



Environmental contamination makes an important contribution to hospital infection

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KEYWORDS

Environmental
contamination;
MRSA;
VRE;
Hydrogen peroxide
vapour

Summary Meticillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant enterococci (VRE) are capable of surviving for days to weeks on environmental surfaces in healthcare facilities. Environmental surfaces frequently touched by healthcare workers are commonly contaminated in the rooms of patients colonized or infected with MRSA or VRE. A number of studies have documented that healthcare workers may contaminate their hands or gloves by touching contaminated environmental surfaces, and that hands or gloves become contaminated with numbers of organisms that are likely to result in transmission to patients. Pathogens may also be transferred directly from contaminated surfaces to susceptible patients. There is an increasing body of evidence that cleaning or disinfection of the environment can reduce transmission of healthcare-associated pathogens. Because routine cleaning of equipment items and other high-touch surfaces does not always remove pathogens from contaminated surfaces, improved methods of disinfecting the hospital environment are needed. Preliminary studies suggest that hydrogen peroxide vapour technology deserves further evaluation as a method for decontamination of the environment in healthcare settings.

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For several decades, there has been considerable controversy over whether or not contaminated environmental surfaces contribute to transmission of healthcare-associated pathogens. This article reviews the evidence that environmental surfaces contaminated with meticillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant enterococci (VRE) contribute to the occurrence of healthcare-associated infections. In addition,

it describes a new strategy that has been used to eliminate environmental contamination by *Clostridium difficile*, another pathogen for which contaminated environmental surfaces serve as a reservoir for transmission.

The potential for contaminated environmental surfaces to contribute to transmission of healthcare-associated pathogens depends on a number of factors, including the ability of pathogens to remain viable on a variety of dry environmental surfaces, the frequency with which they contaminate surfaces commonly touched by

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patients and healthcare workers, and whether or not levels of contamination are sufficiently high to result in transmission to patients.

Pathogens such as MRSA, VRE and *C. difficile* have the ability to remain viable on dry surfaces for days, weeks or even months. For example, strains of MRSA can remain viable for up to 14 days on Formica surfaces, and for up to six to nine weeks on cotton-blanket material.^{1,2} Some epidemic strains of MRSA have been shown to survive longer and at higher concentrations than non-epidemic strains.³ A unique experiment conducted by Colbeck⁴ demonstrated that *S. aureus* can remain virulent and capable of causing infection for at least 10 days after exposure to dry surfaces.

The proportion of hospital surfaces contaminated with MRSA has varied considerably in published reports, ranging from 1% to 27% of surfaces in patient rooms on regular hospital wards, and from a few percent to 64% of surfaces in burn units with MRSA patients. The frequency of contamination has been shown to vary depending on the body sites at which patients are colonized or infected. In one study, 36% of surfaces cultured in the rooms of patients with MRSA in a wound or urine were contaminated, compared to 6% of surfaces in the rooms of patients with MRSA at other body sites.⁵ In a recent study by Otter *et al.*,⁶ ten standardized high-touch surfaces were cultured in the rooms of eight patients with heavy gastrointestinal colonization by MRSA and concomitant diarrhoea (cases) and in the rooms of six patients with MRSA at other body sites, but not in their stool (controls). The investigators found that 59% of surfaces were contaminated with MRSA in the rooms of case patients who had heavy gastrointestinal colonization with MRSA and concomitant diarrhoea. MRSA was recovered most frequently from bedside rails (100% of those cultured), followed by blood pressure cuffs (88%), television remote control devices (75%), bedside tables and toilet seats (63% each), toilet rails and dressers (50% each), door handles (38%) and intravenous pumps (25%). In contrast, significantly fewer (23%) surfaces were contaminated in the rooms of control patients who had MRSA at other body sites, but not in their stool. In the rooms of control patients, bedside rails were also the most frequently contaminated site (67%), followed by toilets and call buttons (33% each). The other seven standardized sites cultured in the rooms of control patients were contaminated less than 20% of the time. In another study, community-acquired strains of MRSA (CA-MRSA), which are becoming increasingly common worldwide, were found to contaminate 19% of

surfaces in an outpatient clinic that cared for patients with human immunodeficiency syndrome.⁷ Two healthcare workers (HCWs) who worked in the clinic developed infections caused by CA-MRSA strains. One of the infected HCWs who did not have direct contact with patients became infected with the same strain that was found on environmental surfaces. Although extensive cleaning effectively removed CA-MRSA from surfaces in the outpatient clinic described by Johnston *et al.*,⁷ routine cleaning of contaminated environmental surfaces does not always eliminate MRSA from high-touch surfaces in hospitals.^{8,9}

