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Erratum: Page 2056, column 1, line 26: ">=10^7 white blood cells" should read "10 white blood cells."

Diversity of Group B Streptococcus Serotypes Causing Urinary Tract Infection in Adults▽

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Serotypes of group B streptococcus (GBS) that cause urinary tract infection (UTI) are poorly characterized. We conducted a prospective study of GBS UTI in adults to define the clinical and microbiological characteristics of these infections, including which serotypes cause disease. Patients who had GBS cultured from urine over a 1-year period were grouped according to symptoms, bacteriuria, and urinalysis. Demographic data were obtained by reviewing medical records. Isolates were serotyped by latex agglutination and multiplex PCR-reverse line blotting (mPCR/RLB). Antibiotic susceptibilities were determined by disc diffusion. GBS was cultured from 387/34,367 consecutive urine samples (1.1%): 62 patients had bacteriuria of >10^7 CFU/liter and at least one UTI symptom; of these patients, 31 had urinary leukocyte esterase and pyuria (others not tested), 50 (81%) had symptoms consistent with cystitis, and 12 (19%) had symptoms of pyelonephritis. Compared with controls (who had GBS isolated without symptoms), a prior history of UTI was an independent risk factor for disease. Increased age was also significantly associated with acute infection. Serotyping results were consistent between latex agglutination and mPCR/RLB for 331/387 (85.5%) isolates; 22 (5.7%) and 7 (1.8%) isolates were nontypeable with antisera and by mPCR/RLB, respectively; and 45/56 (80.4%) isolates with discrepant results were typed by mPCR/RLB as belonging to serotype V. Serotypes V, Ia, and III caused the most UTIs; serotypes II, Ib, and IV were less common. Nontypeable GBS was not associated with UTI. Erythromycin (39.5%) and clindamycin (26.4%) resistance was common. We conclude that a more diverse spectrum of GBS serotypes causes UTI than previously recognized, with the exception of nontypeable GBS.

Group B streptococcus (GBS) is a leading cause of infection in newborns, pregnant women, and older persons with chronic medical illness (3, 8). In addition to maternal cervicovaginal colonization and neonatal infection that results from the vertical transmission of bacteria from mothers to their infants, GBS can also cause urinary tract infection (UTI). The spectrum of GBS UTI includes asymptomatic bacteriuria (ABU), cystitis, pyelonephritis, urethritis, and urosepsis (6, 8, 10, 20, 23, 26). GBS ABU is particularly common among pregnant women, although those most at risk for cystitis due to GBS appear to be the elderly and immunocompromised individuals (8, 9, 25). Despite the uropathogenic nature of GBS, the clinical and microbiological features of GBS UTI, including risk factors for disease and whether there is a tendency for particular GBS serotypes to cause UTI, are poorly understood.

Clinically, UTI due to GBS may be indistinguishable from UTI caused by other uropathogens (25). However, a recent study of multiple uropathogens and host characteristics highlighted unique frequencies of host characteristics in UTI groups defined by the causal organism (37). This suggests that the clinical and microbiological features of UTI may differ depending on the infecting uropathogen. GBS colonization of the urinary tract in women most likely occurs by an ascending route from the vagina, where GBS can persist asymptomatically. While the overall prevalence of GBS UTI in the adult population remains unclear, GBS bacteriuria during pregnancy occurs at rates of between 1 and 3.5% (4, 23, 41). Many of these episodes represent ABU (2, 18); however, GBS ABU is considered to be a surrogate for heavy maternal colonization (29, 42) and is currently recommended for intrapartum antibiotic chemoprophylaxis (23, 34). In addition, up to 7% of pregnancies may be complicated by GBS UTI, and GBS reportedly accounts for approximately 10% of all cases of pyelonephritis during pregnancy (25, 28). GBS UTI may also contribute to chorioamnionitis (1), premature onset of labor (24), and an increased risk of vertical transmission of GBS (29, 42).

