The Role Played by Contaminated Surfaces in the Transmission of Nosocomial Pathogens

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Studies in the 1970s and 1980s suggested that environmental surface contamination played a negligible role in the endemic transmission of healthcare-associated infections. However, recent studies have demonstrated that several major nosocomial pathogens are shed by patients and contaminate hospital surfaces at concentrations sufficient for transmission, survive for extended periods, persist despite attempts to disinfect or remove them, and can be transferred to the hands of healthcare workers. Evidence is accumulating that contaminated surfaces make an important contribution to the epidemic and endemic transmission of *Clostridium difficile*, vancomycin-resistant enterococci, methicillin-resistant *Staphylococcus aureus*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and norovirus and that improved environmental decontamination contributes to the control of outbreaks. Efforts to improve environmental hygiene should include enhancing the efficacy of cleaning and disinfection and reducing the shedding of pathogens. Further high-quality studies are needed to clarify the role played by surfaces in nosocomial transmission and to determine the effectiveness of different interventions in reducing associated infection rates.

Contamination of hospital equipment, medicines, and water supplies with hospital pathogens is a well-recognized cause of common-source outbreaks of infection. Extensive guidance on the prevention and control of such contamination is available from manufacturers, specialist societies, and health departments, and there is often a legal requirement to comply with associated health and safety regulations. In contrast, the degree to which ongoing contamination of the surface environment contributes to the development of healthcare-associated infections is unclear, and approaches to control are uncertain.

Hospital patients shed pathogens into their surrounding environments, but there is debate over the importance of the resulting surface contamination as a source for subsequent transmission. Since the 1950s, hospital design and hygienic practices have been largely directed at controlling nosocomial pathogens contaminating air, hands, equipment, and surfaces. However, several studies in the 1970s and early 1980s suggested that the hospital environment contributed negligibly to endemic transmission. Routine surveillance cultures of the hospital environment were regarded as unjustified, and the significance of environmental cultures performed during outbreaks was questioned. Consequently, the frequency of routine environmental sampling declined from three-quarters of US hospitals in 1975 to virtually none today. Indeed, in recent Centers for Disease Control and Prevention (CDC) guidelines environmental sampling is recommended only during outbreaks. Recently, however, there has been a reassessment of the role played by contaminated surfaces in the transmission of nosocomial pathogens.

Pathogen transfer from an affected patient to a susceptible host occurs most commonly via the hands of healthcare workers (HCWs), but contaminated objects, surfaces, and air can be either directly or indirectly involved in the transmission pathway (Figure 1). Here we review evidence that nosocomial pathogens are shed by patients and can contaminate hospital surfaces at concentrations sufficient for transmission, can survive for extended periods, can persist despite attempts to disinfect or remove them, and can be transferred to the hands of HCWs. We also review evidence that improved environmental hygiene can help to bring outbreaks under control and reduce endemic nosocomial transmission.

**Pathogens are shed into the hospital environment**

Several important pathogens, including *Clostridium difficile*, methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci (VRE), *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and norovirus, have the ability to survive in the dry-surface environment, which may then become a source for transmission. Fungi—in particular *Aspergillus* species—can also contaminate the hospital environment.
and cause healthcare-associated infection; however, fungi are a special group with unusual features that have been well reviewed elsewhere and will not be considered here.10

Bacteria, spores, and viruses are shed from infected and/or colonized patients (and sometimes staff) into the hospital environment. Wide variation in the reported frequency of environmental contamination can be explained by several factors, including the culturability of the organism, the degree of shedding by the patient, the sampling methodology, the ease of contamination (or difficulty of cleaning) of the particular environment, and whether there is an ongoing outbreak at the time of sampling. Methodological differences in sample collection and culture make comparisons between studies difficult, and in some cases the true level of environmental contamination may be underestimated.

Patients are the prime source of contamination, so surfaces in the vicinity of patients that are touched frequently by HCWs and patients—termed “high-touch surfaces”—have a higher frequency of contamination than other sites.11-14 For example, a recent study defined high-touch surfaces as the bed rails, the bed surface, and the supply cart, on the basis of their observed frequency of contact.12 Developing an understanding of which sites are more likely to be contaminated with pathogens can guide infection control practice and direct new innovations.

