J. Hyg., Camb. (1986), **96**, 277–289
Printed in Great Britain

Institutional outbreaks of rotavirus diarrhoea: potential role of fomites and environmental surfaces as vehicles for virus transmission

By SYED A. SATTAR, NELLIE LLOYD-EVANS, V. SUSAN SPRINGTHORPE

From Department of Microbiology and Immunology, School of Medicine, University of Ottawa, Ottawa, Ontario K1H 8M5, Canada

AND RAMA C. NAIR

Department of Epidemiology and Community Medicine, School of Medicine, University of Ottawa, Ottawa, Ontario K1H 8M5, Canada

(*Received 11 June 1985; accepted 31 August 1985*)

SUMMARY

To assess the potential of fomites and environmental surfaces as vehicles in the transmission of rotaviral diarrhoea, disks (1 cm diameter) of various porous and non-porous materials were contaminated with about 10^5 plaque-forming units of the Wa strain of human rotavirus (HRV) suspended in faecal matter. The contaminated disks were then held for 10 days at either room temperature (22 ± 2 °C) or 4 °C with the relative humidity (RH) at the high (85 ± 5 %), medium (50 ± 5 %) or low (25 ± 5 %) level. Survival was longer on non-porous surfaces at the lower temperature and at lower humidity. In contrast, survival on porous surfaces was very variable; better on cotton-polyester than on poster card or paper currency on which HRV survived very poorly. These results suggest that under the right environmental conditions, HRV-contaminated objects could play a role in the transmission of rotavirus infections in hospitals, nursing homes and day-care centres.

INTRODUCTION

Rotaviruses are known as a frequent cause of outbreaks of acute diarrhoea in both the general community (Freij *et al.* 1978; Lycke *et al.* 1978; Morens *et al.* 1979; Echeverria *et al.* 1983) and institutions such as hospitals (Ryder *et al.* 1977; Murphy, Albrey & Crewe, 1977; Middleton, Szymanski & Petric, 1977; Holzel *et al.* 1980; Hoh, Presser & Wigand, 1983), nursing homes (Halvorsrud & Orstavik, 1980; Marrie *et al.* 1982), day-care centres (Haug, Ørstavik & Kveldstad, 1978; Pickering *et al.* 1981) and schools (Hara *et al.* 1976; Sutmoller *et al.* 1982). Rotaviruses are excreted in large numbers in the faeces of infected individuals (Flewett, 1983) for at least 5 days (Davidson *et al.* 1975) and are known to remain viable in such material for prolonged periods (Woode & Bridger, 1975).

Apart from overt cases of diarrhoeal disease, it is well recognized that many asymptomatic cases of rotavirus infection also occur (Murphy *et al.* 1977; Holdaway *et al.* 1982*a*, *b*; Rossi *et al.* 1982; Champsaur *et al.* 1984*a*, *b*). Such individuals may pose a significant hazard to others in their immediate surroundings. This is particularly important in institutional settings where relatively crowded conditions and the use of common facilities may contribute to virus transmission. Keswick *et al.* (1983*b*) have suggested that the prevalence of asymptomatic rotavirus infections in day-care facilities may make them a reservoir of infection for previously uninfected children and their family contacts.

The actual ways by which rotaviruses spread in institutional outbreaks have not been clearly identified, nor do we understand the reasons why such outbreaks in temperate regions normally occur mainly in the winter months (Bryden *et al.* 1974; Middleton, Szymanski & Petric, 1977; Brandt *et al.* 1979; Konno *et al.* 1983). Evidence gathered in some institutional outbreaks of rotavirus diarrhoea (Ryder *et al.* 1977; Halvorsrud & Ørstavik, 1980; Rocci *et al.* 1981), strongly suggests that surfaces may act as vehicles for the spread of the infection. This is further substantiated by the recovery of infectious rotaviruses from environmental surfaces in a day-care centre (Keswick *et al.* 1983*a*), and the detection of rotavirus antigens on hands of persons involved in patient care (Samadi, Huq & Ahmed, 1983).

