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Survival of *Salmonella* in bathrooms and toilets in domestic homes following salmonellosis

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J. BARKER AND S.F. BLOOMFIELD. 2000. The survival and environmental spread of *Salmonella* bacteria from domestic toilets was examined in homes, where a family member had recently suffered an attack of salmonellosis. In four out of six households tested, *Salmonella* bacteria persisted in the biofilm material found under the recess of the toilet bowl rim which was difficult to remove with household toilet cleaners. In two homes *Salmonella* bacteria became incorporated into the scaly biofilm adhering to the toilet bowl surface below the water line. *Salmonella enteritidis* persisted in one toilet for 4 weeks after the diarrhoea had stopped, despite the use of cleaning fluids. Salmonellas were not isolated from normally dry areas such as, the toilet seat, the flush handle and door handle. Toilet seeding experiments were set up with *Salmonella enteritidis* PT4 to mimic environmental conditions associated with acute diarrhoea. Flushing the toilet resulted in contamination of the toilet seat and the toilet seat lid. In one out of three seedings, *Salmonella* bacteria were also isolated from an air sample taken immediately after flushing, indicating that airborne spread of the organism could contaminate surfaces in the bathroom. In the seeded toilet *Salmonella* bacteria were isolated from the biofilm in the toilet bowl below the waterline for up to 50 d after seeding, and also on one occasion from the bowl water. The results suggest that during diarrhoeal illness, there is considerable risk of spread of *Salmonella* infection to other family members via the environment, including contaminated hands and surfaces in the toilet area.

INTRODUCTION

A recent study of infectious intestinal disease in England has indicated that such infections occur in one out of five people each year (Wheeler *et al.* 1999). Estimates show that for every case of infectious intestinal disease reported to the Communicable Disease Surveillance Centre (CDSC) 136 unreported cases occur in the community causing considerable morbidity. Salmonellosis is the second most common cause of bacterial food poisoning reported to CDSC (Evans *et al.* 1998) and for every reported case of infection with *Salmonella*, 3·2 cases probably occur in the community but are unreported (Wheeler *et al.* 1999). A UK study carried out between 1990 and 1994 (Crowley *et al.* 1997) indicated that *Salmonella* was the second most common cause of gastroenteritis in children under 5 years of age.

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Although, the primary cause of salmonellosis is consumption of contaminated foods, there is the potential for secondary spread, from person-to-person and also to other foods. Person-to-person spread within family groups is often associated with poor personal hygiene but there is the opportunity for airborne and surface-to-surface spread within the toilet and bathroom, especially during the diarrhoeal phase. Although the UK system of data collection does not allow for rates of secondary spread of salmonellosis in families to be estimated, it is generally acknowledged that this occurs. Outbreaks involving secondary spread in previously healthy individuals have been recorded. For example, in a *Salm. typhimurium* outbreak amongst male university students, environmental contamination of toilets is thought to have caused secondary cases by hand-to-surface contact (Palmer *et al.* 1981). After a Hospital outbreak of *Salm. typhimurium*, organisms were isolated from ward dust and from sputum of patients, indicating that aerial spread can occur (Datta and Pridie 1960). Several laundry workers and domestic staff became infected when their

only contact was with contaminated bed linen. In another hospital *Salm. typhimurium* outbreak, secondary spread was reported in staff whose only contact with infected patients involved handling sheets and specimen bottles (Steere *et al.* 1975). From a US study of household contacts of infants with *Salmonella* gastroenteritis, Rosenstein (1967) reported that 34·7% had *Salmonella* bacteria in their stools and 19% showed symptoms.

Although audit studies of the home environment, taken at random, show no evidence of the presence of *Salmonella* bacteria in toilets (Finch *et al.* 1978; Scott *et al.* 1982; Josephson *et al.* 1997) there is little documented evidence to show whether, in homes where individuals have or have had salmonellosis, environmental contamination in toilets and bathrooms contributes to the spread of infection. We investigated contamination of surfaces with *Salmonella* bacteria, in toilets and bathrooms, in homes where individuals had suffered from an acute attack of salmonellosis within the previous 2 weeks. Environmental contamination and survival in a domestic toilet experimentally seeded with *Salm. enteritidis* was also examined.