Areas around patients are frequently contaminated with MRSA, VRE, and C. difficile.15-17 The frequency of MRSA and VRE contamination correlates with the number of culture-positive body sites.13,18,19 Infected patients shed more pathogens than those who are only colonized, and diarrhea results in widespread contamination.13,20,21

Contamination of rooms of unaffected patients has been reported for C. difficile, MRSA, and VRE. C. difficile was identified in 16%-17% of samples from the rooms of patients without known C. difficile infection (CDI),22,23 MRSA was cultured from 43% of beds used by patients not known to be MRSA positive,17 and VRE was cultured from 13% of surfaces in the rooms of patients with unknown VRE status.24 Contamination of rooms of unaffected patients is most likely to be due to continued viability of organisms shed by previous occupants,17,25,26 but may also result from importation by HCWs or visitors or from shedding from asymptomatic carriers.27,28

Relatively few prospective studies have been conducted of gram-negative or norovirus surface contamination. The frequency of contamination is approximately 5%-10% of surfaces for gram-negative bacteria29,30 and was found to be highly variable but less than 20% of surfaces for norovirus RNA in one pediatric study.31

Highly variable levels of contamination have been reported during outbreaks. Frequent environmental contamination has been identified and implicated as a contributory factor during continuing outbreaks of C. difficile, MRSA, VRE, A. baumannii, and norovirus.21,32-35

Contamination of air has been reported, but the interchange between contaminated air and surfaces is not well defined.36,37

CONCENTRATION OF CONTAMINATION IS SUFFICIENT FOR TRANSMISSION

In general, colonized or infected patients have a higher concentration of contamination than their surrounding surfaces.14,19 The concentration of VRE is approximately 10³ colony-forming units (CFUs)/50 cm² on the skin of patients,38 whereas the concentration of C. difficile, VRE, and MRSA ranges from 10³ to 10⁹ CFUs/g in stool.20,39,40 The concentration of norovirus can be more than 10¹² particles/g in stool,41 and patients can vomit more than 10⁷ norovirus particles assuming a vomit bolus volume of 20–30 mL and the fact that 10⁶ particles/mL need to be present for detection by electron microscopy.42 In contrast, the concentration of nos-
Nosocomial pathogens is generally in the range of less than 1 to 100 CFUs/cm² on surfaces and is often detected only by broth enrichment. Reports of higher concentrations of surface contamination do occur and include total aerobic counts of more than 200 CFUs/cm² both before and after cleaning and more than 15 to more than 100 MRSA colonies from 23% of sites positive by direct plating in the rooms of MRSA-positive patients with diarrhea.

The presence of a pathogen on a surface does not necessarily represent a transmission risk. However, the infectious dose for most environmentally associated nosocomial pathogens appears to be low. For example, less than 15 S. aureus cells were sufficient to cause infection in experimental lesions, less than 1 CFU/cm² was sufficient to cause C. difficile disease in mice, and a single norovirus particle is thought to have the capacity to cause infection. Importantly, despite the comparatively low concentration of contamination on surfaces compared with that on the skin of patients, touching a VRE-contaminated surface carries approximately the same risk for acquisition of VRE on hands as touching an affected patient. Therefore, the presence of a pathogen on a surface at any concentration may be a risk for transmission, and this is reflected in proposed guidelines for microbiological hygiene standards.

Nosocomial Pathogens Can Survive on Surfaces for Long Periods

Studies investigating the survival of nosocomial pathogens on surfaces were recently reviewed by Kramer et al. Under certain conditions, C. difficile spores, VRE, MRSA, and Acinetobacter species can survive for 4–5 months or more on dry surfaces, and norovirus can survive for a week or more. Large variations in survival times in different reports is partly due to species and strain variation but is also due to differences in experimental conditions, including inoculum size, humidity, the suspending medium, and the surface material.

Limitations of Cleaning and Disinfection

Cleaning is the removal of soil and contaminants from surfaces, whereas disinfection relates to the inactivation of pathogens by use of a disinfectant. Microorganisms vary in their resistance to disinfectants, so agents must be chosen carefully for their effectiveness, particularly for C. difficile spores. Furthermore, the hospital environment is complex and often difficult to clean, and use of a cleaning agent that is not effective against the target organism can spread pathogens to other surfaces.

Liquid disinfectants may damage equipment, especially electronics, and chlorine-containing materials may corrode metals. Disinfectants can potentially harm users, and the discharge of waste biocides into the environment may encourage the development of both biocide and antibiotic resistance and have other, more general environmentally damaging effects. For these reasons, some authorities have questioned the use of routine disinfectant decontamination of the hospital environment and favor instead the use of detergents only. There has been a tendency for disinfectants to be used in the United States and detergents in Europe. Recently, United Kingdom and European workers have moved more toward the use of disinfectants to control MRSA and C. difficile, but the debate continues while we await more evidence for the effective use of particular agents.

