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Assessing the biological efficacy and rate of recontamination following hydrogen peroxide vapour decontamination☆

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Summary The inanimate hospital environment can become contaminated with nosocomial pathogens. Hydrogen peroxide vapour (HPV) decontamination has proven effective for the eradication of persistent environmental contamination. We investigated the extent of meticillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci (VRE) and gentamicin-resistant Gram-negative rod (GNR) contamination in a ward side-room occupied by a patient with a history of MRSA, VRE and GNR infection and colonisation and investigated the impact of HPV decontamination. Fifteen standardised sites in the room were sampled using a selective broth enrichment protocol to culture MRSA, VRE and GNR. Sampling was performed before cleaning, after cleaning, after HPV decontamination and at intervals over the subsequent 19 days on two separate occasions. Environmental contamination was identified before cleaning on 60, 30 and 6.7% of sites for MRSA, GNR and VRE, respectively, and 40, 10 and 6.7% of sites after cleaning. Only one site (3.3%) was contaminated with MRSA after HPV decontamination. No recontamination with VRE was identified and no recontamination with MRSA and GNR was identified during the two days following HPV decontamination. Substantial recontamination was identified approximately one

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week after HPV decontamination towards post-cleaning levels for GNR and towards pre-cleaning levels for MRSA. HPV is more effective than standard terminal cleaning for the eradication of nosocomial pathogens. Recontamination was not immediate for MRSA and GNR but contamination returned within a week in a room occupied by a patient colonised with MRSA and GNR. This finding has important implications for the optimal deployment of HPV decontamination in hospitals.

Introduction

The inanimate hospital environment can become contaminated with nosocomial pathogens including meticillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci (VRE), *Clostridium difficile* and Gram-negative rods (GNR) such as *Acinetobacter baumannii*.[1–5] There is strong evidence that contaminated environmental surfaces are involved substantially in the nosocomial transmission of certain organisms such as *C. difficile*, *Acinetobacter* spp., MRSA and VRE but the degree of contribution is controversial.[1,4–8]

Several factors contribute to the significance of environmental contamination in hospitals. The hands of healthcare workers (HCWs) can become contaminated with nosocomial pathogens, including MRSA, VRE and GNR, through contact with environmental surfaces without direct patient contact, even in rooms that have already been cleaned.[2,9,10] Several studies have highlighted the poor efficacy of manual terminal cleaning for the eradication of nosocomial pathogens from environmental surfaces.[3,5,10–13] In contrast to deficiencies in manual cleaning, hydrogen peroxide vapour (HPV) decontamination, a sporicidal vapour-phase method, has been shown to eradicate nosocomial pathogens from hospital rooms and has been used for terminal decontamination following hospital outbreaks.[3,11,14,15]

HPV is catalytically converted to oxygen and water vapour during the aeration phase of the cycle, so there are no problematic residues and hence no residual biocidal effects. A previous study has demonstrated rapid recontamination with MRSA on an intensive care unit (ICU) following HPV decontamination.[16] Further studies to assess the rate of recontamination are important for deciding upon the optimal deployment of HPV decontamination in hospitals.[17]

We prospectively evaluated the comparative efficacy of conventional terminal cleaning and HPV for the decontamination of environmental surfaces in a ward side-room occupied by a patient with an extensive history of MRSA, GNR and VRE infection and colonisation. We then assessed the rate of recontamination following HPV decontamination.

Methods

North Middlesex University Hospital is a 500-bed teaching hospital in North London. The study was conducted in a single occupancy 35 m^3 ward side-room with an en-suite bathroom. The room was occupied by the same patient throughout the study period, who had been in the room for approximately 18 months prior to the study period.

Microbial contamination was assessed by sampling 15 standard sites in the room for MRSA, GNR and VRE (i) before terminal cleaning, (ii) after terminal cleaning, (iii) after HPV decontamination and (iv) at intervals over the subsequent 19 days. The 15 sites sampled were: floor beside the bed, floor corner, bed-frame, bed-elevation control panel, bedside chair, bedside locker, over-bed table, television (TV) remote control, TV stand, radio remote control, radio, audio cassette holder, room door handle, toilet door handle, and toilet floor. This process was conducted in two distinct experiments separated by five months; the sites were sampled 7 and 19 days after HPV in the first experiment and 1, 2, 5, 6, 7, 8 and 19 days after HPV in the second experiment to assess the rate of recontamination.