Several studies have also reported high rates of GBS UTI in nonpregnant adults (8, 9, 25, 39). In one study, GBS was cultured from 39% of all cases of symptomatic UTI among nursing home residents >70 years of age (40). Other studies reported that GBS UTI may account for up to one-third of all invasive infections due to GBS in adults (9, 12, 19, 26). Several independent surveys have reported the recovery of GBS from between 1 and 2% of all UTI cases (7, 26, 30). GBS UTI may also account for up to 7% of late-onset disease in neonates (43). Thus, while there is an increasing amount of data regarding the prevalence of GBS UTI in adults, little is known re-

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garding the clinical and microbiological features associated with these infections or the GBS serotypes that cause UTI. In this study, we carried out a single-center analysis of adult patients at the University of Alabama at Birmingham Hospital between August 2007 and August 2008 who had GBS cultured from urine during routine assessments for UTI to define the clinical and microbiological characteristics of GBS UTI including which serotypes cause disease.

MATERIALS AND METHODS

Patients. The study subjects were adult patients (>18 years of age) encountered at the University of Alabama at Birmingham Hospital between August 2007 and August 2008 who underwent clinical and microbiological assessments for UTI because of symptoms indicating infection or as part of routine patient screening. Urine samples were obtained as clean-catch voided or catheterized samples from all adult patients who underwent assessment for UTI during the study period. This included inpatients, patients that were evaluated in the emergency department, and patients from various University Hospital outpatient clinics. In cases where GBS was cultured from urine (any count), the medical records for each patient were reviewed for presenting symptoms at the time of sample collection, and demographic data including possible risk factors were recorded. A provisional UTI diagnosis was defined by the presence of single-organism GBS bacteriuria ($>10^7$ CFU/liter) with at least one symptom that included dysuria, increased urinary frequency and/or urgency, fever of >38°C, flank pain, and/or lumbar tenderness. In cases where urinalysis (UA) was performed, UTI was confirmed on the basis of positive urinary leukocyte esterase and pyuria ($\geq 10^7$ white blood cells/high-power field; nonspun). These are the generally accepted criteria for the diagnosis of UTI (11, 14, 21). Patients were grouped into probable GBS UTI where UA was not performed and confirmed cases where (positive) UA data were available. This study was performed in accordance with the ethical standards of the University of Alabama Birmingham committee on human experimentation and the Helsinki Declaration. The need for specific informed consent was waived by the institutional review board of the University of Alabama at Birmingham.

Controls. To conduct an analysis of risk factors for GBS UTI, we identified a group of control subjects who were defined as having GBS isolated from urine incidentally. These individuals were selected on the basis of low-grade GBS bacteriuria ($<10^7$ CFU/liter) in the absence of symptoms. This allowed an assessment of the factors associated with acute GBS UTI versus asymptomatic genitourinary colonization, which is prevalent among healthy adult women. Among the total patient cohort who had GBS isolated from urine, there were 51 individuals who satisfied these criteria, and all were included as controls. We also performed alternative comparisons using a control group of all 325 individuals who did not satisfy the criteria defined for GBS UTI ("all others," subjects who were GBS culture positive but did not have acute symptomatic UTI as defined by the exclusion criteria).

Bacterial isolates. All isolates were cultured from clean-catch voided urine or a catheterized urine sample. Isolates were identified by typical colony morphology on tryptic soy agar–5% sheep blood agar plates (BD), tested for catalase, and grouped using the Remel PathoDx latex agglutination kit. Capsular serotyping of each GBS isolate was performed by latex agglutination using commercial antisera from SSI (Denmark) as described elsewhere previously (36). Antimicrobial susceptibility testing was performed by disc diffusion according to methods of the Clinical and Laboratory Standards Institute (41a). One isolate per patient was analyzed and stored at −80°C in 15% glycerol in Todd-Hewitt broth. Strains were grown at 37°C on Todd-Hewitt agar or in Todd-Hewitt broth.

