Role of hospital surfaces in the transmission of emerging health care-associated pathogens: Norovirus, *Clostridium difficile*, and *Acinetobacter* species

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Health care-associated infections (HAI) remain a major cause of patient morbidity and mortality. Although the main source of nosocomial pathogens is likely the patient's endogenous flora, an estimated 20% to 40% of HAI have been attributed to cross infection via the hands of health care personnel, who have become contaminated from direct contact with the patient or indirectly by touching contaminated environmental surfaces. Multiple studies strongly suggest that environmental contamination plays an important role in the transmission of methicillin-resistant *Staphylococcus aureus* and vancomycin-resistant *Enterococcus* spp. More recently, evidence suggests that environmental contamination also plays a role in the nosocomial transmission of norovirus, *Clostridium difficile*, and *Acinetobacter* spp. All 3 pathogens survive for prolonged periods of time in the environment, and infections have been associated with frequent surface contamination in hospital rooms and health care worker hands. In some cases, the extent of patient-to-patient transmission has been found to be directly proportional to the level of environmental contamination. Improved cleaning/disinfection of environmental surfaces and hand hygiene have been shown to reduce the spread of all of these pathogens. Importantly, norovirus and *C difficile* are relatively resistant to the most common surface disinfectants and waterless alcohol-based antiseptics. Current hand hygiene guidelines and recommendations for surface cleaning/disinfection should be followed in managing outbreaks because of these emerging pathogens.

Key Words: Environmental surfaces; disinfectants; *Clostridium difficile*; norovirus; *Acinetobacter*.

Health care-associated infections (HAI) remain a major cause of patient morbidity and mortality. In the United States, it is estimated that there are 1.7 million HAI each year, which result in approximately 99,000 deaths.[1] The major source of nosocomial pathogens is thought to be the patient's endogenous flora, but an estimated 20% to 40% of nosocomial infections have been attributed to cross infection via the hands of health care personnel.[2] Contamination of the hands of health care workers could in turn result from either direct patient contact or indirectly from touching contaminated environmental surfaces.[3] Less commonly, a patient could become colonized with a nosocomial pathogen by direct contact with a contaminated environmental surface.[3]

For environmental contamination to play an important role in the acquisition of a nosocomial pathogen, the pathogen must demonstrate certain microbiologic characteristics (Table 1). Scientific evidence suggests that environmental contamination plays an important role in the spread of methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant *Enterococcus* spp (VRE).[4,5] For example, admitting a new patient to a room previously occupied by a MRSA- or a VRE-positive patient significantly increases the odds of acquisition for MRSA or VRE.[6] Other pathogens that are capable of surviving in hospital reservoirs

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Table 1. Microbiologic factors that can facilitate surface environment-mediated transmission of selected pathogens

Pathogen able to survive for prolonged periods of time on environmental surfaces (all)
Ability to remain virulent after environmental exposure (all)
Contamination of the hospital environment frequent (all)
Ability to colonize patients (*Acinetobacter, C difficile*, MRSA, VRE)
Ability to transiently colonize the hands of health care workers (all)
Transmission via the contaminated hands of healthcare workers (all)
Small inoculating dose (*C difficile*, norovirus)
Relative resistance to disinfectants used on environmental surfaces (*C difficile*, norovirus)

C difficile, *Clostridium difficile*; *MRSA*, methicillin-resistant *Staphylococcus aureus*; *VRE*, vancomycin-resistent *Enterococcus* spp.

and for which environmental contamination may play a role in nosocomial acquisition are norovirus, hepatitis B virus, *Acinetobacter* spp, *Pseudomonas aeruginosa*, *Clostridium difficile*, and *Candida* spp.[4]

This article will focus on the role of surface contamination in the transmission of 3 emerging nosocomial pathogens: norovirus, *C difficile*, and *Acinetobacter* spp. The article is based, in part, on a lecture presented at a symposium held during the 2009 Annual Meeting of the Association for Professionals in Infection Control and Epidemiology, Inc (APIC).[7] The role of surface contamination in transmission of health care-associated pathogens is an important issue because transmission can be interrupted by appropriate hand hygiene[8,9] and cleaning/disinfection of environmental surfaces.[10-12] For example, improved surface decontamination has been shown to decrease environmental contamination of MRSA and VRE[13] and decrease the likelihood of patients acquiring VRE[14] and developing MRSA infection.[15]