MATERIALS AND METHODS

Environmental sampling in domestic homes

Environmental sampling was carried out in six domestic homes in the West Midlands, UK where *Salmonella* cases had been confirmed. By the time *Salmonella* infections were notified, the infected individuals had recovered from their symptoms. In all cases individuals had been ill at home with symptoms of acute diarrhoea and vomiting. Initial samples were collected between 1 and 2 weeks after the symptoms had subsided. Households were contacted for permission to visit and take samples. When *Salmonella* bacteria were isolated, a second visit was made and further testing carried out before disinfecting the contaminated areas with household bleach (containing $\approx 50\,000$ ppm of free available chlorine).

Surfaces were sampled by a two swab method; the first swab was moistened in test diluent (see below) before use and the second swab was used dry (Holtby *et al.* 1997). Areas of 100 cm^2 were sampled if available. The swab tips were broken off into 1·5 ml diluent for transport to the laboratory. The toilet was flushed before samples were taken of the bowl water and biofilm on the sides of the toilet bowl above and below the water line. A 50-ml sample of water was mixed with 20 ml of test diluent. The biofilm material in the toilet bowl below the waterline was often an adherent scaly material and this was gently scraped off with a sterile scalpel blade and placed in test diluent. Additional sites sampled included: the recess under the rim of the toilet bowl, the toilet seat, flush handle, wash-basin hot and cold tap handles and the sink waste U-bend, the bath hot and cold taps and the bath waste U-bend, the door handle, cleaning cloths and toilet brush (if available).

Diluent and microbiological examination

The diluent used for collecting environmental samples was quarter-strength Ringer solution with peptone 0·1% (w/v), Tween 80, 0·1% (v/v) and sodium thiosulphate 0·2% (w/v) to neutralize residual disinfectant (Tebbutt 1986).

All samples were incubated for 18 h in buffered peptone water (Oxoid, Basingstoke, UK) at 37 °C on an orbital shaker. One millilitre of the pre-enrichment broth was then added to 20 μl Dynabeads® anti-*Salmonella* (Dynal UK Ltd, Bromborough, UK), continuously mixed and incubated at room temperature for 10 min. Placing the tube in a magnetic particle concentrator (Dynal UK Ltd), for 3 min then separated the beads. The supernatant was removed and the beads resuspended in 1 ml of phosphate-buffered saline (PBS) pH 7·4 (Oxoid), containing Tween 80, 0·05% (v/v) and separated once more in the magnetic concentrator. After a further washing procedure the beads were finally resuspended in 100 μl of wash buffer and added to 10 ml of Rappaport-Vassiliadis (RV) broth (Oxoid Ltd). RV broth was incubated at 42 °C for 24 h and plated onto mannitol lysine crystal violet brilliant green agar (MLCB; Oxoid Ltd) before 24 h incubation at 37 °C. Presumptive purple-black *Salmonella* colonies were subcultured onto MacConkey agar (Oxoid Ltd) to confirm that they were non-lactose fermenting. This was done to screen out H_2S-producing strains of *Citrobacter* which gave colonies with a similar appearance to *Salmonella* on MLCB. Most of the environmental *Citrobacter* strains were lactose fermenting. Suspect isolates of salmonellas were confirmed by agglutination with polyvalent antisera to *Salmonella* somatic and flagellar antigens (Mast Laboratories Ltd, Bootle, UK) and biochemical tests (API 20E; BioMérieux, Marcy-l'Etoile, France). The strains were kindly serotyped by the PHLS Food Microbiology Research Unit (Exeter, UK).

Toilet seeding experiments

Seeding experiments were carried in a domestic toilet that was no longer in use and had strictly controlled access, in the home of one of the authors (JB). A human isolate of *Salm. enteritidis*, PT4, strain E as described by Humphrey *et al.* (1995); was used throughout. The organism was grown in a 1 : 20 dilution of tryptose soy broth (Oxoid Ltd) on an orbital shaker at 37 °C for 24 h to give $\approx 10^9$ cfu ml^{-1} of stationary phase cells. The suspension was centri-

fuged (2080 *g* for 20 min) before washing and resuspending in PBS to give 10^8 cfu ml^{-1}. One millilitre of the washed suspension was added to 50 ml of semisolid agar (0·2% w/v) to produce the inoculum for seeding.

