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Effectiveness of deep cleaning followed by hydrogen peroxide decontamination during high *Clostridium difficile* infection incidence

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Background: *Clostridium difficile* infection (CDI) remains an infection control challenge, especially when environmental spore contamination and suboptimal cleaning may increase transmission risk.
Aim: To substantiate the long-term effectiveness throughout a stroke rehabilitation unit (SRU) of deep cleaning and hydrogen peroxide decontamination (HPD), following a high incidence of CDI.
Methods: Extensive environmental sampling (342 sites on each occasion) for *C. difficile* using sponge wipes was performed: before and after deep cleaning with detergent/chlorine agent; immediately following HPD; and on two further occasions, 19 days and 20 weeks following HPD. *C. difficile* isolates underwent polymerase chain reaction ribotyping and multi-locus variable repeat analysis (MLVA).
Findings: *C. difficile* was recovered from 10.8%, 6.1%, 0.9%, 0% and 3.5% of sites at baseline, following deep cleaning, immediately after HPD, and 19 days and 20 weeks after HPD, respectively. *C. difficile* ribotypes recovered after deep cleaning matched those from CDI cases in the SRU during the previous 10 months. Similarly, 10/12 of the positive sites identified at 20 weeks post-HPD harboured the same *C. difficile* ribotype (002) and MLVA pattern as the isolate from the first post-HPD CDI case. CDI incidence [number of cases on SRU per 10 months (January–October 2011)] declined from 20 before to seven after the intervention.
Conclusion: HPD, after deep cleaning with a detergent/chlorine agent, was highly effective for removing environmental *C. difficile* contamination. Long-term follow-up demonstrated that a CDI symptomatic patient can rapidly recontaminate the immediate environment. Determining a role for HPD should include long-term cost-effectiveness evaluations.
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Introduction

Clostridium difficile is highly transmissible in hospitalized patients and control measures to limit cross-infection are part of routine practice.[1] It has become increasingly important to determine how transmission is occurring and to establish effective interventions to minimize these risks.[2] Control methods to limit *C. difficile* transmission in healthcare environments include barrier methods, isolation of infected patients and compliance with hand hygiene measures to minimize the dissemination of spores.[3] *C. difficile* spores represent a particular challenge to effective decontamination because they are shed in high numbers by infected patients and they are resistant to desiccation and to some disinfectants.[4] Strict adherence to environmental cleaning and disinfection policies including surfaces and equipment have been shown to be important in reducing spore contamination and *C. difficile* infection (CDI) rates.[5,6] Admission to a room previously occupied by a patient with CDI is a risk factor for the acquisition of *C. difficile*.[7] Despite implementation of control measures, hospitals still experience CDI case clusters, prompting a search for ways to reduce and limit environmental contamination.

There are many alternatives for decontamination of ward environments, including (sometimes deep) cleaning with various detergents and disinfectants and use of gas/vapour technologies. HPD has recently increased in popularity for decontamination of hospital wards and for the terminal disinfection of rooms.[8–10] Some reports claim that HPD is more effective than manual cleaning for removing environmental microbial contamination, for example by MRSA.[11] Hydrogen peroxide is a powerful oxidizing agent that penetrates microbe cell walls by passive diffusion and inactivates vegetative bacteria and bacterial spores.[12] It causes cell death through mechanisms which, although not fully elucidated, include the production of hydroxyl radicals, causing irreversible damage to bacterial DNA.[12,13]

We aimed to determine, using extensive environmental sampling and ribotyping, the extent of environmental contamination of a hospital ward by *C. difficile*, and to establish the immediate effectiveness of deep cleaning and HPD on microbe prevalence. Additionally, we carried out follow-up extensive sampling to determine the extent of recontamination by *C. difficile* after resumption of clinical activity in a high CDI risk setting.