Molecular serotyping. Molecular serotypes (MSs) were determined at the Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research, Sydney, NSW, Australia, for each GBS isolate (17). The MS identification of 10 serotypes (MS Ia, Ib, and II to IX) was determined by using a multiplex PCR and reverse line blot (mPCR/RLB) hybridization assay targeting a GBS species-specific gene (*cfb*) and serotype-specific sequences in various genes: *cpsH* for MSs Ia, Ib, III, IV, V, VI, and IX; *cpsK* for MS II; *cpsM* for MS VII; and *cpsJ* for MS VIII. Methods for the extraction of GBS genomic DNA were described elsewhere previously (15), along with sequences of oligonucleotides, primers, probes, and optimal PCR and hybridization conditions (17).

Discrepancies between latex agglutination and mPCR/RLB. Isolates were tested independently by the two methods without knowledge of the results of the other method. When discrepancies were identified, both were repeated, and if discrepancies persisted, a portion of the *cpsE-cpsF-cpsG* gene cluster was sequenced (~800 bp) to confirm the result (44). The mPCR/RLB result was accepted as the definitive result.

Statistical analysis. We used SPSS v9.0 for statistical analyses. Differences in categorical variables (i.e., risk factors) between case patients and control subjects were examined by Pearson chi-squared (χ^2) analysis or Fisher's exact test where the expected value of any cell was below 5. For continuous variables without normal distribution (i.e., age), a Mann-Whitney U test was used. Gender comparisons were performed using population data (equal group sizes assumed) from the U.S. Census Bureau for Birmingham, AL (male-to-female ratio, 0.857). The quantitative effects of covariates on variables identified as being significant by univariate analysis were evaluated by logistic regression to identify independent risk factors for GBS UTI. Results are given as adjusted odds ratios (ORs). P values of <0.05 were regarded as being significant.

RESULTS

Study population. A total of 34,367 urine samples were collected from patients who underwent assessment for UTI during the study period. These samples represented a total patient cohort of inpatients (39%), outpatients (19.6%), women in obstetrics care (27.4%), and patients who presented to emergency rooms (14%). GBS was cultured from 387 patients (1.1%), representing both clean-catch voided (346/387 [89.4%]) and catheterized (41/387 [10.6%]) samples. The demographic data for the cohort of 387 GBS-positive patients are summarized in Table 1. The mean age for the 387 patients was 46 years (range, 18 to 95 years), and 322/387 patients (83.2%) were female. The most common comorbid conditions were urological (21.4%), cardiac (17.6%), neurological (15.5%), endocrine (8.5%), and pulmonary (7.8%). The cohort of 387 patients with urine cultures that were positive for GBS included inpatients (18.8%), outpatients (52.4%), women in obstetrics care (21%), and patients who presented to emergency rooms (7.8%). Among the 387 patients with urine cultures that were positive for GBS, 207 (53.5%) had single-organism GBS cultured, and 210 (54.3%) underwent UA. The most commonly found cocultured organisms were *Escherichia coli* (6.7%), coagulase-negative staphylococci (5.7%), diptheroids (4.4%), *Lactobacillus* spp. (3.4%), *Klebsiella pneumoniae* (2.8%), *Staphylococcus aureus* (2.6%), and viridans group streptococci (2.6%).

Cases of GBS UTI. Among 387 patients with urine cultures that were positive for GBS, 62 patients had single-organism GBS bacteriuria with $>10^7$ CFU/liter concurrent with at least one UTI symptom and were defined as having probable GBS UTI. Among these, 50/62 patients (81%) had clinical and microbiological features consistent with cystitis, versus 12/62 (19%) for pyelonephritis (Table 1). All 31 of the 62 patients who had UA performed had positive results based on positive urinary leukocyte esterase and pyuria (Table 1). None of the 62 cases were associated with an indwelling urinary catheter, and only one patient had GBS concurrently isolated from blood, representing probable urosepsis.