Within institutions, the potential of objects and surfaces to act as vehicles would be directly related to both the frequency of handling and the capacity of rotaviruses to survive on them. This study was initiated, therefore, to see how well rotaviruses could survive on certain types of inanimate surfaces and objects commonly found in institutional settings, and to determine whether environmental factors such as humidity and temperature could promote or retard such virus survival.

MATERIALS AND METHODS

Cells. The MA-104 line of rhesus monkey kidney cells was used throughout this study. The techniques for the cultivation, maintenance and passage of these cells have been described in detail elsewhere (Sattar *et al.* 1984). Monolayers for virus plaque assay were prepared in 12-well plastic cell culture plates (Costar, Cambridge, MA., USA) by seeding each well with approximately 5×10^4 cells in 2·0 ml of Eagle's minimal essential medium (MEM; Flow Lab. Inc., Rockville MD., USA) containing 5% foetal calf serum (Flow).

Virus. The cell culture-adapted Wa strain of human rotavirus (Wyatt *et al.* 1980) used in this study was kindly supplied to us by Dr R. G. Wyatt (US National Institutes of Health, Bethesda, MD., USA). The virus was plaque-purified once in MA-104 cells, and the method of Ramia & Sattar (1979) was used for virus quantitation by plaque assay. Virus infectivity was expressed as plaque-forming units (p.f.u.)/ml. For the preparation of the virus pools, each monolayer in a 490 cm^2 roller bottle (Corning Glass; Corning, NY, USA) was inoculated to give a multiplicity of infection of 1 : 100. The inoculated cultures were maintained in MEM (without serum) with 5 μg/ml of trypsin (Nutritional Biochemical Corporation, Cleveland, OH, USA). Complete cytopathic degeneration of the monolayers occurred within 72 h of incubation at 37 °C. The infected cultures were frozen

(−70 °C) and thawed three times to aid in the release of the virus. The virus suspension was clarified of cellular debris by centrifugation (4 °C) at 1000 **g** for 15 min. The virus in the supernatant was subsequently concentrated 10-fold by ultracentrifugation at 100000 **g** for 120 min. The sedimented virus was resuspended in tryptose phosphate broth (TPB) prior to storage at −70 °C.

Faecal samples. The virus to be used for the contamination of the surfaces was suspended in three different faecal samples obtained from children with laboratory-confirmed rotaviral diarrhoea (Children's Hospital of Eastern Ontario (CHEO), Ottawa, Ontario, Canada). The faecal samples were prepared as 1 : 10 suspensions in normal saline and centrifuged at 1000 **g** for 15 min to remove large particulate matter. The indigenous rotaviruses present in the faecal samples did not interfere with the quantitation of the virus used for experimental contamination because the field strains of human rotaviruses were found to be incapable of producing countable plaques in MA-104 cells. Each faecal suspension was also thoroughly checked to rule out the presence of any other enteric virus that could form plaques in our assay system.

Non-porous materials tested. Disks (1 cm diameter) in glass, stainless steel, a smooth plastic (Milar) and a rough plastic (vinyl) were the representative types of non-porous surfaces tested in this study. Glass coverslips (Chance Propper Co., Warley, England) were purchased from Johns Scientific (Mississauga, Ontario, Canada). Sheets of the other three types of materials were purchased from local retail outlets and cut into disks. Prior to contamination with the virus suspension, the disks were cleaned by sonication for 10 min in a detergent solution (7X; Flow) followed by thorough rinsing in running deionized water and soaking for 10 min in 95 % ethanol. The disks were then air-dried before being individually placed in wells of a 24-well plastic plate (Costar); cleaned disks were handled only with a vacuum pick up device or a pair of sterilized forceps.

Porous materials. Pieces (1 cm in diameter) of cloth (cotton-polyester), poster card, Canadian paper currency and four types of writing paper were the porous materials tested in this study. The cloth was taken from a used and laundered gown obtained from an isolation ward of CHEO: the paper and card were new. In all experiments, the disks of currency paper were contaminated with the virus suspension without any prior treatment. On the other hand, and depending on the type of test, the disks of card and paper were either used unwashed or received prior treatment similar to that described for the non-porous surfaces.

