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Trends in Methicillin-Resistant *Staphylococcus aureus* Anovaginal Colonization in Pregnant Women in 2005 versus 2009▽

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In 2005, the prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) anovaginal colonization in pregnant women at our center (Columbia University Medical Center) was 0.5%, and MRSA-colonized women were less likely to carry group B streptococcus (GBS). In this study, our objectives were to identify changing trends in the prevalence of MRSA and methicillin-susceptible *S. aureus* (MSSA) anovaginal colonization in pregnant women, to assess the association between MRSA and GBS colonization, and to characterize the MRSA strains. From February to July 2009, Lim broths from GBS surveillance samples were cultured for *S. aureus*. MRSA strains were identified by resistance to cefoxitin and characterized by MicroScan, staphylococcal cassette chromosome *mec* (SCC*mec*) typing, pulsed-field gel electrophoresis (PFGE), *spa* typing, and Panton-Valentine leukocidin PCR. A total of 2,921 specimens from different patients were analyzed. The prevalences of MSSA, MRSA, and GBS colonization were 11.8%, 0.6% and 23.3%, respectively. GBS colonization was associated with *S. aureus* colonization (odds ratio [OR], 1.9; 95% confidence interval [95% CI], 1.5 to 2.4). The frequencies of GBS colonization were similar in MRSA-positive (34.2%) versus MRSA-negative patients (21.8%) ($P = 0.4$). All MRSA isolates from 2009 and 13/14 isolates from 2005 were SCC*mec* type IV or V, consistent with community-associated MRSA; 12/18 (2009) and 0/14 (2005) isolates were the USA300 clone. Levofloxacin resistance increased from 14.3% (2005) to 55.6% (2009) ($P = 0.028$). In conclusion, the prevalence of MRSA anovaginal colonization in pregnant women in New York City, NY, remained stable from 2005 to 2009, and USA300 emerged as the predominant clone with a significant increase in levofloxacin-resistant isolates.

The rapid spread of community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) across the United States has been associated primarily with the dissemination of a specific clone that has the pulsed-field gel electrophoresis (PFGE) pattern termed USA300 (20). CA-MRSA can cause infections in pregnant and postpartum women and outbreaks in newborn nurseries and neonatal intensive care units (NICUs) (5, 22, 26, 32, 35, 36, 38). CA-MRSA strains, including USA300, have replaced health care-associated (HA)-MRSA as the predominant strains isolated from infants in some NICUs (6, 35). CA-MRSA infections appear to be increasing in otherwise healthy neonates in the nursery (18, 38) who may acquire *S. aureus* from health care workers or from their mothers and other family members (19, 23, 25, 28).

Vertical transmission from mothers to infants at delivery has also been proposed as a possible mechanism of acquisition of CA-MRSA (1, 2, 7, 28). *S. aureus* has been reported to colonize the vagina in 4 to 22% of pregnant women (2, 4, 9, 13, 14). In 2005, following an outbreak of USA300 in postpartum women at our medical center (Columbia University Medical Center), we determined that the prevalence of methicillin-susceptible *S. aureus* (MSSA) anovaginal colonization was 16.6% and the prevalence of MRSA colonization was 0.5% (9). Overall, 93% of MRSA strains were staphylococcal cassette chromosome *mec* (SCC*mec*) type IV or V, which is consistent with CA-MRSA (9). More recent studies conducted in other locales have suggested that the prevalence of anovaginal colonization with MRSA is increasing, with reported rates ranging from 3.5 to 10.4% (2, 13). The association of MRSA colonization with group B streptococcus (GBS) colonization is less clear; some reports have shown an increased rate of MRSA colonization in GBS-positive women (2), while others have shown a decreased rate (8, 32).

Reports suggesting an increasing prevalence of MRSA among pregnant women, coupled with the recent emergence of USA300 in our NICU (6), led us to question whether the epidemiology of MRSA colonization was also changing in pregnant women in our population in New York City, NY. The objectives of this study were to determine the current prevalence of MRSA and MSSA colonization in pregnant women, to assess the association between MRSA and GBS colonization, and to use molecular methods to characterize MRSA strains from the current study and compare those with strains from our 2005 study.