NOROVIRUS

Microbiology and epidemiology

Caliciviruses are single-stranded RNA, nonenveloped, icosahedral viruses that are now recognized as common pathogens of humans and animals.[16,17] *Norovirus*, a genus within the family *Caliciviridae*, is subdivided into 5 genotypes; genotypes GI, GII, and GIV include human pathogens. Understanding viral transmission and pathophysiology has been limited until recently by the lack of a cell culture system for growing norovirus and limited animal models (ie, gnotobiotic pig). Clinical findings associated with norovirus infection include a short incubation period (10-51 hours), variable symptoms of upper (vomiting) and/or lower gastroenteritis (diarrhea), low-grade fever (101°F to 102°F), resolution of symptoms usually in 12 to 72 hours, and prolonged viral shedding.[17] The symptoms of norovirus infection include nausea (79%), vomiting (69%), diarrhea (66%), low-grade fever (37%), and abdominal cramping (30%). Young children, older adults, and immunocompromised persons have higher morbidity and mortality. Only symptomatic treatment is available. Currently, there is no licensed vaccine to prevent norovirus infection.

Noroviruses account for greater than 90% of nonbacterial and approximately 50% of all-cause epidemic gastroenteritis.[16] They are responsible for an estimated 267 million infections annually worldwide and 23 million infections annually in the United States. Modes of transmission include human-to-human transmission via the fecal-oral route from contact with an infected person (direct transmission) or contact with a contaminated surface (indirect transmission) and by consumption of fecally contaminated food or water. In addition, good evidence exists for transmission because of aerosolization of vomitus that presumably results in droplets contaminating surfaces or entering the oral mucosa and being swallowed. No evidence suggests that infection occurs through the respiratory system. A number of features of norovirus biology contribute to its ability to frequently cause outbreaks in humans (Table 2).

Outbreaks are common and have been reported in hospitals, extended care facilities, cruise ships, schools, day care centers, camps, restaurants, hotels, and military installations.[17] Although outbreaks can occur year round, most outbreaks in the Northern Hemisphere occur during winter and spring (hence the term ''winter vomiting disease''). Systematic studies have reported that hospitals and long-term care facilities may account for more than 25% of the outbreaks. Health care-associated outbreaks frequently involve large numbers of patients and staff with high attack rates in affected wards.[18-20] Nosocomial norovirus infections often involves the frail elderly population with limited mobility and may result in prolonged symptoms in this patient population. In extended care facilities, outbreaks have frequently resulted in the need for patients to be hospitalized and have led to patient deaths.[19]

Norovirus outbreaks in health care workers can cause substantial economic losses to hospitals because of absenteeism. Closure of the affected ward may be

Table 2. Microbiologic and epidemiologic features of norovirus that promote epidemics

Large human reservoir of infection
Widespread host susceptibility
Strain-specific immunity is short lived (weeks to months)
Multiple routes of transmission (fecal-oral, foodborne, waterborne, aerosol)
High infectivity
Very low inoculating dose (<10 virions)
Stable in the environment
Prolonged shedding
No vaccine available
No specific chemotherapy

required to contain the outbreak, resulting in inconvenience and additional expense. In fact, in a review of closure of medical departments during a nosocomial outbreak, more than 44% were due to norovirus.[21]

Environmental survival

Because human noroviruses cannot be cultured, most of the data on environmental survival are based on studies using surrogate caliciviruses such as feline calicivirus or murine norovirus or other nonenveloped viruses such as MS2. This is an important limitation in understanding the environmental survival and susceptibility to germicides because these surrogates may not accurately reflect the behavior of human norovirus. Murine norovirus is considered by many as a better surrogate for human norovirus than feline calicivirus. In addition, it is important to understand that human novovirus is detected generally by reverse-transcription polymerase chain reaction (RT-PCR), which will also detect nonviable virus. Thus, this test may not accurately reflect the activity of germicides.

Environmental survival of noroviruses is enhanced by their ability to withstand a wide range of temperatures (from freezing to 60°C) and persist on environmental surfaces, in recreational and drinking water, and in a variety of food items, including raw oysters and vegetables that are irrigated with sewage and are eaten uncooked.[7] Feline calicivirus, a surrogate for human norovirus, was found to persist on berries despite frozen storage. Human norovirus genome cannot be completely degraded despite heating to 72°C for 45 and 60 minutes. Furthermore, it can persist on the surface of refrigerated foods for at least 10 days and in mineral and tap water for over 2 months at 4°C, 25°C, and −20°C. Feline calicivirus can survive in the dried state for 21 to 28 days at room temperature.

