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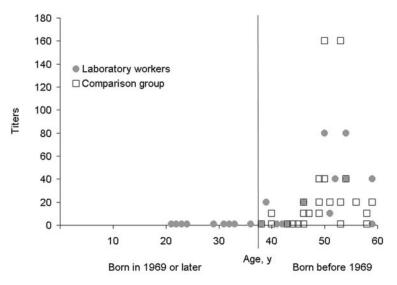


Figure. Titers of antibodies to influenza A H2N2 virus in laboratory personnel (n = 25; 13 born before 1969) and a comparison group born before 1969 (n = 32). The age listed is that in 2005. Titers <10 were assigned a value of 1.

presumably tested in other laboratories involved, but we are not aware of any publications to this end. The lack of evidence for laboratory-acquired infection with A H2N2 in our study suggests that the risk was low under controlled laboratory conditions. However, only a large-scale serologic study (which might still feasibly be undertaken) could further substantiate this finding.

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References

 Eurosurveillance editorial team, Meijer A, Brown C, Paget J. Worldwide laboratory distribution of influenza A/H2N2 virus similar to 1957–58 pandemic strain, labs asked to destroy all samples immediately. Eurosurveillance. 2005;10. [cited 2006 Sep 15]. Available from http://www.eurosurveillance.org/ew/2005/050414.asp

- World Health Organization. International response to the distribution of a H2N2 influenza virus for laboratory testing: risk considered low for laboratory workers and the public. 2005 [cited 2006 May 22]. Available from www.who.int/csr/disease/ influenza/h2n2_2005_04_12/en/.
- Normile D. Mounting lab accidents raise SARS fears. Science. 2004;304:659–61.
- Lim PL, Kurup A, Gopalakrishna G, Chan KP, Wong CW, Ng LC, et al. Laboratoryacquired severe respiratory syndrome. N Engl J Med. 2004;350:1740–5.
- Seidler A, Nienhaus A. Epidemiologie der berufsbedingten tuberkulose: ein literaturreview zu konventionellen studien. In: Nienhaus A, Brandenburg S, Teschler H, editors. Tuberkulose als berufskrankheit: ein leitfaden zur begutachtung. Landsberg (Germany): Ecomed Verlag; 2003. p. 142–69.
- Jacobson JT, Orlob RB, Clayton JL. Infections acquired in clinical laboratories in Utah. J Clin Microbiol. 1985;21:486–9.
- Kao AS, Ashford DA, McNeil MM, Warren NG, Good RC. Descriptive profile of tuberculin skin testing programs and laboratoryacquired tuberculosis infections in public health laboratories. J Clin Microbiol. 1997;35:1847–51.

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Methicillinresistant *Staphylococcus aureus* in Cat and Owner

To the Editor: A 3-year-old, neutered male, domestic shorthaired cat was referred for treatment to a veterinary specialty clinic in San Francisco, California, with a 1-year history of multifocal patches of crusted and well-demarcated ulcers on the trunk. Initially, small crusts suspected to be associated with flea allergy and pyoderma were present; however, response was poor to multiple treatments, including repeated corticosteroid therapy and antimicrobial therapy with amoxicillin-clavulanic acid and enrofloxacin. The owner reported having skin abscesses and pneumonia 3 months earlier, although no microbiologic testing was performed.

Cytologic evaluation of exudate from the cat's lesions identified neutrophils and eosinophils with engulfed cocci. Leukocytosis with eosinophilia was found on a complete blood cell count. No notable abnormalities were present on thoracic radiograph, abdominal ultrasonograph, urinalysis, and tests for feline leukemia and immunodeficiency virus. Skin biopsy specimens were collected for histologic examination, and swabs of the exudates were submitted for bacterial culture. Histopathologic findings demonstrated ulcers and dermal granulation tissue with linearly arranged eosinophils, mast cells, neutrophils, and plasma cells between dense, homogeneous collagen bundles (sclerosing dermatitis). This pattern of inflammation is distinct from most staphylococcal infections of the skin, and it has been suggested that this uncommon histologic finding in cats is associated with methicillin-resistant staphylococcal infection (1).

Methicillin-resistant *Staphylococcus aureus* (MRSA) was isolated from the skin lesions. Identification was confirmed by detection of penicillin-binding protein 2a (PBP2a) by latex agglutination test (PBP2' Test Kit, Oxoid, Hants, UK). Typing was performed by SmaI pulsed-field gel electrophoresis as previously described (2), and the isolate was classified as the USA300 clone. Genes encoding production of the Panton-Valentine leukocidin (PVL) were identified by real-time PCR (3). The isolate was susceptible to chloramphenicol, tetracycline, trimethoprim-sulfamethoxazole, and vancomycin, but resistant to β -lactams, enrofloxacin, and erythromycin. After identification of MRSA in the cat, swabs of the anterior nares were collected from the owner and the cat, and MRSA was identified in specimens from both. All isolates were indistinguishable.

