Original Article

Predictors of *Staphylococcus aureus* Rectovaginal Colonization in Pregnant Women and Risk for Maternal and Neonatal Infections

Karina A. Top,[1,a] Amanda Buet,[1] Susan Whittier,[2] Adam J. Ratner,[1] and Lisa Saiman[1,3]
Departments of [1]Pediatrics, and [2]Pathology, Columbia University, and [3]Department of Infection Control & Prevention, NewYork-Presbyterian Hospital, New York

Corresponding Author: Lisa Saiman, 622 W 168th St, PH4-470, New York, NY 10032. E-mail: ls5@columbia.edu.

[a]**Present affiliation:** Department of Pediatrics, Dalhousie University, Halifax, Nova Scotia, Canada.

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Background. *Staphylococcus aureus* infections are increasing among pregnant and postpartum women and neonates, but risk factors for *S. aureus* colonization in pregnancy and the association between maternal colonization and infant infections are not well defined. We sought to identify risk factors for maternal *S. aureus* rectovaginal colonization and assess colonization as a risk factor for infections among mothers and infants.

Methods. We conducted a retrospective cohort study of pregnant women and their infants. Demographic and clinical data, including *S. aureus* infections that occurred in mothers from 3 months before to 3 months after delivery and in infants during the first 3 months of life, were extracted from electronic medical records. Predictors for maternal *S. aureus* rectovaginal colonization were assessed through multivariable logistic regression analysis.

Results. The cohort included 2702 women and 2789 infants. The prevalence of maternal rectovaginal colonization with methicillin-susceptible *S. aureus* and methicillin-resistant *S. aureus* (MRSA) was 13% and 0.7%. Independent predictors of colonization included multigravidity, human immunodeficiency virus seropositivity, and group B *Streptococcus* colonization. *S. aureus* colonization was associated with an increased risk of infection in mothers (odds ratio [OR], 3.5; 95% confidence interval [CI], 1.4–8.8) but not in their infants (OR, 1.9; 95% CI, .6–5.6). The frequency of *S. aureus* infections was 0.8% in mothers and 0.7% in infants.

Conclusions. *S. aureus* rectovaginal colonization was associated with an increased risk of infections in women but not in their infants. The frequency of MRSA infections was low. These data suggest that routine MRSA screening of pregnant women may not be indicated.

Staphylococcus aureus infections are increasing in pregnant and postpartum women and in healthy neonates and infants hospitalized in neonatal intensive care units (NICUs) [1–4]. Much of this increase has been driven by a rise in methicillin-resistant *S. aureus* (MRSA), specifically community-associated (CA)–MRSA, which most commonly causes infections in patients without traditional risk factors [2, 3, 5]. *S. aureus* infections appear to be more frequent among individuals who are colonized with *S. aureus* in the anterior nares and other sites [6, 7].

S. aureus has been reported to colonize the vagina in 4%–22% of pregnant women [8–12]. The prevalence of MRSA rectovaginal colonization has been reported to range 0.5%–10% [8–12]. We previously conducted a prospective surveillance study of pregnant women undergoing routine screening for colonization with group B *Streptococcus* (GBS) [12]. The prevalence of

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methicillin-susceptible *S. aureus* (MSSA) rectovaginal colonization was 11.8%, and the prevalence of MRSA colonization was 0.6%. All 18 MRSA strains were CA-MRSA strains and 12 of 18 (75%) were the epidemic USA300 clone.

The factors that contribute to *S. aureus* rectovaginal colonization in pregnant women are not well understood. One study identified black race as an independent risk factor for MRSA rectovaginal colonization [10]. We identified younger age and GBS colonization as risk factors for *S. aureus* rectovaginal colonization but did not explore other demographic or maternal factors associated with *S. aureus* colonization [12]. A prior case-control study conducted among 117 women at our institution found that GBS colonization was a risk factor for MSSA rectovaginal colonization but was protective against MRSA colonization and that demographic factors and postpartum complications were not associated with colonization [13].

A few studies have examined the association between *S. aureus* colonization in pregnant women and the frequency of infections in women and/or their infants [9, 10, 14]. In one study, *S. aureus* colonization of the nares and/or vagina was not associated with an increased risk of *S. aureus* infections in the women, but infections in their infants were not assessed [9]. Another study did not demonstrate an increased risk of neonatal infections following neonatal *S. aureus* colonization in the first 48 hours of life, but maternal infections were not assessed [14]. Although vertical transmission has been proposed as a possible mechanism of maternal-to-infant transmission of MRSA [4, 10, 14, 15], the risk of developing early-onset neonatal sepsis caused by MRSA is not increased in infants born to MRSA-colonized women [10].