Environmental contamination may contribute to transmission of healthcare pathogens when healthcare workers contaminate their hands or gloves by touching contaminated surfaces, or when patients come into direct contact with contaminated surfaces. Transmission of MRSA from environmental surfaces to gloves or hands of HCWs has been documented by several investigators. In one study, 42% of 12 nurses who had no direct contact with patients contaminated their gloves by touching objects in the rooms of patients with MRSA in a wound or urine.⁵ In another study, 31% of volunteers who touched bed rails and overbed tables in patient rooms contaminated their hands with *S. aureus* (35% of which were MRSA).⁸ When volunteers touched bed rails and overbed tables in unoccupied rooms that had been terminally cleaned, 7% contaminated their hands with *S. aureus*.⁸

Transmission of MRSA from contaminated environmental sources to patients has occurred in a variety of settings. Schultz *et al.*¹⁰ presented convincing evidence that ultrasonic nebulizers were the source of an MRSA outbreak among patients. Other studies have provided suggestive evidence that contaminated ventilation grills were sources of MRSA outbreaks in hospitals.^{11,12} In a study by Hardy *et al.*¹³ the authors concluded that three patients acquired MRSA from the environment, but did not exclude HCWs as another potential source.

The role of contaminated environmental surfaces in transmission of healthcare-associated pathogens is also supported by the fact that cleaning and/or disinfection of the environment can reduce the incidence of healthcare-associated colonization or infection. Schultz *et al.*¹⁰ demonstrated that cleaning contaminated ultrasonic nebulizers implicated in transmission terminated an outbreak of MRSA. Cleaning contaminated ventilation grills was associated with control of several other MRSA outbreaks.^{11,12} Rampling *et al.*¹⁴ concluded

that increased cleaning of an affected ward was associated with control of an MRSA outbreak.

VRE are also capable of surviving on contaminated environmental surfaces for prolonged time periods. These pathogens have been shown to survive for one week to two months on countertops, for greater than seven days on fabric chairs, for seven days to four months on dry polyvinyl chloride surfaces, and for a few days to more than three months on cloth and plastic surfaces.¹⁵⁻¹⁹

Environmental contamination by VRE occurs as often as, or perhaps more often than, with MRSA. In one study, 46% of environmental cultures obtained in the rooms of VRE patients who had diarrhoea were contaminated, compared to 15% of surfaces cultured in the rooms of VRE patients who did not have diarrhoea.²⁰ Bonten and colleagues²¹ found that 12% of 1294 environmental cultures were positive for VRE in an intensive care unit (ICU). However, in the same study, the frequency of environmental contamination reached 60–70% in the rooms of patients colonized with VRE at three or four body sites.²¹ In a review conducted by Weber *et al.*,¹⁸ 7–29% of environmental sites were positive in areas housing VRE patients. From 36% to 58% of chairs and couches used by VRE patients in outpatient settings were contaminated.²² In hospital settings, sites most commonly contaminated with VRE include bedside rails, bedside tables, blood pressure cuffs, and floors. Less commonly contaminated surfaces include urine containers, intravenous pumps, bed control buttons, nurse call buttons, and pulse oximeters.

Transmission of VRE from environmental surfaces to HCW hands or gloves has been documented by several investigators. In one study, the hand of a HCW who touched a contaminated chair was positive for VRE.¹⁹ Tenorio *et al.*²³ found that three HCWs who touched items in patient rooms without touching patients contaminated their gloves. In two studies conducted by Ray *et al.*²⁴ and by Bhalla *et al.*,⁸ 46% of 13 HCWs who touched bedrails and bedside tables of VRE patients contaminated their gloves, and 20% of volunteers who touched bedrails and bedside tables contaminated their hands with VRE. In a careful study by Duckro *et al.*,²⁵ HCWs touched VRE-positive sites such as the skin of VRE-colonized patients and contaminated environmental surfaces. HCWs then touched 151 VRE-negative sites after touching a contaminated site. VRE were transferred to 16 (10.6%) of negative sites. Touching a contaminated environmental surface resulted in transfer of VRE to another

site about as frequently as touching a colonized patient.

