Are those room finishes and cleaners safe?

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We've all heard that hospitals are "dangerous places." Just what does that mean? The increasing fear of being a patient in even the best hospitals in the United States has to do primarily with two things: avoidable medical errors and hospital-acquired infections due to the rise of "superbugs."

Lest the term conjure some combination of Super Mouse meets *A Bug's Life*, superbugs are no such thing. Superbugs are "opportunistic" bacteria. They lie in wait, silently and usually harmlessly, for the opportunity to take hold in vulnerable patients. Once they do, they kill as many as 90,000 patients annually in the U.S. alone.[1] Superbugs develop when previously innocuous bacteria that we carry around in our gut, such as enterococci, exchange genetic material with harmful bacteria such as staphyolocci in a kind of genetic cross-pollination. Genes giving them resistance to antibiotics can develop in several ways, but once they have them, these new bacteria will survive and pass that resistance on to offspring. Once a strain of bacteria carries several of these resistance genes, it is then referred to as "multiresistant" or, informally, a "superbug."

The rise of superbugs is relatively recent, roughly paralleling in time both the development, and worse, the accelerating evolution, of computers. They were virtually unknown before 1947, when a form of *Staphylococcus aureus* developed resistance to penicillin just four years after penicillin began being mass-produced. After methicillin became the antibiotic of choice, MRSA (methicillin-resistant *S aureus*) was detected in Britain in 1961, where it rose from causing just 4% of fatal cases of blood poisoning in 1991 to 37% in 1999. MRSA has since evolved into a virtual plague in U.S. hospitals, where half of all *S aureus* infections are now "multiresistant" to penicillin, methicillin, tetracycline, and erythromycin.

This left vancomycin, first developed in 1956, as the only effective antibiotic available—until 1997, when vancomycin-resistant *S aureus* was identified in Japan, and has since spread to hospitals in England, France, the United States, and beyond. In our attack-counterattack war with superbugs, a new class of antibiotics—oxazolidinones (first brand name Linezolid) became available in the 1990's, offering comparable effectiveness to vancomycin against MRSA. But then Linezolid-resistant *S aureus* was reported in 2003.

So, almost as fast, or sometimes even faster, than we can develop new antibiotics, new resistant forms of superbugs emerge that foil our efforts to control them. They present a tremendous continuing threat throughout institutional healthcare today.

Let's meet a couple of these "bad actors" up close and personal—the "stars" of our study[2]:

Pseudomonas aeruginosa, nicknamed PSAE, has been called "the epitome of an opportunistic pathogen of humans."[3] Considered a "nosocomial" (or hospital-acquired) pathogen, it infects about 4 patients per 1,000 hospital discharges in the U.S., and accounts for over 10% of all hospital-acquired infections.[3] It almost never infects uncompromised tissues, but will infect almost any tissue whose defenses are compromised. It causes infections of the urinary tract, lungs and respiratory system, soft tissue, bones, joints, skin, and gut, and even the inner linings of the heart, not to mention bacteremia, septicemia, and other systemic infections. PSAE is so resistant to antibiotics that it ends up killing about half of hospitalized cancer, cystic fibrosis, and burn patients unlucky enough to contract it.[3] Most importantly, its nutritional requirements are so low that it can survive for long periods of time outside the body under a great variety of conditions.[3]

Enterococcus faecium, better known as *E Faecium* or just VRE (Vancomycin-resistant enterococcus), is another infamous superbug that infests hospitals across the country and accounts for 12% of all nosocomial infections.[4] Enterococci are part of the flora that naturally inhabit the digestive tract in humans and other animals, so they are able to tolerate not only relatively high salt and acid concentrations, but also to withstand dry conditions (as exist on many environmental surfaces in patient areas).[4] Worse yet, they can also withstand low levels of detergents, which is why inadequate cleaning procedures can actually promote enterococcal infections.[4]

