Invasive Disease Due to Group B Streptococcal Infection in Adults: Results From a Canadian, Population-Based, Active Laboratory Surveillance Study—1996

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In 1996, a population-based surveillance program for invasive adult group B streptococcal (GBS) diseases in Canada was undertaken, to define the epidemiologic and microbiologic characteristics of the disease. Nine public health units across Canada, representing 9.6% of the population, participated in the program. In total, 106 culture-positive cases of invasive adult GBS disease were reported, which represented an incidence rate 4.6 per 100,000 adults (41/100,000 for pregnant and 4.1/100,000 for nonpregnant adults). Sixty-two (58.5%) of the 106 cases occurred in females, and, of these, 15 (14.2%) were associated with pregnancy. Serotype V was the most common, accounting for 31% of the 90 GBS isolates typed (26.7% of nonpregnant and 4.4% of pregnant cases). This was followed by serotypes III (19%), Ia (17%), Ib (10%), II (9%), and VII (1%). Thirteen percent were nontypable. All isolates were susceptible to penicillin, ampicillin, and vancomycin. Resistance to erythromycin and clindamycin was 6.7% and 4.4%, respectively.

Group B streptococcal (GBS) disease is an important cause of illness in newborns, pregnant women, and nonpregnant adults with underlying medical conditions [1–4]. GBS disease also causes severe morbidity during pregnancy and, in the neonate, infection that can lead to meningitis [5–7]. Infections in the elderly include relatively minor urinary tract infections and severe invasive disease manifestations, such as endocarditis and other clinical problems with high mortality [4, 8]. The severity of disease caused by GBS isolates, in all age groups, has made the development of a vaccine a major focus of research [9]. One vaccine component candidate has been the GBS polysaccharide capsule. Efforts to develop a vaccine against severe invasive GBS disease will require extensive knowledge of the population affected and of the capsular serotypes circulating within those target populations.

For Canada there are no published, population-based data that document overall and age-specific incidences or epidemiologic and microbiologic characteristics of invasive GBS disease in adults. Such data are critical for assessing the burden of invasive GBS disease, providing information for cost-benefit assessment of vaccines, monitoring serotype distribution, and assessing the impact of antimicrobial resistance. To provide this information, in 1996 the Canadian Sentinel Health Unit Surveillance System (SHUSS) conducted population-based, active laboratory surveillance for invasive GBS disease. Our objectives were to identify the age-specific incidence of invasive GBS disease in a sample of the Canadian population, to determine the distribution of GBS serotypes, and to evaluate the level of antimicrobial resistance.

Materials and Methods

Epidemiologic and demographic information. The study was conducted from 1 January to 31 December 1996 and involved 9 health units across Canada in the Laboratory Centre for Disease Control’s SHUSS. For this study, adults were defined as anyone ≥15 years old. The health units were located in Halifax, Nova Scotia (adult population, 288,910); Prince Edward Island (adult population, 105,450); Sherbrooke, Quebec (adult population, 224,130); Kingston, Ontario (adult population, 141,950); Guelph, Ontario (adult population, 168,025); Winnipeg, Manitoba (adult population, 495,115); Saskatoon, Saskatchewan (adult population, 150,760); Edmonton, Alberta (adult population, 525,135), and Kelowna, British Columbia (adult population, 172,910) [10]. All units provide mandated public health services to their specific geographic areas. Their participation in the SHUSS was based on interest and on the likelihood that residents in the area would seek all of their health services from within the health unit. Four of the health units...
included large metropolitan centers; the others encompassed smaller urban and rural communities. In 1996, 2,853,975 persons lived within the health units of the SHUSS, and 2,235,461 (78%) were ≥15 years old [10].