The toilet was flushed before seeding with the test suspension, which was discharged from seat height into the bowl water using a 50-ml syringe, to simulate to the force and splashing effects associated with acute diarrhoea. A portable impinger air sampler (MicroBio, FW Parrett Ltd, London, UK) was used to collect 500 l of air onto MLCB agar, immediately after flushing the seeded toilet with the door closed. After intervals of 1 h, second and third seedings were carried out. After the third flush the toilet was left undisturbed except for twice daily flushing. Environmental samples were taken after each of the seedings and for a period of up to 55 d. Contact plates containing MLCB agar were used to detect salmonellae contaminating the surface of the toilet seat after flushing.

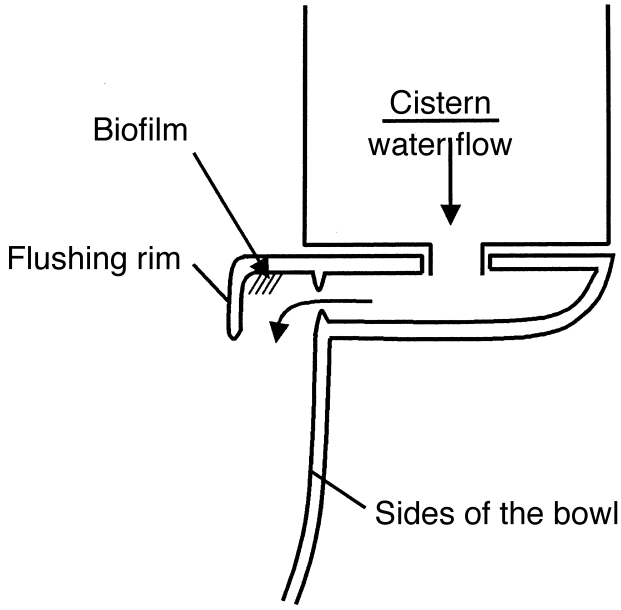

Fig. 1 Cross-sectional view of the domestic toilet bowl

RESULTS

Domestic homes

Salmonella bacteria were isolated from environmental samples in four of the six homes visited but only from locations that were moist (Table 1). In all cases the serotype isolated from the environment was the same as that isolated from the infected person as confirmed by examination of a faecal specimen carried by the local laboratory. In homes where *Salmonella* bacteria had been cultured (A, C, E and F) the organism was isolated from biofilm material removed from the recess under the rear rim of the toilet (see Fig. 1) which showed macroscopic evidence of faecal soiling. In homes C and E *Salmonella* bacteria were only found under the rear rim of the toilet bowl. In homes A and F *Salmonella* bacteria were also found on the sides of the toilet bowl, below the water line, in the scaly biofilm. In home F, but not A, *Salmonella* bacteria were also found in the toilet bowl water.

Salmonella bacteria were not isolated from dry sites such as, toilet seats, flush handles, door handles and tap handles

Table 1 *Salmonella* serotypes isolated from patients and environmental sites in toilets and bathrooms

Household	Patient	Source	*Salmonella* serotype isolated from the patient	*Salmonella* of the same serotype recovered from environmental sites	Cleaning materials used
A	Male 26 years	? Take away chicken meal	*Salm. enteritidis* phage type 6a	Under rear rim of toilet bowl Scale/biofilm in toilet bowl	'Toilet duck' Cleaned every few days
B	Female 25 years	Holiday in Dominican Republic	*Salm. cerro*	Not isolated	Bleach Cleaned daily
C	Female 28 years	Holiday in Dominican Republic and cruise	*Salm. heidelburg*	Under rear rim of toilet bowl	'Toilet duck' Cleaned every few days
D	Male 45 years	? Meal at a local Indian restaurant	*Salm. enteritidis* phage type 4	Not isolated	'Toilet duck' Cleaned weekly
E	Female 18 years	Holiday in Tenerife — ate a lot of chicken	*Salm. enteritidis* phage type 21	Under rear rim of toilet bowl	Bleach Cleaned every few days
F	Male 2 years	Source not identified	*Salm. enteritidis* phage type 7	Under rear rim of toilet bowl Scale/biofilm in toilet bowl Toilet bowl water	Bleach Cleaned weekly