Cleaning and disinfection does not always eradicate pathogens from surfaces. C. difficile was cultured from 44% of 54 surfaces after bleach disinfection in 9 rooms and from 16% of 243 surfaces after bleach disinfection implemented during an outbreak. VRE was cultured from 71% of 102 samples after bleach disinfection in 17 rooms, and it took an average of 2.8 bleach treatments to eradicate VRE in another study. MRSA was cultured from 66% of 124 surfaces in MRSA patient rooms after cleaning with a detergent sanitizer, was cultured from 16% of 65 sites after bleach and steam cleaning during an outbreak in a surgical ward, and was found at a concentration of 0.7 CFUs/plate after phenolic disinfection during an outbreak in a burn unit. Contamination has been identified on apparently clean surfaces during outbreaks due to A. baumannii and viruses. The frequent finding of contamination in empty rooms and in rooms occupied by patients unaffected by pathogens suggests residual contamination from previous occupants. However, the thoroughness of cleaning and disinfection was not evaluated in these studies, meaning that it is difficult to determine whether it is the products, the procedures, or a combination of the two that is responsible for the failure to eradicate pathogens from surfaces.

Nosocomial Pathogens Can Be Transferred From Contaminated Surfaces to the Hands of HCWs

In vitro studies present a picture of rapid dynamic transfer from surfaces to hands and vice versa (Table 1). For example, DNA markers dried onto toys were transferred readily to the hands of researchers and subsequently onto clean toys, and the markers spread rapidly when introduced into a child care center. Similarly, experimentally contaminated fingers serially contaminated multiple surfaces with norovirus. Similar findings have been reported using surfaces experimentally contaminated with bacteria and bacteriophage. Importantly, experimentally contaminated fingers have been shown to transfer more than 30% of inoculated bacteria and bacteriophage to the mouths of volunteers, with clear implications for the fecal-oral transmission of nosocomial pathogens.

Several studies have shown that various bacterial pathogens can be acquired on the hands of HCWs through contact with environmental surfaces in the absence of direct patient contact (Table 1). Patients and contaminated surfaces appear to transfer VRE to the hands of HCWs at similar fre-
<table>
<thead>
<tr>
<th>Reference</th>
<th>Setting, location</th>
<th>Organism(s)</th>
<th>Method</th>
<th>No. ( %) contaminated</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rusin et al&lt;sup&gt;60&lt;/sup&gt; (2002)</td>
<td>Laboratory, USA</td>
<td>Bacteria and phage</td>
<td>Fomites were experimentally contaminated with a mixture of bacteria and phage and touched by volunteers</td>
<td>10–20 ...</td>
<td>Transfer efficiency was higher for non-porous fomites (28%–66%); gram-positive bacteria had the highest transfer efficiency (41%)</td>
</tr>
<tr>
<td>Jiang et al&lt;sup&gt;59&lt;/sup&gt; (1998)</td>
<td>Child care center, USA</td>
<td>Virus surrogate</td>
<td>DNA was dried onto toys, which were passed to researchers to hold</td>
<td>5 5 (100)</td>
<td>Subsequent transfer of DNA to clean toys occurred on 3 of 5 occasions</td>
</tr>
<tr>
<td>Rheinbaben et al&lt;sup&gt;44&lt;/sup&gt; (2000)</td>
<td>Laboratory, Germany</td>
<td>Phage</td>
<td>Volunteers contacted an experimentally contaminated door handle</td>
<td>14 14 (100)</td>
<td>30%–66% of the inoculated virus was recovered from the hands of volunteers</td>
</tr>
<tr>
<td>Boyce et al&lt;sup&gt;13&lt;/sup&gt; (1997)</td>
<td>Ward side rooms, USA</td>
<td>MRSA</td>
<td>Hand cultures were performed after routine patient care without direct patient contact</td>
<td>12 5 (42)</td>
<td>All 12 HCWs wore gloves</td>
</tr>
<tr>
<td>Ray et al&lt;sup&gt;62&lt;/sup&gt; (2002)</td>
<td>Wards side rooms, USA</td>
<td>VRE</td>
<td>Hand cultures were performed after 5-second contact with bed rail and bedside table in rooms of VRE patients</td>
<td>13 6 (46)</td>
<td>5 of 6 hand cultures were indistinguishable from environmental cultures by PFGE</td>
</tr>
<tr>
<td>Barker et al&lt;sup&gt;52&lt;/sup&gt; (2004)</td>
<td>Laboratory, UK</td>
<td>Norovirus</td>
<td>Clean fingertips touched contaminated surfaces and then other objects</td>
<td>30 12 (40)</td>
<td>4 of 10 door handles, 5 of 10 telephones, and 3 of 10 taps became contaminated</td>
</tr>
<tr>
<td>Bhalla et al&lt;sup&gt;43&lt;/sup&gt; (2004)</td>
<td>8 wards, USA</td>
<td>Pathogens</td>
<td>Hand cultures were performed after 5-second contact with bed rail and bedside table</td>
<td>64 34 (53)</td>
<td>Positive hand culture results were obtained for 24% of 25 rooms that had been cleaned after patient discharge</td>
</tr>
<tr>
<td>Hayden et al&lt;sup&gt;11&lt;/sup&gt; (2008)</td>
<td>ICU, USA</td>
<td>VRE</td>
<td>Hand cultures were performed for 44 HCWs who had negative hand culture results at study entry and touched only environmental surfaces during routine patient care</td>
<td>44 23 (52)</td>
<td>Each contact with patient or environmental surface represented a 10% risk of acquiring VRE</td>
</tr>
</tbody>
</table>