Test procedure. Each disk was contaminated with 20 μl of the faecal suspension containing approximately 10^7 p.f.u. of the virus/ml. The inoculum was then allowed to air dry by keeping the plates with the disks for 3 h in a vertical laminar flow hood. The amount of infectious virus remaining after this initial drying period was considered as the input virus level. The plates were then placed in a transparent glass chamber ($21 \times 36 \times 55$ cm^2) kept at 22 ± 2 °C in a room with diurnal fluorescent light cycles or at 4 °C inside a cold-room. Air at the desired level of relative humidity (RH) was circulated through the chamber to give approximately six air changes per hour. The air temperature and RH in the chamber were continuously monitored using a recording hygrothermograph (Cole-Parmer Instrument Co., Chicago. IL., USA) placed inside the glass chamber.

Virus survival was tested at low (25 ± 5 %), medium (50 ± 5 %) and high (85 ± 5 %)

RH levels. Air entering the chamber was passed through a tube containing a dessicant (Drierite; Hammond, Xenia, OH, USA) to obtain the low RH and it was humidified by bubbling it through deionized water in a gas-washing bottle to achieve the high RH. During the period of these experiments, the ambient RH always stayed within the mid-range (45–55%), and, as a result, did not require any further adjustments.

In preliminary experiments using ^{14}C-labelled HRV, we tested a number of proteinaceous solutions for their capacity to elute the virus from the disks. Tryptose phosphate broth was not only completely harmless to rotavirus infectivity but it could also consistently recover 90–100% of the virus added to the disks of non-porous materials. When compared to this, its capacity for virus recovery from the porous materials was somewhat lower (70–90%); none of the other eluents tested, however, could perform any better. TPB was, therefore, selected as the virus eluent in this study.

At the appropriate sampling times, disks of each type was transferred to separate glass vials, and 1·0 ml of TPB was added to each. To effect maximal virus recovery from the surfaces, these vials were then placed in a sonic bath (Bransonic; Johns Scientific, Toronto, Ontario, Canada) and sonicated for 10 min. The eluate was then further mixed using a micropipettor before plaque assay. For any given experiment, samples were collected at least six different times during a 10-day period.

Not less than three replicate experiments were conducted at each of the three RH levels tested using the same faecal sample. In addition, experiments were performed with the other faecal samples to establish whether virus survival was independent of the faecal sample used for suspending the virus.

To determine the effect of changes in RH on virus survival, a separate set of experiments was conducted. Following the above-mentioned protocol, disks were moved between mid and high RH levels during the experiments.

Statistical analysis. Virus inactivation curves were plotted, and linear regression analysis of these gave an inactivation coefficient (K_1) of $\log_{10}$ reduction in virus titre/day for each set of experimental conditions. The Student 't'-test (two-tailed) was used to examine the effect of surface type on rotavirus survival. Subsequently, the effect of relative humidity and temperature was determined using the K_1 values and compared using the 't'-test.

RESULTS

Virus survival on non-porous materials held at 22±2 °C. There was virtually no loss in HRV infectivity on the disks of the non-porous materials during the 3 h period allowed for the initial drying of the virus suspension. However, as shown in Table 1, when the contaminated disks were held over a longer period at 22 °C, RH was found to have a pronounced influence on virus survival. The virus survived equally well at the medium (K_1 0·046–0·072) and low (K_1 0·025–0·048) RH levels and under these conditions, approximately 10% of the virus remained infectious on the disks even after 10 days. On the other hand, at 22±2°C virus survival at the high RH was dramatically different (K_1 1·564–2·408; $P < 0·001$), with > 99·9% reduction in virus titre within 48 h. Fig. 1 is a graphic representation of these results; since there were no significant difference between the results obtained with

Table 1. *Inactivation rates (K_1) of human rotavirus (Wa strain) at various relative humidity levels on four different non-porous inanimate materials*

Air temperature	22±2°C			4°C	
Relative humidity (%)	25±5	50±5	85±5	50±5	85±5
Surfaces					
Stainless steel	0·033	0·046	1·741	0·0008	0·080
Rough plastic	0·025	0·072	1·564	ND*	ND
Smooth plastic	0·031	0·072	1·868	ND	ND
Glass	0·048	0·069	2·408	ND	ND

HRV (Wa strain) suspended in a faecal suspension, was applied to 1 cm diameter disks, allowed to dry for 3 h and sampled at six time intervals over 10 days. The loss of virus infectivity was calculated by regression analysis at each RH level and the K_1 is expressed as loss of virus infectivity in $\log_{10}$ p.f.u./day.

