JOURNAL OF CLINICAL MICROBIOLOGY, Sept. 2008, p. 3112–3115 Vol. 46, No. 9
0095-1137/08/$08.00+0 doi:10.1128/JCM.00400-08

Contamination of the Hospital Environment with Gastroenteric Viruses: Comparison of Two Pediatric Wards over a Winter Season▽

Chris I. Gallimore,[1]* Clive Taylor,[2] Andrew R. Gennery,[3] Andrew J. Cant,[3] Angela Galloway,[4] Jacqueline Xerry,[1] Juliet Adigwe,[1] and Jim J. Gray[1]

Enteric Virus Unit, Virus Reference Department, Centre for Infections, Health Protection Agency, Colindale, London, United Kingdom[1]; Newcastle Laboratory, Health Protection Agency North East, Newcastle General Hospital, Newcastle, United Kingdom[2]; Primary Immunodeficiency Unit, Newcastle General Hospital, Newcastle, United Kingdom[3]; and Department of Microbiology, Royal Victoria Infirmary, Newcastle, United Kingdom[4]

Received 28 February 2008/Returned for modification 27 April 2008/Accepted 1 July 2008

The aims of this study were to examine the extent of gastroenteric virus contamination in a pediatric primary immunodeficiency (PPI) ward and a general pediatric ward over a winter season and to determine whether changes to hospital infection control interventions would have an impact on environmental contamination levels within pediatric units. Environmental swabs were collected weekly from 11 sites in both wards from 15 December 2005 to 3 March 2006 and examined for the presence of norovirus (NoV), astrovirus, and rotavirus (RV) by reverse transcriptase PCR. Viruses were detected in 17% and 19% of swabs from both wards. Virus contamination for NoV and RV decreased from 20% to 6% and 15% to 10% of swabs, respectively, in the PPI ward from the 2004 study by Gallimore et al. (C. I. Gallimore, C. Taylor, A. R. Gennery, A. J. Cant, A. Galloway, M. Iturriza-Gomara, and J. J. Gray, J. Clin. Microbiol. 44:395–399, 2006). Overall, changes to cleaning protocols were deemed to have reduced the level of environmental contamination with gastroenteric viruses, but contamination still occurred due to a breakdown in infection control procedures indicated by contamination in areas frequented by parents but used only occasionally by staff.

Viral gastroenteritis in children is usually caused by rotaviruses (RVs), noroviruses (NoVs), sapoviruses (SaVs), astroviruses (AstVs), and enteric adenoviruses (AdVs). RVs are the leading cause of gastroenteritis in children under 5 years of age worldwide (14). With the introduction of improved detection assays, the roles of NoVs and SaVs are becoming more apparent (12). These viruses are frequently associated with diarrhea and vomiting in children under 5 years of age, and sporadic cases of viral gastroenteritis in hospitalized children (2, 10, 11, 18) and children attending day care centers (15) have been reported. NoVs are a major cause of outbreaks of gastroenteritis in adults in semiclosed institutions, including hospitals (3) and nursing/retirement homes (7).

Transmission of gastroenteric viruses is usually through person-to-person spread by the fecal-oral route. Environmental transmission of NoVs, RVs, and AstVs (4–6) in hospitals has been reported and involves contaminated work surfaces, floors, medical equipment, light switches, taps, door handles, and television/game consoles.

The prevalence of RV on high-risk fomites in day care facilities and RV environmental contamination in a pediatric unit have been reported (17). AstV environmental contamination in hospitals has not been widely reported (5), although several groups have examined the prevalence of AstV in hospitalized children with acute gastroenteritis and children attending day care centers (15, 16). The extent of environmental contamination with multiple gastroenteric viruses in hospital wards and the effect they have on patient morbidity are difficult to determine.

This study was undertaken after a previous study examining environmental contamination with NoVs, AstVs, and RVs in a pediatric primary immunodeficiency unit (PPIU) over a 6-month period in 2004 (4). The same gastroenteric viruses were tested for in the present study, but environmental swabs were collected from the PPIU (ward 23) and a general pediatric ward (ward 24) during the winter of 2005/2006 to determine whether hospital infection control interventions would have an impact on environmental contamination levels within pediatric units. There was no evidence of gastroenteritis in patients in the unit during this study.