This is the first report of isolation of USA300 MRSA from a household pet. USA300 is a community-associated clone that has disseminated widely throughout North America and Europe (4,5) and is reaching epidemic proportions in many regions. MRSA is becoming an important cause of skin and soft tissue infection in persons in the community (4,5) and has also been implicated in invasive infections such as necrotizing pneumonia (6). This clone possesses genes for PVL production, which may be an important factor in its apparent virulence (4,5). Additional characterization of the isolates from this study was not performed; however, USA300 has previously been reported to carry staphylococcal cassette chromosome mec (SCCmec) type IVa and classified as sequence type 8 (ST8) by multilocus sequence typing (4,5).

Reports of MRSA infection and colonization in pets have increased dramatically in the past few years (3,7-9). Although this rise may be partially the result of increased testing and reporting, MRSA is definitely emerging in pet populations through-

out the world. The role of pets in transmission of MRSA is still unclear; however, recent evidence suggests that MRSA can be transmitted between persons and their pets, in both directions (9,10). Reports of MRSA infection and colonization in pets have indicated that pets tend to be infected with isolates that are consistent with clones that are predominant in the human population in their area (7-9). Accordingly, USA100 accounted for initial isolations of MRSA in pets in North America (9). The similarity between pet and human isolates has led to speculation that pet MRSA is closely linked to human MRSA and that the source of MRSA in pets may often be colonized humans. If this is the case, it is not surprising that USA300 would emerge as a cause of disease in pets as it increases in prevalence in the human population. Considering the rapid dissemination of USA300 in humans in the United States, particularly in California, where it is the predominant community-associated clone, finding USA300 in a household pet in that state is not unexpected.

Because indistinguishable isolates were collected from the owner and the infected cat, MRSA likely was transmitted between species in the household. However, while it is tempting to assume that the owner was the source of infection because of the owner's previous history of a soft tissue infection, this cannot be definitively determined on the basis of the timing of sampling in this case.

MRSA appears to be emerging as an important veterinary and zoonotic pathogen, and the epidemiology of MRSA in household pets may take a parallel course to that in humans. Ongoing MRSA surveillance in animals is required, including proper testing of specimens from clinically affected animals and surveillance for colonization. The potential for transmission of this clone between humans and pets should also be evaluated to clarify its epidemiology and to facilitate development of measures to reduce household transmission.

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References

- Ozaki K, Yamagami T, Nomura K, Haritani M, Tsutsumi Y, Narama I. Abscess-forming inflammatory granulation tissue with grampositive cocci and prominent eosinophil infiltration in cats: possible infection of methicillin-resistant *Staphylococcus*. Vet Pathol. 2003;40:283–7.
- Mulvey MR, Chui L, Ismail J, Louie L, Murphy C, Chang N, et al. Development of a Canadian standardized protocol for subtyping methicillin-resistant *Staphylococcus aureus* isolates using pulsed-field gel electrophoresis. J Clin Microbiol. 2001;39: 3481–5.
- Rankin S, Roberts S, O'Shea K, Maloney D, Lorenzo M, Benson CE. Panton Valentine leukocidin (PVL) toxin positive MRSA strains isolated from companion animals. Vet Microbiol. 2005;108:145–8.
- Roberts JC, Krueger RL, Peak KK, Veguilla W, Cannons AC, Amuso PT, et al. Community-associated methicillin-resistant *Staphylococcus aureus* epidemic clone USA300 in isolates from Florida and Washington. J Clin Microbiol. 2006;44: 225–6.
- Tenover FC, McDougal LK, Goering RV, Killgore G, Projan SJ, Patel JB, et al. Characterization of a strain of communityassociated methicillin-resistant *Staphylococcus aureus* widely disseminated in the United States. J Clin Microbiol. 2006; 44:108–18.
- Frazee BW, Salz TO, Lambert L, Perdreau-Remington F. Fatal community-associated methicillin-resistant *Staphylococcus aureus* pneumonia in an immunocompetent young adult. Ann Emerg Med. 2005;46:401–4.
- Loeffler A, Boag AK, Sung J, Lindsay JA, Guardabassi L, Dalsgaard A, et al. Prevalence of methicillin-resistant *Staphylococcus aureus* among staff and pets in a small animal referral hospital in the UK. J Antimicrob Chemother. 2005;56:692–7.
- Rich M, Roberts L. Methicillin-resistant *Staphylococcus aureus* isolates from com-panion animals. Vet Rec. 2004;154:310.