or from toilet brushes. In only two of the six homes were cleaning cloths available for sampling and these did not contain *Salmonella* bacteria. All the households used toilet-cleaning products. Homes A, C and D used 'toilet ducks', a generic term used to describe the shape of the bottle designed for easy cleaning of the toilet bowl. The 'toilet ducks' contained formulations which were either bleach-based or strongly acidic. Homes B, E and F used hypochlorite-based products containing $\approx 50\,000$ ppm of available free chlorine. All of these products would be expected to have a disinfectant action. Household B cleaned the toilet daily whereas the others did it every few days or weekly.

In households C, E and F *Salmonella* bacteria were not isolated from repeat samples taken on a second visit 1 week later. There was evidence in these homes that additional cleaning had been carried out before the second visit because there was a reduction in the amount of visible scaling in the corners of the toilet bowls. However, the householders stated that they had only carried out their normal cleaning procedures.

In household A, *Salm. enteritidis* persisted under the rear rim of the toilet bowl for 3 weeks and on the sides of the bowl, below the waterline, in the scaly biofilm material for 2 weeks. After sampling in week 2, the toilet was thoroughly disinfected with household bleach. However, using the bottle as supplied by the manufacturer, it was impossible to get the bleach into the recess under the rim. On week 3 *Salm. enteritidis* was not detected on the sides of the bowl below the waterline but it persisted under the rear rim. To get bleach under the rim a Pasteur pipette was used to thoroughly disinfect the area. A subsequent sample taken a week later did not contain *Salm. enteritidis*.

Toilet seeding experiments

Samples taken immediately before seeding the toilet confirmed that it was not contaminated with *Salmonella* bacteria. Seeding with a suspension of 10^8 cfu ml^{-1} of *Salm. enteritidis* resulted in $\approx 10^4$–10^5 cfu ml^{-1} in the toilet bowl water. Table 2 shows the results of one out of three experiments which were performed, all of which gave a similar pattern of results. After flushing most of the bacteria in the toilet bowl were washed away, leaving between 10 and 40 cfu ml$^{-1.}$ However, *Salm. enteritidis* was detected in the samples taken from the sides (above the waterline) and the rim of the toilet bowl. The organism was also recovered from the top and the underside of the toilet seat immediately after flushing. Visible splashes found on the toilet seat lid, after the second and third seedings, also contained

Table 2 The results of seeding an experimental toilet with *Salmonella enteritidis*, at 1 h intervals, to stimulate attacks of acute diarrhoea

Sample site	First seeding with *Salm. enteritidis* ($1{\cdot}1 \times 10^5$ cfu ml^{-1} in pan water) followed by flushing	Second seeding with *Salm. enteritidis* ($1{\cdot}3 \times 10^5$ cfu ml^{-1} in pan water) followed by flushing	Third seeding with *Salm. enteritidis* (4×10^4 cfu ml^{-1} in pan water) followed by flushing
Toilet bowl water	*Salmonella* detected (4×10^1 cfu ml^{-1})	*Salmonella* not detected (< 10 cfu ml^{-1})	*Salmonella* detected (10^1 cfu ml^{-1})
Toilet bowl biofilm (front, below water-line)	*Salmonella* detected	*Salmonella* detected	*Salmonella* detected
Toilet bowl biofilm (rear, below water line)	*Salmonella* detected	*Salmonella* detected	*Salmonella* detected
Recess under rear rim	*Salmonella* detected	*Salmonella* detected	*Salmonella* detected
Recess under front rim	*Salmonella* detected	*Salmonella* detected	*Salmonella* detected
Recess under left rim	*Salmonella* detected	*Salmonella* detected	*Salmonella* detected
Recess under right rim	*Salmonella* detected	*Salmonella* detected	*Salmonella* detected
Rim sides (rear)	*Salmonella* detected	*Salmonella* detected	*Salmonella* detected
Toilet seat (top)	*Salmonella* detected (1 cfu per 6 cm^2)	*Salmonella* detected (1 cfu per 24 cm^2)	*Salmonella* detected (1 cfu per 12 cm^2)
Toilet seat (underside)	*Salmonella* detected (6 cfu per cm^2)	*Salmonella* not detected	*Salmonella* not detected
Flush handle	*Salmonella* not detected	*Salmonella* not detected	*Salmonella* not detected
Behind toilet seat	*Salmonella* not detected	*Salmonella* not detected	*Salmonella* not detected
Air sample (500 l)	17 cfu	*Salmonella* not detected	*Salmonella* not detected

