its emergence as a major community pathogen nationally.[8] The concurrent increase in cases of SSIs caused by MSSA, however, was unexpected. This finding suggests that despite the increase in MRSA, MSSA still plays a large role in causing SSIs. Therefore, preoperative screening for *Staphylococcus* spp., not just MRSA, may help guide preoperative antibiotic selection, skin preparation, and postoperative wound care to minimize the risk of infection with either of these organisms.[9]

The predominance of gram-negative organisms in polymicrobial SSIs suggests that external contamination of the wound, (eg, with fecal matter) plays a major role in polymicrobial SSI pathogenesis. This finding highlights the ongoing importance of postoperative wound management and the need for protective barriers to prevent contamination of the wound.[9]

Our conclusions are limited by our inability to account for potential correlations between patient-level characteristics, such as comorbidities, with particular organisms causing SSIs. [10] Another limitation was our inability to assess the direct influence of specific interventions that occurred in our medical center over the study period.[3] Further study is planned to examine such interactions.

Our study findings indicate that among pediatric patients, skin and bowel flora play a significant role in SSIs. Future interventions to target aspects such as preoperative screening and management of MSSA and MRSA colonization and postoperative wound management to prevent fecal contamination may reduce pediatric SSIs. Further study is planned to assess the effect of patient and procedure factors as well as interventions on both the incidence of and the type of pathogens associated with SSIs.

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Jon Woltmann, MD;
Joshua K. Schaffzin, MD, PhD;
Matthew Washam, MD, MPH;
Beverly L. Connelly, MD

Affiliations: Division of Infectious Diseases, Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio.

Address correspondence to Jon Woltmann, MD, Clinical Fellow, Division of Infectious Diseases, Cincinnati Children's Hospital Medical Center, 3333 Burnet Avenue MLC 7017, Cincinnati, Ohio, 45229-3039 (jon.woltmann@cchmc.org).

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Oak in Hospitals, the Worst Enemy of *Staphylococcus aureus*?

To the Editor—Although the infection risk to patients from contaminated healthcare surfaces has long been controversial, it is now recognized that the environment may facilitate transmission of several important healthcare-associated bacteria, including vancomycin-resistant enterococci, *Clostridium difficile*, *Acinetobacter* spp., and methicillin-resistant *Staphylococcus aureus* (MRSA).[1] In addition, the longer a nosocomial pathogen persists on a surface, the longer it may be a source for transmission to a susceptible patient or healthcare worker.[2] Therefore, regular and conscientious cleaning is a necessary measure for keeping surfaces free from microbes. The nature of surfaces can also be considered.[1] Although the use of wood is not banned in hospitals,[3] this material still generates controversy in terms of infection

control.[2,4] Concurrently, the benefits of a wood interior in a hospital room have been acknowledged by hospital staff,[5] and although it was demonstrated that the use of wooden wall panels in hospital rooms had no effect on the amount of volatile organic compounds.[6] Considering those benefits, we aimed to test the potential antimicrobial activity of oak on a panel of *S. aureus* with different resistance patterns to antibiotics.

In total, 8 *S. aureus* clinical isolates (4 MRSA and 4 methicillin-susceptible *S. aureus*) were tested using disc diffusion according to the European Committee on Antimicrobial Susceptibility Testing (EUCAST) recommendations.[7] Of those bacteria, 2 had been isolated from sputum samples from cystic fibrosis patients, 2 from abscesses, 3 from blood cultures, and 1 from a urine sample. Samples of oak (*Querceus* spp.) used for the wood disks were derived from mature trees grown in France. Each oak sample was cut into a10-cm-thick board and was further cut by electric saw (Altendorf-F45, Minden, Germany) into thinner (2.5 mm) sheets with respect to the radial (R) or longitudinal (L) section. These oak sheets were used to prepare circular wood disks using a laser cutting machine (Trotec-SP500, C60, Wels, Austria). The diameter of 9 mm was selected because of the minimum accurate circle-making capacity of the machine. Disks of antibiotics currently used in our lab for clinical microbiology (ie, linezolid, trimethoprim + sulfamethoxazole, kanamycin, tobramycin, gentamicin, ofloxacin, fosfomycin, rifampicin, minocycline, all from Oxoid, Basingstoke, UK) were used for the study. Blank paper disks (ie, without an antimicrobial substance) were included as negative controls.

According to EUCAST break points, 3 isolates were resistant to kanamycin and tobramycin, 1 isolate was resistant to all aminoglycosides tested, 5 isolates were resistant to ofloxacin, and 2 isolates were resistant to rifampicin. All isolates were susceptible to trimethoprim + sulfamethoxazole, linezolid, and minocycline (Table 1). The major result of this report is that oak showed an antimicrobial activity on all the isolates tested. When considering both R and L disks, the inhibition diameters around the disks were ~20 mm and homogeneously distributed (standard deviation < 3 mm). Notably, methicillin resistance did not really influence those diameters. The means of inhibition diameters around oak disks (19.4 ± 2.7 mm) and around aminoglycoside disks (18.8 ± 6.9 mm) were similar. Lastly, diameters around R disks were slightly greater than diameters around L disks.