*ND, not done.

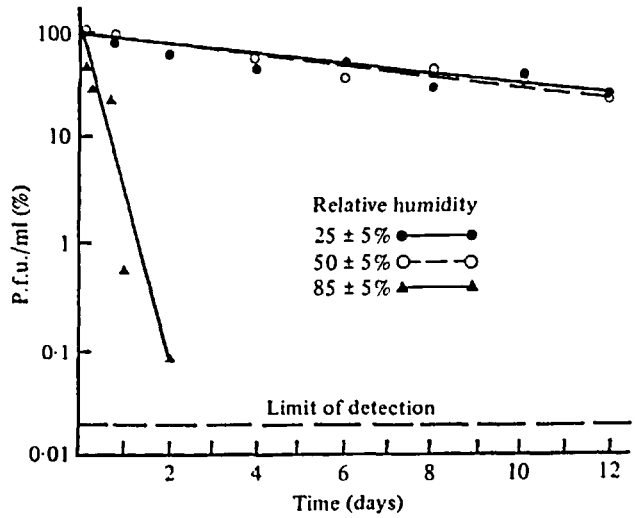

Fig. 1. The effect of various levels of relative humidity on the capacity of human rotavirus (Wa) to survive on disks of stainless steel held at 22±2 °C.

the four types of non-porous materials tested, only the data from the experiments on stainless steel are shown.

Virus survival on non-porous surfaces held at 4 °C. To examine the effect of lower air temperature on HRV survival on non-porous surfaces, a series of experiments were conducted at 4 °C using disks of stainless steel. The results of these tests, conducted at the medium RH level, showed that the capacity of HRV to survive on such materials was further enhanced; however, when compared to the results of the experiments at 22 °C, this increase in virus survival at the low temperature was not statistically significant. In contrast to this, the rate of virus inactivation ($K_1 = 0{\cdot}080$) at the high RH level and 4 °C was significantly lower ($P < 0{\cdot}001$) than at the same RH and 22 °C. Furthermore, at 4 °C, the observed

https://doi.org/10.1017/S0022172400066055 Published online by Cambridge University Press

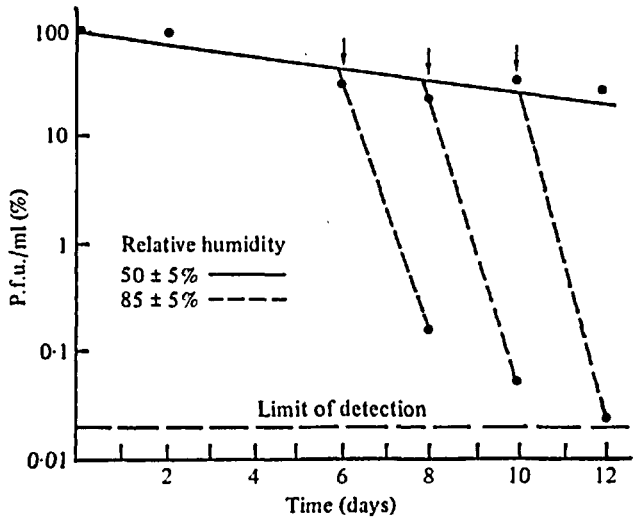

Fig. 2. Inactivation of human rotavirus (Wa) on stainless steel disks (22±2 °C) when relative humidity is shifted from the medium to the high level.

difference between the rates of virus inactivation at mid and high RH levels was not statistically significant.

Virus survival on non-porous materials held at 36±1 °C. Preliminary experiments conducted at 36 °C and at the medium and low RH levels indicated that, although the rate of virus inactivation (K_1 approximately 0·197) was higher than at 22°C, HRV could survive on non-porous materials for at least 6 days under these conditions.