Terminal cleaning conducted after the discharge of the patient differed from standard cleaning in that a quaternary ammonium compound (QAC)-containing disinfectant-detergent (HP800, PVA Hygiene Ltd, Weston-super-Mare, Somerset, UK) was used instead of detergent; the cleaning process complied with NHS standards and local protocols. HPV decontamination was conducted using the Room Bio-Decontamination Service (RBDS™) (BIOQUELL (UK) Ltd. Hampshire, UK) as described previously.[3] Eight *Geobacillus stearothermophilus* biological indicators (BIs) with a $>1.0 \times 10^6$ loading on 10 mm stainless steel discs in Tyvek pouches (Apex Laboratories, Apex, USA) were located around the room to verify the efficacy of both HPV cycles and analysed as described previously.[3]

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Patient details

The patient was a 36-year-old male who had undergone extensive general surgery. The patient had a considerable history of infection and colonisation over the 18 months prior to the study, including persistent colonisation and infection with MRSA at multiple sites, transient colonisation with gentamicin-resistant GNR such as *Acinetobacter* sp., *Escherichia coli*, *Serratia* sp. and *Pseudomonas aeruginosa*, and various other infections including a VRE wound infection, a *Streptococcus* sp. wound infection and *E. coli* bacteraemia. We suspected that persistent environmental contamination was a factor in the patient's recurrent infection so we used HPV decontamination of the patient's room as a novel adjunct to standard decolonisation methods. The patient had a full microbiological screen on the day of intervention, at one week and two weeks post-intervention for the first experiment and regularly throughout the second experiment.

Environmental sampling methods

All media were obtained from BioConnections (Wetherby, Leeds, UK). Three sterile cotton-tipped swabs, for MRSA, gentamicin-resistant GNR and VRE, respectively, were moistened in nutrient broth (NB) and used to sample each site. (Gentamicin resistance in GNR was used as a marker of clinical significance.) Separate 25 cm^2 areas were used for each swab so that the same area was not sampled more than once before cleaning, after cleaning and after HPV decontamination. The same area on each site was then sampled repeatedly to assess recontamination. The swab tips were cut into separate 4 mL vials of NB. All broths were incubated at 37 °C for 24 h as an enrichment step. The MRSA enrichment broth was plated onto Baird—Parker (BP) agar containing 8 mg/L ciprofloxacin. *S. aureus* were identified by colonial morphology on BP and meticillin resistance was tested by a strip diffusion test (Mast, Bootle, UK). The GNR enrichment broth was plated onto MacConkey agar containing 4 mg/L gentamicin from which GNR were identified and speciated by standard methods.[18] The VRE enrichment broth was plated onto 'Staph/Strep' agar containing 5 mg/L vancomycin from which VRE was identified by standard methods. No sampling for VRE was conducted in the second experiment based on the results of the first experiment.

A selection of MRSA environmental isolates from the second experiment were compared to a patient isolate by pulsed-field gel electrophoresis (PFGE), which was conducted by the Health Protection Agency's Staphylococcal Reference Laboratory.

Results

MRSA and GNR were cultured from 60.0 and 30.0% of the 30 sites, respectively, before cleaning in the two experiments combined. MRSA and GNR were cultured after terminal cleaning from 40.0 and 10.0% of the sites, respectively, which took approximately 4 h from ordering to completion on both occasions. VRE was cultured from the floor beside the bed before cleaning and from the bed frame after cleaning in the first experiment only (Table I).

HPV was injected for 30 min at 20 g/min and a further 30 min at 12 g/min in both the first and second cycles. Peak HPV concentrations determined by an HPV sensor situated inside the room were 530 and 540 ppm for the first and second cycles, respectively, and the rate of change in HPV concentration tended towards zero for both cycles, indicating that microcondensation had occurred on the surfaces in the room. All 16 BIs exposed to HPV were deactivated and no GNR or VRE were cultured from the room immediately after HPV decontamination (Table I). MRSA was cultured from the radio remote following HPV decontamination in the second experiment, representing 3.3% of the 30 sites in the two experiments combined (Table I).