MATERIALS AND METHODS

Study design. A prospective surveillance study of *S. aureus* and MRSA colonization was conducted from February to July 2009 in pregnant women receiving care at Columbia University Medical Center (CUMC), NewYork-Presbyterian Hospital, who underwent routine screening for GBS colonization, according to Centers for Disease Control and Prevention (CDC) recommendations (34). The clinical microbiology laboratory at CUMC processes approximately 6,000 GBS

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screening cultures annually, received from the prenatal clinics at CUMC, including those at a community hospital and several off-site clinics affiliated with CUMC. The research protocol was approved by the Institutional Review Board at CUMC, and a waiver of consent was granted.

Culture methods. GBS screening cultures were inoculated in selective broth medium (BBL Lim broth; BD Diagnostics, Sparks, MD) and processed according to CDC guidelines (34). GBS culture results were obtained from the microbiology laboratory database.

The residual Lim broths from GBS screening cultures were cultured for *S. aureus*. Inocula of approximately 10 μl of culture were streaked onto a selective differential chromogenic agar plate approved for isolation of *S. aureus* from clinical specimens (BBL CHROMagar Staph aureus; BD Diagnostics, Sparks, MD) (3). *S. aureus* appears on CHROMagar as mauve colonies. After a 20- to 24-hour incubation period, colonies were picked from each individual CHROMagar plate and restreaked to a blood agar plate for further identification. *S. aureus* was confirmed by latex agglutination (Staphaurex; Remel Europe, Dartford, United Kingdom), and MRSA strains were identified by the MicroScan WalkAway instrument (Siemens Healthcare Diagnostics, Deerfield, IL) and the cefoxitin disk diffusion screen (11). Formal antimicrobial susceptibility testing was performed on all MRSA strains using the MicroScan system. MICs were determined for a standard panel of antibiotics and reported as susceptible, intermediate, or resistant following current Clinical and Laboratory Standards Institute recommendations (10, 11). The nonsusceptible results were confirmed by the Etest method (bioMérieux, Marcy l'Etoile, France). Erythromycin-inducible clindamycin resistance was assessed with a double-disk diffusion test (D-test) according to current guidelines (11).

Molecular typing. MRSA strains were further characterized by multiplex PCR of the SCC*mec* gene, as described elsewhere (27, 39).

(i) PFGE. MRSA strains isolated in 2005 and 2009 were genotyped by pulsed-field gel electrophoresis (PFGE) with the restriction enzyme SmaI using the GenePath system (Bio-Rad, Hercules, CA) as previously described (31). Relatedness of strains based on PFGE patterns was calculated using the Dice coefficient (Bionumerics; Applied Maths, Austin, TX) with a 1.5% molecular weight position tolerance. Strains were considered clonal if they demonstrated 85% or greater similarity. Strains demonstrating less than 85% similarity by PFGE were defined as closely related, possibly related or unrelated according to established criteria (37).

(ii) *spa* typing. Isolates were characterized by comparative DNA analysis of the variable number of tandem repeats region of the *S. aureus* protein A (*spa*) gene similar to a previously described method (21), but using M13-tagged primers, either 5′-GTA AAA CGA CGG CCA GTG AGA AGC TAA AAA GCT AAA CGA TGC TCA AG-3′ (product size, 200 to 600 bp) or 5′-TGT AAA ACG ACG GCC AGT GCC AAA GCG CTA ACC TTT TA-3′ (product size, 700 to 900 bp) as the forward primer and 5′-CAG GAA ACA GCT ATG TCC AGC TAA TAA CGC TGC AC-3′ as the reverse primer for PCR amplification. Bacterial DNA was extracted from pure cultures using the BioRobot EZ1 Magtration system 68G (Qiagen, Basel, Switzerland) and the Qiagen EZ1 DNA blood 350-μl kit. PCR amplification was performed in a MJ Research PTC-200 Peltier thermal cycler (Bio-Rad, Hercules, CA) using the following program: an initial denaturation step of 5 min at 95°C, followed by 35 cycles, with 1 cycle consisting of 1 min at 95°C, 1 min at 65°C, and 1 min at 72°C, and ending with a final elongation step of 10 min at 72°C. PCR products and a 100-bp DNA ladder (Invitrogen, Carlsbad, CA) were visualized by 2% agarose gel electrophoresis and using the E-Gel system (Invitrogen). Images were captured using the Gel Doc XR (Bio-Rad) digital image capture system and the Quality One one-dimensional analysis software version 4.6.0 (Bio-Rad). Sequencing was accomplished using the appropriate standard M13 forward and M13 reverse primers. Direct sequencing of PCR fragments was performed at the Columbia University DNA Sequencing Lab (https://www.dnasequencing.hs.columbia.edu) using a BigDye terminator kit (PE Applied Biosystems, Foster City, CA) and an ABI 3700 DNA sequencer. The *spa* type of each strain was determined by inputting the sequencing data to the *spa* type tool of the eGenomics website (eGenomics, New York, NY).