Human norovirus RNA has been shown to persist on experimentally contaminated surfaces of stainless steel, Formica (Formica Corporation, Cincinnati, OH), and ceramic coupons for up to 7-days postinoculation.[22] Feline calicivirus was found to survive for 8 to 12 hours on a computer keyboard and brass, 1 or 2 days on a computer mouse, and for up to 3 days on telephone buttons and receivers.[23] The time for 90% virus reduction was less than 4 hours on the computer keyboard, mouse, brass, and telephone wire; 4 to 8 hours on a telephone receiver; and 12 to 24 hours on telephone buttons. Murine norovirus has been shown to survive for more than 40 days with less than 2-$\log_{10}$ decrease in survival on both gauze and diaper material.[24] Virus survived better in a stool suspension than on the surface of gauze or diaper material.

Hospital contamination

As described above, health care-associated outbreaks of norovirus are now common. Widespread environmental contamination of the hospital rooms of ill patients has been described.[7] The most common contaminated site was the toilet tops. Environmental contamination outside of the room of the infected patient has been demonstrated; however, the immediate environment of symptomatic patients is more likely to yield norovirus as detected by polymerase chain reaction (PCR).

Barker et al using a human challenge study demonstrated that human noroviruses could be consistently transferred via contaminated fingers to surfaces such as toilet tops, door handles, and telephone receivers.[25] Furthermore, they demonstrated that contaminated fingers could sequentially transfer virus as detected by PCR to up to 7 clean surfaces.

Evidence of the role of environmental contamination in transmission

The evidence to support the role of surface environmental surface contamination for norovirus is circumstantial (Table 3). Food and waterborne transmission, as well as direct person-to-person transmission, are well described. The best evidence comes from the serial occurrence on cruise ships of norovirus infections caused by identical strain of norovirus. More than 5 waves of infection have been reported, despite ship-wide sanitization between cruises. Evans et al described an outbreak of norovirus in attendees of a metropolitan concert hall over a 5-day period.[26] The index case was a concert attendee who vomited in the auditorium and in an adjacent male toilet for males. Gastroenteritis occurred among 8 of 15 school parties who attended a concert on the following day. Children who sat on the same level of the auditorium as the index case were more likely to be ill than those seated elsewhere (relative risk, 7.1).

In hospitals, widespread environmental contamination of surfaces by norovirus has been found in outbreaks. Experimental human challenge studies have demonstrated that fingertips can be contaminated from the environment and transfer norovirus subsequently to multiple surfaces. Furthermore, health care workers not providing direct care to infected patients have become ill, most likely via acquisition of virus from contaminated surfaces outside the patient rooms.

Interventions to control surface contamination

General methods. The general methods to prevent and control norovirus outbreaks in health care facilities have been well described.[18,19] To prevent norovirus

Table 3. Evidence supporting role of environmental contamination in transmission of emerging health care-associated pathogens

Characteristic	Norovirus	*Clostridium difficile*	*Acinetobacter* spp
Able to survive for prolonged periods in the environment	Yes	Yes	Yes
Environmental contamination frequently found in rooms of infected patients	Yes	Yes	Yes
Contaminated environmental reservoir demonstrated to be source of an outbreak	—	Yes	Yes
Contamination of health care worker hands demonstrated	—	Yes	Yes
Human challenge studies demonstrate that contaminated health care worker hands can transfer pathogen	Yes	—	Yes
Level of environmental contamination associated with frequency of health care worker hand contamination	—	Yes	—
Prevalence of environmental contamination associated with incidence of patient acquisition/infection	—	Yes	—
Admission to a room previously occupied by an infected patient associated with risk of colonization/infection	—	Yes	—
Enhanced cleaning demonstrated to reduce hospital incidence of infection	—	Yes	Yes

outbreaks, it is crucial for health care providers to use Standard Precautions with all patients (gloves for contact with any body secretions except sweat, hand hygiene before and after all patient contacts), especially those with a diarrheal illness. Patients with known or suspected norovirus infection should be placed on Contact Precautions (single room, don gloves and gown prior to entering room) until the patient has been asymptomatic for 48 to 72 hours. Hand hygiene should be performed using soap and water or water and an antiseptic (eg, chlorhexidine). Other key aspects of control include preventing visitation of sick persons, eliminating sharing of food and drinks, and identifying and furloughing sick employees for 48 to 72 hours after symptoms have resolved. Because few laboratories possess the ability to rapidly diagnose norovirus infection, the Kaplan criteria (ie, stool cultures negative for bacterial pathogens, vomiting in >50% of cases, mean/median incubation period of 24-48 hours, mean/median duration of illness of 12-60 hours) should be employed to aid in early identification of outbreaks.[27]