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- Weese JS, Dick H, Willey BM, McGeer A, Kreiswirth BN, Innis B, et al. Suspected transmission of methicillin-resistant *Staphylococcus aureus* between domestic pets and humans in veterinary clinics and in the household. Vet Microbiol. 2006;115: 148–55.
- van Duijkeren E, Wolfhagen MJHM, Box ATA, Heck MEOC, Wannet JB, Fluit AC. Human-to-dog transmission of methicillinresistant *Staphylococcus aureus*. Emerg Infect Dis. 2004;10:2235–6.

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Communityassociated Methicillinresistant Staphylococcus aureus, Colombia

To the Editor: Methicillin-resistant *Staphylococcus aureus* (MRSA) is an established nosocomial pathogen worldwide but more recently has emerged as a highly virulent organism in the community, particularly in the United States (1-3). In Latin America, community-associated MRSA (CA-MRSA) has only been described in the southern area of the continent (Uruguay and Brazil) (4,5). No reports from the Andean region are available. We describe 2 cases of CA-MRSA causing soft-tissue infections (1 severe) in Colombia.

The first case was in a 19-year-old man with a history of trauma to the left side of his body 1 week before admission after a fall. On admission, he complained of 2 days of fever, malaise, erythema and induration in the left hemithorax extending to the left thigh, and purulent secretion from an excoriation on the anterior aspect of the left thigh. He had no previous medical history. No previous hospitalizations or antimicrobial drug prescriptions were documented, nor did he report relatives with history of recent hospitalizations. Vital signs at admission were normal except for fever (39°C), and physical examination showed induration and erythema in the region of left hemithorax extending to the thigh, with an area of excoriation in the same thigh with purulent discharge. Laboratory evaluation showed a leukocyte count of 23.1×10^{9} /L (86% neutrophils with 2% band forms) and elevated C-reactive protein levels.

The patient was hospitalized. Because necrotizing fascitis was suspected, intravenous ampicillin-sulbactam (12 g per day) was started, and surgical evaluation was requested. The patient underwent surgical debridement of the left thigh, left hemiabdomen, and hemithorax, which confirmed the diagnosis of necrotizing fascitis. Intravenous vancomycin (1 g every 12 h) was added to the regimen, and the patient was transferred to the intensive care unit. After several surgical debridements, the patient underwent placement of cutaneous-muscle grafts. He was discharged from the hospital without complications after completing 14 days of antimicrobial agents.

The second case involved a 53year-old man with no history of previous hospitalizations who reported to the emergency department with a 3-day history of fever, pain, swelling, and warm sensation on the posterior aspect of the left thigh. A diagnosis of cellulitis was made, and cephalexin (500 mg every 6 h) and gentamicin (160 mg intramuscularly every 24 h) were administered for 7 days without improvement. He returned to the hospital with worsening symptoms, an area of induration of 4×4 cm in the thigh, and purulent discharge. Drainage of the lesion was performed, and oral trimethoprim and sulfamethoxazole (160 and 800 mg, respectively, every 12 h) was started. His clinical signs and symptoms completely resolved after 7 days of therapy.

Tissue culture from secretions from both patients showed gram-positive cocci in clusters on the Gram stain, and subsequent cultures yielded MRSA. Species identification and presence of the mecA gene were confirmed by PCR, as described previously (6). MICs were determined by using the agar diffusion test, according to Clinical and Laboratory Standards Institute recommendations (7). Both organisms were susceptible to vancomycin, teicoplanin, chloramphenicol, linezolid, ciprofloxacin, gentamicin, and rifampin. The isolate from the second patient was resistant to erythromycin and susceptible to clindamycin, exhibited the M phenotype on the double-disk diffusion assay (D test), and harbored the msrA gene, encoding an efflux pump (8). In contrast, the first isolate was susceptible to both erythromycin and clindamycin and resistant to tetracycline (MIC >64 μ g/mL). Because infections caused by CA-MRSA isolated elsewhere are associated with the presence of the *lukF* gene encoding the Panton-Valentine leukocidin toxin and the staphylococcal chromosome cassette mec (SCCmec) type IV, the presence of both was evaluated by PCR, as described previously (9). Both isolates were positive for lukF and harbored the SCCmec type IV.

The molecular epidemiology of healthcare-related MRSA in Colombia has changed during the past 3 years (10), but no reports of CA-MRSA had emerged. We believe these to be the first reports of CA-MRSA in Colombia with similar characteristics to those reported elsewhere. No risk factors associated with healthcareassociated MRSA were found in either of these patients, and the patients were not epidemiologically