To date, the clinical significance of *S. aureus* rectovaginal colonization as a predictor of subsequent infection in mothers and their infants has not been systematically examined in a large cohort. The objectives of this study were to identify risk factors for *S. aureus* rectovaginal colonization, to assess maternal *S. aureus* colonization as a risk factor for infection among mothers and infants, and to determine the frequency of *S. aureus* infections in pregnant and postpartum women and their infants.

METHODS

Study Design and Subjects

We conducted a retrospective observational cohort study. Subjects included pregnant women screened for GBS and assessed for *S. aureus* rectovaginal colonization, as previously described, [12] who delivered an infant at NewYork-Presbyterian Hospital/Columbia University Medical Center (NYP/CUMC) and their infants. Women were screened from February 2009 to July 2009 and delivered their infants from February 2009 to November 2009. Women who delivered an infant at another institution (and their infants) were excluded. This study was approved by the Institutional Review Board at CUMC and was conducted in accordance with the ethical standards of the Helsinki Declaration. A waiver of informed consent was granted.

Study Procedures

As previously described, pregnant women in the cohort underwent routine screening for GBS at 35–37 weeks gestation with rectovaginal swabs [12] using established guidelines [16]. These specimens were cultured for *S. aureus*; 10 µL of the broth was streaked onto a selective differential chromogenic agar plate (BBL CHROMagar Staph aureus, BD Diagnostics) [12]. *S. aureus* was confirmed by latex agglutination (Staphaurex, Remel Europe), and methicillin susceptibility was determined by the cefoxitin disk diffusion screen [17].

Data Collection Procedures

The electronic medical records (EMRs) of pregnant women and their infants were reviewed by 2 members of the study team (K. T. and A. B.) who were blinded to maternal *S. aureus* colonization status. Demographic characteristics, prenatal and obstetrical history, including sexually transmitted infections, gravidity, parity, peripartum complications, and mode of delivery, and neonatal outcomes, including Apgar scores, birthweight, and disposition (eg, admission to the newborn nursery or NICU), were collected.

Identifying *Staphylococcus aureus* Infections. Both MSSA and MRSA infections were identified by positive clinical cultures for *S. aureus* and/or by *International Classification of Diseases* (ICD)–9 diagnosis codes pertaining to *S. aureus* infections, using previously validated ICD-9 codes [18]. To maximize ascertainment of *S. aureus* infections in infants, the clinical microbiology laboratory generated an epidemiology report of all positive *S. aureus* cultures sent from pediatric inpatient units, the pediatric emergency department (ED), and pediatric

outpatient clinics from 1 February 2009 to 1 February 2010. Cultures were reviewed to identify infants born to women in the study cohort.

Case Definitions. A definite *S. aureus* infection was defined as isolation of *S. aureus* from a sterile site (eg, blood, cerebrospinal fluid) or from a nonsterile site (eg, wound, respiratory culture) in the presence of signs and symptoms of infection (eg, fever, erythema, purulent drainage). A probable *S. aureus* infection was defined if, in the absence of a positive culture, there were signs and symptoms of skin or soft tissue infection (SSTI), including postoperative infections and mastitis, and a clinical response to antistaphylococcal antibiotics was documented in the EMR.

Outcomes

In order to assess colonization as a risk factor for infection, the primary outcomes were *S. aureus* infections occurring from 3 months prior to delivery to 3 months after delivery in the mothers and *S. aureus* infections in their infants within the first 3 months of life. The secondary outcomes were perinatal complications including intrapartum maternal fever (>38°C), cesarean delivery, preterm birth, birthweight <2500 g, 5-minute Apgar <7, admission to the NICU, and readmission within 3 months after delivery.

Statistical Analysis

Multivariable logistic regression analysis was conducted to identify independent risk factors for maternal *S. aureus* rectovaginal colonization. Hierarchical model building was conducted, and the effect of each additional variable on the model was assessed using the –2 log-likelihood test. Odds ratios (OR) and 95% confidence intervals (CIs) were computed for each variable in the model. The χ^2 test and Fisher's exact test were used to test for associations between categorical variables when appropriate. Student *t* test was used to analyze continuous variables. Statistical analysis was conducted using SAS version 9.1 software.