Salm enteritidis. The organism was not detected on the flush, handle, the door handle, light switch, toilet roll holder or carpet. A 500-l air sample taken immediately after flushing the first seeding revealed 17 cfu of *Salm. enteritidis*, although subsequent air samples taken immediately after flushing the second and third seedings did not contain the organism.

Six days after seeding, water in the toilet bowl was not contaminated with *Salm. enteritidis*, although the organism was detected in the biofilm at the front and rear of the toilet bowl below the waterline (Table 3). *Salmonella* bacteria were not detected on the toilet seat or lid, which were contained with the organism immediately after the first seeding. After 12 d, *Salmonella* bacteria were found in the toilet bowl water and in the biofilm at the rear of the bowl, below the waterline. *Salmonella* bacteria continued to be isolated from the biofilm, below the water level at the rear of the toilet bowl, for up to 50 d. After 50 d the toilet was disinfected with household bleach and thoroughly cleaned with a toilet brush. Subsequent samples taken 5 d after disinfection were not contaminated with *Salmonella* bacteria.

Table 3 Persistence of *Salmonella enteritidis* in an experimental toilet after seeding

Sample site (taken after flushing)	6 d after seeding	12 d after seeding	30 d after seeding	50 d after seeding*	55 d after seeding
Toilet bowl water	*Salmonella* not detected	*Salmonella* detected	*Salmonella* not detected	*Salmonella* not detected	*Salmonella* not detected
Toilet bowl biofilm (front, below water-line)	*Salmonella* detected	*Salmonella* not detected	*Salmonella* not detected	*Salmonella* not detected	*Salmonella* not detected
Toilet bowl biofilm (rear, below water line)	*Salmonella* detected	*Salmonella* detected	*Salmonella* detected	*Salmonella* detected	*Salmonella* not detected

*Hypochlorite disinfection carried out after sampling.

DISCUSSION

The purpose of this investigation was to evaluate the environmental spread of *Salmonella* from an infected family member via the domestic toilet. Studies of *Salmonella* cases showed that the organism persisted in household toilets for several weeks after individuals suffering from salmonellosis had recovered from their symptoms. This contrasts with previous audits of control homes in the UK and US visited and sampled at random (Finch *et al*. 1978; Scott *et al*. 1982; Josephson *et al*. 1997) which did not show evidence of *Salmonella* in any of the toilets (although it is unlikely that samples were taken of scale from toilet bowls).

Since it was not possible to gain access to homes of *Salmonella* cases during the diarrhoeal stage of the infections, experiments were set up using toilets seeded with a clinical isolate of *Salm. enteritidis* PT4 to mimic conditions associated with acute diarrhoea. The toilet was seeded with 10^8 cfu of *Salm. enteritidis*, whereas an infected person may shed up 10^{11} cfu per stool (Thomson 1954). Even so, we found that flushing the toilet resulted in contamination of the toilet seat and toilet seat lid. In one out of three seedings, *Salmonella* bacteria were isolated from an air sample taken immediately after flushing, indicating that airborne spread of the organism could contaminate surfaces in the bathroom. Visible splashes containing *Salmonella* bacteria, detected on the toilet seat lid after flushing, showed how surfaces might become contaminated during an attack of acute diarrhoea. These results are consistent with previous studies of Gerba *et al*. (1975), using *Escherichia coli* as an indicator organism, which showed that flushing of seeded

toilets produces bacteria-laden aerosols which settle on toilet and bathroom surfaces.