Every 2 weeks between 1 January and 31 December 1996, site coordinators in each health unit contacted all microbiology laboratories within their jurisdictions, to identify GBS isolates in adults. All laboratories that process clinical microbiologic specimens in each health unit were surveyed. The survey included specimens from hospital inpatients and outpatients. Only isolates recovered by culture from a sterile body site—including soft tissue (necrotic tissues, abscesses, ulcers, wounds, and cellulitis), blood, cerebrospinal fluid, and pleural, articular, or peritoneal fluid—were included. Isolates from urine, sputum, or bronchoalveolar lavage were not accepted. Epidemiologic and demographic information about each case was collected by chart review and recorded on a standardized data collection instrument (Bureau of Surveillance and Field Epidemiology, Laboratory Centre for Disease Control, Ottawa) that was forwarded to the Laboratory Centre for Disease Control for collation and analysis. In 1998, a validation study was carried out to assess ascertainment of eligible cases of GBS disease during the active surveillance period. This involved the laboratories’ accessing their records for 1996 and comparing cases of invasive GBS disease for 1996 to the cases originally captured during the active surveillance period. This comparison was done either by the laboratory or by the SHUSS site coordinator. The validation study was feasible in 7 of the 9 health units (Kelowna, Edmonton, Winnipeg, Guelph, Sherbrooke, Halifax, and Prince Edward Island). Epidemiologic and demographic data were analyzed by Epi Info 6.04b [11]. Age-specific calculations used population estimates from the 1996 Canadian census obtained from Statistics Canada; live birth and stillbirth information also was obtained from Statistics Canada [12].

Identification and typing of GBS isolates. Isolates were forwarded to the National Centre for Streptococcus (NCS) in Edmonton for confirmation of group and for serotyping and antimicrobial susceptibility testing. Grouping was performed by the capillary precipitin test by using Lancefield hot acid extracts and group-specific antisera (Difco Laboratories, Detroit) [13]. Serotyping was done on the hot acid extracts by Ouchterlony immunodiffusion by using type-specific antisera prepared in rabbits at the NCS [14]. The typing set included antisera specific for polysaccharide antigens Ia, Ib, II, III, IV, V, VI, VII, and VIII.

Antimicrobial susceptibility testing. Antibiotic susceptibility testing was performed by the agar dilution method according to National Committee for Clinical Laboratory Standards (NCCLS) by using Mueller Hinton agar supplemented with 5% sheep blood [15]. The MIC was determined for penicillin, ampicillin, clindamycin, erythromycin, and vancomycin. Interpretive standards published by the NCCLS were used to categorize the MICs as susceptible, intermediate, or resistant [16].

Results

Invasive GBS disease in Canada. From 1 January through 31 December 1996, 106 cases of adult invasive GBS disease were identified by SHUSS. This represents an annual incidence rate in adults of 4.6 per 100,000. The annual incidence rate for nonpregnant adults was 4.1 per 100,000 (table 1), and the maternal GBS infection rate was 41 per 100,000 live and stillbirths. Thirty-four percent of all cases occurred between July and September, followed by April–June (26.4%), January–March (21.7%), and October–December (17.9%). The site-specific incidence rates for nonpregnant adults (cases of nonpregnant adult invasive GBS disease/population of nonpregnant adults ≥15 years old) per 100,000 are as follows: Kelowna, 5.3 (9/170,710); Edmonton, 3.9 (20/516,081); Saskatoon, 4.0 (6/147,892); Winnipeg, 5.6 (27/486,871); Guelph, 3.0 (5/165,015); Kingston, 6.4 (9/140,007); Sherbrooke, 2.7 (6/220,825); Halifax, 2.5 (7/284,312); and Prince Edward Island, 1.9 (2/103,748).

GBS disease in nonpregnant adults. There were 91 cases of GBS disease among nonpregnant adults. Of these, 51.6% of the nonpregnant cases occurred in women. GBS were isolated from blood, soft tissue, or joint/articular fluid in 98% of the nonpregnant cases (table 2). A review of the clinical records of the 91 adult cases found the most frequent clinical presentations were soft-tissue infections (necrotic tissues, abscesses, cellulitis, ulcers, and wounds), bacteremia with no defined site of infection, arthritis/bursitis, and pneumonia or an unspecified lower respiratory tract infection (table 2).