Environmental swabs were collected weekly from 11 similar, but not identical, sites in both wards (identical swabs could not be taken, as the ward layouts were different) from 15 December 2005 to 3 March 2006 (Tables 1 and 2). Detection of NoV, AstV, and RV was performed using previously published protocols (4). Briefly, nucleic acid was extracted from swabs using a guanidinium isothiocyanate-silica procedure, and then the nucleic acid was converted to cDNA using random primers. Specific nested and heminested PCR assays for genogroup II (GII) NoV strains, AstVs (genotypes 1 to 8), and group A RVs were performed on cDNA (4). Primer sets GIIFB1, GIIFB2, GIIFB3/GIISKR (9), and GIIFBN/GIISKR (1) were used to detect NoVs, primer pairs Mon269/Mon270 (13) and Mon269N/Mon270 (5) were used to detect AstVs, and primer pairs VP6-F/VP6-R (8) and VP6NF/VP6NR (4) were used to detect group A RVs. Genotyping for group A RV was not performed. PCR machine conditions were as previously stated (4). Standard control measures and unidirectional work flow were used for all nested PCRs with the second-round PCR setup per-

* Corresponding author. Mailing address: Enteric Virus Unit, Virus Reference Department, Centre for Infections, Health Protection Agency, Colindale, London NW9 5HT, United Kingdom. Phone: 44-208-327-7795. Fax: 44-208-205-8195. E-mail: christopher.gallimore@hpa.org.uk.

▽ Published ahead of print on 9 July 2008.

TABLE 1. Combined enteric virus PCR results on environmental swabs for NoV, AstV, and RV detected in a pediatric primary immunodeficiency ward (ward 23)

Environmental swab site	Combined enteric virus PCR result for swab batch collected on the indicated date (day/mo/yr)[a]											
	A (15/12/05)	B (22/12/05)	C (30/12/05)	D (6/1/06)	E (13/1/06)	F (18/1/06)	G (27/1/06)	H (3/2/06)	I (7/2/06)	J (17/2/06)	K (24/2/06)	L (3/3/06)
Staff telephone[b]	−	−	−	−	−	+[e]	−	+[f]		−	−	−
Room 4 outside flow switch[b,h]	−	−	−	−	−	−	−	+[f]	−	−	−	−
Room 3 outside flow switch[b,h]	−	−	−	−	−	−	+[f]	−	−	+[d]	−	−
Parent's room handle[c]	−	−	−	−	−	−	−	−	−	−	−	−
Parent's television[c]	−	−	−	−	−	−	+[f]	+[f]	+[f]	+[d]	−	−
Parent's mobile telephone[c]	+[d]	−	−	−	−	−	−	−	−	+[d]	−	−
Parent's toilet door handle[c]	−	−	−	−	−	−	+[f]	−	−	−	−	−
Parent's toilet tap[c]	−	−	−	−	−	−	+[f]	+[f]	+[f]	−	−	−
Staff toilet tap[b]	−	−	−	−	−	−	−	+[f]	−	−	−	−
Staff toilet light switch[b]	−	−	−	−	−	−	−	−	−	+[d]	−	−
Microwave oven[c]	−	−	−	−	−	−	+[f]	+[f,g]	−	+[d]	−	−

[a] Combined enteric virus PCR result for NoV, AstV, and RV on environmental swabs for batchs A to L. Symbols: −, negative; +, positive.
[b] Environmental swab site frequented by staff.
[c] Environmental swab site frequented by parents.
[d] NoV GII-4 detected.
[e] NoV GII-1 detected.
[f] RV detected.
[g] Positive result in single-round PCR.
[h] Flow switch refers to an outside laminar flow curtain syringe pump switch.

formed in a PCR workstation with UV decontamination (4). Water controls were used in each assay as negative controls. No fecal specimens were collected for PCR during this study.