The potential for *Salm. enteritidis* PT4 to form biofilms is assisted by the expression of discrete fimbrial structures projecting from the bacterial cell surface (Turcotte and Woodward 1993; Collinson *et al.* 1996). At least two of these, type 1 fimbriae and SEF17 fimbriae possess adhesive properties and play a role in the formation of biofilms on Teflon™ and stainless steel (Austin *et al.* 1998). SEF17 fimbriae are expressed after growth at temperatures ≈ 20 °C, i.e. common environmental temperatures in the UK. In the four positive households *Salmonella* bacteria survived in moist areas of the toilet associated with biofilm formation, in particular the recess under the rear rim of the toilet bowl. In most toilets this recess was about 50 mm deep and 10 mm wide. This often harboured thick biofilm material, with macroscopic evidence of faecal soiling. When an individual suffers from acute diarrhoea there is often a splashing effect and this may contaminate the bowl sides and the recess under the rim. The design of the toilet means that parts of the rim are protected from the flushing action of the water which is discharged from outlets at set intervals around the underside of the rim and thus offers an ideal habitat for establishment of persistent biofilms. Unlike the results of the field studies, we did not find *Salmonella* bacteria under the rim recess of the experimental toilet 6 d after seeding.

In two homes (A and F) *Salmonella* bacteria became incorporated into the scaly biofilm material adhering to the toilet bowl surface below the water line. This material was found in the corners of the toilet bowl at the front and the rear, both areas that are difficult to clean with a toilet brush. Although in household A, *Salm. enteritidis* was found in the toilet bowl biofilm for 3 weeks after the patient's symptoms had subsided, the organism was not isolated from the bowl water. This indicates that salmonellae are able to survive as part of the biofilm flora which may give protection from cleaning agents and the mechanical effects of flushing. Similar results were found in the seeded toilet where *Salm. enteritidis* was isolated on four occasions from the scaly biofilm in the toilet bowl for up to 50 d after seeding, and also on one occasion from the bowl water. Previous studies have shown that biofouling in toilets is greater below the waterline where biofilms up to 20 μm thick may form (Pitts *et al.* 1998).

In one of the four positive households (F), the presence of *Salmonella* bacteria in the toilet bowl biofilm was accompanied by the presence of the organism in the toilet water. In the toilet seeding experiments, *Salm. enteritidis* was also isolated from toilet water 12 d (although not 6 d) after seeding. The sporadic nature of these isolations suggests that it may result from periodic detachment of scale particles from the bowl surface into the toilet water. It is possible, however, that the detection of *Salmonella* bacteria on one occasion and not on another reflects the problems associated with recovery methods. Detection of salmonellae is influenced by a variety of factors such as the ability to recover damaged cells from mixed populations of environmental bacteria. Because of the necessity to use selective techniques for recovery from heavily contaminated areas it was not possible to quantify the numbers of salmonellae on surfaces, except on toilet seats. Despite this it is clear that salmonellae are able to survive for many weeks in environments where there are likely to be wide fluctuations in conditions, such as temperature, pH and nutrients.

Although simulation of a patient with diarrhoea using a toilet showed that flushing produced detectable contamination on the toilet seat and lid, there were no isolations of salmonellae from any normally dry areas such as the toilet seat, the flush handle and door handle in the longer term (i.e. after 6 d or more from the initial contamination). This suggests that there was little spread of contamination by splashing or aerosol formation during this time, or that the organisms, which were spread, did not survive. Either way it is concluded that contamination of surfaces outside the toilet bowl is probably only a potential hazard during the acute phase of illness. Nevertheless, Newsom (1972) demonstrated the potential for long-term persistence of *Salmonella* bacteria on surfaces because the organism could be recovered for up to 9 d after faeces containing 10^9 cfu ml^{-1} of *Salm. typhimurium* were dried onto toilet seats.