Sixty-three (69.2%) of the 91 nonpregnant case patients had ≥1 underlying medical condition or predisposing factor. In decreasing order of frequency, these were diabetes, residence in a nursing home, hospitalization, and cancer (table 2). More than one-third (34.2%) of the case patients with soft-tissue infections had diabetes mellitus as an underlying condition. Four of the 8 case patients with osteomyelitis reported diabetes mellitus. Among the 11 nonpregnant cases associated with nosocomial transmission, 5 (45.5%) patients had undergone a medical procedure (surgery, cystoscopy, or biopsy) before developing the GBS infection. Two others had central catheters, and 1 had a urinary catheter. The other 3 cases had been hospitalized ≥48 h before the first positive culture, but the exact mechanism of transmission was not determined. No human immunodeficiency virus infections were reported.

GBS disease associated with pregnancy. There were 15 cases of GBS disease during pregnancy, an incidence rate of 41 per 100,000 live and stillbirths (95% confidence interval, 23.6–68.7). The 15 pregnancy-related case patients ranged in age from 19.3 to 45.1 years (mean, 30.3 years). Eleven (73.3%) of these women delivered a live child, 2 (13.3%) had a stillbirth, 1 (6.6%) had
Clinical presentation

<table>
<thead>
<tr>
<th>Source of GBS isolate</th>
<th>All (n = 91)</th>
<th>&lt;5 years old (n = 49)</th>
<th>≥65 years old (n = 42)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>53 (58.2)</td>
<td>19 (38.8)</td>
<td>34 (80.9)</td>
</tr>
<tr>
<td>Soft tissue</td>
<td>13 (14.3)</td>
<td>13 (26.5)</td>
<td></td>
</tr>
<tr>
<td>Joint (articular fluid or tissue)</td>
<td>9 (9.9)</td>
<td>7 (14.3)</td>
<td>2 (4.8)</td>
</tr>
<tr>
<td>Blood and soft tissue</td>
<td>5 (5.5)</td>
<td>3 (6.1)</td>
<td>2 (4.8)</td>
</tr>
<tr>
<td>Blood and joint</td>
<td>2 (2.2)</td>
<td>2 (4.1)</td>
<td></td>
</tr>
<tr>
<td>Joint and soft tissue</td>
<td>3 (3.3)</td>
<td>1 (2.0)</td>
<td>2 (4.8)</td>
</tr>
<tr>
<td>Blood and bile/wound</td>
<td>1 (1.1)</td>
<td>1 (2.4)</td>
<td></td>
</tr>
<tr>
<td>Blood and urine</td>
<td>1 (1.1)</td>
<td>1 (2.4)</td>
<td></td>
</tr>
<tr>
<td>Blood, soft tissue, and joint</td>
<td>1 (1.1)</td>
<td>1 (2.0)</td>
<td></td>
</tr>
<tr>
<td>Cerebrospinal fluid, blood, and soft tissue</td>
<td>1 (1.1)</td>
<td>1 (2.0)</td>
<td></td>
</tr>
<tr>
<td>Axillary lymph node</td>
<td>1 (2.2)</td>
<td>1 (2.0)</td>
<td></td>
</tr>
<tr>
<td>Not specified</td>
<td>1 (1.1)</td>
<td>1 (2.0)</td>
<td></td>
</tr>
</tbody>
</table>