Gehrke et al tested the efficacy of several alcohols (ethanol, 1-propanol, 2-propanol) using 70% or 90% concentrations with 30-second contact time against feline calicivirus that had been used to experimentally contaminate fingertips.[28] For each alcohol, the 70% concentration was more effective than the 90% concentration. The most effective germicide was 70% ethanol (3.78-$\log_{10}$ reduction), followed by 70% 1-propanol (3.58-$\log_{10}$ reduction), and 70% 2-propanol (2.15-$\log_{10}$ reduction). Barker et al demonstrated, using RT-PCR, that 1 minute of handwashing with soap and water, followed by rinsing for 20 seconds and drying with a disposable towel completely removed human norovirus from hands contaminated with norovirus containing feces.[25] More recently, Liu et al used the American Society of Testing and Materials (ASTM) standard finger pad method and a modification (with rubbing) to study the effectiveness of water, an antibacterial liquid soap treatment, and a waterless hand antiseptic (62% ethanol) against human norovirus. As measured by reverse-transcription quantitative polymerase chain reaction and using the modified ASTM method, the water rinse was slightly more effective (1.58-$\log_{10}$ reduction) then the liquid soap (1.20-$\log_{10}$ reduction), and both were significantly more effective than the ethanol-based hand sanitizer (0.20-$\log_{10}$ reduction).[29] It therefore appears that hand hygiene with soap and water is more effective than hand hygiene with a waterless alcohol-based hand sanitizer against human norovirus. The results of human challenge studies with human norovirus by Barker et al[25] and Liu et al[29] suggest that handwashing for at least 1 minute may be more effective in removing norovirus than handwashing for 10 to 20 seconds. The studies noted above also suggest that human norovirus is less susceptible to alcohols than feline calicivirus.

Environmental disinfection. Only limited data are available on the activity of germicides against caliciviruses. Because of the inability to culture noroviruses, data are based on the use of surrogates such as murine norovirus or feline calicivirus or on assessment for the presence of human norovirus genome by RT-PCR. Both methods have important drawbacks. The surrogate viruses may not mimic the susceptibility of human noroviruses to germicides. The use of RT-PCR may detect nonviable norovirus.

The efficacy of germicides against calicivirus using a suspension test has been evaluated.[7] Ethanol and quaternary ammonium products have not proved effective. Hypochlorite has been demonstrated to be effective, although concentrations of 300 ppm are less effective than higher concentrations (ie, 3000 ppm). Importantly, human norovirus appeared more resistant than feline calicivirus. Other investigators have evaluated the efficacy of germicides using a carrier test. In

a quantitative test with stainless steel discs, peracetic acid, glutaraldehyde, 50% ethanol, and 30% 1-propanol were able to inactivate ≥4-$\log_{10}$ murine norovirus under clean conditions within 5 minutes.[30] Whitehead and McCue studied the activity of germicides against feline calicivirus at a 1-minute exposure time.[31] Hypochlorite (1000 ppm) and acid-based disinfectants were very effective in eliminating virus. Inactivation of feline calicivirus by alcohol, phenolics, and quaternary compounds depended on how these agents were formulated as disinfectants. However, Malik et al demonstrated that ethanol (70%-90%) and isopropanol (40%-60%) were able to kill 99% of feline calicivirus with a short contact time of 1 minute.[32] Jimenez and Chiang reported that hypochlorite (1000 ppm but not 100 ppm) was effective in eliminating >6-$\log_{10}$ feline calicivirus within 10 minutes.[33]

