocols described previously [19]. The sequences were analysed using Omega 2.0 (Oxford Molecular Ltd., Oxford, UK). One unopened bag of mussels was tested for HuCVs by RT-PCR. No other food served to the groups was available for testing.

## RESULTS

### Epidemiologic investigation

A total of 69 (63%) attendees of group A completed the questionnaire. Thirty-four met the case definition for illness for an attack rate of 49%. Ill persons ranged in age from 37–62 years with a median of 52 years and 20 (59%) were female. Twenty-seven (28%) of the 95 attendees of group B completed the questionnaire and 11 (41%) satisfied the case definition. The median age of ill persons was 31 years and 8



**Fig 2.** Onset of acute gastroenteritis among three guest groups

(73%) were female. Group C had 15 persons who satisfied the case definition. The median age of the ill persons was 71 years and 9 (60%) were female. Three peaks of cases occurred which matched the arrival in the hotel by each group (Fig. 2). The first case in each group occurred within 24 h of arrival and the case numbers peaked at 24–48 h. Illness among employees occurred during the 2 week period from 27 October to 10 November. Predominant symptoms of ill persons among the three groups of guests and the employees were similar (Table 1). The median duration of illness was 31 h (range: 18–72 h). No interviewed attendees were symptomatic before the event.

#### Mode of transmission of illness

Group A arrived in the hotel on 1 November. The hotel catered a reception for group A on 1 November (Fig. 1). Twenty-seven of the 45 (60%) people who attended the reception became ill compared with 7 of the 24 (29%) who did not attend the reception (Relative risk = 2.1,  $P = 0.03$ ; 95% CI = 1.1, 4.0). No other event for group A had a statistically increased risk of illness. The explosive nature of the outbreak, with large numbers of cases within a 24–48 h cluster, suggested a common food source exposure. The investigation, however, did not implicate any specific food item. Group B arrived at the hotel on 3 November. The hotel catered breakfast and a picnic lunch for the group on 4 November. The group ate together at an outside restaurant on 4 November. The interview participation rate for attendees in group B was poor (only 28% completed the questionnaire). There was a significant association between eating coleslaw at the picnic on 4 November and becoming ill. Nine of the 15 (60%) attendees who ate coleslaw

at the picnic became ill compared to 2 of the 10 (17%) who did not eat coleslaw (RR = 3.6,  $P = 0.03$ ; 95% CI = 1.0, 13.6). Fifteen ill members of group C were interviewed. Because of the difficulties in recalling foods eaten, a food history was not obtained for group C.

#### Role of employees in transmission of the illness

During the initial outbreak investigation, only three employees claimed to be ill. Two of these were food handlers. On 9 November the health director met with hotel management to arrange interviews for all employees. The management agreed to pay ill employees while out of work and to encourage ill employees not to work. A total of 150 hotel employees were interviewed on 9 and 10 November. Forty (27%) satisfied the case definition for illness in the 2-week period of the outbreak. The highest attack rates were among persons who worked at the front desk (7/9, 78%) followed by housekeeping staff (9/22, 41%). Thirteen of 67 (19%) food handlers and 1/6 (17%) dishwashers were ill. Because infected hotel employees could transmit the disease, it was requested that ill employees not work if symptomatic. Personal hygiene was reinforced and food handlers were particularly instructed on the importance of not working while ill.

#### Environmental investigation

The environmental investigation revealed no major deficiencies in food storage, preparation, or service. Food handlers seemed knowledgeable about safe food handling techniques.

#### Laboratory investigation

Stool specimens obtained from 3 ill attendees in group A, 2 ill attendees and 1 family member in group B, 3 ill attendees in group C, and 1 ill employee were positive for Norwalk-like virus by RT-PCR. PCR results were confirmed by the recombinant EIA. All stool specimens were negative for bacterial pathogens. The frozen mussels tested negative for Norwalk-like virus by RT-PCR.