RESULTS

Of 2921 women included in the prior study of the prevalence of *S. aureus* rectovaginal colonization [12], the current study cohort consisted of 2702 women (93%) who delivered 2789 infants at our institution. The prevalence of maternal rectovaginal colonization with GBS, MSSA, and MRSA was 24%, 13% and 0.7%, respectively, among these 2702 women.

Risk Factors for Rectovaginal Colonization

The demographic and obstetrical characteristics associated with *S. aureus* colonization in the maternal cohort are shown in Table 1. In this univariate analysis, the factors most strongly associated with *S. aureus* rectovaginal colonization included black race, government insurance, being unmarried, multigravidity, GBS colonization, and human immunodeficiency virus (HIV) seropositivity. In the multivariable regression analysis, government insurance, multigravidity, HIV seropositivity, and GBS colonization were identified as independent risk factors for *S. aureus* rectovaginal colonization in the full model analysis (Table 2). Maternal *S. aureus* colonization status was not associated with adverse peripartum or neonatal outcomes (Table 3).

Association Between Colonization and Infection

The frequency of *S. aureus* infections in women with *S. aureus* rectovaginal colonization was 2.1% (7 of 340), compared with 0.6 % (14 of 2362) in uncolonized women. There was a significant association between maternal colonization and risk of infection when women with probable and definite infections were considered (OR, 3.5; 95% CI, 1.4–8.8; $P = .004$). However, when only definite *S. aureus* infections were considered in women with concordant colonization and infection based on methicillin-susceptibility, the association between maternal colonization and infection was no longer statistically significant ($P = .07$). The frequency of *S. aureus* infections in infants was 1.2% (4 of 340) among those born to *S. aureus*–positive mothers and 0.6% (15 of 2362) among those born to *S. aureus*–negative mothers (Table 3), but there was no significant association between maternal *S. aureus* rectovaginal colonization and the risk of *S. aureus* infection in their infants (OR, 1.9; 95% CI, 0.6–5.6; $P = .29$).

Maternal *Staphylococcus aureus* Infections

During the study period, 14 (0.5%) of 2702 women developed definite *S. aureus* infections and 7 (0.3%) women had probable *S. aureus* infections. Surgical site infections were most common (Table 4). Eighty-one percent (17 of 21) of infections occurred within 3 months after delivery, and 19% (4 of 21) occurred in

Table 1. Univariate Analysis of Demographic and Clinical Risk Factors for Maternal *Staphylococcus aureus* Rectovaginal Colonization

Characteristics	*S. aureus* Positive N = 340 No. (%)	*S. aureus* Negative N = 2362 No. (%)	OR (95% CI)	*P* Value[a]
Maternal age (years)				
Mean, SD	29.6 (6.6)	30.4 (6.7)	…	.03
≤ 25	106 (31)	672 (28)	1.41 (1.05–1.89)	.02
26–35	176 (52)	1147 (49)	1.53 (1.10–2.11)	.01
≥ 36[b]	58 (17)	543 (23)	…	
Race/Ethnicity				
Hispanic	143 (42)	943 (40)	1.57 (1.15–2.13)	.004
Black (non-Hispanic)	59 (17)	297 (13)	2.05 (1.41–2.99)	<.001
Other	86 (25)	517 (22)	1.75 (1.22–2.49)	.002
White (non-Hispanic)[b]	52 (15)	595 (25)	…	
Insurance				
Government/Self pay	235 (69)	1280 (54)	1.89 (1.48–2.41)	<.001
Private	105 (31)	1082 (46)	…	
Marital Status				
Unmarried[c]	207 (61)	1208 (51)	1.50 (1.18–1.88)	<.001
Married/Life partner	133 (39)	1154 (49)	…	
Gravidity				
Multigravid (≥2)	262 (77)	1591 (67)	1.63 (1.25–2.13)	<.001
Primigravid	78 (23)	771 (33)	…	
Parity				
Multiparous (≥1)	192 (56)	1183 (50)	1.30 (1.03–1.62)	.03
Nulliparous	148 (44)	1179 (50)	…	
GBS colonization				
Yes	119 (35)	518 (22)	1.92 (1.50–2.44)	<.001
No	221 (65)	1844 (78)	…	
HIV status				
Positive	4 (1.2)	4 (0.2)	7.01 (1.74–28.15)	.001
Negative	336 (98.8)	2358 (99.8)	…	
Chlamydia trachomatis				
Yes	12 (3.8)	55 (2.5)	1.50 (0.80–2.83)	.21
No	308 (96.2)	2115 (97.5)	…	
Syphilis				
Yes	3 (0.9)	15 (0.6)	1.39 (0.40–4.84)	.61
No	337 (99.1)	2347 (99.4)	…	