**Note.** HCW, healthcare worker; ICU, intensive care unit; MRSA, methicillin-resistant *Staphylococcus aureus*; PFGE, pulsed-field gel electrophoresis; VRE, vancomycin-resistant enterococci.
Evidence that Surface Contamination Contributes to Nosocomial Cross-Transmission

A number of studies have identified the previous presence of a colonized or infected patient in a side room as a risk factor for the acquisition of the same pathogen by a new occupant, presumably because of residual room contamination that is not removed through terminal cleaning and disinfection. This effect has been shown for VRE, MRSA, C. difficile, multidrug-resistant P. aeruginosa, and A. baumannii (Table 2). A further strand of evidence suggesting that the contaminated surface environment contributes to the transmission of nosocomial pathogens is the effect of improved cleaning and disinfection on infection rates.

The next sections will review evidence that contaminated surfaces are important in the transmission of C. difficile, VRE, MRSA, norovirus, and certain gram-negative rods (Table 3).

C. difficile

C. difficile outbreaks were first linked to contaminated surfaces in the 1980s. Samore et al conducted a detailed 6-month prospective study of all C. difficile cases in a US hospital. The frequency of positive hand culture results and clinical cultures that matched the pulsortype of the index case patient among contacts (either roommates, neighbors, or subsequent room occupants of index case patients) correlated with the intensity of environmental contamination, suggesting that the transmission risk was related to the intensity of contamination.

Several studies have investigated the effect of improved environmental hygiene on the incidence of CDI. Mayfield et al showed that switching from quaternary ammonium compounds to bleach disinfection reduced the incidence of CDI for high-risk bone marrow transplant patients. However, no significant reduction in infection rates occurred for lower-risk patient groups, and environmental contamination was not quantified. Wilcox et al conducted a crossover study in elderly care wards to compare the effect of routine cleaning with detergent versus bleach and demonstrated a significant reduction in infection rates on one ward. No significant reduction was demonstrated on the other ward and the frequency of environmental contamination was not reduced in any study arm, suggesting that other factors were involved. More recently, Boyce et al found that the use of hydrogen peroxide vapor (HPV) to decontaminate rooms after the discharge of patients with CDI reduced the incidence of CDI in 5 high-incidence wards. The hospital-wide incidence of CDI was also reduced, but this was statistically significant only when the analysis was limited to the months when the epidemic NAP1 C. difficile strain was known to be present. Another before-after study of HPV by Manian et al also reported a significant hospital-wide reduction in the incidence of CDI.

Recent evidence from an in vitro model provides proof of concept that C. difficile spores can be transmitted via environmental surfaces. Mice exposed to an experimentally contaminated enclosure became colonized in a dose-dependent manner, and oxidizing agents, including a chlorine-containing liquid disinfectant and HPV, effectively reduced the level of contamination and blocked transmission. A recent study found that prior room occupancy by patients with CDI increased the risk of C. difficile acquisition, providing evidence that the in vitro concept translates into the clinical setting (Table 2).

VRE

Evidence from several studies suggests that hospital acquisition of VRE is associated with environmental contamination. Huang et al found that admission to a room previously occupied by a VRE-infected patient significantly increased the risk of acquiring VRE (Table 2). In this study, other routes of transmission (which may involve contaminated surfaces indirectly) accounted for the majority of nosocomial transmission. More recently, Drees et al found that VRE acquisition was associated with a positive room culture result before admittance to the room, with a prior room occupant positive for VRE, and with any VRE-positive room occupants within the 2 weeks before admission (Table 2). A recent cohort study of HPV decontamination in 6 intensive care units found that patients admitted to rooms decontaminated by HPV were less likely to acquire VRE than were patients admitted to rooms cleaned using standard methods when the prior room occupant was positive for VRE (incidence rate ratio, 0.22).