We demonstrated that wooden materials, and more particularly oak in this study, have an antimicrobial activity against a small but diverse panel of *S. aureus*. These results are somewhat discordant with those of some preceding reports. In a study comparing the recoverable proportion of MRSA from wood-free paper (containing < 5% wood pulp and therefore essentially composed of cellulose pulp) and paper containing wood, Kacmaz et al[4] demonstrated that the counts of recoverable bacteria were significantly higher in paper containing wood at the different point measures (ie, 24 h, 48 h, 120 h,

TABLE 1. Inhibition Diameters Recorded Using the Disk Diffusion Method With 4 Methicillin-Resistant *Staphylococcus aureus* (MRSA)and 4 Methicillin-Susceptible *Staphylococcus aureus* (MSSA) Isolates[a]

Isolates	Oak (R)	Oak (S)	NC	LNZ (19–19)[b]	SXT (14–17)[b]	K (14–18)[b]	TM (18–18)[b]	GM (18–18)[b]	OFX (20–20)[b]	RIF (23–26)[b]	MN (20–23)[b]
16514759301 (MSSA)	20	20	6	25	34	24	22	28	26	32	30
15015019102 (MSSA)	24	20	6	30	43	6	6	24	10	6	23
16006004401 (MSSA)	25	21	6	24	26	24	22	23	28	34	28
16513145401 (MSSA)	18	14	6	21	32	22	27	22	36	30	27
Means ± SD (MSSA)	21.8 ± 3.3	18.8 ± 3.2	6	25.0 ± 3.7	33.8 ± 7.0	19.0 ± 8.7	19.3 ± 9.1	24.3 ± 2.6	25.0 ± 10.9	25.5 ± 13.1	27.0 ± 2.9
16005880401 (MRSA)	18	16	6	24	23	6	9	8	14	32	26
16009349201 (MRSA)	20	17	6	30	33	6	6	25	14	30	28
14528592702 (MRSA)	22	22	6	29	31	23	24	23	10	6	28
16518868104 (MRSA)	18	16	6	26	30	24	23	24	8	30	26
Means ± SD (MRSA)	19.5 ± 1.9	17.8 ± 2.9	6	27.3 ± 2.8	29.3 ± 4.3	14.8 ± 10.1	15.5 ± 9.3	20.0 ± 8.0	11.5 ± 3.0	24.5 ± 12.4	27.0 ± 1.2
Means ± SD (overall)	20.6 ± 2.8	18.3 ± 2.9	6	26.1 ± 3.3	31.5 ± 5.9	16.9 ± 9.0	17.4 ± 8.8	22.1 ± 6.0	18.3 ± 10.3	25.0 ± 11.8	27.0 ± 2.1

NOTE. NC, negative control; LNZ, linezolid; SXT, trimethoprim + sulfamethoxazole; K, kanamycin; TM, tobramycin; GM, gentamicin; OFX, ofloxacin; RIF, rifampicin; MN, minocycline; SD, standard deviation.

[a]All data are presented in millimeters.

[b]EUCAST 2016 break points (in mm) are indicated under the name of each antibiotic.

144 h, and 168 h after the initial contamination). They proposed the use of paper containing wood to a lesser degree and for shorter periods in hospitals, especially when the compliance for hand hygiene is poor. By using a model of bacterial transmission from wood fomites artificially contaminated with MRSA USA300 to pigskin at different times after the initial contamination, Desai et al[8] demonstrated that USA300 was transmitted from wood to skin up to 3 days. Lastly, in a study conducted in 3 intensive care units (16 rooms in total) with weekly measures over a 43-month period, Schmidt et al[2] demonstrated that 61% of wooden chair arms were contaminated by high bacterial loads (microbial burden, >250 CFU/100 cm^2). Our results are more consistent with those reported by Da Costa et al.[9] By observing the spontaneous contamination of tiles cut from oak, stainless steel, and high-density polyethylene, they demonstrated that wooden tiles were contaminated significantly less often than plastic tiles (10.3% vs 33.3%; $P = .028$) and were less often contaminated than metal tiles (10.3% vs 30.1%; $P = 0046$). They concluded that oak is a more hostile environment for bacteria than the other surfaces tested.

The difference of the results between R and L could be explained by a difference in the diffusion of antimicrobial products in the agar medium depending upon the wood-cutting method. This finding is also consistent with the existence of antimicrobial products inside oak. Another interesting result is absence of impact of methicillin-resistance on the diameters around L and R. A hypothesis to explain this result could be the diversity of effective antimicrobial molecules that can be potentially present in vegetal resources like essential oils.[10]

These results should be completed by testing other bacteria potentially isolated from environmental surfaces to evaluate the microbial safety of using oak in the hospital setting.

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Hélène Pailhoriès, PharmD, PhD;[1]
Muhammad Tanveer Munir, MSc;[2]
Florence Aviat, PhD;[2]
Michel Federighi, DVM, PhD;[3]
Christophe Belloncle, PhD;[2]
Matthieu Eveillard, PharmD, PhD[1]

Affiliations: 1. ATOMycA, Inserm Equipe Avenir, L'UBL Université Bretagne-Loire, Angers, France; 2. Laboratoire Innovation Matériau Bois Habitat Apprentissage (LIMBHA), Ecole Supérieure du Bois, Nantes, France; 3. Hygiène et qualité des aliments, Oniris, Nantes, France.

Address correspondence to Matthieu Eveillard, PharmD PhD, L'UBL Université Bretagne-Loire, ATOMycA, Inserm Equipe Avenir, CRCNA, Inserm U892, 6299 CNRS, IRIS, CHU, 4 rue Larrey, 49933 Angers cedex, France (MaEveillard@chu-angers.fr).

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