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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 phage-encoded PVL genes (*5*). Although phage-mediated dissemination of PVL genes into MRSA lineages does not seem to be the preeminent 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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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

potentially misleading. This term could perpetuate a line of thinking that is all too common among clinicians and leads to unnecessary antimicrobial drug use, thereby ironically aggravating the problem of antimicrobial drug resistance.

Although the first paragraph of their report implicitly acknowledges the distinction between infection and colonization, the rest of the report (including the abstract) uses the terms infection or infected interchangeably with colonization or colonized. Examples include "Subsequently, infection spread by healthcare worker contact with other neonates," "a healthcare worker also was infected," and "a urinary tract infection developed...."

One wonders why, in the absence of genitourinary symptoms, the (postpartum) mother's urine was cultured and why the positive culture prompted antimicrobial drug therapy. This seeming misinterpretation by the mother's providers of what probably was a harmless colonization state as representing acute disease, and their all too typical response (i.e., antimicrobial drug therapy), are to be discouraged (*2*). More cautious use of terminology, to emphasize the distinction between colonization and infection (which have radically different therapeutic implications), may help refine clinicians' thinking and practice in this regard, thereby promoting improved antimicrobial drug stewardship and slowing the antimicrobial drug resistance epidemic.

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In Response: We thank James Johnson for the issue that he has raised in his letter (*1*). We agree that distinction of the terms colonization and infection is crucial to prevent misinterpretation of clinical findings and subsequently unnecessary antimicrobial drug use. The outbreak occurred in the hospital; therefore, definitions of nosocomial infections were used throughout the article (*2*).

Nosocomial urinary tract infection is defined by the Centers for Disease Control and Prevention as asymptomatic bacteriuria or symptomatic infection (urinary tract infection–symptomatic urinary tract infection; www.cdc.gov/nhsn/PDFs/pscManual/17pscNosInfDef_current.pdf). (*3*,*4*). Therefore, the term nosocomial urinary tract infection in our report is correct. However, we agree with the author that the term asymptomatic bacteriuria is less than optimal and it was removed when we submitted our report. We agree that the term infection is misleading for describing spread to health care workers and that colonization should have been used. However, the article clearly states that invasive infection did not occur in any of the neonates or health care workers found to be colonized. In addition, the focus of the article was to describe the mode of transmission rather than the distinction between colonization and infection.

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