In addition to patient-level analysis, several studies have shown that improved environmental hygiene can reduce the general incidence of VRE. Hayden et al investigated the effect of environmental and hand hygiene improvements on VRE infections in an intensive care unit. An educational improvement program for environmental cleaning reduced the frequency of contamination in the rooms of patients with and without VRE infection, and the incidence of VRE acquisition fell. The reduction in contamination was sustained through a “washout” period during which no further intervention occurred and through a subsequent hand hygiene educational improvement program. The study by Manian et al demonstrated a reduction in CDI following the use of HPV environmental decontamination also demonstrated an association with a significant hospital-wide reduction in the incidence of VRE.
<table>
<thead>
<tr>
<th>Reference</th>
<th>Setting (study design)</th>
<th>Findings</th>
<th>Variables</th>
<th>Acquired</th>
<th>Did not acquire</th>
<th>Percentage difference</th>
<th>Adjusted ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Martinez et al</td>
<td>ICU, USA (9-month retrospective control study)</td>
<td>Placement within a room from which VRE had been cultured was associated with VRE acquisition in the subsequent room occupant</td>
<td>Admitted to room from which VRE had been cultured</td>
<td>13% of 30</td>
<td>2% of 60</td>
<td>87.5</td>
<td>OR: 81.7 (2.2–3,092)</td>
</tr>
<tr>
<td>Drees et al</td>
<td>ICU, USA (14-month prospective cohort study)</td>
<td>Positive VRE room culture results or previous VRE-positive room occupants were associated with VRE acquisition</td>
<td>Positive culture before admission or acquisition</td>
<td>8.0% of 50</td>
<td>4.8% of 588</td>
<td>40.5</td>
<td>HR: 4.3 (1.5–12.5)</td>
</tr>
<tr>
<td>Nseir et al</td>
<td>ICU, France (12-month prospective cohort study)</td>
<td>Admission to a room previously occupied by an A. baumannii- or P. aeruginosa-positive patient was associated with acquisition of these pathogens</td>
<td>Prior room occupant with A. baumannii</td>
<td>28.1% of 57</td>
<td>7.9% of 454</td>
<td>71.8</td>
<td>OR: 4.2 (2.0–8.8)</td>
</tr>
<tr>
<td>Huang et al</td>
<td>ICU, USA (20-month retrospective cohort study)</td>
<td>Admission to a room previously occupied by a MRSA- or VRE-positive patient was associated with acquisition of these pathogens</td>
<td>VRE status of prior room occupant</td>
<td>4.5% of 1,291</td>
<td>2.8% of 9,058</td>
<td>37.1</td>
<td>OR: 1.4 (1.0–1.9)</td>
</tr>
<tr>
<td>Shaughnessy et al</td>
<td>ICU, USA (18-month retrospective cohort study)</td>
<td>Admission to a room previously occupied by a C. difficile-positive patient was associated with C. difficile acquisition</td>
<td>C. difficile status of prior room occupant</td>
<td>11.0% of 91</td>
<td>4.6% of 1,679</td>
<td>58.3</td>
<td>HR: 2.3 (1.2–4.5)</td>
</tr>
</tbody>
</table>

Note. Martinez et al, Drees et al, and Nseir et al compared associations listed in the Variables column in patients who did acquire the pathogen with patients who did not acquire the pathogen; Huang et al and Shaughnessy et al compared the frequency of patients who acquired the pathogen depending on the status of the prior room occupant. CI, confidence interval; HR, hazard ratio; ICU, intensive care unit; MRSA, methicillin-resistant Staphylococcus aureus; OR, odds ratio; VRE, vancomycin-resistant enterococci.
MRSA

Dancer recently reviewed evidence that environmental contamination makes an important contribution to the transmission of MRSA. That review summarized evidence that staphylococci are carried by people and shed into the environment, can survive for extended periods on surfaces, and can spread between people and the environment and that improved hygiene reduces staphylococcal infection rates. Dancer reviewed prospective studies investigating the role played by contaminated surfaces in the transmission of MRSA (Table 3). More recently, Dancer et al conducted a ward crossover study to investigate the effect of an extra cleaner, focusing on hand-touch sites. The enhanced cleaning was associated with a significant reduction in the total aerobic counts on surfaces and in the number of failures to reach a hygienic standard considered to be acceptable (more than 2.5 CFUs/cm²) and with a significant reduction in MRSA acquisition by patients. However, there was no significant reduction in surface contamination with methicillin-susceptible S. aureus and MRSA admission screening was not universally applied, so the true MRSA acquisition rate was uncertain. Nonetheless, the study provides further evidence to support the view that reducing surface contamination reduces MRSA nosocomial transmission.