Effect of faecal matter. Only minor differences in HRV survival were observed among the faecal samples used in this study. To determine if faecal material itself had an effect on HRV survival, experiments were performed under ambient conditions (50% RH, 22 °C) with the virus suspended in TPB rather than faecal material. The rate of virus inactivation here was greater ($K_1 = 0{\cdot}233$) than that observed under identical conditions but with faeces as the virus suspending medium.

Effect of RH changes on virus survival. In many natural settings there are wide variations in RH levels during the course of a day. Fig. 2 shows the results from experiments where virus-contaminated disks of stainless steel were moved from mid to high RH. On days 6, 8 and 10, some disks held at mid-range RH were transferred to the high RH level, and kept there for a 2-day period. Whereas the virus continued to survive well on those disks left at the mid-range, there was a 3–4 $\log_{10}$ reduction in the virus titre on those moved to the high RH. No virus reactivation was observed when disks held at the high RH for 2 days were returned to mid-range RH for a further 2-day period. This indicated that, under our experimental conditions, virus inactivation occurring at the high RH was irreversible.

Virus survival on porous materials. Virus survival on the porous materials tested was very variable. No infectious HRV could be recovered in any of the tests from experimentally contaminated samples of poster card, even immediately after the

drying period. This indicated that virus inactivation on this material was rapid. The anti-viral property of the card could not be eliminated by soaking it for 2 h in either TPB or 95 % ethanol. The virus was not inactivated by the TPB obtained after soaking the card, indicating that the anti-viral component(s) in this material were non-water soluble. As mentioned earlier TPB, when used as a virus eluent, could recover between 70 and 90 % of the radioactivity contained in HRV added to disks of such porous materials. Therefore, the drop in virus infectivity seen in these experiments was not due to the irreversible binding of infectious virus to the matrix of the card.

Infectious virus particles were occasionally recovered from experimentally contaminated samples of currency paper, but the titres were too low and variable for accurate quantitation and statistical analysis. Between 50 % and 80 % of infectious HRV particles were recovered from the four writing papers after the 3-h drying period but long-term survival on them was not studied.

The variability of HRV survival on the laboratory-contaminated pieces of cloth was such that, in some instances, no infectious virus could be recovered, whereas, at other times the inactivation rates were comparable to those of the non-porous surfaces tested under identical conditions. For example, in one set of experiments, infectious HRV could be recovered from them up to 2 days at 22 °C, and up to 10 days at 4 °C. When working with this material, the influence of high RH on virus survival at 22 °C also appeared to be less marked than was observed for the non-porous surfaces. This wide variation in HRV survival on cloth could not be eliminated by prior washing of the disks.

DISCUSSION

If fomites and environmental surfaces play a role in the transmission of HRV infections, the virus must be able to survive on them. Therefore, using materials usually found in institutional settings, the capacity of HRV (Wa strain) to survive on such materials under different environmental conditions was evaluated.

The results of this study clearly demonstrate that HRV in faecal matter can survive for prolonged periods on several types of materials commonly found in institutions and domestic environments. The stability of the virus was clearly influenced by environmental factors such as relative humidity, temperature and the type of surface contaminated.

The surfaces most likely to be exposed to direct or indirect contamination with virus-containing matter include our selection of porous and non-porous surfaces. Because of the difficulty in obtaining appropriate disks of enamel and porcelain, materials commonly used for bathroom fixtures, these two types of surfaces could not be included in this study. However, based on the data reported here, it is considered highly unlikely that HRV survival on them would be any different from that on the other non-porous surfaces tested. The temperatures studied (22 ± 2 °C and 4 °C) are similar to the air temperature in climate-controlled buildings and cold-storage. The low and high RH levels selected represent extremes in the indoor RH due to seasonal variations in many temperate and tropical regions. In most modern air-conditioned buildings, on the other hand, the RH is generally maintained at the mid-range throughout the year.

Poster card was included here because such material is commonly used in day-care centres and schools and is often carried into the home. This material was also similar to that used for patient records in hospitals and nursing homes. Paper currency was selected as a potential vehicle for rotaviruses because it frequently forms a link between the outside community and institutions.

