

whether they exclude a null effect (say, a relative risk of 1), since doing so creates the same problems noted by Proestel for significance tests.

One important consideration for subgroup analysis is how subgroups are formed. Kent and Hayward recommend a specific way of forming subgroups on the basis of multiple, rather than individual, baseline characteristics. In their approach, patients are divided into separate groups according to their risks of a disease outcome, which are calculated from a prespecified, externally validated formula involving multiple baseline characteristics. The purpose of such subgroup analyses is to assess whether the treatment effect is homogenous across patients with different risks. We agree that such an approach can provide valuable information to guide individualized patient care. Moreover, when a specific risk-score algorithm is unavailable, it still could be appropriate to assess the heterogeneity of the treatment effect with the use of a prespecified clinically meaningful categorization based on multiple baseline characteristics. In other settings, interest in the heterogeneity of treatment effects may

be motivated by metabolic, physiological, anatomical, genetic, or other independently identifiable features of the patients or their disease, not by their risk of the disease outcome under study. These considerations should be the main determinants of how subgroups are formed.

Finally, we do not believe that journals should dictate the scientific questions that investigators address, including whether and how they undertake subgroup analyses of any specific type. Rather, investigators should use a well-reasoned and fully described approach to subgroup analyses and report them in accordance with the guidelines offered in our article.

Rui Wang, M.S.

Stephen W. Lagakos, Ph.D.

Harvard University
Boston, MA 02115

Jeffrey M. Drazen, M.D.

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Methicillin-Resistant *Staphylococcus aureus* in a Family and Its Pet Cat

TO THE EDITOR: Many isolates of community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) produce Pantón-Valentine leukocidin (PVL), increasing the virulence of the bacteria, which can cause disseminated deep abscesses and necrotizing pneumonia.¹ We report the transmission of PVL-positive MRSA between a symptomatic woman and both her asymptomatic family and their healthy pet cat.

An otherwise healthy woman presented with recurrent multiple deep abscesses. Swabs from several abscesses and nasal cultures grew MRSA that was resistant to both beta-lactam and fusidic acid antibiotics. Polymerase-chain-reaction assays for the PVL genes *lukS-PV* and *lukF-PV* were positive. The genotype of the staphylococcal chromosomal cassette was SCCmec type IV. Nasal, axillary, and inguinal cultures from her husband and their two children yielded MRSA on several occasions. Mupirocin nasal ointment and antiseptic washes were recommended for all family members. Although

the patient's husband and children became MRSA-negative, the patient remained MRSA-positive. Therefore, her three apparently healthy cats were screened. Pharyngeal culture from one cat grew MRSA with the same antimicrobial resistance pattern as that of the human isolates. The clonal identity of the isolates from the family and the cats was found by typing of the *spa* gene repeat region and multilocus sequence typing,^{2,3} which showed *spa*-type t131 and ST80 in all isolates. This sequence combination does not correspond with that of clone USA300 (<http://spa.ridom.de>).²

A veterinarian recommended topical decolonization of the MRSA-positive cat with ciprofloxacin and rifampin. Four weeks after the cat's treatment, screening tests of the family were negative for MRSA. Moreover, the patient's deep abscesses completely resolved. Further MRSA screening of the asymptomatic cat was declined by the family.

There is evidence that companion animals, mainly dogs, harbor MRSA,⁴ and interspecies

MRSA transmission has been shown in the members of a family and their dog.⁵ This case illustrates that MRSA transmission also occurs between humans and cats. The abscesses in our patient cleared only after antibiotic treatment of the cat. It remains unclear whether the cat was the source of the patient's infection or vice versa, although *spa*-type t131 is extremely rare in humans.² We conclude that pets should be considered as possible household reservoirs of MRSA that can cause infection or reinfection in humans.

Andreas Sing, M.D.

Christian Tuschak, Ph.D.

Stefan Hörmansdorfer, Vet.D.

Bavarian Food and Health Safety Authority
85764 Oberschleißheim, Germany

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