Abbreviations: CI, confidence interval; GBS, group B *Streptococcus*; HIV, human immunodeficiency virus; OR, odds ratio; SD, standard deviation.

[a] Pearson χ^2 or Student *t* test *P* value comparing *S. aureus*–positive and *S. aureus*–negative women.

[b] Reference group.

[c] Includes never married, widowed, divorced, separated.

the last 3 months of pregnancy. Three (21%) definite infections were caused by MRSA, and 11 (79%) were caused by MSSA. Seven women developed probable infections with *S. aureus*: 5 developed incisional erythema 2–4 days after cesarean section and were treated successfully with cephalexin, and 2 were treated in the ED for mastitis with either dicloxacillin or amoxillin-clavulanic acid. Neither of the 2 women was readmitted to the ED or hospital.

Women who had a cesarean delivery were at significantly higher risk of developing a *S. aureus* infection compared with women who had a vaginal delivery (14 of 990 [1.4%] vs 7 of 1712 [0.4%]; OR, 3.5; 95% CI, 1.4–8.7; *P* = .008). Women who were HIV-seropositive were also significantly more likely to develop a *S. aureus* infection than women who were HIV-seronegative (2 of 8 [25%] vs 19 of 2694 [0.7%]; *P* = .002). There was no association between

insurance status, gravidity, or GBS colonization and *S. aureus* infections.

Neonatal *Staphylococcus aureus* Infections

Twelve (0.4%) of 2789 infants had 13 definite *S. aureus* infections, and 8 (0.3%) infants had probable *S. aureus* infections. The median age at diagnosis of the first *S. aureus* infection was 27 days (range, 8–84 days). Of the 12 infants with definite *S. aureus* infections, 1 had an MRSA SSTI and 11 had MSSA infections, including 1 infant with MSSA bacteremia followed by an SSTI 6 weeks later. Two infants in the NICU with a history of MSSA colonization based on positive cultures from the conjunctiva or respiratory tract without concomitant signs and symptoms of infection developed MSSA infections 3–6 weeks after colonization was detected. The types of infant infections are shown in Table 4. Ten of 20 (50%) infants with *S. aureus* infections were born preterm (<37 weeks gestational age), 7 (35%) were of multiple gestation, and 15 (75%) were admitted to our NICU. These 3 factors were all significantly associated with *S. aureus* infection ($P < .001$).

Table 2. Multivariable Logistic Regression for *Staphylococcus aureus* Rectovaginal Colonization Among 2692 Pregnant Women

Characteristics	*S. aureus* Colonized OR (95 % CI)[a]	*P* Value
Age, years		
≤ 25	1.25 (0.85–1.84)	.26
26–35	1.28 (0.94–1.74)	.12
≥ 36 *(reference)*	…	…
Black race	1.32 (0.96–1.80)	.08
Government insurance/Self pay	1.63 (1.19–2.25)	.003
Unmarried[b]	1.04 (0.78–1.39)	.80
Multigravid (≥2)	1.70 (1.19–2.41)	.003
Multiparous (≥1)	0.92 (0.67–1.26)	.59
HIV positive	6.32 (1.54–25.96)	.01
GBS colonization	1.82 (1.42–2.33)	<.001

Abbreviations: CI, confidence interval; GBS, group B *Streptococcus*; HIV, human immunodeficiency virus; OR, odds ratio.

[a] ORs comparing *S. aureus*–positive and *S. aureus*–negative women.

[b] Includes never married, widowed, divorced, separated.

DISCUSSION

This large retrospective cohort study identified risk factors for *S. aureus* rectovaginal colonization in late pregnancy and assessed the risk of peripartum and neonatal infections. Independent predictors of *S. aureus* rectovaginal colonization included multigravidity, HIV infection, government insurance, and GBS colonization. Although maternal rectovaginal *S. aureus* colonization may be a risk factor for maternal infections, maternal colonization was not significantly associated with neonatal infections. Overall, the frequency of serious *S. aureus* infections in pregnant and postpartum women and infants was low.