Clinical presentation^a

- Soft tissue: 38 (41.7), 23 (46.9), 15 (35.7)
- Bacteremia with no defined site of infection: 18 (19.8), 6 (12.2), 12 (28.6)
- Arthritis/bursitis: 16 (17.6), 9 (18.4), 7 (16.7)
- Pneumonia/unspecified lower respiratory tract infection: 13 (14.3), 6 (12.2), 7 (16.7)
- Urinary tract infection: 8 (8.8), 2 (4.1), 6 (14.3)
- Osteomyelitis: 8 (8.8), 5 (10.2), 3 (7.1)
- Central catheter: 2 (2.2), 1 (2.0), 1 (2.4)
- Meningitis: 1 (1.1), 1 (2.0)
- Peritonitis and septic shock: 1 (1.1), 1 (2.0)
- Infected echinococcal cyst: 1 (1.1), 1 (2.0)
- Pancreatitis: 1 (1.1), 1 (2.0)
- Lymph node infection: 1 (1.1), 1 (2.0)
- Diskitis: 1 (1.1), 1 (2.0)
- Vascular (aneurysm) infection: 1 (1.1), 1 (2.4)

Underlying conditions^a

- Diabetes mellitus: 18 (19.8), 14 (28.6), 4 (9.5)
- Resident in nursing home: 13 (14.3), 1 (2.0), 12 (28.6)
- Hospital acquired infection: 11 (12.1), 4 (8.2), 7 (16.7)
- Cancer: 11 (12.1), 8 (16.3), 3 (7.1)
- Cardiac disease: 9 (9.9), 3 (6.1), 6 (14.3)
- Gastrointestinal disease: 7 (7.7), 2 (4.1), 5 (11.9)
- Previous trauma to infected site: 5 (5.5), 4 (8.2), 1 (2.4)
- Joint prosthesis: 5 (5.5), 2 (4.1), 3 (7.1)
- Immune disorder: 5 (5.5), 1 (2.0), 4 (9.5)
- Liver disease: 4 (4.4), 1 (2.0), 3 (7.1)
- Vascular disease: 4 (4.4), 1 (2.0), 3 (7.1)
- Neurologic disorders: 4 (4.4), 2 (4.1), 2 (4.8)
- Respiratory disease: 3 (3.3), 1 (2.0), 2 (4.8)
- Hydramnios: 1 (1.1), 1 (2.0)
- Obstetric renal disease: 2 (2.2), 1 (2.0), 1 (2.4)
- Unknown/not specified: 28 (30.8), 19 (38.8), 9 (21.4)

^a Patient may have ≥1 clinical presentation or underlying condition.

GBS disease. 

Five (33.3%) of the pregnancy-related cases presented as endometritis, 4 (26.7%) with chorioamnionitis, 1 (6.6%) with a surgical abortion 2 days before diagnosis, and 1 (6.6%) was diagnosed with GBS infection after a dilation and curettage after a spontaneous abortion. No live births were infected with GBS, and there were no deaths among pregnant women with GBS disease.

Outcome of infection in nonpregnant adults. Seven of the 91 case patients died while hospitalized. Five deaths were attributed to GBS infection (overall case-fatality rate, 5.5%). No single site had ≥2 fatalities attributed to invasive GBS disease (Charlottetown, Guelph, and Winnipeg, 1 death each; Kelowna, 2 deaths). The deaths included 4 men (a 45-year-old who died of pneumonia, a 60-year-old who died of peritonitis and septic shock, a 74-year-old with aortic aneurysms who died of sepsis, and a 36-year-old with cardiomyopathy who presented with abdominal cellulitis and died of sepsis) and a 102-year-old woman who died of acute renal failure and sepsis. The mean interval between positive culture and death was 4.4 days. Six of the 99 case patients who survived the infections were treated as outpatients. Fifty-one (54.8%) of the 93 hospitalized patients were still hospitalized 10 days after their diagnosis with GBS disease, including 2 of the pregnancy-related cases.

Validation study. In 1998, a validation study was performed to capture cases of GBS disease that may have been missed during the active surveillance period, when 94 cases were found. The study was done in 7 of the 9 participating sites from which 77 (82%) of the 94 original cases were identified. Twelve additional cases were found—4 in pregnant women and 8 in nonpregnant adults. Seven (58%) of the additional cases were from 1 health unit; 2 health units identified no additional cases. Isolates were not available for 11 of the 12 cases.