When managing norovirus infection, it has been recommended that health care facilities ensure consistent environmental cleaning and disinfection with a focus on restrooms even when apparently unsoiled and that hypochlorite solutions may be required when there is continued transmission.[34] The Centers for Disease Control and Prevention (CDC) has recommended the use of a chlorine beach solution (1000-5000 ppm) or another agent approved for noroviruses by the Environmental Protection Agency. Experts have also recommended more frequent environmental cleaning with disinfection of high-touch surfaces (eg, doorknobs, light switches, tables, computer keyboards) every shift and room disinfection every 24 hours. Separate toilet facilities should be provided for ill and nonill patients. Any supplies left in a patient's room should be discarded after the infected patient's release. The floors should be cleaned with an approved disinfectant and the disinfecting solution and mop head changed every 3 rooms. Furthermore, after cleaning the room of a patient with diarrhea and/or vomiting, the disinfecting solution and mop head should be changed. Curtains should be removed and replaced if soiled or contaminated. Persons who clean areas heavily contaminated with feces or vomitus may benefit from wearing a mask to protect against contamination of one's oral mucosa because virus can be aerosolized for short distances (droplet transmission) from feces or vomitus. However, there is currently no evidence that utilization of these enhanced interventions will aid in controlling a norovirus outbreak.

C DIFFICILE

Microbiology and epidemiology

C difficile is an anaerobic, gram-positive, spore-forming, toxin-producing bacillus.[35] It is part of the normal intestinal flora in humans and is carried by approximately 3% of healthy adults and 20% to 30% of hospitalized adults. *C difficile* exists in both vegetative and spore forms; in the colon, it exists as a vegetative cell, whereas, outside the colon, it survives in spore form. *C difficile* is the causative agent of antibiotic-associated colitis. Colonization of the intestinal tract occurs via the fecal-oral route. *C difficile* infection (CDI) occurs in a colonized patient when antibiotic therapy disrupts the colonic microflora leading to proliferation of *C difficile* with release of toxin A (enterotoxin) and/or toxin B (cytotoxin), leading to mucosal injury and inflammation. Antibiotic use is the most commonly recognized risk factor for CDI. Recently, a new strain of *C difficile* emerged in the United States with increased virulence, resistance, or both.[36,37] This new strain, which was initially reported from Canada, has been characterized as restriction endonuclease analysis group B1, North American pulsed-field gel electrophoresis type 1, ribotype 027, and toxinotype III. In recent years, an increased incidence of CDI has been reported along with an increase in *C difficile*-related hospitalizations and an increase in the case-fatality rate.

C difficile is acquired by fecal-oral transmission. In the health care setting, 3 mechanisms of transfer of *C difficile* are possible: first, direct transfer of *C difficile* from a colonized or infected patient to the environment (eg, rectal thermometer, commode) and contact by another patient with inoculation into the mouth or directly into the colon; second, direct transfer via hands to a noncolonized or noninfected patient; and finally, indirect transfer via health care worker contact (or any other person) with the contaminated environment and transfer to a noncolonized or noninfected patient.

Environmental survival

The vegetative form of *C difficile* survives for only 15 minutes on dry surfaces in room air, although cells may remain viable for up to 6 hours on moist surfaces.[7] On the other hand, bacterial spores are highly resistant to drying, heat, and chemical and physical agents. In 1981, Kim et al reported that *C difficile* inoculated onto a hospital floor persisted for 5 months.[38] Neither storage temperature (4°C, −20°C) nor multiple cycles of refrigeration/freezing and thawing have been found to affect the viability of *C difficile* vegetative cells or spores.

Hospital contamination

In 1989, McFarland et al reported that 49% of rooms occupied by symptomatic patients with *C difficile* were contaminated and that 29% of room occupied by asymptomatic patients were contaminated.[39] Since

that study, many other studies have demonstrated widespread environmental contamination with *C difficile* in the rooms of patients with CDI with a range from 2.9% to 75%.[7] Moreover, *C difficile* has been isolated from surfaces in rooms of patients not colonized or infected with *C difficile*, although with lower frequency. *C difficile* spores have been isolated from the air, and aerosol dissemination of spores may, in part, account for widespread environmental contamination.

C difficile has commonly been isolated from the hands of infected patients and the hands of their health care providers.[7,40] The frequency of positive personnel hand culture has been shown to be strongly correlated with the intensity of environmental contamination.[41] For example, hand contamination was 0% when environmental contamination was 0% to 25%, 8% when environmental contamination was 26% to 50%, and 36% when environmental contamination was greater than 50%.