The RT-PCR products from positive specimens selected from each of the three groups of guests and the employee had an identical sequence identified as VA750. This strain shares 100% and 96% nucleic acid identities with a Japanese strain (AB05308) and a Hungarian strain (MOH), respectively. Similar strains

Table 1. Frequency of patients with acute gastroenteritis symptoms among the three groups of guests and the hotel employees in the outbreak

Symptoms	Group A No. = 34 (%)	Group B No. = 11 (%)	Group C No. = 15 (%)	Employees No. = 40 (%)
Diarrhoea	32 (94)	9 (82)	11 (73)	37 (93)
Vomiting	25 (74)	11 (100)	13 (87)	29 (73)
Nausea	30 (88)	11 (100)	8 (57)	ND*
Fever	8 (24)	6 (55)	2 (13)	23 (58)
Abdominal cramp	21 (64)	9 (82)	ND*	ND*

ND, not documented.

also were identified in other Virginia outbreaks recently (our unpublished data) and in the UK in the early 1990s (NLV/Hillingdon/90/UK). Genetic analysis of these related strains showed that they belong to the genogroup II of HuCVs, currently the most commonly circulating genogroup in many areas around the world [21–25].

#### Measures to control the outbreak

On 3 and 7 November the Peninsula Health Department requested that all ill employees go home and not return to work until free of symptoms for 24 h. A training session on personal hygiene, proper glove use, and food protection measures was given to workers on 7 November. On 9 November all employees were interviewed. Those who were ill in the past 2 weeks or had an ill child in diapers were excluded from work for 1 day. Those employees who were currently ill with vomiting or diarrhoea were told not to work for 1 day after resolution of symptoms. All employees were paid for time off while ill. All employees were instructed about hygiene and hand washing. The facility was closed for 8 h to permit thorough cleaning of all food service areas and guest rooms. New guests were not accepted until all guestrooms, bathrooms, and common rooms were thoroughly cleaned. All cold food requiring hand-preparation was excluded from the menu. No open bowls of food such as chips or popcorn were served. On 14 November the hotel reported no further ill guests or employees.

#### DISCUSSION

This report describes an outbreak of acute gastroenteritis among three groups of guests and the employees in a large hotel. The first cases occurred within 24 h after the first group arrived in the hotel

and the numbers of cases increased sharply within 24–48 h. Forty (27%) of the 150 hotel employees also were sick. Ill guests and employees had similar symptoms. The outbreak finally stopped after a major cleaning in the hotel followed by an 8 h closure to guests. The following is a discussion about possible transmission modes of the virus and recommendations for future outbreak control and prevention.

The explosive nature of the outbreak suggested that a common source of illness occurred for groups A, B and C. Water was not considered, because the hotel is on a municipal water system and no other outbreaks were reported in the surrounding community. Food was the likely source for most hotel guests as shown by the epidemiologic investigation. Direct evidence of food contamination was not obtained because most of the foods had been discarded when the investigation started. One unopened bag of frozen mussels tested negative for Norwalk-like virus. Thirteen hotel food handlers became ill in the 2-week period with vomiting or diarrhoea. Sub-clinical carriers also can shed virus. The link between illness and food contamination can be explained for the guests, but not the employees, because most employees did not dine in the hotel. Furthermore, some guests who did not eat in the hotel also became ill. Therefore, other pathways, such as person-to-person transmission and environmental surface contamination probably contributed to the spread of disease. The highest attack rates of employees were those who had direct contact with guests, including front desk receivers (78%) and housekeepers (41%). Contamination of the guest bathrooms is a probable source of infection. The housekeepers were asked to clean the surface of the bathrooms. However, wiping of surfaces without disinfectant and using the same rag elsewhere could actually help to spread the pathogen. The practice observed was to use the same cleaning materials and gloves between rooms.

