

Global Spread of the Community-Associated Methicillin-Resistant *Staphylococcus aureus* USA300 Latin American Variant

TO THE EDITOR—We read with interest the recent article by von Dach et al [1] on the

comparative genomics of community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) in Switzerland. Von Dach et al nicely highlight what remains one of the great biological and epidemiological conundrums regarding

the spread of CA-MRSA, which is that the same strain can cause an epidemic in one geographic location and fail to spread in another despite multiple introductions and a high volume of travel between locations. The failure of the

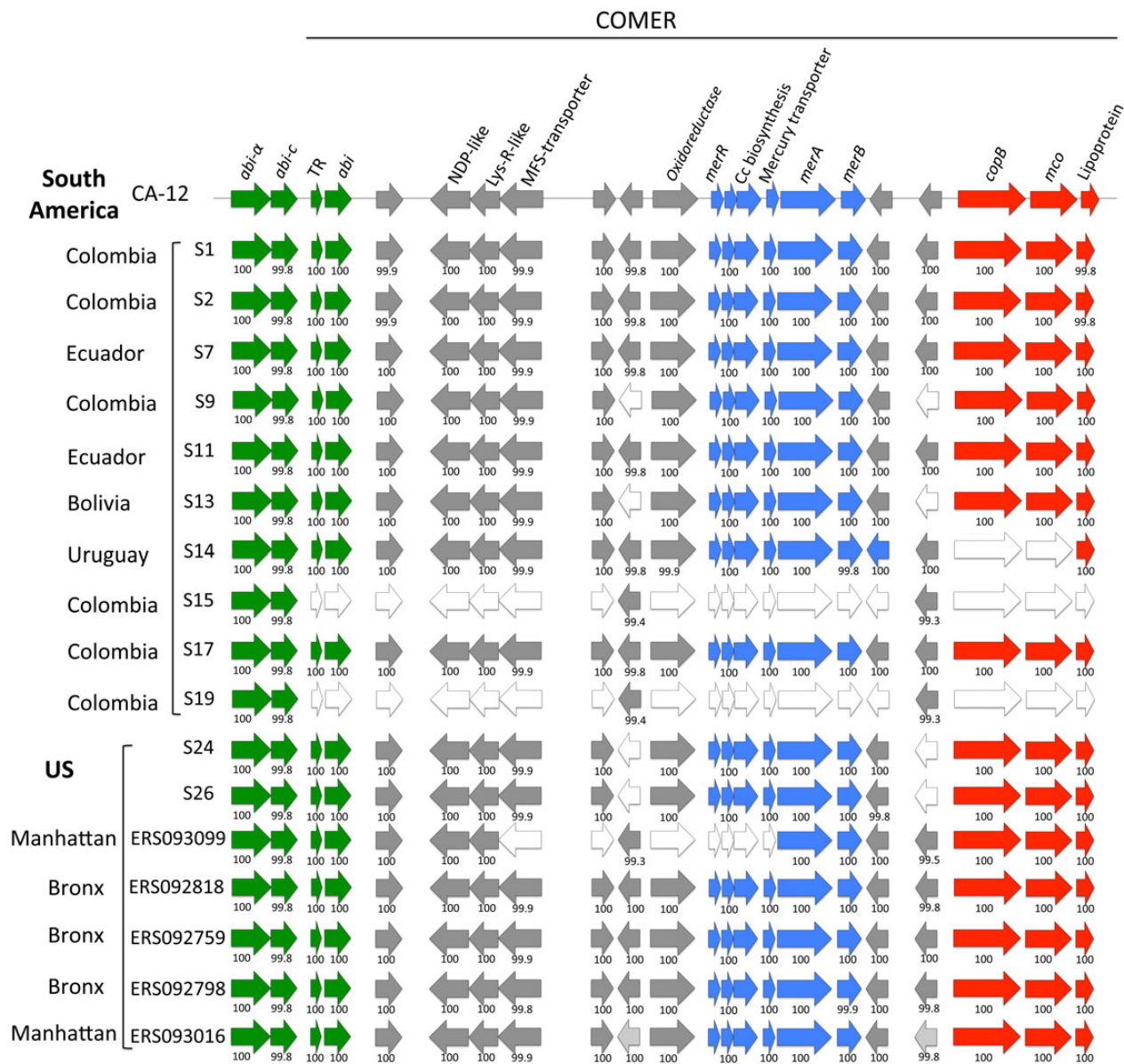


Figure 1. Presence of the coding sequences belonging to the copper and mercury resistance factor (COMER) region in methicillin-resistant *Staphylococcus aureus* (MRSA) genomes. Schematic representation for the presence of coding sequences of the copper (red arrows) and mercury (blue arrows) resistance factors belonging to the COMER region and predicted abortive phage genes (green) on the MRSA genomes reported by von Dach et al, using CA12 as a reference genome (accession number CP007672). Additional coding sequences present in the DNA region of CA12 are represented in dark gray arrows. White arrows represent the undetected coding sequences. The reported geographical association of the strains is indicated by the brackets on the left. The percentage of nucleotide identity is indicated below each arrow. All results had a coverage percentage of >99% and an *e* value of 0 with the exception of 2 coding sequences, represented with light gray arrows, in strain ERS093016, with a coverage of 89%. Abbreviations: Cc, cytochrome C; TC, transcriptional regulator.

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predominant CA-MRSA strain in the United States (USA300) to spread in Europe was also recently documented, using whole-genome analysis, by Glaser et al [2] and Toleman et al [3] in France and the United Kingdom, respectively. Both studies demonstrated that USA300 genomes isolated in Europe are phylogenetically interspersed with US isolates, suggesting multiple introductions and multiple failures to establish endemic transmission.

Of particular interest in the article by von Dach et al was an apparent link of some CA-MRSA isolates to recent travel or prior residence in South America. Specifically, several isolates were associated with the northern region of South America, specifically Colombia and Ecuador. The genomes of these isolates formed a robust clade, were susceptible to fluoroquinolones and erythromycin, possessed a variant of the methicillin resistance cassette (IVc), and lacked the arginine catabolic mobile element (ACME) that is commonly found in USA300 strains from North America. These characteristics are all features of the so-called Latin American variant (LV) of USA300 that was first isolated in 2005 [4, 5]. We recently reported that a specific clade of USA300-LV constitutes a parallel USA300 epidemic affecting predominantly northern South American countries [6]. This South American epidemic clade, the USA300-SAE lineage, is characterized by a unique genomic region encoding copper and mercury resistance factors (COMERs).

Because of the strong similarities of USA300-SAE to the Swiss isolates associated with South America, we were surprised that von Dach et al did not find evidence of the COMER region in their whole-genome sequences. Using a basic BLAST strategy on the publically available genomes from the study by von Dach et al, we were able to identify the COMER region in 10 of 12 genomes from the ACME-negative isolates (Figure 1). The only 2 genomes that did not have COMERs (labeled S15 and S19) were isolates from a single family. The

COMER locus was also present in all the other publically available genomes that grouped in the ACME-negative clade in the article by von Dach et al. These genomes, in fact, were isolated in northern Manhattan and the Bronx, which both have large communities of individuals of Latin American origin [7]. These findings show that the USA300-SAE clade has now been isolated in both North and South America, as well as in Europe, and the detection of the COMER locus could serve as a tool to monitor its spread. This finding may also support the idea that the *copB* locus (the only region found both in ACME and COMER) may be an important contributor to the enhanced fitness and success of this lineage. These findings also highlight the importance of detailed whole-genome characterization of epidemic strains that could help explain evolutionary and phylogeographic determinants of virulence and transmission.

Notes

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