Methods

Background to the outbreak and setting

Leeds Teaching Hospitals NHS Trust is a large UK teaching hospital and tertiary referral centre with ~2000 beds. At the time of testing the stroke rehabilitation unit (SRU) contained 30 beds and was divided into male and female sections. The male section comprised three four-bedded bays, three single rooms, a small day-room and bathroom facilities. In the female section there were four four-bedded bays and bathroom facilities. In the central area of the ward there was a large day-room which was used for multiple activities including meetings with therapists or carers, serving food and storage of equipment. The single rooms did not have en-suite facilities, and so patients nursed here in isolation for infection control purposes used in-room commodes.

Between January 2011 and October 2011, 20 cases of CDI were diagnosed in patients in the SRU. Investigations led to the findings that, of these cases, 11 represented transmission between patients, eight were probably imported infections, and one represented a patient with recurrent CDI. Ribotyping provided evidence suggestive of transmission between cases and a number of clusters of cases were identified. During this period a number of practice changes were implemented within the ward, including reviews of antimicrobial prescribing, transfer of patients with CDI to an alternative ward and further staff training. Despite this, it was concluded that the measures which had been taken to reduce the incidence of CDI in the SRU had been ineffective. A decision was made in October 2011 to close the ward for 10 days and decant the patients into alternative accommodation so that HPD could be used.

Deep cleaning

The deep cleaning of the SRU took place over one week and involved an intensive, prolonged, manual clean, which aimed to restore all surfaces to the best possible condition, leaving them free from ingrained dirt, debris and marks. This involved a dedicated team of six trained personnel, and included cleaning the walls, vents, radiators, floors and all patient shared equipment, and changing of all curtains. The chlorine-based sporicidal disinfectant 'Chlor-Clean' (Guest Medical Ltd, Aylesford, UK) at a chlorine concentration of 1000 ppm (from Chlor-Clean tablets) with launderable microfibre mops (one per single room or bay) and cloths (number used per room determined on the level of soiling encountered, amount of equipment, items contained within the room and the nature of the surface being cleaned) were used for surface decontamination. Cleaning was carried out in a systematic manner with the single rooms being cleaned first, followed by the bed bays, then the general areas, corridor areas and out to the ward entrance.

Hydrogen peroxide decontamination

Hydrogen peroxide decontamination was achieved using the Deprox system (Hygiene Solutions, Kings Lynn, UK). Mobile generators using high frequency ultrasound to atomize hydrogen peroxide were placed into the ward following closure and deep cleaning. All windows and doors were sealed and the generators were used to release hydrogen peroxide droplets of size 2 μm into the environment to achieve a hydrogen peroxide concentration of 87 ppm for a predetermined length of time, dependent on the size of the area to be decontaminated. According to the manufacturer the small size of the droplets enables them to be hypermobile and to achieve a high level of spatial distribution through natural convection currents throughout the space. Fans were also used to circulate the air throughout the decontamination time. Air was then blown into the environment which displaced all remaining hydrogen peroxide. Entry was allowed into the ward when levels had fallen to 1 ppm, which was measured by equipment sensors.

Organization of environmental sampling

Environmental sampling was carried out on five separate occasions: sampling 1 was carried out immediately following the move of all patients and staff into an alternative ward,

sampling 2 was carried out immediately following the deep cleaning (day 8), and sampling 3 the day after HPD (day 10). Further sampling was also carried out to examine the extent of recontamination following reopening of the ward: sampling 4 (day 28, i.e. 19 days post HPD), and sampling 5 (day 150, i.e. 20 weeks post HPD).

Environmental sampling

As far as possible the same sites were sampled on each of the five separate sampling sessions. These were divided into three groups consisting of high sites ($N = 74$), medium height sites ($N = 180$) and low sites ($N = 88$). The high sites included areas infrequently touched including curtain tracks, wall trunking, patient line boxes, and the tops of hoist rails. Medium sites were the areas likely to be frequently touched within the ward by patients, staff and visitors including the beds, hand-wash basins, glove dispensers, bins, tables, and chairs. Low sites were infrequently touched areas including sections of the floor, the bases of beds, the bases of tables and the bases of other pieces of equipment. Sites sampled covered all areas within the ward environment including both male and female multi-occupied bays, single occupancy rooms, bathrooms, equipment stores, day rooms, kitchen, stores, and the therapy room.