MRSA and GNR were cultured from 26.7 and 13.3% of sites, respectively, after both 7 and 19 days in the first experiment, which was approximately the post-cleaning level of contamination. To investigate further the rate of recontamination, the sampling frequency was increased during the second experiment and it was found that MRSA recontamination had not occurred by day 2 after HPV decontamination, but 46.7% of the sites were contaminated with MRSA by day 5 and 73.3% by day 6. In contrast, no GNR contamination was detected in the room until day 8, when 13.3% of the sites were contaminated with GNR (Table I).

The patient remained colonised and infected with MRSA during the course of the first and second experiments at multiple locations. PFGE analysis found that 12 environmental isolates recontaminating the room during the second experiment were grouped as three variants of epidemic (E)MRSA-15 whereas the patient isolate from 12 months previously was EMRSA-16 (data not shown). At the time of the second experiment, the patient had a wound infected with MRSA resistant to ciprofloxacin and erythromycin in addition to resistance to the β-lactams, which is the same antibiogram type as the 12 environmental isolates and a common antibiogram type for EMRSA-15.

Fifty-three percent of the GNR cultured from the environment were *Acinetobacter* spp. and 32% were

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Table I Proportion of sites contaminated with meticillin-resistant *Staphylococcus aureus* (MRSA), gentamicin-resistant Gram-negative rods (GNR) and vancomycin-resistant enterococci (VRE) before cleaning, after cleaning, after hydrogen peroxide vapour (HPV) decontamination and at intervals over the subsequent 19 days during two separate experiments in a ward side-room[a]

		Before cleaning	After cleaning	After HPV	Days after HPV decontamination						
					1	2	5	6	7	8	19
No. of sites sampled	1st experiment	15	15	15	—	—	—	—	15	—	15
	2nd experiment	15	15	15	15	15	15	15	15	15	15
	Total	30	30	30	15	15	15	15	30	15	30
MRSA	1st experiment	7 (46.7)	3 (20.0)	0	—	—	—	—	4 (26.7)	—	4 (26.7)
	2nd experiment	11 (73.3)	9 (60.0)	1 (6.7)	1 (6.7)	0	7 (46.7)	11 (73.3)	8 (53.3)	4 (26.7)	6 (40.0)
	Total	18 (60.0)	12 (40.0)	1 (3.3)	1 (6.7)	0	7 (46.7)	11 (73.3)	12 (40.0)	4 (26.7)	10 (33.3)
GNR	1st experiment	4 (26.7)	2 (13.3)	0	—	—	—	—	2 (13.3)	—	2 (13.3)
	2nd experiment	5 (33.3)	1 (6.7)	0	0	0	0	0	0	2 (13.3)	1 (6.7)
	Total	9 (30.0)	3 (10.0)	0	0	0	0	0	2 (6.7)	2 (13.3)	3 (10.0)
VRE	1st experiment	1 (6.7)	1 (6.7)	0	—	—	—	—	0	—	0

Values in parentheses are percentages.

[a] Fifteen sites in a ward side-room were sampled before cleaning, after cleaning, after HPV decontamination and at intervals over the subsequent 19 day period in two separate experiments, which were separated by five months.

Klebsiella spp. but the patient was not colonised or infected with the GNR species that was cultured from the patient environment after HPV decontamination during the first or second experiments (data not shown).

The sites that were contaminated after cleaning did not always match those contaminated before cleaning. For example, three (25%) of the 12 sites found to be contaminated with MRSA after cleaning (the floor corner, the bedside locker and the TV stand) were not found to be contaminated before cleaning (data not shown).

Certain sites were contaminated more frequently with MRSA and GNR, e.g. the floors and certain hand-touch sites such as the TV remote control and the bed-frame (Figure 1).

Discussion

We found that conventional terminal cleaning did not eradicate MRSA, GNR and VRE from the hospital environment. Several other studies have demonstrated that MRSA, VRE and GNR are not eradicated by conventional terminal cleaning although before-and-after studies investigating the impact of conventional terminal cleaning for the eradication of GNR are lacking.[3,5,11–13,19] Our finding that 10% of 30 sites remained contaminated with GNR despite terminal cleaning provides further evidence that GNR can persist despite terminal cleaning.