Changes were introduced in the cleaning regimen for patients' rooms in ward 23. Traditionally, nursing staff damp dusted the patient area and washed the floor in each room with the support and assistance of the child's carers. Disposable detergent wipes (Cutan; DEB Ltd., Belper, United Kingdom)

TABLE 2. Combined enteric virus PCR results on environmental swabs for NoV, AstV, and RV detected in a general pediatric ward (ward 24)

Environmental swab site	Combined enteric virus PCR result for swab batch collected on the indicated date (day/mo/yr)[a]											
	A (15/12/05)	B (22/12/05)	C (30/12/05)	D (6/1/06)	E (13/1/06)	F (18/1/06)	G (27/1/06)	H (3/2/06)	I (7/2/06)	J (17/2/06)	K (24/2/06)	L (3/3/06)
Staff telephone[b]	−	−	−	−	−	−	−	−	−	−	−	−
Room 2 light switch[b]	+[d]	+[f]	−	−	−	−	−	+[f,h]	+[e]	+[f,h]	−	−
Treatment room tap[b]	−	−	−	−	−	−	−	+[f]	+[f]	+[i]	−	−
Parent's room door handle[c]	−	−	−	−	+[f]	−	−	−	−	+[e]	−	−
Parent's room television[c]	−	−	−	−	−	−	−	−	−	−	−	−
Public telephone[c]	−	+[g,h]	−	−	−	−	−	+[f]	−	−	+[e]	−
Parent's toilet door handle[c]	−	−	−	−	−	−	−	+[f]	−	−	−	−
Parent's toilet tap[c]	−	−	−	−	−	−	−	+[f]	−	−	−	−
Staff toilet tap[b]	+[d]	−	−	−	−	−	+[f]	+[f]	−	−	−	−
Staff toilet light switch[b]	+[e]	−	−	−	−	−	−	+[f]	+[f]	−	−	−
Microwave oven[c]	−	−	−	−	−	−	+[f]	−	+[f]	−	−	−

[a] Combined enteric virus PCR result for NoV, AstV, and RV on environmental swabs for batchs A to L. Symbols: −, negative; +, positive.
[b] Environmental swab site frequented by staff.
[c] Environmental swab site frequented by parents.
[d] AstV-2 detected.
[e] NoV GII-4 detected.
[f] RV detected.
[g] AstV-1 detected.
[h] Positive result in single-round PCR.
[i] NoV GII-7 detected.

were used. The floor mop was used in the patient's area (within the laminar flow curtain) and then in the remainder of the patient's room. Ancillary staff washed surfaces outside the patient's area and mopped the floor twice daily with detergent (Youngs Hospec; The Darcy Group, Warrington, United Kingdom) using washable mops.

This was changed to allow ancillary staff access to the patient's area for the first time (due to restrictions of access). The floor was cleaned daily, the surfaces inside and outside the patient's area were cleaned twice daily using microfiber cloths (Ecolab, Swindon, United Kingdom), and glass screens and windows were cleaned weekly. All equipment within ward 23 (infusion pumps, etc.) were cleaned daily by the health care assistants, and a sign off list of tasks was placed in each room to indicate who cleaned what.

Viruses were detected by PCR in 20/121 (17%) and 23/121 (19%) of environmental swabs taken from wards 23 and 24 during the course of this study, respectively. In ward 23, NoV GII genotype 4 (GII-4) was detected in 6/121 (5%) swabs, NoV GII-1 was detected in only 1 swab, and RV was detected in 13/121 swabs (10%). No AstVs were detected in ward 23. Similarly, in ward 24, NoV GII-4 was detected in 4/121 (3%) swabs, NoV GII-7 was found in only 1 swab, RV was detected in 15/121 swabs (12%), AstV genotype 1 (AstV-1) was found in only one swab, and AstV-2 was detected in two swabs. In four swabs, enteric viruses (one RV from ward 23 and two RVs and one AstV-1 from ward 24) were detected by single-round PCR. No enteric viruses were detected in swabs taken from ward 23 between 22 December 2005 and 13 January 2006 and between 24 February 2006 and 3 March 2006; similarly, in ward 24, no enteric viruses were detected in swabs taken between 30 December 2005 and in swabs taken 6 January 2006, 18 January 2006, and 3 March 2006 (Tables 1 and 2). In ward 23, the most contaminated swabs were taken 27 January 2006 and 3 February 2006, with five and six swabs in which RV was detected, respectively, and 17 February 2006, with five NoV GII-4-positive swabs. Similarly, in ward 24, seven swabs collected 3 February 2006 were positive for RV, and four swabs collected 7 February 2006 were positive for RV.