Serotype distribution. Isolates from 90 cases were available and were submitted to the NCS for serotyping. Type V was the most frequent serotype (31.1% of isolates) that caused invasive GBS disease in adults, followed by types III, Ia, Ib, and II (table 3). Serotypes IV, VI, and VIII were not detected. Of the serotype V isolates, 26.7% were from nonpregnant cases and 4.4% were from pregnant cases. Because of the high percentage of serotype V isolates, associations between serotype V and clinical presentations or underlying conditions were investigated. Serotype V was isolated from 5 (62.5%) of the 8 case patients who presented with osteomyelitis. The odds ratio of developing osteomyelitis associated with serotype V in patients with a GBS infection was 3.7. No other serotype was associated with any clinical presentation or underlying condition in any significant magnitude.

Antimicrobial resistance. The 90 GBS isolates tested were susceptible to penicillin (MIC₉₀, 0.06 μg/mL), ampicillin (MIC₉₀, 0.125 μg/mL), and vancomycin (MIC₉₀, 0.5 μg/mL). Six isolates (6.7%) were resistant to erythromycin (eryR), and 3 of these 6 also were resistant to clindamycin (clindR). One isolate was resistant to clindamycin only. Isolates susceptible to erytho-
mycin and clindamycin had MIC₉₀ of 0.125 μg/mL for both antibiotics. The serotype distribution among all resistant cases was as follows: type III, 2 (1 eryR + clindR, 1 eryR); type V, 2 (1 eryR + clindR, 1 eryR); type Ia, 1 (clindR); type Ib, 1 (eryR); nontypeable, 1 (eryR + clindR).

### Table 3. Distribution of group B streptococcal serotypes.

<table>
<thead>
<tr>
<th>Serotype</th>
<th>Pregnant (n = 11)</th>
<th>Nonpregnant (n = 79)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia</td>
<td>3 (27.3)</td>
<td>12 (15.2)</td>
</tr>
<tr>
<td>Ib</td>
<td>1 (9.1)</td>
<td>8 (10.1)</td>
</tr>
<tr>
<td>II</td>
<td>0</td>
<td>8 (10.1)</td>
</tr>
<tr>
<td>III</td>
<td>2 (18.2)</td>
<td>15 (19)</td>
</tr>
<tr>
<td>V</td>
<td>4 (36.4)</td>
<td>24 (30.4)</td>
</tr>
<tr>
<td>VII</td>
<td>1 (9.1)</td>
<td>0</td>
</tr>
<tr>
<td>Nontypeable</td>
<td>0</td>
<td>12 (15.2)</td>
</tr>
</tbody>
</table>

## Discussion

This study is the first population-based, prospective surveillance of invasive GBS disease in Canadian adults. We found an incidence rate of 4.1 per 100,000 nonpregnant adults in Canada in 1996. In comparison, a series of retrospective and prospective surveillance studies of invasive GBS disease in metropolitan Atlanta found an incidence rate of GBS invasive disease in nonpregnant adults of 2.4 per 100,000 in 1982–1983, 4.3 per 100,000 in 1989–1990, and 5.9 per 100,000 in 1992–1993 [6, 7, 17]. These Atlanta-based surveys document a steadily increasing trend in the incidence of GBS invasive disease from 1982 to 1993 in this population. Another surveillance program in Maryland recently reported an incidence rate of 6.5 per 100,000 in nonpregnant adults, slightly higher than that in Atlanta during the same year [18].

The incidence of GBS invasive disease in pregnant women in Atlanta has also risen steadily from 22 per 100,000 in 1982–1983 to 66.4 per 100,000 in 1992–1993 [6, 7, 17]. Our invasive disease rate was 41 per 100,000 live and stillbirths in Canada during 1996. Even though our disease rate is not as high as that in the US-based surveys, given the trend of GBS disease in the United States, the need for continued surveillance for this organism in Canada is clear. In relation to fetal mortality, 2 stillbirths occurred during our survey period. One was suspected to be caused by GBS disease, on the basis of positive cord tissue and vaginal/rectal cultures. In the second stillborn case, the role of GBS disease is unclear, although this agent is the most probable cause.