Risk factor analysis. When 62 case patients were compared with 51 controls (Table 1), the mean age of case patients was significantly greater (53 ± 19 years versus 30 ± 12 years; $P < 0.001$). However, there were no significant differences in the prevalence rates of previously identified (and several other potential) risk factors including limited mobility, diabetes mellitus, chronic kidney disease, and the presence of an indwelling urinary catheter between case patients and control subjects

TABLE 1. Adult patients who had GBS isolated from urine during routine assessment for UTI at the University of Alabama Hospital between August 2007 and August 2008

Parameter	Value for group[c]					P value (all cases vs controls)
	Total specimens (n = 387)[a]	GBS UTI cases[b]			Controls[d] (n = 51)	
		UA +ve[c] (n = 31)	UA ND[k] (n = 31)	All (n = 62)		
Age (yr) (mean; range)	46; 18–95	54; 19–82	52; 19–93	53; 19–93	30; 18–64	<0.001[g]
No. (%) of females	322 (83)	25 (81)	27 (87)	52 (84)	46 (90)	0.002[h]
No. (%) of patients with symptom:						
Dysuria	68 (17.6)	18 (58.1)	17 (54.8)	35 (56.5)	0 (0)	ND
Frequency	57 (14.7)	11 (35.5)	12 (38.7)	23 (37.1)	0 (0)	ND
Flank pain	35 (9.0)	7 (22.6)	7 (22.6)	14 (22.6)	0 (0)	ND
Fever	15 (4.0)	4 (13.0)	2 (6.0)	6 (10.0)	0 (0)	ND
C/W cystitis[e]	130 (33.6)	25 (80.6)	24 (77.4)	50 (81.0)	0 (0)	ND
C/W pyleonphritis[f]	65 (16.8)	6 (19.4)	7 (22.6)	12 (19.0)	0 (0)	ND
No. (%) of pregnant patients	99 (30.1)	1 (4)	5 (18.5)	6 (11.5)	35 (76)	
No. (%) of patients with possible risk factor of:						
Limited mobility	13 (3.4)	1 (3.2)	0 (0)	1 (1.6)	0 (0)	1.000
Diabetes mellitus	91 (23.5)	5 (16.1)	4 (12.9)	9 (14.5)	9 (17.6)	0.651
Chronic kidney disease	72 (18.6)	4 (12.9)	0 (0)	4 (6.5)	7 (13.7)	0.219
Indwelling urinary catheter	4 (1.0)	0 (0)	0 (0)	0 (0)	0 (0)	ND
Altered mental status	14 (3.6)	3 (9.7)	2 (6.5)	5 (8.1)	0 (0)	0.063[i]
Prior history of UTI	76 (19.6)	9 (29.0)	9 (29.0)	18 (29.0)	6 (11.8)	0.032[j]
Pure GBS isolated	207 (53.5)	31 (100)	31 (100)	62 (100)	15 (29.4)	ND
No. (%) of patients with GBS counts of $>10^7$ CFU/liter	319 (82.4)	31 (100)	31 (100)	62 (100)	0 (0)	ND
Mean GBS count ($\times 10^7$ CFU/liter) ± SD	4.7 ± 3.6	7.4 ± 3.5	6.1 ± 3.1	6.7 ± 3.4	0.5 ± 0.3	ND
No. (%) of patients with UA done	210 (54.3)	31 (100)	0 (0)	31 (50)	9 (17.3)	ND
No. (%) of patients with finding:						
Pyuria	114 (54.3)	31 (100)	ND	31 (100)	0 (0)	ND
Leukocyte esterase	122 (58.1)	31 (100)	ND	31 (100)	0 (0)	ND
Hematuria	74 (35.2)	21 (67.7)	ND	21 (67.7)	2 (22)	ND
+ve and C/W UTI[e,f]	91 (43.3)	31 (100)	ND	31 (100)	0 (0)	ND

