Table. Clinical and biological characteristics of mothers and children with congenital syphilis, Réunion Island, 2010*

Year of diagnosis	Mother					Child	
	Age, y	Time of syphilis screening	Duration of treatment, d	Presence and stage of disease	Positive serologic titer test results	Gestation, wk	Clinical signs
2008	16	≥13 wk gestation	5 d BD	Probable secondary syphilis at first trimester	TPHA, VDRL, FTA-ABS	34	None
2008	25	Unknown	2 d AD	NA	TPHA, VDRL, FTA-ABS	34	None
2008	16	AD	14 d AD	Primary syphilis at third trimester	TPHA, VDRL, FTA-ABS	38	None
2009	26	≥13 wk gestation	17 d BD	NA	TPHA, VDRL, FTA-ABS	31	Present
2009	18	≥13 wk gestation	2 d BD	NA	TPHA, VDRL	32	Present
2009	22	≥13 wk gestation	1 d BD	NA	TPHA, VDRL	32	Present
2009	37	After delivery	1 d AD	NA	TPHA, VDRL	38	None

*BD, before delivery; TPHA, *Treponema pallidum* hemagglutination assay; VDRL, Venereal Disease Research Laboratory; FTA-ABS, fluorescent treponemal antibody absorption; AD, after delivery; NA, not applicable.

Our report highlights an alarming situation in Réunion Island. Reemergence of CS after the increase of early syphilis in women of childbearing age must be considered as a public health alert, especially in countries where health care is supposed to be efficient. CS is easy to prevent with adequate screening of the mother and good follow-up of seropositive parturients.

The results of our study permitted reinforcement of the syphilis mass screening and awareness campaign regarding this sexually transmitted infection in the general population and medical corps. Although it is unrealistic to expect complete eradication of primary and secondary syphilis in communities, a minimal increase of CS rates should trigger reinforcement of these prevention policies.

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Reduced Susceptibility to Vancomycin in *Staphylococcus aureus*

To the Editor: I read with interest the article by Aguado et al. (*1*). I congratulate the authors for their high-quality research and would like to make 2 brief comments.

My first point regards the mechanism by which methicillin-susceptible *Staphylococcus aureus* (MSSA) with reduced susceptibility to vancomycin would also acquire decreased susceptibility to β-lactams. The authors "hypothesize that certain structural modifications might also occur in the cell wall of strains with high vancomycin MIC, including a thicker cell wall as it has been described in MRSA [methicillin-resistant *S. aureus*]." In addition to cell wall thickening, possible

mechanisms could include reduction in autolysis (*2*,*3*) and in the cell wall content of penicillin binding protein 4 (PBP4) (*3*). A study of MSSA isolates has shown a reduction in autolysis and in the bactericidal activity of oxacillin after development of intermediate vancomycin susceptibility (*2*). Lower content of PBP4 and decreased autolysis were reported in MRSA isolates after reduced susceptibility to vancomycin developed after exposure to this antimicrobial drug (*3*). Decreased PBP4 has been associated with reduced methicillin susceptibility in *S. aureus* (*4*).

Second, knowing whether the authors had the exact vancomycin MIC of the isolates by broth microdilution to compare with the Etest results would be interesting. In the article, they only state that "all 99 MSSA strains were susceptible to vancomycin (MIC ≤2 μg/mL) by the broth microdilution method." Although difficult to compare (because Etest dilution progression is arithmetic and broth microdilution is performed with a geometric dilution), some authors have found substantial differences between the vancomycin MIC results given by these 2 methods (*5*). These differences could have major laboratory and clinical implications.

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In Response: We appreciate the comments by M. J. Mimica (*1*). We agree that mechanisms other than increased cell wall thickness, such as decreased autolysis, metabolic changes, and reduction in the cell wall content of penicillin binding protein 4, characterize *Staphylococcus aureus* isolates, and they could explain this reduced susceptibility not only to vancomycin but also to β-lactam antimicrobial drugs. A recently published report by Holmes et al. (*2*) confirms this hypothesis. That study showed that *S. aureus* vancomycin MIC ≥1.5 μg/mL, determined by Etest, was associated with a significantly higher death rate for patients with methicillin-susceptible *S. aureus* bacteremia irrespective of the type of antimicrobial drug used.

Unfortunately, the vancomycin MIC determined by broth microdilution could not be compared with the Etest results because all our isolates had a MIC by broth microdilution that oscillated between 0.5 and 1.0 μg/mL. No isolate had a MIC of 2 μg/mL. In the study by Holmes et al., vancomycin MICs were higher by Etest than by broth microdilution (*1*); however, all isolates were considered vancomycin susceptible by Clinical and Laboratory Standards Institute broth microdilution methods. The authors pointed out that they also found that increased mortality rate was associated with increased broth microdilution MIC (data not shown), although the trend was not so prominent (*2*).

A prospective study would be required to specifically investigate the relationship between vancomycin MIC and outcome of *S. aureus* bacteremia. Our group is performing such a prospective study that we hope will enable us to shed light on this relevant topic.

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