Thought to be another strain of streptococci, or "Strep" until the 1930s, enterococci were given the status of a separate genus only back in 1984. An enterococcal infection can cause complicated abdominal, skin, skin structure, wound, pelvic, and urinary tract infections, "super-infections" (infections that occur while a patient is being treated for another infection), and bacteremia and septicemia (infections of the blood stream, local and systemic respectively).[4]

If it's any indication of the dynamic evolution of these bugs it was only back in 1983 that the resistance to penicillin of *E faecium* was first noted. And, again, it was only in 1988 that the first case of resistance to vancomycin, the "antibiotic of last resort," was first detected.[4] Between 1995 and 1997 15,000 sample isolates of *E faecium* were analyzed; of these, 83% were resistant to ampicillin and 52% were resistant to vancomycin![4] Worst of all, it appears likely that we will soon be faced with increasing numbers of enterococci (along with other strains of superbugs, as well) for which there are no adequate remedies at all.

With such dangerous critters always keeping patients at high risk, it is vital that we develop an understanding of not only how superbugs are spread, but where they hide—which, in many cases is in the wide open. We know that caregiver's hands have long been implicated as the major culprit in spreading infection.[5-8] We know that superbugs can survive for long periods of time on dry surfaces such as countertops, bedrails, and healthcare equipment.[9,10] But we know less about the role that environmental surfaces in the medical environment—upholstery, wall and flooring—play in infection.

How successfully do these su

Products and materials might have been reformulated since the time of this study. Publication of this article should not be construed as endorsement or condemnation of particular products by *HEALTHCARE DESIGN*.

Table 1. Materials tested.

Upholstery	
	1. 100% woven solution-dyed nylon fabric (*A-M)
	2. Polyester and acrylic blend woven Crypton® (*A-M)
	3. 100% jacquard woven polyester with proprietary water-based polymeric coating (*A-M)
	4. Vinyl upholstery (*A-M)
Flooring	
	5. Tufted 6,6 solution-dyed carpet with woven synthetic backing (*A-M)
	6. Tufted 100% solution-dyed proprietary nylon carpet with recycled vinyl backing
	7. Vinyl composition tile
	8. Linoleum
	9. Vinyl sheet goods
	10. Rubber tile
Wall finishes	
	11. Latex paint with eggshell finish
	12. Vinyl wallcovering, Type II, 20 oz.
	13. Vinyl microvented perforated vinyl wallcovering Type II, 20 oz.
	14. Continuous monofilament polyethylene textile paper-backed wallcovering

*A-M indicates antimicrobial treatment claims

faces harbor superbugs? Do such surfaces actually support or even enhance their survival? And over what intervals of time do they host these organisms before they weaken or die? How "clean" are these surfaces actually after disinfecting according to manufacturers' instructions and recommendations? How well do they support or inhibit the action of disinfectants? How well do these dangerous bacteria recover after cleaning? And, finally, how well do these surfaces allow the transmission of infection?

These are—or should be—vital questions for healthcare designers and architects, hospitals, insurers, and the healthcare industry in general. So CHER—the Coalition for Health Environments Research—commissioned investigators at Chicago's Northwestern Memorial Hospital to find out. The study's title is "Limiting the Spread of Infection in the Healthcare Environment," with Mary G. Lankford, RN, BSN as the principal investigator, along with Susan Collins, MT, Larry Youngberg, MT, Denise M. Rooney, RN, BSN, John R. Warren, MD, and Gary Noskin, MD.[2] The study was funded by the Global Vinyl Council and the Chemical Fabrics and Film Association.

These researchers evaluated three categories of environmental surfaces: upholstery, flooring, and wall finishes; 14 materials in all (table 1) were assembled according to manufacturers' installation and construction specifications, and then blind-tested.

For testing ("contamination"), researchers used bacterial strains (called "isolates") of the two superbugs described above: VRE and PSAE. Samples of each of these superbugs, roughly at concentrations one would find in infected urine, were dripped onto each of the surfaces to simulate soiling by contaminated body fluids.