[a] Consecutive urine specimens sent for culture from which GBS was isolated.
[b] Patients with at least one symptom of UTI and pure growth of GBS of $>10^7$ CFU/liter.
[c] +ve (consistent with UTI), positive for leukocyte esterase and pyuria.
[d] Subjects without symptoms from whose urine GBS was isolated in counts of $<10^7$ CFU/liter.
[e] Symptoms consistent with (C/W) cystitis are dysuria and/or frequency.
[f] Symptoms consistent with pyelonephritis are dysuria and/or frequency plus flank pain and/or fever of >38°C.
[g] Determined by Mann-Whitney U test.
[h] Determined by Pearson χ^2 analysis. Gender comparisons were performed using population data (equal group sizes) from the U.S. Census Bureau for Birmingham (male-to-female ratio, 0.857).
[i] Determined by Fisher's exact test.
[j] Determined by forward stepwise logistic regression subsequent to Pearson χ^2 analysis.
[k] ND, not done.

(Table 1). GBS UTI case patients were significantly more likely to have had a prior history of UTI than controls (OR, 3.1; 95% confidence interval [CI], 1.1 to 8.7) and to be female (OR, 3.8; 95% CI, 1.6 to 8.7). A prior history of UTI was highly significant among pyelonephritis case patients (50.0% [6/12] versus 11.8% [6/51]; $P = 0.007$) compared to controls and was an independent risk factor for GBS pyelonephritis (OR, 10.1; 95% CI, 1.9 to 54.4). Significantly fewer women of child-bearing age (defined as <40 yrs) with GBS UTI were pregnant than women of child-bearing age who were controls (37.5% [6/16] versus 81.4% [35/43]; $P = 0.003$). No significant effects of race were observed (data not shown). Overall, these results were consistent with an alternative analysis where we compared 62 GBS UTI case patients to "all other patients" who were GBS culture positive but did not have acute symptomatic GBS UTI as defined by the exclusion criteria (n = 325) (data not shown).

GBS serotypes and capsular sequence types. Twenty-two of 387 (5.7%) isolates were nontypeable (NT) by latex agglutination, and 7 (1.8%) were NT by mPCR/RLB. Results were consistent between the two methods for 331/387 (85.5%) isolates. After retesting by both methods, 45/56 (80.4%) isolates for which results were discrepant were serotyped by mPCR/RLB as being serotype V after being NT (16) or identified as other serotypes (serotype VIII, 15 isolates; serotype II, 6 isolates; and serotypes Ia, Ib, IV, and III, 2 isolates each) by latex agglutination. Four isolates that were NT by mPCR/RLB were

TABLE 2. Molecular serotyping data for GBS UTI isolates in this study

GBS serotype	No. (%) of GBS isolates[c]				
	All specimens (n = 387)[a]	GBS UTI cases[b]			Controls[d] (n = 51)
		UA +ve[c] (n= 31)	UA ND[g] (n = 31)	All (n = 62)	
Ia	81 (21)	6 (19)	8 (26)	14 (23)	8 (16)
Ib	31 (8)	2 (7)	3 (10)	5 (8)	5 (10)
II	69 (18)	5 (16)	3 (10)	7 (11)	12 (24)
III	48 (12)[e]	8 (26)	5 (16)	13 (21)[e]	5 (10)
IV	24 (6)	2 (7)	1 (3)	3 (5)	5 (10)
V	125 (32)	8 (26)	12 (37)	20 (32)	10 (20)
VI	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
VII	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
VIII	2 (1)	0 (0)	0 (0)	0 (0)	1 (2)
NT[f]	7 (2)	0 (0)	0 (0)	0 (0)	5 (10)
IX	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)

[a] Consecutive urine specimens sent for culture from which GBS was isolated.
[b] Patients with at least one symptom of UTI and pure growth of GBS of $>10^7$ CFU/liter.
[c] +ve (consistent with UTI), positive for leukocyte esterase and pyuria.
[d] Subjects without symptoms from whose urine GBS was isolated in counts of $<10^7$ CFU/liter.
[e] Difference between the prevalence of serotype III among all GBS UTI case patients and all other non-GBS UTI cases (n = 325), determined to be significant by Pearson χ^2 analysis (P = 0.026).
[f] These isolates were identified using antisera as being NT (three isolates) or belonging to serotypes II (two isolates), IV (one isolate), and V (one isolate).
[g] ND, not done.