(iii) PVL. Panton-Valentine leukocidin (PVL), a possible virulence factor commonly detected in CA-MRSA, was detected by real-time PCR of the *lukS-PV* and *lukF-PV* genes as described previously (17).

Demographic data. Subjects' ages were determined from the year of birth as entered in the microbiology laboratory database. A chart review was conducted to collect the ethnicity and insurance status of MRSA-positive subjects.

Statistical analysis. The χ^2 test was used to test for associations between categorical variables. Fisher's exact test was used when expected cell frequencies were 5 or fewer. Logistic regression was used to examine the association between age, GBS colonization, and *S. aureus* colonization status. Odds ratios (ORs) were calculated with 95% confidence intervals (95% CIs).

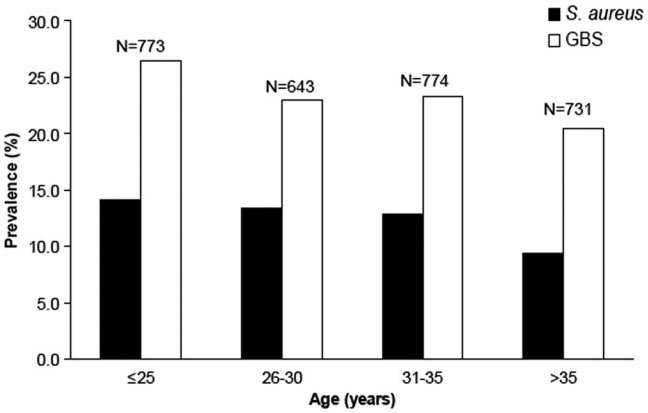

FIG. 1. Prevalence of *S. aureus* and GBS colonization by subject age. The total number of subjects in each age group is shown above the bars.

RESULTS

Prevalence of *S. aureus*. From 1 February to 31 July 2009, 3,099 Lim broth specimens were processed by the clinical microbiology laboratory and 3,030 (97.8%) specimens were retrieved for this study. More than one specimen was obtained from 107 patients, but only the last specimen collected was analyzed for this study. Similarly, samples from two additional patients not of child-bearing age (5 years and 75 years of age) were excluded. Thus, 2,921 samples from different patients were analyzed for the presence of *S. aureus*. *S. aureus* was cultured from 363 specimens (12.4%), of which 18 were identified as MRSA. The prevalences of MSSA colonization, MRSA colonization, and GBS colonization were 11.8%, 0.6% and 23.3%, respectively.

Risk factors associated with MRSA colonization. The prevalences of GBS colonization in patients who were MSSA positive, MRSA positive, and *S. aureus* negative were 34.2%, 33.3%, and 21.8%, respectively. There were significant associations between GBS and *S. aureus* colonization (OR, 1.9; 95% CI, 1.5 to 2.4) and MSSA colonization (OR, 1.9; 95% CI, 1.5 to 2.4), but not between GBS and MRSA colonization (OR, 1.7; 95% CI, 0.6 to 4.4; $P = 0.4$).

There was an inverse correlation between age and *S. aureus* colonization status ($P = 0.008$) (Fig. 1). Among 773 subjects who were ≤25 years old, 14.1% were colonized with *S. aureus* compared to a colonization rate of 9.4% among 732 subjects who were >35 years old. The frequency of GBS colonization was also inversely correlated with age: 26.5% of younger subjects versus 20.5% of older subjects were colonized with GBS. After adjustments for GBS colonization status were made, *S. aureus* colonization was significantly higher in younger patients than in older patients (OR, 1.4; 95% CI, 1.1 to 2.0). Comparisons between the other age groups, i.e., subjects 26 to 30 years old and 31 to 35 years old, were not significantly different. The demographic characteristics of patients colonized with CA-MRSA in 2005 versus 2009 are compared in Table 1; no sig-

TABLE 1. Demographic characteristics of patients with CA-MRSA colonization

Characteristic	No. of patients (%) or parameter value		*P* value
	2005[a] ($n = 13$[b])	2009 ($n = 18$)	
Mean age (yr) (SD)	28.3 (7.5)	26.7 (5.9)	0.77
Hispanic ethnicity	7 (53.8)	13 (72.2)	0.45
Medicaid insurance	12 (92.3)	14 (77.8)	0.37

[a] Data from 2005 from reference 8.
[b] The numbers of women (n) in the two studies (2005 and 2009) are shown. One of 14 MRSA-colonized women (2005) carried an SCC*mec* type II strain, a health care-associated strain, and was excluded from the demographic analysis.

nificant differences between these groups with regard to age, ethnicity, and insurance status were demonstrated.