Gram-Negative Rods

A recent prospective cohort study showed that prior room occupancy by a patient colonized or infected with A. baumannii or P. aeruginosa was a significant risk factor for the acquisition of these pathogens. This was the first evidence from an endemic setting that contaminated surfaces contribute to the transmission of gram-negative rods (Table 2). Numerous outbreaks of A. baumannii have been associated with contaminated inanimate fomites, which resolve once the common source is identified and removed, replaced, or adequately disinfected. Several outbreaks in which environmental surfaces were contaminated but a common source was not identified offer limited evidence that surface contamination also plays a role in continued transmission. For example, during a A. baumannii outbreak in the United Kingdom affecting 19 patients in a neurosurgical unit, 53% of 51 surfaces in the unit were contaminated with the outbreak strain, and monthly screens showed that the frequency of contamination correlated with the number of affected patients in the unit. Crucially, failure to maintain low levels of contamination resulted in increases in patient colonization, suggesting that the contamination was contributing to the outbreak. However, it was not possible to prove causality because neither molecular epidemiological analysis nor hand cultures were performed.

Further evidence for the role played by contaminated surfaces in the transmission of A. baumannii comes from an investigation of a multi-institutional outbreak of A. baumannii among war-wounded US soldiers. In this study, outbreak strains of A. baumannii were cultured from 21% of 175 surfaces in 7 field hospitals, but a very low frequency of contamination was identified in soil samples and on the skin of healthy soldiers.

The continued emergence of multidrug-resistant gram-negative rods—in particular those producing carbapenemases such as Klebsiella pneumoniae carbapenemase and New Delhi metallo-ß-lactamase—means that there is an urgent need to understand the epidemiology of these pathogens, including the extent to which contaminated surfaces contribute to their transmission.

Norovirus

Compelling evidence for the role played by surface contamination in the transmission of norovirus comes from outbreaks affecting epidemiologically distinct cohorts of passengers on boat trips. For example, an outbreak affected 74% of guests on 3 consecutive houseboat trips. An environmental investigation identified norovirus RNA on 71% of surfaces in bathrooms, kitchens, and door handles, and fomite contamination appeared to contribute to continuation of the outbreak over the 3 trips.

Several investigations have identified surface contamination with norovirus in the absence of other potential reservoirs during continuing outbreaks. During one outbreak in a long-term care facility, norovirus RNA was identified on 5 of 10 environmental sites collected after phenolic disinfection, suggesting widespread persistent contamination. Positive sites included an elevator call button used only by staff. The outbreak resolved following a second, more thorough facility-wide disinfection, suggesting that environmental contamination contributed to transmission. Various community outbreaks have been linked to shared computer keyboards, specific episodes of vomiting (for example, a kitchen assistant vomited into a sink that was used to prepare vegetables), contamination of carpets after a hospital outbreak, and persistent widespread contamination in a United Kingdom hotel. However, a key limitation of these studies is that the role played by symptomatic or asymptomatic staff carriage in transmission often was not investigated. Thus, prospective studies are required to quantify the role played by surface contamination in the spread of norovirus.