Our results suggest that during diarrhoeal illness, there is considerable risk that pathogens responsible for gastrointestinal tract infections can be spread to other family members via the environment, including contaminated bathroom surfaces as well as hands. This could arise either through direct hand-to-mouth transfer, hand-to-surface-to-mouth transfer or by transfer to foods where the organism multiplies because of inappropriate storage. Although direct transfer of salmonellae to the mouth may occur, it probably involves only small numbers of organisms and transfer to food may be a greater risk. Nevertheless, although it is generally acknowledged that the infective dose of salmonellae is $\approx 10^6$ cfu, it can be as low as 10–100 cfu depending on the strain involved (Craven *et al.* 1975; Lipson 1976; Gill *et al.* 1983; Greenwood and Hooper 1983; D'Aoust 1985; Hockin *et al.* 1989). Although the study reported here focused on homes of *Salmonella* cases, it also highlights the potential for other faecal–oral pathogens which have low infective doses such as *Shigella*, *Campylobacter*, Rotavirus, Caliciviruses and *E. coli* O157 to be transmitted via contaminated toilet/bathroom surfaces (Dupont *et al.* 1972; Tauxe 1992; Griffin *et al.* 1994; McDonnell *et al.* 1995). Parry and Salmon (1998) have estimated that the household transmission rate for sporadic *E. coli* O157 in the UK to be 4–15%. Thomas and Tillett

(1973) and Khan (1982) have described the role of poor hygiene in school toilets in aiding the spread of sonne dysentery amongst pupils. Hutchinson (1956) demonstrated contamination of toilet seats with *Shigella sonnei* when heavily infected loose bulky stools were flushed away. In a recent case control study of domestic homes, Wilson *et al.* (1998) showed that where there was a confirmed *Salmonella* case, the same organisms could be isolated from the dishcloths in 6% of the homes. Although it is not certain whether the contamination of the cloth derived from the food which caused the infection or the presence of the infected person in the home, this highlights the potential for cross-contamination in homes where there is a *Salmonella* case. In our study salmonellae were not isolated from any of the cleaning cloths taken from case homes.

In case households there was obvious concern about infection risk but there was no evidence of infection spreading to other family members. By the time the visits were made the positive toilets were unlikely to have been a major hazard because hands would not normally touch the contaminated areas. Even so, this did not reassure householders who wanted the organism eradicated. Indeed, a contaminated toilet could be a risk to young children who might inadvertently put their hands into the toilet bowl. Recent findings suggest that *Salmonella* bacteria surviving in various environmental sites in the home might be responsible for spread of infection to children (Schutze *et al.* 1999).

Scott *et al.* (1984) and Rusin *et al.* (1998) have shown that hypochlorite cleaners are effective in reducing levels of faecal coliforms in toilets and surfaces in the bathroom. Scott and Bloomfield (1985) also found that a continuous release system disinfectant produced a sustained reduction in faecal contamination of the toilet itself (water, toilet bowl and rim) and some reduction in contamination of sites surrounding the toilet (seat, floor and air). Although under normal conditions there may be little risk from the toilet, it could be argued that the introduction of an infected person in the home may not be detected until it is too late to act and that toilet hygiene should function to guard against this eventuality. Our study demonstrates that effective scale removal is of equal importance in achieving hygiene of the toilet. Use of disinfectant without physical or chemical treatment to disrupt the scaly biofilm will probably have little effect. Removal of scale in the toilet bowl can be difficult and in the field studies it was necessary to use repeated scrubbing with a toilet brush and household bleach, into the corners of the toilet bowl, to remove the scale. Disinfecting the recess under the toilet bowl rim was also difficult until a Pasteur pipette was used to spray bleach under the recess. This suggests the need for properly designed 'delivery systems' if effective toilet disinfection is to be achieved.

The results of the first visit were telephoned to the households and they had the opportunity to carry out cleaning before the next visit. In three out four households, salmonellae were not detected on the second visit and it is not clear whether this was a result of additional cleaning or whether the organism disappeared naturally.

The home represents an important component in the chain of infection transmission in the community. As infections can spread easily in the domestic home it would seem prudent that during an episode of acute gastroenteritis, toilets (and other items such as surfaces and cleaning cloths) are rigorously descaled and disinfected to eliminate any pathogenic flora. It must be stressed, however, that decontamination of the toilet and surface disinfection during and after an outbreak of *Salmonella* can only be an adjunct to careful personal hygiene which is probably the most critical factor in preventing transmission of gastrointestinal pathogens.

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