Evidence of the role of environmental contamination in transmission

It is widely accepted that environmental contamination plays an important role in the transmission of *C difficile* in the hospital setting (Table 3). The key evidence is as follows. The frequency of *C difficile* acquisition has been linked with the level of environmental contamination.[42] Patients admitted to a room previously occupied by a patient with *C difficile* have a higher risk for *C difficile* acquisition.[43] Finally, improved room disinfection has led to decreased rates of *C difficile* infection.[44,45] In addition to a strong relationship between surface contamination and *C difficile* transmission in hospitals, several medical devices have been linked to transmission of *C difficile* in the hospital, including a portable bed commode and electronic rectal thermometers.[7]

Interventions to control surface contamination

General guidelines. The general methods to control *C difficile* are available from experts and from position statements/recommendations by professional societies.[34,46-48] Patients with known or suspected *C difficile* infection should be placed on Contact Precautions (single room, don gloves and gown prior to entering room) until the patient has been asymptomatic for 48 to 72 hours. Handwashing with soap and water or soap and an antiseptic is preferred because of the absence of sporicidal activity of alcohol in waterless antiseptic hand rubs. Furthermore, hand hygiene with soap and water has been shown to be superior to an alcohol rub for removal of *C difficile*.[49] Soap and water and water and chlorhexidine have been shown to be equally effective in the removal of *C difficile* from bare hands. Because chlorhexidine is not sporicidal, it is the physical removal of *C difficile* from the hands by vigorous washing that is key to preventing hand contamination. In addition, the use of disposable gloves has been shown to significantly reduce hand contamination of health care workers. Importantly, the use of alcohol-based hand rubs in the endemic setting has not been shown to result in an increase in *C difficile* infection.[50] Because alcohol-based hand rubs increase hand hygiene compliance, their use should be encouraged in health care facilities. The use of alcohol-based waterless products for hand hygiene in hospitals have been demonstrated to not affect the rates of CDI in the institution.

Environmental disinfection. The CDC and the Hospital Infection Control Practices Advisory Committee recommend that environmental cleaning and disinfection should be ensured to aid in preventing *C difficile* transmission.[34] They also state that hypochlorite solutions may be required for disinfection of noncritical items and environmental surfaces. The recent guideline by the CDC, the Society for Healthcare Epidemiology of America, and the Infectious Disease Society of America recommends that facilities consider using a 1:10 dilution of sodium hypochlorite for environmental disinfection in outbreak settings and settings of hyperendemicity in conjunction with other infection prevention and control measures.[47] Use of 1:10 diluted hypochlorite solutions for surface disinfection has been demonstrated to reduce CDI rates when used either in outbreak settings or when high rates of CDI have been documented. Surface disinfectants such as 70% isopropanol, phenols, and quaternary ammonium compounds should not be used because they are not sporicidal.[7]

Whereas the use of sodium hypochlorite for surface disinfection has demonstrated benefit when used as part of an intervention program to control outbreaks or in cases of high endemicity, the routine use of hypochlorite to reduce *C difficile* infection rates has not been evaluated. Recently, the routine use of hydrogen peroxide vapor room decontamination was shown to reduce the epidemic rate of CDI.[45]

ACINETOBACTER SPECIES

Microbiology and epidemiology

Acinetobacter spp are strictly aerobic, gram-negative, nonfermentative, coccobacillary rods. In recent years, the frequency of multidrug-resistant (MDR) *Acinetobacter* spp has been increasing, and multiple outbreaks have been reported.[51-53] Once established, outbreak strains may become endemic within an institution. The crude mortality rate for *Acinetobacter* infections has ranged up to 50%,

whereas attributable mortality has ranged from 8% to 23% for hospitalized patients and from 10% to 43% for intensive care unit patients.

Environmental survival

Multiple clonal outbreaks of *Acinetobacter* have been reported, most commonly in intensive care units.[7] The potential to cause outbreaks is enhanced by the ability of *Acinetobacter* to survive in the environment on both dry surfaces and in water for prolonged periods of time (weeks).[7] In vitro experiments have demonstrated that *Acinetobacter* can survive on multiple surfaces including Formica, ceramic, stainless steel, rubber, and polyvinyl chloride. Wendt et al tested 10 strains of *A baumannii* on 4 surfaces (ceramic, polyvinyl chloride, rubber, and stainless steel); 50% of survival curves showed survival at relevant colony counts of more than 10^2 colony-forming units (CFU) per sample for at least 2 weeks.[54] Some curves demonstrated survival for 16 weeks. Higher relative humidity promotes survival. Both sporadic and outbreak strains of *A baumannii* exhibited prolonged survival on dry surfaces (mean survival time, 21 to 31 days).[55] In one outbreak, the outbreak strain of *Acinetobacter* was isolated from a bed rail 9 days after the infected patient had been discharged.[56] In a human challenge study, *Acinetobacter* survived on fingertips for 60 minutes.[57]