identified, using antisera, as being serotypes II (two isolates), IV (one isolate), and V (one isolate). Mismatches for other isolates were as follows (latex agglutination-mPCR/RLB shown): serotype II-serotype Ia (two isolates), serotype II-serotype III (one isolate), serotype IV-serotype II (one isolate), NT-serotype Ia (two isolates), and NT-serotype Ib (one isolate). A total of three isolates were identified as being NT by both methods. Discrepant results for the most common mismatch (serotype VIII-serotype V) were confirmed by sequencing of the mPCR amplicon. The mPCR/RLB serotyping results were accepted as definitive and are shown in Table 2.

The most prevalent serotypes among the 62 cases of GBS UTI were serotypes V, Ia, and III (Table 2), which together account for 76% of cases. Serotypes II, Ib, and IV accounted for the remaining 24% of cases. Serotype III and V GBS were the only serotypes that were more frequently isolated from UTI case patients than from controls (21% versus 10% and 32% versus 20%, respectively) (Table 2), but the differences were not significant. The trend of an increased prevalence of serotype III GBS was statistically significant when we compared 62 GBS UTI case patients with "all other patients" who were GBS culture positive but did not have acute symptomatic GBS UTI as defined by the exclusion criteria (21% [13/62] versus 10.8% [35/325]; P = 0.026), but there was no difference in the prevalence of serotype V GBS between case patients and "all other patients" (32% [20/62] versus 32.3% [105/325]; P = 0.994).

Antibiotic susceptibilities. Disc diffusion analysis of all 387 GBS isolates demonstrated that they were uniformly susceptible to amoxicillin (amoxicilline) with clavulanic acid, penicillin, linezolid, and chloramphenicol. A considerable proportion of isolates was resistant to clindamycin (102/387 [26.4%]) and erythromycin (153/387 [39.5%]), and the majority was resistant to tetracycline (311/387 [80.4%]), cefoxitin (343/387 [88.7%]), and trimethoprim with sulfamethoxazole (380/387 [98.3%]).

DISCUSSION

The uropathogenic potential of GBS prompted the current study to better define the clinical and microbiological features of GBS UTI and the serotypes that are predominately associated with disease. Among 387 patients who had GBS cultured from urine, we identified 62 UTI cases on the basis of single-organism bacteriuria and symptoms; diagnosis was confirmed in half of these cases where UA was performed. Diagnostic strategies for UTI vary substantially between clinicians (11, 14, 21); however, patients with a combination of symptoms have a high probability of UTI (5, 27). Pyuria concurrently with bacteriuria may constitute diagnostic criteria (35), as does $>10^6$ CFU/liter of a uropathogen for cystitis (27, 33). We regarded single-organism GBS bacteriuria of $>10^7$ CFU/liter and at least one UTI symptom as being a probable case of UTI and used urinary leukocyte esterase with significant pyuria as being confirmatory for diagnosis. Our criteria excluded contaminated samples and low-grade ABU, which are especially common among urine cultures that are positive for GBS (2, 28, 29) and elderly individuals (13). The majority of 62 GBS UTI cases in this study were uncomplicated cystitis, although diagnostic features for pyelonephritis (27) identified this in almost 20% of cases overall. It is noteworthy that our exclusion criteria limited the GBS UTI case definition to only those patients with single-organism GBS bacteriuria. Based on the prevalence of single-organism cultures and the ratio of GBS UTI to ABU (1:2), it is likely that up to one-third and one-quarter of the 387 urine cultures that were positive for GBS in this study represent GBS UTI and ABU, respectively. The total isolation rate of GBS from 34,367 urine cultures of 1.1% in this study is consistent with data from previous reports on the prevalence of GBS UTI among adults (7, 26, 30).