ENVIRONMENTAL CLEANING, DISINFECTION, AND INFECTION CONTROL

Improving the Efficacy of Cleaning and Disinfection

Several studies have demonstrated that focused efforts can improve the efficacy of cleaning. For example, Eckstein et al found that a research team was able to eradicate persistent VRE and C. difficile from surfaces, whereas a housekeeping team was not. In addition, improved monitoring of cleaning by means of markers invisible to the naked eye or routine quantitative microbiological culture and educational interventions
<table>
<thead>
<tr>
<th>Reference</th>
<th>Setting, location</th>
<th>Organism</th>
<th>Study design</th>
<th>Key findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Samore et al⁹ (1996)</td>
<td>Hospital-wide, USA</td>
<td>C. difficile</td>
<td>6-month prospective observational study</td>
<td>Frequency of transmission correlated with the intensity of environmental contamination</td>
</tr>
<tr>
<td>Mayfield et al⁹ (2000)</td>
<td>3 units, USA</td>
<td>C. difficile</td>
<td>18-month prospective before-after study of a switch from QAC to bleach disinfection</td>
<td>Significant reduction in CDI incidence in the highest-risk unit (from 8.6 to 3.3 cases per 1,000 patient-days)</td>
</tr>
<tr>
<td>Wilcox et al¹⁰ (2003)</td>
<td>2 units, UK</td>
<td>C. difficile</td>
<td>2-year prospective ward crossover study of a switch from detergent to bleach disinfection</td>
<td>Significant reduction in CDI incidence in one of the units (from 8.9 to 5.3 cases per 100 admissions) but not in the other</td>
</tr>
<tr>
<td>Boyce et al¹⁰ (2008)</td>
<td>Hospital-wide, USA</td>
<td>C. difficile</td>
<td>20-month prospective before-after study of routine use of HPV decontamination</td>
<td>Significant reduction in CDI incidence in 5 high-incidence units (from 2.3 to 1.3 cases per 1,000 patient-days); lesser reduction in CDI incidence hospital-wide</td>
</tr>
<tr>
<td>Manian et al⁷² (2010)</td>
<td>Hospital-wide, USA</td>
<td>C. difficile/VRE</td>
<td>24-month prospective before-after study of routine use of HPV decontamination</td>
<td>Significant reductions in C. difficile (from 1.0 to 0.5 cases per 1,000 patient-days) and VRE (from 0.3 to 0.15 cases per 1,000 patient-days); substantial but not statistically significant reductions in MRSA and Acinetobacter species</td>
</tr>
<tr>
<td>Bonten et al¹⁹ (1996)</td>
<td>ICU, USA</td>
<td>VRE</td>
<td>2-month prospective observational study</td>
<td>Frequency of environmental contamination was reduced; patient acquisition of VRE was reduced from 33 to 17 acquisitions per 1,000 patient-days during the improved cleaning phase</td>
</tr>
<tr>
<td>Hayden et al⁷² (2006)</td>
<td>ICU, USA</td>
<td>VRE</td>
<td>9-month prospective before-after study of educational improvement of cleaning and hand hygiene</td>
<td>HPV was protective against VRE acquisition when the prior room occupant had VRE (IRR for patients admitted to rooms decontaminated using HPV vs standard methods, 0.22)</td>
</tr>
<tr>
<td>Passaretti et al⁷³ (2008)</td>
<td>ICU, USA</td>
<td>VRE</td>
<td>12-month prospective cohort study investigating the effect of HPV decontamination</td>
<td>Frequency of transmission correlated with the intensity of environmental contamination</td>
</tr>
<tr>
<td>Hardy et al⁷⁶ (2006)</td>
<td>ICU, UK</td>
<td>MRSA</td>
<td>14-month prospective observational study</td>
<td>More than 10% of acquisitions were likely to have been directly from the environment</td>
</tr>
<tr>
<td>Mahamat et al⁷⁶ (2007)</td>
<td>Hospital-wide, UK</td>
<td>MRSA</td>
<td>8-year prospective interrupted time-series analysis of multiple infection control interventions</td>
<td>Introduction of bleach disinfection, environmental sampling, alcohol gels, and admission screening all reduced the prevalence of MRSA</td>
</tr>
<tr>
<td>Dancer et al⁷⁶ (2009)</td>
<td>2 wards, UK</td>
<td>MRSA</td>
<td>12-month prospective crossover study of the effect of 1 extra cleaner</td>
<td>Enhanced cleaning was associated with significant reductions in surface contamination, hygiene failures, and MRSA acquisition</td>
</tr>
</tbody>
</table>

*CDI, *Clostridium difficile* infection; HPV, hydrogen peroxide vapor; ICU, intensive care unit; IRR, incidence rate ratio; MRSA, methicillin-resistant *Staphylococcus aureus*; QAC, quarternary ammonium compound; VRE, vancomycin-resistant enterococci.*
have been shown to improve cleaning efficacy. However, the long-term effect of educational cleaning improvements has not yet been evaluated fully.

Dancer proposed setting standards for environmental hygiene audited by routine microbiological culture—a process currently not recommended by the CDC. The introduction of routine environmental sampling was associated with a reduction in MRSA prevalence in one study and standards exist for the quality of air in operating theaters, so why not for surfaces in healthcare settings? Regardless of whether we return to routine microbiological sampling in hospitals, as was done in the 1970s, improved sampling methods to assess the frequency and concentration of surface contamination would be useful.

