SCCmec Typing in Methicillin-Resistant Staphylococcus aureus Strains of Animal Origin

To the Editor: Van Loo et al. described the presence of staphylococcal cassette chromosome mec (SCCmec) type III in some methicillin-resistant Staphylococcus aureus sequence type (ST) 398 isolates related to pig farming (1). SCCmec types are based on the allotype of ccr genes and the mec gene complex. Class A mec has intact mecI/R regulator genes. Type III SCCmec has type 3 ccr genes and class A mec complex, whereas type V SCCmec contains ccrC and class C mec (2,3). The authors typed SCCmec for the isolates by the method of Zhang et al. (4), in which type III is defined by amplification of a 280-bp fragment located in the junkyard region. This fragment is found in SCCmer that is associated with SCCmec type III.

We have typed SCCmec of the same 4 isolates that were reported to be SCCmec type III positive by using the primer sets defined by Ito et al. (2,3) and Lim et al. (5) for ccr types 1–3 and ccrC and 4 additional primers developed at our institute (Table) in single PCRs. All ST398 isolates were PCR negative when primers specific for SCCmec type III were used, but PCR positive with the ccrC-specific primers. DNA sequencing confirmed the product as ccrC. Further, the isolates did not have a class A mec complex, a requisite for SCCmec type III, because a mecI-specific PCR was negative for these isolates. In addition, Southern hybridizations with digoxigenin-dUTP-labeled PCR fragments obtained with our primer pair specific for ccr3 and primers for ccrC (3) showed no hybridization with the ccrA/B3 probe (except for the positive control). All of the ST398 isolates hybridized with the ccrC-specific probe.

We conclude that on the basis of generally accepted definitions SCCmec type V is present in these ST398 pig-farming–related isolates, not SCCmec type III. Therefore, researchers should be aware that some typing methods may lead to inadequate results.

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References

1. van Loo I, Huijsdens X, Tiemersma E, de Neeling A, van de Sande-Bruinsma N, van Loo M, Tiensasitorn C, et al. Emergence of methicillin-resistant Staphylococcus aureus in many published papers. Fluit et al. questioned whether the SCCmec type III isolates were correctly typed (1). To prove that the results of typing these 4 isolates were incorrect, these researchers performed several different SCCmec PCRs, including a PCR with

<table>
<thead>
<tr>
<th>Genes</th>
<th>Primer name</th>
<th>Primer sequence (5' → 3')</th>
</tr>
</thead>
<tbody>
<tr>
<td>ccrA/B1</td>
<td>ccr1B-for</td>
<td>CTT TCA CGA TAG ACA CAG</td>
</tr>
<tr>
<td>ccrA/B1</td>
<td>ccr1B-rev</td>
<td>TAA AAG AAC TTA ATA GCC GTT AAA TTG G</td>
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<td>ccr2B-for</td>
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</tr>
<tr>
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<td>ccr2B-rev</td>
<td>CTA TAA CCT TCT GTG CTT TGC A</td>
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<td>ccr3B-for</td>
<td>TTC GTA ATA AGA AGC AAC TTC AC</td>
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<td>CTG CAC CAC ATT TTG GCC AC</td>
</tr>
</tbody>
</table>

*SCCmec, staphylococcal cassette chromosome mec; MRSA, methicillin-resistant Staphylococcus aureus; ST, sequence type.
primers they developed themselves. In addition, Southern hybridization was done. The results showed that SCCmec III ST398 MRSA isolates should be typed as SCCmec type V. In this conclusion we agree with the authors. It seems clear that Zhang’s method incorrectly identified 4 of the animal-related ST398 isolates as SCCmec type III instead of SCCmec type V. Whether all ST398 MRSA are SCCmec type IV or V remains unclear. Recently, an article by Nemati et al. was published in which ST398 MRSA was also typed as SCCmec III (3). However, in that study the SCCmec typing method of Zhang was also used.

In conclusion, the choice of SCCmec typing method is directly related to obtaining accurate SCCmec results for ST398 isolates. To date, almost all animal-related ST398 MRSA isolates are SCCmec types IV and V.

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School Closure to Reduce Influenza Transmission

To the Editor: Cowling et al. reported on the effects of school closure in Hong Kong, People’s Republic of China, during March 2008 in response to influenza-related deaths of children (1). The influenza epidemic started in January 2008 and peaked in late February, but the 2-week school closure did not begin until March 12. Consequently, the school-based epidemic was on the decline by the time the health officials closed schools. Other studies have suggested that early school closures can help reduce influenza illness in the community and among school children, especially during a pandemic (2–6). However, surveillance systems that rely on school absenteeism or deaths would likely provide information too late during the outbreak for school closure to effectively reduce influenza transmission.

The Centers for Disease Control and Prevention (CDC) has recommended early closure of schools as a community mitigation measure in the event of a severe pandemic (7). Specifically, CDC recommends rapidly initiating activities such as advising sick persons to stay home, dismissing children from schools, closing childcare facilities, and initiating further social distancing measures within a state or a community at the beginning of the upslope of a pandemic wave (acceleration interval), i.e., when cases are initially identified and community transmission begins to occur (8). We concur with the authors that the 2007–08 influenza season was already waning by the time the decision was made to close schools (deceleration interval).

School closure used as a single pandemic control measure is predicted to be less effective than early, concurrent use of multiple measures. Socially disruptive measures like early school closure and keeping children from congregating in the community would likely reduce community transmission of pandemic disease, but would also create secondary challenges (9,10). Therefore, to ensure maximal benefit for reducing disease transmission, interventions should be implemented early and concomitantly with other nonpharmaceutical and pharmaceutical measures, accompanied by public education, and used judiciously based on pandemic severity.

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References