To simulate closely natural conditions of environmental contamination, the virus used was suspended in faeces diluted in normal saline. This dilution was necessary to make the stool specimens less inhibitory for the virus used for experimental contamination. Moreover, the particulate matter present in the undiluted faeces interfered in the accurate dilution and quantitation of the virus. From the results in this study, diluted faecal matter in the virus suspension protected virus infectivity better than TPB. Hence the virus may survive even better on fomites and environmental surfaces if it is contained in undiluted faecal matter.

In the only other comparable study, Moe & Shirley (1982) showed that a field strain of HRV could survive better on contaminated glass coverslips when the RH was kept either at the low (12–34%) or high (75–94%) level as compared to the medium level (51–59%). Even at the medium RH level, however, they found the virus capable of surviving for 9 days. The results of our study using a laboratory-adapted strain of HRV also showed that this virus can persist for protracted periods on a variety of surfaces.

The factors influencing the survival of HRV on the cloth and papers tested in this study could not be conclusively determined. Chemical preservatives or surfactants that may be present in such porous materials, and not removed by the washing procedure, could have virucidal activity. It is also possible that such chemical residues may not be uniformly distributed throughout the fabric (e.g. detergents and fabric softeners used in laundering) and may account for the variations observed on the cloth. On the other hand, when HRV did survive on the cloth, the observed inactivation rate was comparable to that on the non-porous surfaces. Moe & Shirley (1982) also found no significant differences in HRV survival on glass or cotton swabs at all levels of RH tested.

Our observation that HRV survived least well at high RH levels disagrees with the results reported by Moe & Shirley (1982). The reasons for this disparity between the two studies are unknown. One difference between the design of the two studies was the method of generating and maintaining the humidity level. In their experiments, coverslips contaminated with rotavirus-positive faeces were placed in closed containers and the RH inside them was maintained with the help of saturated salt solutions. We circulated air at the desired RH level through the chamber containing the disks to give approximately six air changes per hour. Furthermore, in our study the virus-contaminated disks were allowed to dry before exposure to the various humidity levels whereas it is not clear if the same procedure was adopted by the other investigators. In spite of these differences in experimental protocols, the possibility of variations in survival characteristics between HRV strains remains. In support of our data, a similar relationship has been found between RH and the capacity of three different types of rotaviruses to survive in the airborne state (Sattar *et al.* 1983, 1984; Ijaz *et al.* 1985). Our finding that moving the virus-contaminated surfaces from mid to high RH level caused rapid and

irreversible inactivation of the virus provide further evidence that high RH is detrimental to virus survival at ambient temperature.

The greater stability of HRV at 4 °C observed in this study indicates a hazard from handling objects contaminated before cold storage.

The seasonal factors which predispose susceptible individuals in the general community to rotaviral gastroenteritis are not known. Nevertheless, it appears that cases of rotaviral infections (Murphy, Albrey & Crewe, 1977; Cubitt & Holzel, 1980; Holzel *et al.* 1980; Noone & Banatvala, 1983; Keswick *et al.* 1983b) occur mainly during the cooler months of the year. Therefore such cases entering institutions could initiate outbreaks of acute diarrhoea. Factors such as the congregation of susceptibles for prolonged periods in restricted areas, the inadvertent contamination of the surroundings by infected persons, and the capacity of human rotaviruses to survive on surfaces and commonly used objects may promote spread of the infection.

In the general community, food (Hara *et al.* 1978) and drinking water (Lycke *et al.* 1978; Harris, Cohen & Lippy, 1983; Hung *et al.* 1984) have been incriminated as vehicles in some outbreaks of acute rotavirus diarrhoea. However, institutions in urban areas generally receive their drinking water from well-monitored municipal facilities, and their food from centralized kitchens. Therefore, it is considered highly unlikely that these vehicles would be important in institutional outbreaks of rotavirus diarrhoea.

Rotaviruses can survive well while airborne (Sattar *et al.* 1984; Ijaz *et al.* 1985), but the role of air as a vehicle in the direct spread of rotaviral infections remains to be shown. It is conceivable, however, that airborne infectious rotavirus particles could also lead to the indirect spread of rotaviral infections through the contamination of fomites and environmental surfaces in the immediate surroundings of individuals excreting the virus (Totterdell, Chrystie & Banatvala, 1976).