Several patients in this study with multiple symptoms of UTI and positive UA findings had GBS bacteriuria of between 10^7 and 10^8 CFU/liter, which is consistent with reports that up to 30% of women with cystitis present with bacteriuria of $<10^8$ CFU/liter. In addition, there were several symptomatic patients who presented with clinical features consistent with acute cystitis including positive UA findings but who were excluded from our case definition of GBS UTI because of single-organism bacteriuria of only between 10^6 and 10^7 CFU/liter at the time of analysis. For one such patient, GBS bacteriuria increased fourfold over a 6-h period, highlighting the dynamic nature of bacteriuria as an indicator of GBS UTI. These findings support the notion that low-grade bacteriuria ($>10^6$ CFU/liter) of a uropathogen including GBS may be indicative of cystitis in symptomatic individuals (5, 27, 33).

Urinary tract abnormalities, chronic renal failure (26), diabetes mellitus (32), and corticosteroid use (9) are among risk factors previously associated with GBS UTI. In this study, the rate of diabetes and chronic kidney disease among GBS UTI case patients did not differ significantly from the rate among control subjects who had GBS isolated from urine in the absence of symptoms. Multivariate analysis, however, revealed significant effects of a prior history of UTI, which is compara-

ble to data from studies that have identified a history of UTI as being a risk factor for disease caused by other uropathogens including *Enterobacter cloacae* (37). Increased age was also significantly associated with GBS UTI.

Nontypeable and serotype III GBS have been identified as being causes of UTI (23, 29). In this study, serotype III was the only type that was more frequently associated with acute disease than other serotypes. Future molecular studies will be important to determine the clonal types of serotype III uropathogenic GBS since only a few clones cause early- and late-onset neonatal disease (38). Recovery of multiple additional serotypes from case patients, particularly of serotypes II, V, and Ia, shows for the first time that a broader spectrum of GBS serotypes causes UTI than previously recognized. Notably, NT GBS accounted for no cases of GBS UTI in this study, in contrast to data from previous reports (23, 29). Molecular serotyping is more specific than antiserum-based assays (17), which are confounded by variable expression and partial sharing of GBS capsular antigens. It is possible that high prevalence rates of NT GBS among patients with UTI in previous studies may represent novel MSs that could not be classified by agglutination approaches. Very few GBS isolates are NT by mPCR/RLB; those that are can usually be shown to have mutations in the serotype-specific target region. The high proportion of MS V isolates that were NT or gave conflicting results with latex agglutination is consistent with results of a recent study (16). In a large collection of NT GBS isolates, we showed that serotype V was the most common MS identified. Reactions of these isolates with discordant antisera (especially serotype VIII) may reflect low titers of antisera and/or cross-reactivity. These findings underscore the limitations of agglutination serotyping approaches for some GBS in epidemiological surveys (15).

A high rate of resistance to macrolides was observed among GBS isolates in this study. These findings are consistent with a recent survey of GBS in the United States, where Manning et al. demonstrated higher-than-expected frequencies of macrolide resistance among GBS in nonpregnant women (22). Trends of increasing antibiotic resistance may reflect clonal dissemination and horizontal transfer of resistance genes among GBS, which occurs among certain GBS serotypes (31).

In summary, GBS UTI in this study occurred mostly as uncomplicated cystitis in women over the age of 50 years in the absence of chronic underlying disease but was associated with a prior history of UTI. GBS UTI was caused by a more diverse spectrum of serotypes than previously recognized, with the exception of NT GBS. Relatively high rates of treatment failure and poor clinical outcomes have been associated with GBS UTI (26). Further longer-term surveillance studies will help to better define the clinical features and serotypes associated with these important infections.

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Diversity of Group B Streptococcus Serotypes Causing Urinary Tract Infection in Adults

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Volume 47, no. 7, p. 2055–2060, 2009. Page 2056, column 1, line 26: "$\geq 10^7$ white blood cells" should read "≥ 10 white blood cells."

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