Antibiotic susceptibility. The antibiograms of MRSA isolates from 2005 and 2009 are shown in Table 2. The proportion of strains resistant to levofloxacin increased significantly from 14.3% in 2005 to 55.6% in 2009 ($P = 0.028$). The rates of resistance to the other antibiotics tested remained unchanged.

Molecular analysis. A comparison of SCC*mec* type, *spa* type, and the presence of PVL among strains isolated in 2005 versus 2009 is shown in Table 3. All 18 strains isolated in 2009 and 13/14 strains isolated in 2005 were SCC*mec* type IV or V. The distribution of SCC*mec* type IV subtypes was significantly different in 2005 versus 2009 ($P = 0.004$). The proportion of SCC*mec* type IV isolates that were subtype IVa increased from 41.7% in 2005 to 93.8% in 2009.

(i) Pulsed-field gel electrophoresis. Analysis of MRSA strains from 2009 showed that 12/18 (66.7%) strains were the USA300 clone (97 to 100% similarity). In contrast, all 13 strains analyzed from 2005 were less than 85% similar to USA300. One strain from 2005 was untypeable by PFGE, but the results of molecular analysis were not consistent with USA300; it was SCC*mec* type V, *spa* type 162, and negative for PVL. Two isolates from 2005 were closely related to USA400 (two- or three-band difference). The remaining 11 isolates analyzed from 2005 and 6 isolates from 2009 were unrelated to both USA300 and USA400. The PFGE patterns of a representative subset of MRSA isolates from 2005 and 2009 are presented in Fig. 2.

TABLE 2. Antimicrobial resistance profiles of MRSA strains isolated from pregnant women in 2005 versus 2009

Antibiotic	No. (%) of isolates resistant to antibiotic		*P* value
	2005 ($n = 14$)[a]	2009 ($n = 18$)	
Clindamycin	5 (35.7)	2 (11.1)	0.19
Erythromycin	8 (57.1)	15 (83.3)	0.13
Levofloxacin	2 (14.3)	10 (55.6)	0.028[b]
Linezolid	0 (0)	0 (0)	
Tetracycline	2 (14.3)	3 (16.7)	>0.99
Trimethoprim-sulfamethoxazole	0 (0)	0 (0)	
Vancomycin	0 (0)	0 (0)	

[a] The numbers of strains (n) in the two studies (2005 and 2009) are shown.
[b] Statistically significant.

TABLE 3. Results of molecular analysis of strains isolated in 2005 versus 2009

Type or factor	No. (%) of strains isolated in:		*P* value[c]
	2005[a] ($n = 14$)[b]	2009 ($n = 18$)	
SCC*mec* type			0.004
IV	12/14 (85.7)	16/18 (88.9)	
IVa	5/12 (41.7)	15/16 (93.8)	
IVb	4/12 (33.3)	0/16 (0)	
Other	3/12 (25.0)	1/16 (6.2)	
spa type			0.002
1	2 (14.3)	9 (50.0)	
2	2 (14.3)	1 (5.6)	
59	0 (0)	5 (27.8)	
Other	10 (71.4)	3 (16.7)	
Presence of PVL	3 (21.4)	15 (83.3)	<0.001

[a] SCC*mec* type results for 2005 are from reference 9.
[b] The numbers of strains (n) in the two studies (2005 and 2009) are shown.
[c] Fisher's exact test was used to measure the difference in SCC*mec* type IV subtypes (IVa, IVb, and IV other) between 2005 and 2009 and to examine differences in the distribution of *spa* types between 2005 and 2009.