Outbreaks of rotaviral diarrhoea, particularly those associated with hospitals and nursing homes, can result in increased mortality and morbidity (Halvorsrud & Ørstavik, 1978; Marrie *et al.* 1982; Yolken *et al.* 1982). For example, in a prospective study rotaviruses were found to be an important cause of infectious gastroenteritis in patients with bone marrow transplants (Yolken *et al.* 1982); the mortality rate among such infected patients was 55 % as compared to 13 % in those who remained uninfected. Nosocomial outbreaks of rotaviral diarrhoea have also been shown to add greatly to the cost of hospitalization for each affected individual (Ryder *et al.* 1977).

Rotaviruses also represent a potential health threat at work. Published data are available to show that, in hospitals at least, medical and nursing staff frequently contract the infection during nosocomial outbreaks of rotaviral gastroenteritis (Hildreth, Thomas & Ridgeway, 1981; Hoh, Presser & Wigand, 1983). There is evidence to show that such infected adults can be a source of rotaviruses for infants and young children (Kim *et al.* 1977). It is also important to note that children who acquire rotaviral diarrhoea in day-care centres or schools can carry the virus to their families (Haug, Ørstavik & Kveldstad, 1978; Pickering *et al.* 1981; Grimwood *et al.* 1983; Keswick *et al.* 1983*b*).

In an attempt to control institutional outbreaks of rotaviral diarrhoea, a thorough disinfection of the affected wards is usually carried out (Halvorsrud &

Ørstavik 1978). However rotaviruses have been found to be relatively resistant to inactivation by the chemical disinfectants and antiseptics commonly used in hospitals and other institutions (Tan & Schnagl, 1981; Sattar *et al.* 1983; Springthorpe *et al.* 1986). A number of products that could readily inactivate a human rotavirus in a 'suspension test' (adding virus suspension to produce under test), failed to do so in a 'carrier test' where the disinfectant or antiseptic was applied to a non-porous surface experimentally contaminated with the virus in a faecal suspension (Lloyd-Evans, Springthorpe & Sattar, 1986). These findings emphasise the potential role of inanimate surfaces and fomites as vehicles for rotaviral diarrhoea. Further evidence in support of this is the interruption in the appearance of new cases during an outbreak of rotavirus diarrhoea among a closed herd of Friesian dairy cattle following thorough cleansing and disinfection of the calf house (McNulty & Logan, 1983).

Although we found no difference in the capacity of the virus to survive at the low and medium RH levels, the lack of statistical significance seen in our data may have been due to variations in individual observation and the lack of a much larger sample size.

The relatively rapid inactivation of human rotaviruses at the high RH level suggests that humidification of the atmosphere in an isolation ward, for instance, may represent a simple and safe means of 'disinfection'. Further work is, however, required to test the feasibility of such a procedure under actual conditions.

The importance of contaminated hands in the dissemination of infectious rotavirus particles in institutions cannot be over-emphasized. This study has concentrated on inanimate surfaces, but work now in progress in our laboratory is aimed at studying rotavirus survival on hands using both laboratory-adapted as well as field strains of human rotavirus. A number of chemical disinfectants and antiseptics are also being tested to find those suitable for use on rotavirus-contaminated hands, fomites and environmental surfaces. Rotavirus survival on surfaces held at a higher air temperature (30–37 °C) is also being further studied in order to simulate the environmental conditions found in many tropical settings.

Although it is not known whether an individual would become clinically infected by the quantity of HRV that may be acquired from handling a contaminated surface, the findings reported here will help in understanding why institutional outbreaks of rotaviral diarrhoea occur frequently. They should also help in the formulation of more effective strategies for the prevention and control of such outbreaks.

This study was supported by grants from the International Development Research Centre, Ottawa, Canada and the Ontario Ministry of Health. Complimentary supplies of antibiotics were received from Roussel Canada Inc. We thank Mrs M. E. Kennedy and Mr L. McClelland for their helpful comments and criticisms throughout this study. We also gratefully acknowledge the excellent technical assistance of Jodi Grenier, Hanne White, Christine Chudzio and Linda Therrien. The computer program used in this study was designed for us by Dr A. Springthorpe.

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