The frequency of contamination with GNR was lower than the frequency of contamination with MRSA, which is consistent with other studies (Figure 1).[20,21] Although we did not evaluate hand contamination, several hand-touch sites in the near-patient environment were contaminated frequently with MRSA and GNR and present a risk of contaminating HCW hands (Figure 1).

The origin of the VRE sampled from the room before and after cleaning in the first experiment (Table I) is unknown because no VRE had been cultured from the patient for six months prior to the first experiment and the patient was screen negative for VRE throughout the study period. It is possible that the VRE persisted from a VRE wound infection six months before the first experiment since VRE is very uncommon in our hospital. VRE can survive for extended periods on environmental surfaces, hence this explanation is plausible.[22]

In our study, HPV was an effective method for the decontamination of nosocomial pathogens from surfaces, which corroborates data from other studies.[3,11,15] Our study found that one site was contaminated following HPV decontamination (Table I). French *et al.* also found that one (1.2%) of 85 sites remained contaminated with MRSA following HPV decontamination.[3] The site was contaminated by enrichment in both instances, which will detect a low level of contamination. Surfaces should be cleaned prior to HPV decontamination because biological soiling reduces the efficacy of HPV.

We found that the level of MRSA and GNR contamination had returned towards pre-cleaning levels after one week. However, no MRSA recontamination had occurred by day 2 after HPV decontamination in the second experiment (Table I). A recent study investigated MRSA recontamination following HPV decontamination on an ICU.[16] MRSA contamination to the post-cleaning level occurred by 24 h post-HPV

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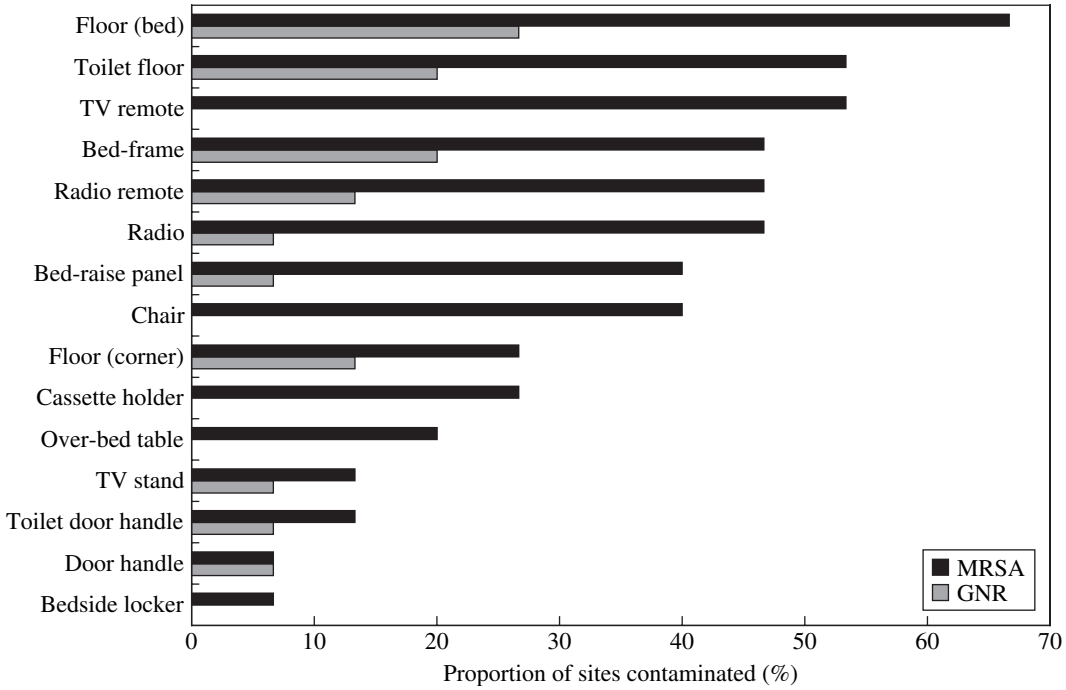