Polywipe sponges (Medical Wire & Equipment, Corsham, UK) were used for sampling of all surfaces as described previously.[14] After sampling an area of $\sim 25\,cm^2$ the sponge was placed using sterile forceps into a sterile screw-top container; 20 mL Robertson's cooked meat broth (E&O Laboratories, Bonnybridge, UK) was added to each sponge and this was incubated anaerobically (37 °C for 48 h). The liquid was sub-cultured on to plates containing Brazier's cycloserine–cefoxitin–egg yolk agar (Bioconnections, Knypersley, UK) supplemented with 5 mg/L lysozyme (CCEYL; not pre-reduced) and incubated as above.

Control tests were set up to verify the sampling process on each occasion; this included the use of sponges spiked with a known *C. difficile* strain, which were enriched within the screw-top container and cultured as above. Negative controls (unused swab sponges) were also set up for each sampling session; all our own environmental sampling equipment (trolleys, boxes) were also sampled before each sampling session to ensure that there was no extraneous *C. difficile* present.

Polymerase chain reaction (PCR) ribotyping and multilocus variable-number tandem repeat analysis (MLVA)

Polymerase chain reaction ribotyping was performed on all *C. difficile* isolates as described previously.[15] Isolates sharing the same ribotype were further investigated using MLVA, as described previously using seven loci (A6, B7, C6, E7, F3, G8 and H9).[16] Fragments were analysed using Genemapper software (version 4.0 Applied Biosystems) and copy numbers were determined. The summed absolute difference between two MLVA-typed isolates was the calculated summed tandem repeat difference (STRD) at all seven loci.[17] Isolates with MLVA STRD ≤ 2 were indicative of a high degree of genetic relatedness.

Results

Environmental sampling results

For the pre-deep clean, 342 sites were sampled and 37 (10.8%) were positive for *C. difficile*. This included 15, 11 and 11 positives from low, medium and high height sites, respectively. Following the deep clean, 21 (6.1%) sites remained *C. difficile* positive which included 13, three and five positives from low, medium and high height sites respectively. Post HPD the number of positives was reduced to only three (0.9%), comprising positives from a wall-mounted glove dispenser, a nurse call button, and glass panels on corridor dividing doors. We found no *C. difficile* in the fourth environmental sampling exercise, which was carried out 19 days after HPD. At the fifth sampling exercise, carried out 20 weeks following HPD, we found a total of 12 (3.5%) sites positive for *C. difficile* (Table I and Figure 1).

Ribotyping results

Five different ribotypes (001/072, 014, 078, 002 and 046) were isolated from CDI-positive patients staying in the SRU during the 10-month period (January 2011 to October 2011). Four of these ribotypes (001/072, 014, 078 and 046) were recovered from the SRU during environmental sampling. Ribotypes 078, 014 and 046 were found in pre- and post-deep-clean samples, but not following HPD (Figure 2). Ribotype 001/072 was found during the post-deep clean and immediately following HPD sampling points. Non-toxigenic *C. difficile* ribotypes including 035 and 010 were also found.

Twelve samples were positive in the long-term follow-up at 20 weeks post HPD (Table I and Figure 1). These included 10 positives of ribotype 002 and two positives of ribotype 087. This sampling episode coincided with the presence on the ward of a patient who had been isolated in a side room because of diarrhoea and a suspicion of CDI. This patient was confirmed CDI positive the day after we sampled, and typing confirmed a ribotype 002. The 002 strains isolated from environmental and patient specimens were indistinguishable by enhanced fingerprinting (MLVA profile 25-14-25-7-6-10-2) (Table I).