Women who were multigravid had 1.7 times the odds of being colonized with *S. aureus* in the rectum

Table 3. Maternal and Infant Outcomes by *Staphylococcus aureus* Rectovaginal Colonization Status

Outcomes	*S. aureus* Positive N = 340 No. (%)	*S. aureus* Negative N = 2362 No. (%)	OR (95% CI_{95})	*P* Value[a]
Gestation <37 weeks	30 (8.8)	200 (8.5)	0.96 (.64–1.43)	.83
Mode of delivery				
Vaginal	215 (63)	1497 (64)	1.00 (.79–1.27)	.96
Cesarean	125 (37)	865 (37)		
Any peripartum complication[b,c]	74 (22)	571 (24)	0.87 (.66–1.15)	.33
5-minute Apgar <7[c]	3 (1)	32 (1)	0.65 (.20–2.13)	.61
Birth weight <2500 g[c]	22 (6)	177 (7)	0.85 (.54–1.35)	.50
Infant disposition at 24 hours[c]				
Well baby nursery	297 (87)	2087 (88)	NA	.70
NICU	41 (12)	255 (11)		
Infant demise	2 (0.6)	20 (0.8)		
Infant *S. aureus* infections[d]	4 (1.2)	15 (0.6)	1.86 (.61–5.63)	.29

Abbreviations: CI, confidence interval; NA, not applicable; NICU, neonatal intensive care unit; OR, odds ratio.

[a] Pearson χ^2 or Student *t* test *P* value comparing *S. aureus*–positive and *S. aureus*–negative women and their infants.

[b] Peripartum complications analyzed included maternal fever >38°C, chorioamnionitis, nonreassuring fetal status.

[c] For multiple births, the outcome of baby A was used in the analysis of categorical variables.

[d] Two infants with *S. aureus* infections were twins, and only baby A was included in the analysis.

Table 4. Types of *Staphylococcus aureus* Infections in Mothers and Infants

Type of Infection[a]	Mothers N = 21 No. (%)	Infants[b] N = 20 No. (%)
Mastitis	3 (14)	0 (0)
Surgical site	10 (48)	5 (25)
Other skin and soft tissue	4 (19)	10 (50)
Bacteremia/meningitis	0 (0)	5 (25)
Conjunctivitis	0 (0)	1 (5)
Urinary tract infection	4 (19)	0 (0)

[a] Infection types are as reported in the electronic medical record.
[b] One infant experienced 2 *S. aureus* infections during the study period.

and genital tract, an association that has not been previously reported. A prior study showed that multiparous women had a higher rate of CA-MRSA infections, which may be attributed to transmission from young children in the home [1]. However, in the current study, multiparous women were not at increased risk of *S. aureus* colonization, suggesting that another factor was important among multigravid women. We speculate that the discrepancy between multigravidity and multiparity could be due to a higher frequency of prior spontaneous or therapeutic abortions among *S. aureus*–colonized women, although this was not specifically addressed in the current study. Multigravid women may have increased contact with the healthcare system (eg, outpatient visits and medical procedures) and thus have increased opportunities to contract MRSA or MSSA [19]. Further studies may help to clarify the importance of an increased number of pregnancies and increased contact with the healthcare system on *S. aureus* transmission and colonization.

Prior studies have shown an association between HIV infection and *S. aureus* colonization and infection, particularly in those with lower CD4+ T-cell counts and higher viral loads [20, 21]. Asymptomatic HIV patients have also been shown to be at increased risk of *S. aureus* and MRSA colonization and infection compared with HIV-negative controls [21]. In the current study, HIV-positive women were at significantly greater risk of both rectovaginal *S. aureus* colonization and *S. aureus* infection than HIV-negative women. Of note, there were only 8 HIV-positive women in the current study, and no information was available on the status of their HIV infection (eg, CD4 + T-cell counts or HIV RNA levels). However, *S. aureus* rectovaginal colonization has not been previously demonstrated in pregnant women with HIV infection, suggesting the need for further studies in different populations.