The three groups of guests had only a 1-day overlap in the hotel; each had its own social activities and menus, but the strains detected from all three groups were identical and matched the strains among the hotel employees. The initial public health interventions were not effective in controlling the outbreak because ill food handlers were not initially identified and those identified were not kept out of work for longer than 24 h after becoming asymptomatic. In addition, 72 h would have been more effective in stopping the disease transmission based on the current literature [26]. The investigating team relied on the food handlers to voluntarily remove themselves from work. This was not effective and finally when they were interviewed on 9 November it was discovered that 13 food handlers had been ill during the time of the outbreak. They did not admit to illness either because they were asymptomatic at the time of interview or because they did not want to miss work. Prolonged viral shedding (up to 2 weeks) can increase the risk of a food handler infecting others [27]. In addition a 1994 study of NLV showed that of 50 volunteers infected with NLV, 32% asymptomatic and viral shedding was observed in both symptomatic and asymptomatic persons [28].

Recommendations to control future outbreaks are as follows. Interviews on all hotel employees, particularly food service workers, should be performed at the time the outbreak is recognized. All persons involved in food handling, who experience vomiting or diarrhoea, should be required not to work for at least 72 h after becoming free of symptoms. Hotel bathrooms should be thoroughly cleaned with phenolic compounds with particular attention to those used by ill guests [29].

Housekeeping personnel should be instructed on safe personal hygiene when cleaning contaminated guest bathrooms and the importance of not cross-contaminating guest bathrooms. Both hotel staff and attendees should practice effective hand washing after using the bathroom. Safe food handling should be practiced particularly for foods served cold or requiring hand preparation.

#### ACKNOWLEDGEMENTS

We wish to acknowledge D. Warren, A. Jindal, J. Monroe, P. Pickard, P. Parham, L. Rose, S. Windley, L. Nycum, D. Jordan, D. Toney, M. Mismus, J. Sharp, and C. Mitchell for their contributions to this investigation.

We thank Ms Helen Frankum for her technical support of the study.

#### REFERENCES

- Green KY, Ando T, Balayan MS, et al. Taxonomy of the caliciviruses. *J Infect Dis* 2000; **181** (Suppl. 2): S322–30.
- Ang LH. An outbreak of viral gastroenteritis associated with eating raw oysters [published erratum appears in *Commun Dis Public Health* 1998 Jun; 1(2): 140]. *Commun Dis Public Health* 1998; **1**: 38–40.
- Appleton H. Small round viruses: classification and role in food-borne infections. *Ciba Found Symp* 1987; **128**: 108–25.
- Appleton H. Control of food-borne viruses. *Br Med Bull* 2000; **56**: 172–83.
- Bean NH, Goulding JS, Lao C, Angulo FJ. Surveillance for foodborne-disease outbreaks – United States, 1988–1992. *MMWR* 1996; **45**: 1–73.
- Caul EO, Ashley CR, Curry A. Foodborne gastroenteritis due to Norwalk virus in a Winnipeg hotel. *Can Med Assoc J* 1990; **142**: 290.
- Clover DO. Virus transmission via food. *World Health Stat Q* 1997; **50**: 90–101.
- Fleet GH, Heiskanen P, Reid I, Buckle KA. Foodborne viral illness-status in Australia. *Intl J Food Microbiol* 2000; **59**: 127–36.
- Glass RI, Noel J, Ando T, et al. The epidemiology of enteric caliciviruses from humans: a reassessment using new diagnostics. *J Infect Dis* 2000; **181** (Suppl. 2): S254–61.
- Baron RC, Murphy FD, Greenberg HB, et al. Norwalk gastrointestinal illness: an outbreak associated with swimming in a recreational lake and secondary person-to-person transmission. *Am J Epidemiol* 1982; **115**: 163–72.
- Beller M, Ellis A, Lee SH, et al. Outbreak of viral gastroenteritis due to a contaminated well. *International consequences*. *JAMA* 1997; **278**: 563–8.
- Berg DE, Kohn MA, Farley TA, McFarland LM. Multi-state outbreaks of acute gastroenteritis traced to fecal-contaminated oysters harvested in Louisiana. *J Infect Dis* 2000; **181** (Suppl. 2): S381–6.
- Brugha R, Vipond IB, Evans MR, et al. A community outbreak of food-borne small round-structured virus gastroenteritis caused by a contaminated water supply. *Epidemiol Infect* 1999; **122**: 145–54.
- Caceres VM, Kim DK, Bresee JS, et al. A viral gastroenteritis outbreak associated with person-to-person spread among hospital staff. *Infect Control Hosp Epidemiol* 1998; **19**: 162–7.
- Corwin AL, Soderquist R, Edwards M, et al. Shipboard impact of a probable Norwalk virus outbreak from coastal Japan. *Am J Trop Med Hyg* 1999; **61**: 898–903.
- Daniels NA, Bergmire-Sweet DA, Schwab KJ, et al. A foodborne outbreak of gastroenteritis associated with Norwalk-like viruses: first molecular traceback to deli sandwiches contaminated during preparation. *J Infect Dis* 2000; **181**: 1467–70.