Hospital contamination

Extensive environmental contamination has been demonstrated in numerous outbreaks. Colonized sites have included bed rails, bedside tables, surfaces of ventilators, sinks, suction equipment, mattresses, resuscitation equipment, curtains, slings for patient lifting, mops, buckets, door handles, stethoscopes, incubators, and computer keyboards. The colonization of respiratory tract equipment and devices has been common. The frequency of environmental contamination in outbreak settings has been reported by investigators to range from 3% to 50%.[7] Colonization of the hands of health care workers with *Acinetobacter* has been demonstrated. For example, Markogiannakis et al recovered *Acinetobacter* from 12 of 42 (28.6%) hand cultures.[58]

Evidence of the role of environmental contamination in transmission

Environmental contamination is thought to play an important role in hospital outbreaks because clinical isolates of *Acinetobacter* spp are capable of surviving for prolonged periods in the environment, many outbreaks have been associated with extensive environmental contamination, and contamination of the hand of health care workers has been demonstrated (Table 3). Enhanced environmental cleaning and disinfection have often been part of intervention programs for controlling *Acinetobacter* outbreaks. In some outbreaks, enhanced environmental disinfection was temporally associated with control of the outbreak, whereas, in other outbreaks, the unit was closed to allow thorough disinfection. Enhanced environmental disinfection was part of a comprehensive ''bundle'' successfully used to lower the endemic rate of MDR-*Acinetobacter*.[59]

Interventions to control surface contamination

General guidelines. Common measures to control *Acinetobacter* outbreaks have included emphasizing hand hygiene, use of Contact Precautions for colonized or infected patients, cohorting colonized or infected patients, cohorting staff when taking care of colonized or infected patients, use of surveillance cultures to identify colonized patients, and unit closure. Investigations have occasionally revealed an environmental reservoir, which was most commonly respiratory equipment. In a human challenge study, 4 hand antiseptics (liquid soap, 70% ethanol, 10% povidone-iodine, and 4% chlorhexidine) were equally effective (>99.8%) in removing *Acinetobacter* from lightly contaminated (ie, 10^3 CFU)/fingertip) hands.[60] However, when fingertips were heavily contaminated (ie, 10^6 CFU)/fingertip), 70% ethanol and 10% povidone-iodine were more effective.

Environmental disinfection. Enhanced environmental cleaning/disinfection is recommended as part of a ''bundle'' when managing an outbreak of *Acinetobacter*. Improved cleaning frequency and efforts to clean all surfaces are critical as well as sterilization/disinfection of potentially contaminated respiratory/water sources/devices such as humidifiers, pressure transducers, spirometers, temperature probes, and ventilators. *Acinetobacter* has been shown to be susceptible to phenols, quaternary ammonium compounds, a 0.5% accelerated hydrogen peroxide product, and ultraviolet light.[7] For this reason, standard Environmental Protection Agency-approved hospital disinfectants are recommended for surface disinfection during *Acinetobacter* outbreaks. As always, surface disinfectants need to have contact with all contaminated surfaces, and they should be applied in the appropriate concentrations for the correct time.

CONCLUSION

The CDC/Hospital Infection Control Practices Advisory Committee guidelines for environmental infection control in health care facilities[10] and sterilization and disinfection in health care facilities[11] should form the basis for institutional policies regarding surface disinfection. The scientific evidence has strongly suggested

that contamination of surfaces in hospital rooms plays an important role in the transmission of MRSA and VRE. Recent evidence also strongly suggests that contaminated surfaces are important in the spread of the emerging health care-associated pathogens norovirus, *C difficile*, and MDR-*Acinetobacter*. For all 3 pathogens, as well as all MDR pathogens, enhanced cleaning and disinfection of all room surfaces are highly recommended when managing outbreaks. Studies have demonstrated that many room surfaces are not adequately cleaned, but that validated methods can be used to improve cleaning such as improved training of environmental service workers, use of checklists, and use of marker fluorescent dyes. Alternatively, the use of no touch disinfection methods such as ultraviolet light and vaporized hydrogen peroxide may be used. For norovirus and *C difficile*, the use of hypochlorite solutions (usually 1:10 diluted household bleach) has often been recommended for surface disinfection in hospital rooms as part of an intervention ''bundle'' to control a health care-associated outbreak.

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