Figure 1 Frequency of contamination with meticillin-resistant *Staphylococcus aureus* (black bars) and gentamicin-resistant Gram-negative rods (grey bars) from 15 standard sites sampled on 15 occasions during the two separate experiments in a ward side-room.

decontamination on the open-plan ICU. The relatively rapid MRSA recontamination identified in this study compared with ours could be explained by the fact that multiple (two of nine) patients admitted to the ICU following HPV decontamination were MRSA positive. Interestingly, no MRSA was identified on the next two screens of the ICU at 48 h and one week post-HPV despite the presence of MRSA-colonised patients on the ICU and the frequency of MRSA contamination did not return to pre-cleaning levels until four weeks after HPV. The aerobic colony counts on workstations returned to post-cleaning levels by 24—48 h and towards pre-cleaning levels by 1 week post-HPV, which is similar to the MRSA recontamination pattern identified in our study.

Studies in the 1960s demonstrated rapid staphylococcal recontamination of floors and clinical areas within a matter of hours.[23,24] Compared with these investigations, our study did not identify rapid recontamination. These studies investigated principally floor contamination with meticillin-susceptible *S. aureus*, which is a common component of the natural skin flora; thus more frequent contamination, especially on the floor, is to be expected.

The patient remained colonised with MRSA during and after both experiments so HPV decontamination did not result in the decolonisation of the patient through removal of the environmental reservoir. It is likely that the MRSA recontamination originated from the patient because he remained heavily colonised and infected with MRSA at multiple sites. PFGE identified three variants of EMRSA-15 in the environment, which was consistent with the antibiogram of the patient's infecting strain.

It is unlikely that the GNR recontamination originated from the patient because the GNR species of the environmental contamination did not match the GNR species infecting or colonising the patient. Several studies have identified bacterial contamination of liquid cleaning solutions, especially with GNR, which can 'seed' environmental surfaces.[25—27] It is possible therefore that the GNR recontaminating the room originated from other parts of the ward and were introduced into the room through contaminated cleaning materials, though we did not sample the cleaning materials used in this study. We noted that certain of the sites that were not contaminated before cleaning were contaminated after cleaning. This could be explained by the fact that the sampling methods did not consistently identify contamination or that the cleaning process altered the profile of environmental contamination in the room.

Our study had several limitations. First, we investigated one patient who had an unusually extensive history of infection and colonisation with a variety of nosocomial pathogens. Certain patients and types of infections are likely to result in more frequent environmental contamination.[2,28] Further research is

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required to investigate recontamination rates in different patient groups.

Second, we used a non-quantitative enrichment protocol, which will detect low-level contamination. The critical level of environmental contamination that is significant for cross-transmission is unknown, but very low numbers of staphylococci are capable of causing infection under certain conditions.[29] Dancer (2004) has recently proposed a microbiological standard of <1 cfu/cm^2 for MRSA, certain species of GNR and VRE.[30] We took the view that the presence of MRSA, gentamicin-resistant GNR or VRE represents a potential risk for cross-transmission.

The findings of our research present several challenges for the implementation of HPV decontamination in hospitals, which has been discussed recently.[17] Further work is required to determine whether less frequent HPV decontamination possibly combined with the use of antimicrobial materials and enhanced cleaning will reduce the risk of infection acquisition. Another potential application of HPV decontamination in hospitals is the disinfection of mobile medical equipment, which could be conducted in a dedicated room. Work is underway at this hospital to investigate the impact of HPV decontamination on mobile medical equipment.

Our study has shown that a ward side-room was contaminated with MRSA, GNR and VRE, which was not eradicated by terminal cleaning. HPV proved to be effective for the eradication of contamination with nosocomial pathogens. When an MRSA- and GNR-colonised patient was in the room, substantial recontamination with MRSA occurred within five days, but not before two days, whereas substantial recontamination with GNR was not evident until one week after HPV decontamination. These data provide useful information for determining the optimal deployment of HPV decontamination in the healthcare setting.

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Conflict of interest statement
M.C., F.A., C.v.T., Y.J.D. have no potential conflicts. J.A.O. is employed part-time by BIOQUELL (UK) Ltd. All work was conducted collaboratively.

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