Discussion

Several studies have reported the effectiveness of HPD (using droplets, aerosols and vapour) for *C. difficile* decontamination of clinical areas.[18–20] These studies have predominantly examined single rooms, and have employed modest microbiological testing and often little or no follow-up. For example, Passaretti *et al*. carried out a 30-month prospective cohort intervention study of HPD on rooms in three units after discharge of patients known to be infected or colonized with multidrug-resistant organisms (MDROs).[21] They compared room HPD with standard disinfection methods and found that HPD was associated with a 64% reduced risk of patients acquiring any MDRO ($P < 0.001$). However, the reduction in risk of acquiring *C. difficile* (and meticillin-resistant *Staphylococcus aureus* and multidrug-resistant Gram-negative rods) individually was not statistically significant. The total yield of environmental *C. difficile* was two rooms out of 1039 sampled. Similarly, Boyce described their use of HPD in association with

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Table I
C. difficile positives for each sampling session with locations and ribotype results

Sampling session	Area of ward	Sample type	Location within ward	Positive site	Ribotype	MLVA (if applicable)
Pre deep clean	Male	High	Side room 1	Patient line box	078	
			Bathroom	Curtain rail	078	
			Bathroom	Window ledge	035	
			Bay 6	Patient line box (L)	078	
				Patient line box (R)	078	
			Bay 5	Window ledge	035	
		Medium	Side room 3	Bins	014	
			Bay 6	Bins	035	
				Towel dispenser	035	
			Bay 5	Handwash basin	035	
				Towel dispenser	035	
				Bed rail (L)	035	
				Bed rail (R)	035	
				Bed control	035	
		Low	Side room 2	Bed base	046	
				Floor	078	
			Side room 3	Floor	035	
				Bed base	078	
				Table	014	
			Day room 2	Radiator	078	
				Floor	035	
			Bay 7	Table	035	
			Bay 6	Floor	035	
				Bed base	014	
	Female	High	Bay 1	Curtain rail	005	
				Patient line box	046	
		Medium	Bay 2	Bins	014	
			Bathroom	Pop-up cone	027	
			Bay 3	Bed rail	014	
		Low	Bathroom	Pipe work	035	
				Floor	078	
			Bay 2	Floor	035	
				Door ledges	014	
	General	High	Nurses station	Air con unit	046	
			Day room	Top of drinks machine	078	
			Store 1	Top of shelves	046	
		Low	Store 2	Trolley wheels	010	
Post deep clean	Male	High	Day room 2	Lights	078	
			Bay 6	Patient line box	078	
			Bay 5	Patient line box (L)	078	
				Patient line box (R)	078	
		Medium	Side room 2	Trunking	046	
			Bay 5	Bedside table	035	
		Low	Side room 2	Floor	035	
				Bed base	046	
				Table base	014	
			Corridor	Floor	035	
			Bathroom	Floor	078	
				Pipe work	078	
			Corridor 2	Floor	078	
	Female	Low	Bay 3	Bed base	046	
			Bathroom	Floor	078	
			Toilet	Partition	078	
			Corridor	Floor	001/072	
	General	High	Day room	Picture frame	078	
		Medium	Physio room	Table	046	

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Table I (*continued*)

Sampling session	Area of ward	Sample type	Location within ward	Positive site	Ribotype	MLVA (if applicable)
		Low	Nurses' station	Trolley wheels	078	
			Corridor	Door stop	078	
Post HPD	Male	Medium	Side room 2	Bed control	046	
			Bay 5	Glove dispenser	027	
	Female	Low	Corridor	Doors	001/072	
20 weeks post HPD	Male	High	Side room 1	Patient line box	002	25-14-25-7-6-10-2
			Bay 5	Window ledge	087	
		Medium	Side room 1	Medicine cabinet	002	25-14-25-7-6-10-2
				Nurse call button	002	25-14-25-7-6-10-2
				Locker	002	25-14-25-7-6-10-2
				Bins	002	25-14-25-7-6-10-2
			Side room 3	Handwash basin	087	
			Bay 5	Nurse call	002	25-14-25-7-6-10-2
			Bay 6	Bed rail	002	25-14-25-7-6-10-2
				Nurse call button	002	25-14-25-7-6-10-2
		Low	Side room 1	Floor	002	25-14-25-7-6-10-2
				Table	002	25-14-25-7-6-10-2

