measures, as well as useless treatment with toxic entities such as amphotericin B, which would have been used in these cases. Although *F verticillioides* was identified as a contaminant in this episode, it cannot always be assured to be the consequence of laboratory contamination. For this reason, it is of fundamental importance to conduct a careful examination both of the patient's clinical records and of the work protocol in other wards and in the laboratory, so that the results of the cultures may be critically interpreted in a clinical way. It also seems opportune to identify the microorganism at the species level and, in case of outbreak, to use a molecular typing method to establish a link with an environmental source.

Only close, constructive, and timely collaboration between the microbiology laboratory and the diagnostic and nursing wards can permit prompt identification of such episodes and establish the common denominator that unites patients who are quite different but are linked by the isolation of an often unusual microorganism. Furthermore, close collaboration with the hospital pharmacy is needed to establish suitable containment measures for such an episode, adopting precautionary measures (such as withdrawing the contaminated equipment) and proposing administrative tasks (such as notifying the suppliers and the Ministry of Health) in order to bring about systematic quality control of commercial sterile products.[9]

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Molecular Epidemiology of VRE in New York

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Bopp and colleagues determined the distribution of vancomycin-resistant enterococci (VRE) within and between six New York State hospitals, using antibiotic susceptibility testing, pulsed-field gel electrophoresis (PFGE) fingerprinting, plasmid profile analysis, *van*A and *van*B PCR, and DNA: DNA hybridization with *van*A and *van*B probes.

PFGE and plasmid typing generally agreed, but plasmid profiles were more variable. Genetic heterogeneity among isolates from within each of the six hospitals varied considerably. Among 23 *Enterococcus faecium* isolates from one hospital, there were only 3 PFGE types, and 20 isolates had the same type. However, in another hospital, each isolate was genetically distinct. Closely related strains were not found in separate hospitals. VRE strains with *van*A genes and strains with *van*B genes were found in three hospitals. Both plasmid and chromosomal carriage of these genes was detected. PFGE typing showed that nosocomial VRE transmission had occurred in some hospitals. However, there was no evidence for it in others; neither was there evidence for intrahospital transmission or for emergence of an endemic strain.

These observations demonstrate that it is important to evaluate genetic heterogeneity among VRE before implementation of infection control measures. PFGE is the method of choice for epidemiological typing, but PCR, plasmid, and hybridization studies can provide important information concerning the presence and potential for transfer of vancomycin resistance genes.

FROM: Bopp LH, Schoonmaker DJ, Baltch AL, Smith RP, Ritz WJ. Molecular epidemiology of vancomycin-resistant enterococci from 6 hospitals in New York state. *Am J Infect Control* 1999;27:411-417.