

Table 1. Sample Report for Heart-Failure Performance Measure, with Exceptions Delineated.*

Percentage of patients with diagnosis of heart failure and LVSD for whom ACE-inhibitor or ARB therapy prescribed	Patients with heart failure and LVSD — $N_{\text{total}} = \sum n_1 \dots n_4$ ACE-inhibitor or ARB therapy prescribed — n_1 Therapy not prescribed for medical reason — n_2 Therapy not prescribed for patient reason — n_3 Therapy not prescribed for system reason — NA Therapy not prescribed, reason not specified — n_4 Patients without valid exception, therapy should be prescribed — $N_{\text{eligible}} = N_{\text{total}} - (n_2 + n_3)$ Performance rate = $n_1 / N_{\text{eligible}}$ Exception rate = $n_1 / N_{\text{total}}, n_3 / N_{\text{total}}$
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* The sample report is based on the heart-failure performance measure of the American College of Cardiology/American Heart Association/Physician Consortium for Performance Improvement.² ACE denotes angiotensin-converting enzyme, ARB angiotensin-receptor blocker, LVSD left ventricular systolic dysfunction, and NA not applicable.

rized, and reported to delineate appropriate and inappropriate variations in care. Exceptions flag important clinical conditions (e.g., allergies) and patient preferences; they permit transparency by accounting for every patient (Table 1). In the United States, the Physician Consortium for Performance Improvement includes categories of exceptions and codes for reporting them in its performance measures.³ The Physician Quality Reporting Initiative provides physicians with reports that include rates of categorical exceptions.⁴ Researchers are exploring exception data in electronic health records.^{5,6} We are learning how to use these data to improve patient care and encourage continued use of exception reporting.

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MRSA USA300 Clone and VREF — A U.S.–Colombian Connection?

TO THE EDITOR: In the United States, the dissemination of a major clone of community-associated methicillin-resistant *Staphylococcus aureus* (MRSA), designated USA300, and outbreaks of vancomycin-resistant *Enterococcus faecalis* (VREF) have been described.^{1,2} Community-associated MRSA infections emerged in Colombia in 2005,³ and a total of 15 community-associated MRSA infections were documented in four cities in 2006 and 2007. All the patients presented with severe skin and soft-tissue infections, which were often complicated

by necrotizing fasciitis, bacteremia, paraspinal abscess, arthritis, or meningitis, with a mortality rate of 20%. The first known Colombian VREF isolate was recovered in a hospital in Bogotá in 2001. Since then, additional isolates have been identified from 50 patients in seven Bogotá hospitals.

The Colombian MRSA isolates were susceptible to most antistaphylococcal antibiotics, although 40% were resistant to tetracycline. All isolates had staphylococcal chromosomal cassette *mec* (SCC*mec*) type IV, the Pantón–Valentine leukocici-