MLVA, multi-locus variable repeat analysis; HPD, hydrogen peroxide decontamination.

the control of an outbreak of CDI.[22] HPD was efficacious in eradicating *C. difficile* from contaminated surfaces, but these data were based on only 35 rooms plus eight open wards versus 29 plus eight environmental samples, taken before versus after the HPD intervention, respectively. The claim that HPD reduced the incidence of CDI is doubtful as the outbreak appeared to have ended before the intervention started. Havill *et al.* evaluated the reduction in bacterial counts on pre-contaminated carrier discs placed in 15 patient rooms.[18] HPD was more effective than UVC light at killing *C. difficile*; the mean $\log_{10}$ reduction was >6 for HPD and ~2 for UVC. Not surprisingly, UVC was significantly less effective for sites that are out of direct line of sight. Barbut *et al.* showed that HPD was significantly more effective than hypochlorite cleaning in removing *C. difficile* from rooms occupied by CDI cases.[23] They examined a total of 360 samples from rooms treated with hydrogen peroxide and 388 from rooms treated with hypochlorite. Sampling was carried out 1 h after decontamination, but not beyond. Holmdahl *et al.* carried out an in-vitro head-to-head comparison of a hydrogen peroxide vapour and aerosol, and concluded that the former was more effective and faster at killing in studies using *G. stearothermophilus* biological indicator.[9] Fu *et al.* reached similar conclusions in favour of hydrogen peroxide vapour.[8]

Our long-term study shows that HPD, after deep cleaning, was a more effective way of reducing *C. difficile* levels in the environment than the use of deep cleaning using a chlorine-detergent agent alone. We have previously shown, using a ward cross-over design, that the same chlorine-detergent agent significantly reduced *C. difficile* environmental contamination and CDI in one of two study elderly medicine wards, in comparison with detergent-based cleaning.[6] The use of HPD was associated with a reduction of CDI incidence, from 20 cases in the 10 months before the intervention to seven in the following equivalent period; the latter were isolated cases and not considered to be due to cross-infection. The reduction in incidence was possibly a result of improved cleaning practices on the ward, more comprehensive staff training and a heightened awareness of good hand hygiene and infection prevention.

In the first environmental sampling exercise, carried out before deep cleaning, we recovered *C. difficile* from 10.8% of sites in the SRU, including the same ribotype that had been isolated from CDI cases in this ward. As in most hospitals, sampling of the environment is not routine and therefore contamination levels remain largely unknown. It is only when problems arise, typically persistent evidence of *C. difficile* transmission, that environmental sampling is sometimes used to guide enhanced decontamination.[24] As the SRU underwent routine daily cleaning until the day on which the patients and staff were relocated, the finding of 10.8% of sites to be *C. difficile* positive suggests that routine manual cleaning and disinfection (with a combined detergent/chlorine release product) does not reliably remove environmental spores. Previous studies have reported that up to one-quarter of sites in *C. difficile* high-risk areas were positive despite regular environmental cleaning.[25,26] Speight *et al.* recently reported that, of 32 disinfectants tested against spores of *C. difficile in vitro*, 16 products achieved $>10^3$ reduction in viability after 60 min under both clean (0.3% albumin) and dirty (3% albumin) conditions. However, only eight products achieved $>10^3$ reduction in viability within 1 min under dirty conditions. Our in-situ sampling data, both at baseline and following deep cleaning with a chlorine-detergent agent, support such observations, although it is not possible to determine whether suboptimal removal of *C. difficile* is due to reduced disinfectant killing, poor cleaning technique or a combination of these. Notably, Rupp *et al.* have recently reported that there was no significant correlation between the amount of time taken to clean a room and the effectiveness of cleaning frequently touched surfaces.[27] It has been shown that simple educational interventions directed at housekeeping staff may improve the decontamination of environmental surfaces.[28,29] However, we point out here the relatively poor efficacy of deep cleaning at removing *C. difficile* was despite cleaning by trained staff.