Technological developments to assist with cleaning and disinfection include the introduction of microfiber cleaning materials, which may be more effective than standard cloths for removing pathogens from surfaces. The use of adenosine triphosphate analysis as a rapid surrogate test for the assessment of surface hygiene is becoming a popular monitoring method but does not necessarily correlate with microbial contamination. Designers and manufacturers of hospital equipment can help by producing hospitals that are easier to clean. For example, the Design Bugs Out initiative (http://www.designcouncil.org.uk/designbugsout) in the United Kingdom aims to design hospital furniture and equipment that is easier and quicker to clean.

New liquid disinfectants boast improved efficacy and practicability, reducing the risk for human error during formulation. However, liquid detergents and disinfectants rely on the operator to ensure adequate distribution and contact time. Area decontamination methods such as HPV, aerosolized hydrogen peroxide, and UV radiation do not rely on the operator to ensure distribution and contact time and are generally more efficacious than conventional terminal disinfection. However, they require areas to be vacated and pre-cleaning to remove visible soil, and they take longer and are more expensive than conventional terminal disinfection. Further research is required to determine when use of these methods is cost-effective.

Reducing and Controlling the Extent of Environmental Contamination

In addition to improving the efficacy of cleaning and disinfection once contamination has occurred, steps can be taken to prevent or reduce the incidence of recontamination. Rapid identification and isolation of affected patients could reduce contamination of bays and open ward areas shared by unaffected patients. Identification and isolation of asymptomatic shedders may also play a role. Asymptomatic carriers of C. difficile were a source of widespread contamination in one study, and asymptomatic fecal carriage of small round virus (probably norovirus) was common in another study in a long-term care facility. Further work is required to determine the extent and length of time that patients continue to shed pathogens into the environment after the resolution of symptoms, particularly for C. difficile and norovirus.

A novel approach to reducing environmental contamination is “source control” by daily washing of the skin of patients with chlorhexidine. This process has been shown to reduce contamination of patient skin, environmental surfaces, and the hands of HCWs and to reduce the incidence of VRE transmission. It is possible that controlling contamination at the source may be effective in reducing the incidence of transmission of other pathogens as well. Source control could be used in conjunction with complementary strategies aimed at improving cleaning and disinfection to further reduce transmission. However, chlorhexidine is not effective against spores and resistance may emerge, so novel source control strategies are warranted.

Improvements in hospital design and surface science can help to reduce the potential for contamination. Copper, silver, and other antimicrobial-impregnated materials reduces bacterial survival in vitro and numerous antibacterial surface materials or treatments are now available. However, such treatments do not remove the need for cleaning, and there is very limited evidence that they have any significant effect on hospital infection rates. The results of carefully designed trials are awaited.

Improving the Quality of the Evidence

The quality of evidence supporting the role played by surface contamination in transmission has improved from outbreak reports to large, well-designed prospective studies (Tables 2 and 3). Structured reporting of future outbreaks—for example, by using the ORION guidance—will help, but large, prospective controlled trials are needed to properly elucidate the role played by surface and air contamination (decontamination) in the transmission of nosocomial pathogens.

Most of the evidence investigating the role played by contaminated surfaces in transmission comes from acute care facilities. However, environmental contamination may play an important role in transmission in long-term care facilities and other nonacute healthcare facilities. There is considerable evidence that contaminated environmental surfaces are involved in the transmission of norovirus in the community, and there is emerging evidence that contamination with pathogens such as community-associated MRSA may be involved in transmission in outpatient and community settings. Environmental interventions that are effective for the prevention and control of the transmission of pathogens in acute healthcare facilities may not be effective in community and nonacute settings. Therefore, further research is required to investigate the role played by surface contamination in the transmission of pathogens in nonacute and community settings.
CONCLUSION
The historical perspective that contaminated surfaces contribute negligibly to nosocomial transmission has been re-evaluated in light of new information. There is now compelling evidence that contaminated surfaces make an important contribution to the epidemic and endemic transmission of C. difficile, VRE, MRSA, A. baumannii, and P. aeruginosa (Tables 2 and 3) and to the epidemic transmission of norovirus. However, few studies have quantified the link between contaminated surfaces and the risk of transmission. This is in part due to the difficulties in conducting research in this area because of the multifaceted nature of nosocomial transmission (Figure 1). In addition, the widespread view that contaminated surfaces are relatively unimportant in transmission has meant that fundholders and administrators have not commissioned research in this area until relatively recently. There is now sufficient evidence to support further studies in this area to identify the best methods of achieving and maintaining clean hospitals and to evaluate the cost and effectiveness of such interventions with respect to reducing the incidence of hospital-associated infections. In particular, there is a need to conduct large, high-quality prospective controlled trials to identify interventions that significantly reduce surface contamination and transmission.

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