To assess the potential of each of these now-contaminated 14 surfaces both to harbor superbug infection over time and to allow it to be picked up on a provider's hands (and subsequently passed on to a patient), healthy volunteers touched the VRE-contaminated surfaces with the freshly washed palms of their hands after intervals of 5 minutes (essentially immediately after contamination), after 1 day, after 3 days, and after 1 week. They then imprinted their palms onto culture plates, which were incubated to show contamination. Table 2 shows the results.

As a control, all surfaces were initially cultured to see if there was any measurable bacterial growth noted on any of the surfaces. There was not, but *after* contamination, all surfaces seeded with VRE remained contaminated for an entire week. While contamination levels declined for 11 materials, 3 surfaces— vinyl composition tile (7), microvented perforated vinyl wall covering (13), and continuous monofilament polyethylene textile paper-backed wall covering (14)—had virtually no reduced contamination at 1 week. In contrast, surfaces contaminated with PSAE had overall lower bacterial growth. Impressively, one surface—woven Crypton®(2) —showed no PSAE at any of the testing intervals past the initial one.

Many manufacturers represent their products as being "antimicrobial," and the first 5 materials listed make similar claims—or at least did so at the time of the study. In all fairness, it is possible that some of these products have been reformulated since. At the time of the study, however, none of them was apparently effective in preventing the growth of VRE, and only the vinyl and the Crypton® upholstery showed no continuing growth of PSAE at 72 hours and beyond.

What these results show is that *any* surface, regardless of the material, may act as a reservoir for the transfer of infection.

To assess how effectively the surfaces could be cleaned ("decontaminated") and the degree to which and duration that samples would remain infectious, our superbug-contaminated surfaces were first cleaned according to manufacturers' instructions (table 3), then cultured to detect remaining superbug contamination at intervals of 5 minutes, then at 1 day, 3 days, and 1 week. Results are also in table 3.

This part of the testing yielded some surprising results:

- VRE are in general hardy organisms that can survive long periods of time on surfaces within patient rooms.
- Cleaning with detergents, quaternary solutions, 70% alcohol, and vinegar solutions effectively cleaned only 7 (50%) of the VRE-contaminated surfaces and 9 (64%) of the PSAE-contaminated surfaces. In other words, detergents and quaternary compounds, as recommended by manufacturers of the surface materials, weren't effective in disinfecting 5 of the 14, or almost 36% of the surfaces.
- One interesting and highly counterintuitive variable related to the texture of surfaces is that while smooth surfaces would seem to be easier to sanitize, smoother surfaces were on the whole (with the notable exception of vinyl upholstery) less effectively cleaned and/or more likely to transmit infection to hands.

There are several plausible explanations:

1. different cleaning compounds were used for each, according to their manufacturer's recommendations,
2. absorptive surfaces retain disinfectants in contact with the pathogens longer, or
3. smooth surfaces provide much better transmission surfaces for hands.

Conclusion

Several conclusions emerge from these findings:

- Cleaning of environmental surfaces is important, with differing results depending on the cleaning products used, their concentrations, and the amount of time they're left on the surface.
- Complying with standard hand-hygiene recommendations before and after patient contact is vital.
- Most designers aren't microbiologists and rely on manufacturers' recommendations. Their general impression is that "antimicrobial" goods don't permit germs to grow on or in them. In fact, the antimicrobial label does not appear to ensure safe clinical performance.
- Some manufacturers seemed not to have done their homework specifying effective cleaning means consistent with the types of cleaning products actually being used or not to have tailored their products to withstand today's clinical housekeeping realities.

Table 2. Contamination levels at varying intervals for each of 2 containments. No cleaning.