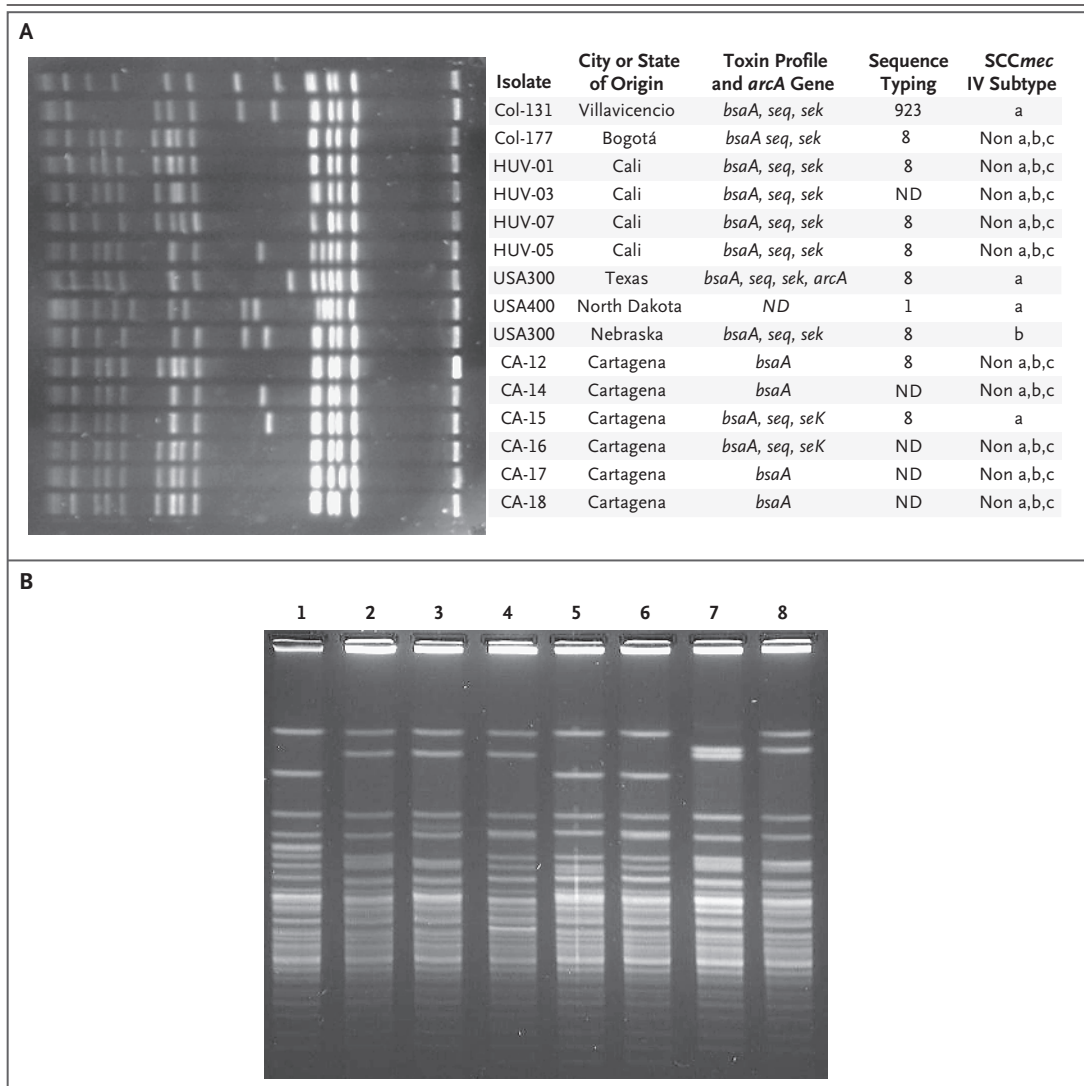


Figure 1. Representative Isolates of Community-Associated Methicillin-Resistant *Staphylococcus aureus* (MRSA) from Colombia and the United States.

Panel A shows the results of pulsed-field gel electrophoresis (PFGE) after digestion with *Sma*I enzyme, the city or state of origin, the toxin profile (including the presence of *arcA* as a marker of the ACME [arginine catabolic mobile element] island), multilocus sequence typing, and the presence or absence of staphylococcal chromosomal cassette *mec* (SCC*mec*) IV and subtypes (a, b, and c) of representative isolates of community-associated MRSA from Colombia. The strains from Texas (USA300, SCC*mec* IVa), North Dakota (USA400), and Nebraska (USA300, SCC*mec* IVb) were used as controls for typing experiments. The presence of *arcA* has been associated with USA300 isolates carrying the SCC*mec* type IVa cassette but not others, a finding that is consistent with the possibility that ST8 community-associated MRSA in Colombia acquired the SCC*mec* DNA independently from the U.S. isolates. ST923 is a single-nucleotide variant of ST8 in the *yclL* gene. ND denotes not determined. Panel B shows PFGE results with the use of *Apal* enzyme of representative isolates of vancomycin-resistant *Enterococcus faecalis* (VREF) ST2 from seven hospitals in Bogotá from 2001 to 2006 and isolate TX2486, the index isolate of strain HV1 (ST2) recovered from a Houston hospital in 1994, as follows: lane 1, TX2486; lanes 2 and 3, ERV-25 and ERV-31, respectively, which are representatives of the first Colombian VREF isolates recovered in 2001 (VREF isolates from 2002 and 2003 had a PFGE pattern that was identical to that of isolate ERV-31 and are not shown); lanes 4, 5, and 6, isolates ERV-62, ERV-63, and ERV-65, respectively, recovered in 2004; lane 7, ERV-81, isolated in 2005; and lane 8, ERV-116, recovered in 2006.

din genes, and at least one of the toxins associated with USA300⁴ but did not have the *arcA* gene as a marker of the ACME (arginine catabolic mobile element) island. Pulsed-field gel electrophoresis and multilocus sequence typing revealed that all but one of the clinical isolates were ST8 and

clonally related to USA300. The remaining isolate was a single-locus variant of ST8 (ST923) (Fig. 1A). The majority of isolates carried SCC mec subtypes other than IVa, which suggests that a similar lineage of virulent, ST8, methicillin-susceptible *S. aureus* that independently acquired various SCC mec subtypes existed in both Colombia and the United States. To our knowledge, this is the first documentation of the USA300 community-associated MRSA lineage as the predominant (and exclusive) clone in a country other than the United States. No community-associated MRSA isolates belonging to a different clonal cluster have been documented by multilocus sequence typing in Colombia so far.

Pulsed-field gel electrophoresis also indicated that a single clone of *vanB* VREF has been disseminated in Bogotá (Fig. 1B). This clone is genetically related to an ST2 outbreak strain (HV1) of VREF described in Houston in 1994.⁵ In addition, the allelic profile of the antigenic or resistance-associated genes *ace*, *sala*, and *lsa* (encoding an adhesin, a cell-wall antigen, and quinupristin-dalfopristin resistance, respectively) was identical in the two strains. Furthermore, the Houston and Colombian isolates had the same pathogenicity island profile, which has been suggested as an epidemiologic marker of more virulent clones of *E. faecalis*.⁵ To our knowledge, the only two epidemic strains of ST2 *vanB*-type *E. faecalis* that have been described are the Houston and Bogotá strains. Our findings suggest a close epidemiologic relationship between Colombia and the United States in these two pathogenic and resistant species.

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