VRE had also been transmitted directly from contaminated equipment to patients. For example, contaminated electronic rectal thermometer probes and electronic ear probe thermometers have been implicated as sources of several VRE outbreaks.^{26,27} A contaminated EKG lead was the source of continuing transmission of VRE in a burn unit outbreak in which molecular typing was used to confirm the genetic relatedness of VRE recovered from equipment items and affected patients.²⁸ Transmission of VRE from environmental sources other than medical equipment has also been documented. In a prospective study carried out in an ICU revealed that three (23%) of 13 patients who were VRE-negative became positive after their room became contaminated.²¹ Two of 10 VRE-negative patients whose bedside rails became contaminated acquired the same strain of VRE that was present on the bedside rail. In a retrospective case-control study that included multivariate analysis, patients who acquired VRE were significantly more likely than controls to have occupied a room with persisting VRE environmental contamination, suggesting that inadequately cleaned rooms served as a source of transmission to patients.²⁹

Eliminating the contaminated environmental source, i.e., contaminated electronic rectal or tympanic thermometers, reduced transmission of VRE in several outbreaks.^{26,27,30} In one study, enhanced environmental cleaning was associated with a 26–34% reduction in VRE transmission on an affected ward.³⁰ Also, enhanced environmental cleaning, when used in combination with other control measures, was considered a major factor in terminating an outbreak of VRE in a burn unit.²⁸ Recently, Hayden *et al.*³¹ conducted a 9-month prospective study in a medical ICU (MICU) to assess the impact of improved environmental cleaning on VRE transmission. The study included screening of patients upon admission to the unit and daily thereafter. The study was divided into four time periods: a baseline period, a period that included education to improve environmental cleaning, a wash-out period with no specific intervention, and a multimodal hand hygiene intervention period. Enhanced cleaning with a detergent-disinfectant was found to independently contribute to reduced VRE environmental contamination and hand contamination, and significantly reduced VRE acquisition rates.³¹

Since environmental contamination is also felt to contribute to nosocomial transmission of *C. difficile*, a recently conducted study assessed the

impact of hydrogen peroxide vapour (HPV) room decontamination on environmental contamination and nosocomial transmission by *C. difficile*.³² Hydrogen peroxide vapour decontamination was selected for evaluation because it has been shown to effectively reduce environmental contamination caused by MRSA.⁹ A 10-month, prospective collaborative trial was conducted in a university-affiliated hospital affected by the epidemic NAP-1 strain of *C. difficile* in conjunction with the Centers for Disease Control and Prevention (CDC) and Bioquell LLC (Andover, UK). A pre- and post-intervention study design was used. HPV was injected into sealed patient rooms or entire wards using methods previously described.⁹ The perimeter of the enclosures was monitored during the cycle using hand-held Drager Pac III HPV sensors. Initially, three entire wards (including nursing stations) were decontaminated, and all patient rooms on two additional wards were decontaminated during the first two months of the trial period. Subsequently, priority was given to decontaminating rooms recently vacated by patients with *C. difficile*-associated disease (CDAD). Swab cultures of environmental surfaces obtained immediately before and after HPV decontamination were plated directly and after broth enrichment onto cycloserine-cefoxitin-fructose agar with lysozyme. Additional samples of surfaces were obtained before and after HPV by using moistened sterile cellulose sponges, which were sent to CDC for culture with and without an alcohol-shock procedure. Infection control personnel performed surveillance for new cases of nosocomial laboratory-confirmed CDAD, and hospital-wide antimicrobial usage of each antimicrobial agent was expressed as defined daily doses (DDDs) of each agent/1000 patient-days.

Four (2.4%) of 165 swab cultures obtained before HPV decontamination yielded *C. difficile*, compared to none of 155 swab cultures obtained after HPV decontamination ($p=0.12$). Eleven (25.6%) of 43 sponge cultures obtained before HPV decontamination yielded *C. difficile*, compared to none of 37 sponge cultures obtained after HPV decontamination ($p=0.0006$). The incidence of new nosocomial cases of CDAD decreased from 1.36 cases/1000 patient-days in the 10-month pre-intervention period to 0.84 cases/1000 patient-days during the 10-month intervention period, a reduction of 39% ($p=0.26$). If the analysis was confined to only those months when the epidemic NAP-1 strain was present both during the pre-intervention period and during the intervention period, the incidence of new nosocomial CDAD decreased from 1.89 cases/1000 patient-days to

0.88 cases/1000 patient-days, a reduction of 53% ($p=0.047$). The decreased incidence of CDAD could not be attributed to changes in antimicrobial usage patterns.

In summary, healthcare-associated pathogens such as MRSA, VRE and *C. difficile* can survive for days to weeks on environmental surfaces. Items frequently touched by HCWs or patients are often contaminated by such pathogens in the rooms of affected patients. Contaminated surfaces contribute to transmission of healthcare-associated pathogens by serving as sources of hand (or glove) contamination among HCWs, and by direct spread of pathogens to susceptible patients. An increasing body of evidence suggests that enhanced cleaning/disinfection of environmental surfaces can reduce transmission of these pathogens.

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