(ii) *spa* typing. The *spa* types identified in the 2009 MRSA isolates were primarily *spa* types 1 (Y1-H1-G1-F1-M1-B1-Q1-B1-L1-O1) (9/18 strains) and 59 (Y1-H1-G1-G1-F1-M1-B1-Q1-B1-L1-O1) (5/18 strains), which differ only by the duplication of the G1 repeat in type 59. Only two isolates (14.3%) from 2005 were *spa* type 1, and none was *spa* type 59. The USA300 PFGE pattern was identified in 7/11 isolates with *spa* type 1 and all 5 isolates with *spa* type 59.

(iii) Panton-Valentine leukocidin. In 2005, 21.4% (3/14) of MRSA strains were positive for the PVL toxin compared to 83.3% (15/18) of MRSA strains from 2009 ($P < 0.001$). PVL was detected in all 12 strains from the USA300 clone.

DISCUSSION

This is the first study, to our knowledge, to evaluate the epidemiology of MRSA anovaginal colonization in pregnant women over time at a single institution. The prevalence of MRSA anovaginal colonization among pregnant women in our New York City population remained stable in 2009 compared with 2005. However, molecular analysis provides evidence of a significant epidemiologic shift with USA300 emerging as the predominant MRSA clone. *spa* type 59 was a common *spa* type identified in 2009, and it was associated with the USA300 PFGE pattern and with the presence of PVL, an association not previously reported to our knowledge. There was a significant increase in levofloxacin resistance between 2005 and 2009 (from 14.3% to 55.6%; $P = 0.028$), with the highest frequency of resistance noted in strains from the USA300 clone (75% [9/12]). Risk factors for *S. aureus* colonization included younger age and GBS colonization.

The prevalence of MRSA anovaginal colonization reported herein is consistent with previous reports in which the prevalence has ranged from 0.3 to 1% (19, 28, 30), although the rate of MRSA anovaginal colonization appears to vary widely by geographic location, with centers in Alabama and Tennessee reporting significantly higher rates (2, 13). While the rate of

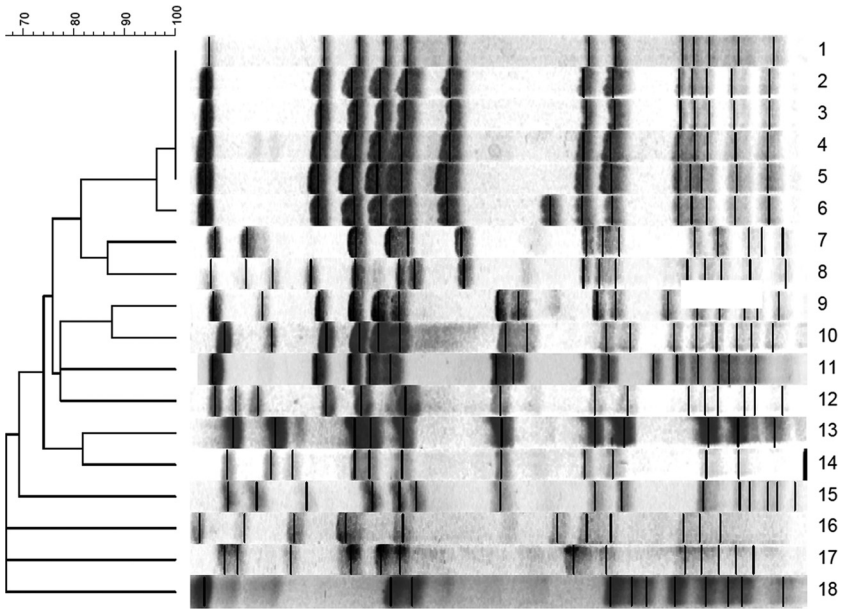

FIG. 2. (Right) PFGE analysis of a representative subset of MRSA strains from pregnant women with anovaginal colonization isolated in 2005 and 2009. Lane 1, USA300 reference strain; lane 11, USA400 reference strain; lanes 2 to 6, MRSA strains isolated in 2009 belonging to the USA300 clone; lanes 7 to 10 and 12 to 15, MRSA strains isolated in 2005; lanes 16 to 18, MRSA strains isolated in 2009. (Left) Dendrogram, with the scale representing percent similarity as calculated by the Dice coefficient.

anovaginal colonization has been shown to be lower than colonization of the anterior nares when assessed in the same subjects (0.3 to 1% versus 2 to 4%) (19, 28, 30), a recent study of *S. aureus* nasal colonization in northern Manhattan, New York City, NY, the catchment area for our institution, demonstrated that the prevalence of MRSA colonization of the anterior nares was 0.4% (24). Thus, MRSA anovaginal colonization may be lower in our population due to a lower overall prevalence of MRSA in our community.