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Figure 1. Plan of the stroke rehabilitation unit showing the locations where the positive samples were found. Corridor areas (yellow), bathroom areas (blue), male area bed bays (purple), female area bed bays (red) and single-bed side rooms (green).

Our results demonstrate that *C. difficile* was distributed similarly in low, medium and high height sites. This may reflect the known potential for airborne dissemination of *C. difficile*, especially around newly symptomatic patients.[14] Interestingly, in the environmental samples prior to the HPD, the lowest locations remained the least clean. This may be as a result of a focus by the cleaners on cleaning the more frequently touched middle and high site areas, but also because the lower sites (e.g. bed bases, table bases) may be more difficult to clean. We found a number of positive floor samples; it is plausible to consider that the floor could be acting as a reservoir for spread of infection within the ward, as the continual contact and the disturbance of debris might be adding to airborne dissemination of spores.[14] Clusters of sites positive for the same *C. difficile* ribotype were recovered from frequently touched areas. We found seven positives of ribotype 035 in two adjacent bays from the bed, towel dispenser and bins, and ribotype 014 was found in two adjacent bays from the bins and a bed rail. Ribotypes 014, 078, 046, 001/072 and 002 were recovered from CDI cases on the SRU in the 10-month period prior to testing. Only ribotype 014 was recovered from the medium height sites, compared with the low sites where ribotypes 014 and 078 were recovered, and from the high sites where ribotypes 078 and 046 were recovered. This suggests that whereas the medium height sites are the most frequently touched sites — and so can act as an immediate source of transmission to patients — they may be areas most likely to be frequently cleaned. By contrast, we were able to recover *C. difficile* ribotypes associated with other CDI cases, suggesting that these may have persisted on out-of-reach sites, such as the tops of a drinks machine, shelf unit, and patient line boxes, and on bathroom pipe work and a radiator base. It is likely that these areas could have been missed during routine cleaning. Airborne dissemination of *C. difficile* spores may have seeded such rarely touched sites in the same manner that airborne dissemination may infect patients.[14]

The isolation of ribotype 002 from a single patient and from 10 environmental samples in the final episode of sampling demonstrates the relative ease and extent to which the environment can become recontaminated with *C. difficile* from just a single infected patient. The patient was admitted to bay 5 of the SRU 15 days before the sampling episode (20 weeks post HPD) and moved into side room 1 three days later, because of a previous history of CDI with *C. difficile* ribotype 078. The

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patient had no symptoms of diarrhoea on admission to the side room, but six days afterwards had a single episode of diarrhoea, which was discarded untested. No further diarrhoea was recorded until seven days later, the day after environmental sampling had been carried out, when persistent symptoms began. This patient was *C. difficile* toxin positive due to ribotype 002, which was indistinguishable by MLVA from isolates recovered from seven sites in side room 1, two sites in bay 5 (directly across a clinical corridor from side room 1) and one site in bay 6 (adjacent to bay 5) (Figure 2). Although *C. difficile* ribotype 002 had been isolated from CDI cases occurring prior to HPD, it had not been isolated from any of the subsequent environmental samples; it is unlikely, therefore, that he acquired this strain from the SRU environment. It may be concluded that the patient's room became heavily contaminated with *C. difficile* 002 strain, despite the absence of reported diarrhoea in the early stages of his CDI. The presence of the same *C. difficile* strain in bays 5 and 6 represents either contamination by this patient before symptoms became evident and/or the transfer of spores from the side room.

Deep cleaning reduced the numbers of *C. difficile* positives by 43%, but had almost no effect at removing *C. difficile* from low sites (15 versus 14 positives, pre versus post deep clean, respectively). On further investigation four of these positives, from the bathroom floor, were *C. difficile* 078, suggesting that these bacteria could possibly have been redistributed during floor mopping. There were only three positives from medium height sites, suggesting again that these were the areas where cleaning was more concentrated/effective. Six high sites were positive, all with the same ribotype (078), one of which was positive with the same strain pre deep cleaning; this implies that this surface (patient-operated flat-screen video display unit) had been inadequately cleaned during deep cleaning.

