Lethal Necrotizing Pneumonia Caused by an ST398 Staphylococcus aureus Strain

To the Editor: The prevalent colonization of livestock with methicillin-resistant Staphylococcus aureus (MRSA) sequence type (ST) 398 in many countries is a cause for consternation. However, understanding of the emergence of these organisms and their public health implications is embryonic. The perceptions that all MRSA found in livestock are of ST398 lineage or that livestock are the only reservoir requires systematic investigation of livestock workers as yet have been minimal. Understanding the public health implications of ST398 oversimplify a complex epidemiology, and therefore, prudence is required when attributing human infections with S. aureus ST398 to livestock reservoirs. The fatal infection of a young girl with ST398 methicillin-susceptible S. aureus (MSSA) is tragic (1). However, the conclusion by the authors that “the spread of S. aureus ST398 among livestock is a matter of increasing concern because strains of this sequence type were able to acquire PVL [Panton-Valentine leukocidin] genes” is misleading.

The authors report no history of livestock exposure and the spa type reported (t571) is relatively rare among livestock isolates (2,3). The isolate from the fatal case was tetracycline-susceptible and positive for PVL toxin, while livestock ST398 isolates have been almost uniformly tetracycline resistant and PVL negative. Notably, spa type t571 ST398 MSSA was detected in 9 families from the Dominican Republic living in Manhattan, New York, without contact with livestock (4). Furthermore, t571 was the only spa type of MSSA identified in a study in the Netherlands of ST398 isolates, including 3 independent cases of nosocomial bacteremia in Rotterdam with no apparent livestock contact (5). spa type t571 was the predominant (11%) MSSA type in patients at a Beijing, China, hospital (6). More recently, a study of t571 MSSA strains from cases of bloodstream infections in France determined that the isolates differed from pig-born strains and shared similarities with strains from humans in China and virulent USA300 strains (7). These observations concur with a hypothesis that ST398 strains of diverse genotype and geographic origin may also be epidemiologically distinct (8), and livestock contact is a notably inconsistent feature of invasive ST398 infections (5,7–10).

The possibility that variants of the ST398 lineage may persist in human populations without livestock contact should not be indiscriminately attributed to livestock, particularly if isolates are genotypically dissimilar to those occurring commonly in animals.

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References


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In Response: We thank Davies et al. (1) for their interest in our report of a lethal case of necrotizing pneumonia caused by a sequence type (ST) 398 Staphylococcus aureus strain (2). We fully agree with their request that ST398 S. aureus infections not be systematically attributed to contact with livestock. They correctly pointed out that several characteristics of the incriminated strain, including methicillin and tetracycline susceptibility, spa type, and the presence of genes encoding the Panton-Valentine leukocidin (PVL), differed from the usual genetic features of strains isolated from livestock (3,4). However, we did not state or suggest in our report that the case originated from livestock contact. Our aim in reporting this case was to warn that S. aureus of the ST398 lineage, regardless of its host specificity, is able to acquire PVL genes and provoke severe PVL-related infection in humans. This observation adds support to the need for controlling the increasing animal reservoir of ST398 methicillin-resistant S. aureus (MRSA). Indeed, the recent whole-genome analysis of an ST398 strain by Schijffelen et al. (5) highlighted several specific features of the ST398 genetic background, including the absence of a type I restriction and modification system. Such features have been proposed to promote horizontal gene transfer and the uptake of mobile genetic elements such as the plasmid-encoded PVL genes (5). Although phage-mediated dissemination of PVL genes into MRSA lineages does not seem to be the prevalent pathway leading to the emergence of highly epidemic PVL-positive MRSA (6), this eventuality should not be dismissed with respect to the ST398 lineage, which possesses all the required features to become the next MRSA “superbug” (7).

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References

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Extended-Spectrum β-Lactamase–producing Escherichia coli in Neonatal Care Unit

To the Editor: Tschudin-Sutter et al. provide convincing evidence of transfer of an extended-spectrum β-lactamase–producing Escherichia coli strain from a mother to her vaginally delivered twins, then from the neonates to a health care worker and other neonates in a neonatal care unit (1). This finding advances our understanding of how extended-spectrum β-lactamase–positive (and, by extension, other antimicrobial drug–resistant or virulent strains) E. coli can spread within the community. However, the authors’ use of the term infection for the asymptomatic colonization that was observed, including in the mother (who had asymptomatic bacteriuria), is