Women with government insurance had 1.6-fold greater odds of *S. aureus* colonization. Another large cohort of pregnant women also identified government insurance as a possible risk factor for MRSA rectovaginal colonization in the univariate but not in the multivariable analysis [10]. Studies in other patient populations have similarly shown an association between MRSA colonization in the anterior nares and lower household income [19]. Crowding and poor hygiene may facilitate *S. aureus* transmission among populations of lower socioeconomic status, which could include those with government insurance [19].

Concurrent colonization with GBS was independently associated with an increased risk of *S. aureus* rectovaginal colonization in the multivariable regression model, confirming our prior finding [12]. Previous studies have demonstrated an association between GBS and *S. aureus* in the vaginal flora of both pregnant and nonpregnant women [10, 22]. Isolation of GBS and *S. aureus* as part of the vaginal flora has been associated with asymptomatic alterations in the microbiota and with symptomatic vaginitis [23, 24]. In both settings, elevated levels of cytokines and chemokines including interleukin 1, interleukin 6, and interleukin 8 have been detected in vaginal secretions [23, 24]. In vitro studies have shown that GBS inhibits growth of other streptococcal but not staphylococcal species [25]. The inhibition of competing bacterial species may provide an opportunity for *S. aureus* to proliferate in the setting of GBS colonization and thus contribute to alterations in the vaginal flora [13]. The impact of bacterial inhibition by GBS on the vaginal microbiota in vivo and the extent to which GBS and *S. aureus* contribute to mucosal inflammation are unclear.

Colonization with *S. aureus*, and MRSA in particular, has been associated with an increased risk of infections [3, 6, 7]. In the current study, women with *S. aureus* rectovaginal colonization had 3.5-fold greater odds of infection ($P = .004$). However, infants born to *S. aureus*–colonized mothers did not appear to be at higher risk of *S. aureus* infections ($P = .29$). Prior reports have demonstrated that infants born to mothers with *S. aureus* rectovaginal colonization can develop colonization of the anterior nares and skin [10, 14, 15], but neonatal *S. aureus* colonization within the first 48 hours of life was not associated with an increased risk of infection [14]. Two studies that employed active surveillance to identify

postpartum and neonatal infections, respectively, reported higher frequencies of *S. aureus* infections of 6.3% and 2.4% [9, 14]. In both studies, all of the reported infections were SSTIs, most commonly surgical site infections or postpartum mastitis. No patient in either study developed an invasive *S. aureus* infection, supporting our finding of a low frequency of invasive *S. aureus* infections in mothers and infants. Infants who became infected with *S. aureus* were more likely to have been hospitalized in the NICU, suggesting unique risk factors for infection in this population.

The study had limitations. This was a single-center study conducted at an academic referral center located in an area of relatively low prevalence of MRSA colonization [26], thereby limiting the generalizability of the results. The study did not examine colonization at body sites other than the genital tract; thus, some women with *S. aureus* colonization may have been missed. All data collection, excluding *S. aureus* colonization and GBS status, was conducted retrospectively through the hospital EMR and relied on existing documentation. There may have been inaccuracies in the medical record, including in recording ICD-9 diagnosis codes. However, if the study team noted discrepancies in the EMR, multiple data sources (eg, laboratory results, clinical notes written by multiple providers) were reviewed in efforts to resolve the discrepancies. Ascertainment of *S. aureus* infections, particularly minor infections, may have been incomplete because patients may have sought care at another facility or from a physician who did not use the institution's EMR or they may have been managed by telephone. Approximately 45% of infants had an outpatient visit recorded in the EMR. Among these infants, all infections requiring a physician visit should have been captured in the study. In addition, serious infections requiring an ED visit or hospitalization would have been captured for infants followed by private practices affiliated with our medical center. Due to the low frequency of *S. aureus* infections observed in the current study there was reduced statistical power to identify risk factors for *S. aureus* infections.

In conclusion, this large cohort study identified several independent risk factors for maternal *S. aureus* rectovaginal colonization, notably multigravidity and HIV infection, which have not been previously associated with rectovaginal colonization. Further studies in other populations of pregnant women are needed to characterize the association between *S. aureus* colonization, multigravidity, and HIV infection. The frequency of serious *S. aureus* infections in *S. aureus*–colonized women and their infants was low. Maternal rectovaginal colonization was associated with an increased risk of *S. aureus* infections in women but was not associated with a significantly increased risk of infection in their infants. Furthermore, MSSA infections were more common than MRSA infections in both mothers and infants in our population. Together, these data suggest that routine surveillance for MRSA colonization in pregnant women may not be indicated.

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