Hydrogen peroxide decontamination further reduced the number of *C. difficile* sites that were still *C. difficile* positive after deep cleaning by 86%; overall, deep cleaning plus HPD reduced the number of positive sites by 92%. Of the three sites that remained positive for *C. difficile*, one was the bed control button where a ribotype 046 was recovered. As ribotype 046 had been found from both the previous environmental samples within the same side room and from the bed, this suggests that this site may not have been fully exposed to hydrogen peroxide. Ribotype 001/072 was recovered from glass panels on the corridor dividing doors; the same ribotype was recovered in the previous environmental sampling from the adjacent floor in the corridor, suggesting this may have been redistributed after deep cleaning, while the ward was prepared for HPD. The third positive (ribotype 027) was recovered from a wall-mounted gloves dispenser. This ribotype had not been recovered from any earlier environmental samples and had not been found in recent CDI cases on SRU. HPD was effective at decontaminating high sites, with no *C. difficile* isolated post exposure. There may be differences in the efficacy of HPD according to the porosity of surfaces.[30]

HPD offers potential benefits as a decontamination method as it offers complete surface coverage in contrast to

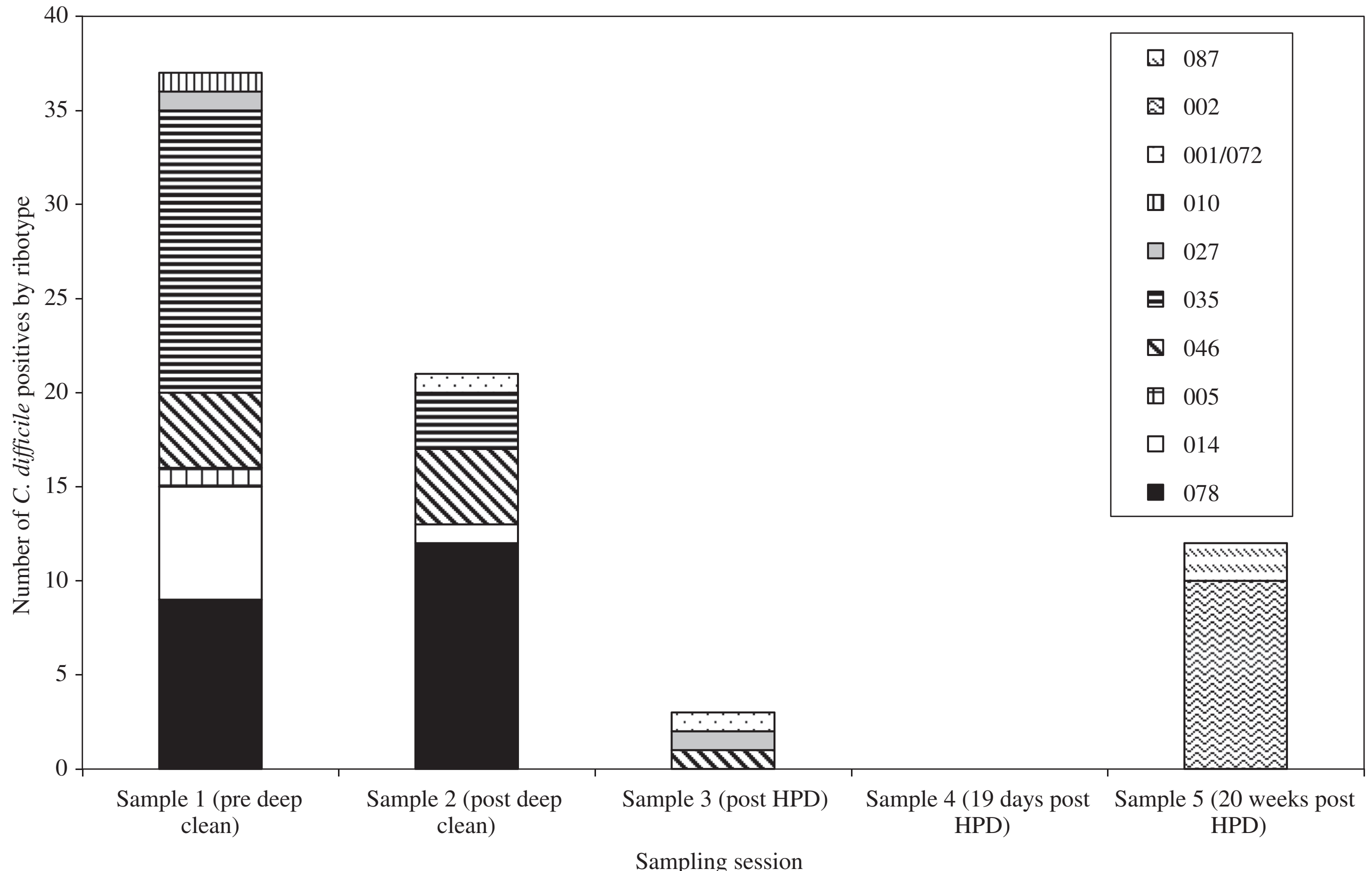

Figure 2. Total number of *C. difficile* positive at each sampling session by ribotype. HPD, hydrogen peroxide decontamination.

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conventional (deep) cleaning that is prone to operator error and/or suboptimal disinfectant contact times, and which also may miss bacteria in cracks and crevices or in ventilation systems.[23,27,28] Additionally, HPD can be used on electrical equipment, which may be damaged through the use of some liquids and cleaning chemicals. There are, however, limitations to HPD. These include the need to first remove debris and organic matter from surfaces that may prevent droplets of hydrogen peroxide from accessing micro-organisms. The whole ward has to be moved to alternative accommodation, which is a major undertaking and is dependent on the availability of decant space, an increasingly rare resource in some hospitals. This is a prime reason why most reports of HPD relate to its application to rooms as opposed to whole wards. Even when applied to rooms, HPD is relatively time-consuming. For example, HPD required a mean of 2 h and 20 min per room, which was about four times longer than needed for conventional cleaning.