The only environmental swabbing site in ward 23 not to have enteric virus detected during the study was the parent's room door handle, whereas in ward 24, the staff telephone and the parent's room television were the only negative sites. Interestingly, the swab sites that were most frequently contaminated in ward 23 were the parent's room television, parent's toilet tap, and microwave, and in ward 24, they were room 2 light switch, treatment room taps, and the public telephone, all of which had three or four positive swabs. The public telephone in ward 24 had NoV GII-4, RV, and AstV-1 detected on at least one occasion; all other swab sites that had positive results had only one or two enteric viruses detected.

NoV GII-4 v3 (variant 3) strains were genotyped from swabs; there were different strains of these viruses based on the gene encoding the S-domain sequencing. When these three subtypes were compared to outbreak strains cocirculating in the United Kingdom, only one strain found on swabs JN3, JN5, JN6, JN10, JN11, and KN17 was seen commonly in outbreaks throughout the United Kingdom. Comparisons between ward 23, the PPIU, from the previous published study (4) and in this study show a reduction in the level of NoV environmental contamination from 20% to 6% for the sampling period March 2004 to October 2004 (4) and December 2005 to March 2006 (this study), respectively. There was also a reduction in RV contamination from 15% to 10%. No comparable data for ward 24 were available for the 2004 study.

It was noted in this study that ward 23 had a peak of environmental contamination from 27 January 2006 to 17 February 2006, with 41% of swabbing samples positive for one of the three enteric viruses; 90% of the ward 23 positive results were within this period. During the same period, ward 24 also had its peak of enteric virus environmental contamination, with 36% of swabs positive; however, only 70% of all the positive swabs were detected during this time. In both wards during this peak that lasted 3 weeks, 26/34 (76%) of the swabs were positive for RV.

Seven environmental swab sites out of 20 (35%) positive for enteric viruses were frequented by staff, whereas in those sites more likely to be frequented by parents, virus was found in 13/20 swab sites (65%) (Table 1).

Overall, it was shown that environmental sites contaminated with NoV were often associated with parents trafficking throughout the unit, which may indicate that hand washing was not performed. In contrast, there was a reduction in NoV at environmental sites frequented mainly by staff, suggesting that contamination by staff had been reduced through the introduction of improved hygiene measures since the first study. Ward staff had the impression that some parents were more diligent in washing their hands when visiting their own children, as they wanted to protect them from infection, but they were less rigorous about washing their hands when leaving their children, as they could see no direct benefit. Although the two surveys were conducted at different times of the year, the reduction in environmental contamination appears significant, as this reduction was seen during a period of high incidence of gastroenteritis in the community.

Nucleotide sequence accession numbers. GenBank accession numbers are EU735553 for NoV GII-7 strain JN14/2006/UK (ward 24 treatment room taps), EU735554 for GII-1 strain FN1/2006/UK (ward 23 staff phone), EU735555 for GII-4 strain JN3/2006/UK (ward 23, room 3, outside flow switch), EU735556 for GII-4 strain JN15/2006/UK (ward 24, parent's room door handle), and EU735557 for GII-4 strain AN6/2005/UK (parent's mobile phone). These strains are representatives of the diversity of NoV strains genotyped in this study.

We thank Lesley Wilson and Angela Reed, Infection Control Nurses in Newcastle upon Tyne Hospitals NHS Foundation Trust for carrying out the environmental swabbing.

REFERENCES

1. **Gallimore, C. I., J. S. Cheesbrough, K. Lamden, C. Bingham, and J. J. Gray.** 2005. Multiple norovirus genotypes characterised from an oyster-associated outbreak of gastroenteritis. Int. J. Food Microbiol. **103:**323–330.
2. **Gallimore, C. I., D. Cubitt, A. Richards, and J. J. Gray.** 2004. Diversity of enteric viruses detected in patients with gastroenteritis in a tertiary referral paediatric hospital. J. Med. Virol. **73:**443–449.
3. **Gallimore, C. I., J. Green, D. Lewis, A. F. Richards, B. A. Lopman, A. D. Hale, R. Eglin, J. Gray, and D. W. G. Brown.** 2004. Diversity of noroviruses cocirculating in the North of England from 1998 to 2001. J. Clin. Microbiol. **42:**1396–1401.
4. **Gallimore, C. I., C. Taylor, A. R. Gennery, A. J. Cant, A. Galloway, M. Iturriza-Gomara, and J. J. Gray.** 2006. Environmental monitoring for gas-