Material	VRE-contaminated				PSAE-contaminated			
	5 min	1 day	3 days	1 week	5 min	1 day	3 days	1 week
Upholstery								
1.	major	spotty	spotty	spotty	major	spotty	spotty	spotty
2.	major	spotty	spotty	spotty	major	none	none	none
3.	major	spotty	?	?	major	spotty	spotty	spotty
4.	major	spotty	spotty	spotty	major	spotty	none	none
Flooring								
5.	major	spotty	spotty	spotty	major	spotty	spotty	spotty
6.	major	spotty	spotty	spotty	major	spotty	none	spotty
7.	major	major	major	major	major	spotty	none	none
8.	major	major	spotty	spotty	major	spotty	spotty	spotty
9.	major	major	spotty	spotty	major	spotty	none	none
10.	major	major	spotty	spotty	major	spotty	none	none
Wall finishes								
11.	major	major	major	spotty	major	spotty	none	none
12.	major	major	major	spotty	major	spotty	none	none
13.	major	major	major	major	major	spotty	none	none
14.	major	major	major	major	major	spotty	spotty	spotty

Table 3. Contamination of VRE- and PSAE-contaminated samples before & after cleaning (according to manufacturer's instructions).

Material	Mfr's recommended cleaning mat'ls/methods. Mat'ls/methods used in ( )'s.	VRE		PSAE	
		BC	AC	BC	AC
Upholstery					
1.	Water-based cleaning agent (Mild detergent foam shampoo, hot water extraction)	major	none	major	none
2.	Proprietary upholstery cleaner pre-treatment, followed by enzyme powder laundry detergent	major	spotty	major	spotty
3.	5 minutes 70% Isopropyl alcohol, hot water extraction, repeated in 24 hours	major	spotty	major	none
4.	Mild soap solution (Energetic washing, mild soap)	major	none	major	none
Flooring					
5.	Quaternary solution; hot water extraction	major	none	major	none
6.	Full-strength proprietary cleaning agent, agitation	major	none	major	none
7.	Proprietary detergent floor cleaner	major	spotty	major	spotty
8.	Neutral pH detergent (Quarternary solution)	major	spotty	major	none
9.	Proprietary detergent floor cleaner	major	spotty	major	spotty
10.	Neutral pH cleaner	major	spotty	major	spotty
Wall finishes					
11.	Mild detergent and water with soft brush	major	spotty	major	spotty
12.	Strong soap solution (Quaternary solution, hot water extraction)	major	none	major	none
13.	70% Isopropyl alcohol and specified spray-on degreaser	major	none	major	none
14.	Proprietary upholstery cleaner followed by vinegar and rinsed with water	major	none	major	none

BC=before cleaning. AC=after cleaning.

- Conventional wisdom about vinyl upholstery in healthcare may be correct: It is perhaps hotter, sweatier, and sometimes less attractive than textiles, but it appears that it can be effectively disinfected.

Again, in fairness, the results of this study might have differed had a different concentration of superbug "brew" been used for testing, had standardized cleaning methods been used rather than following manufacturers' recommendations, had different microorganisms been used for testing, and had similar categories of products from different manufacturers been used.

It should also be noted that, in order to transmit an infection, an "agent" is needed. In our hospitals, that agent is all too frequently the healthcare worker who fails to adequately clean his or her hands. That infection may be carried from another patient, from an infected bedrail, or from leaning with one's hands against a contaminated wall. Or, it may be the patient himself who touches a contaminated bedrail, seat cushion, armrest, doorknob, or remote control, and whose own hands become the vector. This is why handwashing is such a key to infection control. Because this requirement is not always met, however, it is vital that surfaces—particularly those likely to be touched—effectively be kept as free of infection as possible.

Specific Recommendations

For architects/designers: Pay particular attention to location and usability of hand-washing stations within the patient room. Do everything you can to make it easy and convenient or even unavoidable to use.

For manufacturers: Start finding out which disinfecting agents the hospitals likely to use your products are actually stocking. Then begin testing to develop a range of alternatives for safely cleaning your products. Get together with your competition and with manufacturers whose products are likely to be cleaned with the same agents—the healthcare goal justifies this—and standardize methods and materials for cleaning; start promoting those standardized recommendations. A good place to begin is reviewing the Center for Disease Control and Prevention's "Guidelines for Environmental Infection Control in Healthcare Facilities," available at **www.cdc.gov/ncidod/dhgp/pdf/guidelines/Enviro_guide_03.pdf.**

For all stakeholders: Start demand products that will not harbor infectio "Antimicrobial", as presently defined, is good enough. **HD**

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