[27,30,31] Safety concerns, the isolation of fire alarm systems, and the need for desorption time are added drawbacks. Finally, HPD is expensive (with costs amounting to about £7,000 per ward, including necessary staff costs and materials), and therefore may only be justifiable in some cases.

There are several limitations to the study. It was not possible for us to test the effect of HPD alone without the prior deep clean; thus, we were not able to directly compare each method. It would be interesting to test the effect of HPD on environments with different levels of soiling. In general, as HPD is a disinfection process, it is always applied after cleaning within an environment with little debris and soiling. If it could be applied without the need to wait for a deep clean to take place, the HPD process may become easier, less time-consuming and more cost-effective for healthcare use. Also, we did not test the activity of HPD on porous surfaces. It would be useful to ascertain if it could be used for other surfaces such as fabric hospital curtains, which may be contributing to microbe dissemination.

There is little doubt that HPD is effective at killing bacteria including *C. difficile* in the environment. The key issues are whether this enhanced environmental antimicrobial effectiveness of HPD is long-lived and whether removal of *C. difficile* translates into reduction in CDI incidence. The answer is likely to be influenced by the rate of CDI, including whether a ward is in an outbreak or non-outbreak setting. In the outbreak setting nosocomial transmission of *C. difficile* is likely to be occurring frequently, but so will (re)contamination of the healthcare environment; conversely, emerging evidence shows that only a minority of CDI cases is linked to other cases when endemic as opposed to epidemic infection rates prevail.[32] There may therefore be an optimum level of CDI at which HPD is most likely to be cost-effective. Our results demonstrate that HPD may be a useful method for decontamination of a hospital ward with a high CDI incidence.

Conflict of interest statement

The Deprox system and hydrogen peroxide vapour for this study were provided free of charge by Hygiene Solutions, Kings Lynn, Norfolk, UK. Hygiene solutions had no input into the study protocol, results analysis or manuscript.

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