troenteric viruses in a primary immunodeficiency unit. J. Clin. Microbiol. **44:**395–399.
5. **Gallimore, C. I., C. Taylor, A. R. Gennery, A. J. Cant, A. Galloway, D. Lewis, and J. J. Gray.** 2005. Use of a heminested reverse transcription PCR assay for detection of astrovirus in environmental swabs from an outbreak of gastroenteritis in a pediatric primary immunodeficiency unit. J. Clin. Microbiol. **43:**3890–3894.
6. **Green, J., P. A. Wright, C. I. Gallimore, O. Mitchell, P. Morgan-Capner, and D. W. G. Brown.** 1998. The role of environmental contamination with small round structured viruses in a hospital outbreak investigated by reverse-transcriptase polymerase chain reaction assay. J. Hosp. Infect. **39:**39–45.
7. **Green, K. Y., G. Belliot, J. L. Taylor, J. Valdesuso, J. F. Lew, A. Z. Kapikian, and F. Y. Lin.** 2002. A predominant role for Norwalk-like viruses as agents of epidemic gastroenteritis in Maryland nursing homes for the elderly. J. Infect. Dis. **185:**133–146.
8. **Iturriza Gómara, M., C. Wong, S. Blome, U. Desselberger, and J. Gray.** 2002. Molecular characterization of VP6 genes of human rotavirus isolates: correlation of genogroups with subgroups and evidence of independent segregation. J. Virol. **76:**6596–6601.
9. **Kageyama, T., S. Kojima, M. Shinohara, K. Uchida, S. Fukushi, F. B. Hoshino, N. Takeda, and K. Katayama.** 2003. Broadly reactive and highly sensitive assay for Norwalk-like viruses based on real-time quantitative reverse transcription-PCR. J. Clin. Microbiol. **41:**1548–1557.
10. **Kirkwood, C. D., and R. F. Bishop.** 2001. Molecular detection of human calicivirus in young children hospitalized with acute gastroenteritis in Melbourne, Australia, during 1999. J. Clin. Microbiol. **39:**2722–2724.
11. **Kirkwood, C. D., R. Clark, N. Bogdanovic-Sakran, and R. F. Bishop.** 2005. A 5-year study of the prevalence and genetic diversity of human caliciviruses associated with sporadic cases of acute gastroenteritis in young children admitted to hospital in Melbourne, Australia (1998–2002). J. Med. Virol. **77:**96–101.
12. **Moreno-Espinosa, S., T. Farkas, and X. Jiang.** 2004. Human caliciviruses and pediatric gastroenteritis. Semin. Pediatr. Infect. Dis. **15:**237–245.
13. **Noel, J. S., T. W. Lee, J. B. Kurtz, R. I. Glass, and S. S. Monroe.** 1995. Typing of human astroviruses from clinical isolates by enzyme immunoassay and nucleotide sequencing. J. Clin. Microbiol. **33:**797–801.
14. **Parashar, U. D., E. G. Hummelman, J. S. Bresee, M. A. Miller, and R. I. Glass.** 2003. Global illness and deaths caused by rotavirus disease in children. Emerg. Infect. Dis. **9:**565–572.
15. **Rosenfeldt, V., T. Vesikari, X. L. Pang, S. Q. Zeng, M. Tvede, and A. Paerregaard.** 2005. Viral etiology and incidence of acute gastroenteritis in young children attending day-care centers. Pediatr. Infect. Dis. J. **24:**962–965.
16. **Shastri, S., A. M. Doane, J. Gonzales, U. Upadhyayula, and D. M. Bass.** 1998. Prevalence of astroviruses in a children's hospital. J. Clin. Microbiol. **36:**2571–2574.
17. **Soule, H., O. Genoulaz, B. Gratacap-Cavallier, M. R. Mallaret, P. Morand, P. Francois, D. Luu Duc Bin, A. Charvier, C. Bost-Bru, and J. M. Seigneurin.** 1999. Monitoring rotavirus environmental contamination in a pediatric unit using polymerase chain reaction. Infect. Control Hosp. Epidemiol. **20:**432–434.
18. **Zintz, C., K. Bok, E. Parada, M. Barnes-Eley, T. Berke, M. A. Staat, P. Azimi, X. Jiang, and D. O. Matson.** 2005. Prevalence and genetic characterization of caliciviruses among children hospitalized for acute gastroenteritis in the United States. Infect. Genet. Evol. **5:**281–290.