17. Hedberg CW, Osterholm MT. Outbreaks of foodborne and waterborne viral gastroenteritis. *Clin Microbiol Rev* 1993; **6**: 199–210.
18. Cheesebrough JS, Green J, Gallimore CI, Wright PA, Brown DWG. Widespread environmental contamination of Norwalk-like viruses (NLV) detected in a prolonged hotel outbreak of gastroenteritis. *Epidemiol Infect* 2000; **125**: 93–8.
19. Farkas T, Jiang X, Guerrero ML, et al. Prevalence and genetic diversity of human caliciviruses (HuCVs) in Mexican children. *J Med Virol* 2000; **62**: 217–23.
20. Jiang X, Huang PW, Zhong WM, Farkas T, Cubitt DW, Matson DO. Design and evaluation of a primer pair that detects both Norwalk- and Sapporo-like caliciviruses by RT-PCR. *J Virol Meth* 1999; **83**: 145–54.
21. Jiang X, Wilton N, Zhong WM, et al. Diagnosis of human caliciviruses by use of enzyme immunoassays. *J Infect Dis* 2000; **181** (Suppl. 2): S349–59.
22. Fankhauser RL, Noel JS, Monroe SS, Ando T, Glass RI. Molecular epidemiology of Norwalk-like viruses in outbreaks of gastroenteritis in the United States. *J Infect Dis* 1998; **178**: 1571–8.
23. Fankhauser RL, Monroe SS, Humphrey CD, Glass RI. Molecular epidemiology of 'Norwalk-like viruses' (NLV) in outbreaks of gastroenteritis in the U.S. 19th Annual Meeting of the American Society for Virology, 2000.
24. Vinje J, Koopmans MP. Molecular detection and epidemiology of small round-structured viruses in outbreaks of gastroenteritis in the Netherlands. *J Infect Dis* 1996; **174**: 610–5.
25. Vinje J, Altena SA, Koopmans MPG. The incidence and genetic variability of small round-structured viruses in outbreaks of gastroenteritis in The Netherlands. *J Infect Dis* 1997; **176**: 1374–8.
26. LeBaron CW, Furutan NP, Lew JF, et al. Viral agents of gastroenteritis: public health importance and outbreak management. *MMWR* 1990; **39** (RR-5): 1–24.
27. Okhuysen PC, Jiang Xi, Ye L, Johnson PC, Estes MK. Viral shedding and fecal IgA response after Norwalk virus infection. *J Infect Dis* 1995; **171**: 566–9.
28. Graham DY, Jiang X, Tanaka T, Openkun AR, Madore HP, Estes MK. Norwalk virus; infection of volunteers: new insights based on improved assays. *J Infect Dis* 1994; **170**: 34–43.
29. Gulati BR, Allwood PB, Hedberg CW, Goyal SM. Efficacy of commonly used disinfectants for the inactivation of calicivirus on strawberry, lettuce, and a food-contact surface. *J Food Protect* 2001; **64**: 1430–4.