The increasing prevalence of USA300 in pregnant women, coinciding with the increased incidence in the NICU and in newborn nurseries (6, 18, 35), suggests that MRSA may be vertically transmitted from mother to infant. We recently reported that infants admitted to the NICU who developed MRSA infections were younger at presentation than infants with MSSA infections (median age of 23 days versus 32 days, respectively; $P = 0.03$), suggesting potential evidence of vertical transmission (7). Using a variety of methods in different populations, several authors have recently studied the risk of vertical transmission of MRSA, resulting in infant colonization, from mothers detected by prenatal anovaginal cultures (2) or by culture of the anterior nares and vagina during labor (19, 28, 30). The rates of infant MRSA colonization detected by culture of the nares and skin sites (e.g., axilla, umbilicus, and groin) at birth and at 48 h of age ranged from 0.6 to 3.6% (19, 28, 30). On the basis of PFGE analysis, up to 12% of colonized infants carried a strain identical to their mothers (19, 28, 30). Fewer studies have examined the risk of infection; among 252 mother-infant pairs, 6 of whom were concordant for *S. aureus* colonization in both mothers and infants in the early neonatal period, no infant subsequently developed infection with the same strain with which they were colonized (28). In a study of 5,732 pregnant women who underwent prenatal anovaginal cultures for GBS screening and MRSA culture, no cases of early-onset invasive neonatal MRSA infection were identified among the infants of the 202 MRSA-colonized women (2). Thus, current data suggest that MRSA can be transmitted from mothers to their infants but that the risk of infection in infants is low. Further studies are needed to define the role of vertical transmission and risk of neonatal colonization and/or infection. At this time, data do not support routine screening of pregnant women for colonization with MRSA.

The increase in levofloxacin resistance observed in the present study, particularly in strains from the USA300 clone, is consistent with recent reports showing increasing fluoroquinolone resistance in CA-MRSA strains, including USA300, isolated from infected patients in the community (64%) and from men who have sex with men (71%) (15, 16). The emergence of multidrug-resistant CA-MRSA is likely related to selective pressure due to the increased use of fluoroquinolones and other non-β-lactam antibiotics for the treatment of confirmed and suspected MRSA infections (16).

We noted an inverse association between age and *S. aureus* anovaginal colonization status, with women who were ≤25 years old having a significantly higher rate of *S. aureus* colonization than women who were >35 years old (OR, 1.4; 95% CI, 1.1 to 2.0). This is consistent with a prior study showing that MRSA-positive women were younger than MRSA-negative women (2). Because younger individuals are more likely to have multiple sexual partners and are at higher risk of sexually transmitted infections, we speculate that *S. aureus* may be sexually transmitted. Notably, heterosexual transmission of MRSA has been reported recently (12).

The relationship between GBS and *S. aureus* colonization in the female genital tract remains uncertain. Two reports demonstrated a decreased frequency of MRSA colonization in the

setting of GBS colonization (8, 33). Conversely, MSSA colonization was more frequent in GBS-positive women (8). Of note, the rates of GBS colonization in the control groups were 46 to 50% (8, 33), which is higher than the generally reported rates of 10 to 30% (29). In contrast, others have reported that both MSSA and MRSA colonization were significantly more common in GBS-positive women (2). In the current study, GBS-colonized women were significantly more likely to be colonized with *S. aureus* and specifically, MSSA, but no significant association was observed with MRSA. It is possible that colonization with one organism may promote the acquisition and growth of the other, but further research is needed.

This study had limitations. It was a single center study at a hospital with a catchment area that includes a high proportion of Hispanic patients and patients with Medicaid insurance. Thus, our findings may not be generalizable. Further, we studied only 32 MRSA strains over time, and the molecular characteristics of these isolates may not accurately represent changes in the epidemiology of MRSA in our community.

In conclusion, the prevalence of anovaginal colonization with MRSA in our population remains stable and low. However, the epidemiology of MRSA strains is changing; USA300 has emerged as the predominant clone colonizing pregnant women, and MRSA strains associated with anovaginal colonization have increasing fluoroquinolone resistance. The rise in USA300 could lead to an increase in MRSA infections in pregnant and postpartum women and their newborns. Ongoing studies are needed to monitor the rate of vertical transmission resulting in neonatal colonization and to determine the risk of MRSA infections in colonized women and their newborns.

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