To augment our assessment of GBS disease incidence, we conducted a validation study of 7 of the 9 SHUSS. The survey resulted in 12 additional cases of invasive adult GBS disease. Of interest, 7 of the 12 were from one health unit and 3 from another. These increases in cases of GBS disease resulted in incidence rates at these 2 sites that approached that seen for the national average. The 2 sites that did not participate in the validation had incidence rates similar to the national average.

The nonpregnant incidence rates for the SHUSS varied between sites and ranged from a low of 1.93 per 100,000 to a high of 6.43 per 100,000. Many explanations are possible for this range. One is related to the population in each health unit. Five sites had populations below 200,000. Therefore, an increase or decrease of 2 or 3 cases can lead to large swings in the incidence rates for these sites. Another possibility relates to how physicians in each health unit treat suspected cases of invasive GBS disease. Physicians in one health unit may request a microbiologic investigation on suspected cases, whereas others may elect to treat the cases without microbiologic information. Therefore, it is possible that differing physician practices by site may result in variations in incidence. A third possibility may be different microbiology practices of the laboratories that isolate and identify GBS. We realize that processes such as accreditation exist to address this issue; however, not all microbiology laboratories go through the same accreditation process. Therefore, there is a potential for variation in GBS isolation and identification between laboratories, which may partially explain the variations seen in incidence rates. There may be other factors that we have not addressed, such as socioeconomic factors and ethnicity. However, it is likely that the 3 factors previously discussed account for most of the rate variation between sites.

The case-fatality rate in this SHUSS for invasive GBS disease in nonpregnant adults was 5.5%. No single site had >2 fatalities attributable to invasive GBS infection. Farley et al. [7], in an Atlanta-based survey, reported a mortality rate of 21% among nonpregnant adults with invasive GBS infections. Explanations for the different mortality rates between the 2 surveys are most likely multifactorial and would require a detailed analysis of the 2 populations surveyed. The case-fatality rate of 5.5% in Canada is significant enough to highlight the importance of developing strategies to prevent GBS infection in this country.

The most common clinical presentation in adults with GBS invasive disease was soft-tissue infections (41.7%). The ability of GBS disease to manifest as skin and soft-tissue infections is well documented and is the most frequent GBS infection in nonpregnant adults [4, 8, 19]. These infections typically present as cellulitis, decubitus ulcers, or abscesses, as was seen in the patients in this study. Of the patients with soft-tissue infections, 34.2% reported diabetes mellitus as an underlying condition, which suggests that this clinical condition may predispose an individual to GBS infections. Others have reported that GBS infections tend to occur in patients with significant underlying disease [6–8, 19]. Age ≥65 years is also a risk factor for GBS infection. In our study, the incidence doubled from age group 50–64 years (5.9/100,000) to ≥65 years (11.9/100,000). This probably is due to the increase in underlying conditions associated with increasing age, which predispose persons to GBS invasive disease.
Gardam et al. [20] recently described 3 cases of GBS disease in southern Ontario and Quebec in 1996 and 1997 that caused necrotizing fasciitis [20]. Our surveillance study failed to find any cases of necrotizing fasciitis. This probably is due to the rarity of this type of GBS disease manifestation.

Determination of the circulating GBS serotypes that cause invasive GBS disease is important if a vaccine derived from the polysaccharide capsule is to be used to protect high-incidence populations such as the elderly and neonates. Recognized serotypes are Ia, Ib, II, III, IV, V, VI, VII, and VIII [21–25]. Serotypes Ia, Ib, II, III, and, most recently, V have been isolated from neonates with severe invasive disease [17, 26]. Serotype V is one of the more frequent serotypes isolated in adults with invasive disease in the United States and is included in a vaccine being evaluated in a clinical trial [17, 26–28]. The remaining serotypes are infrequently isolated in North America. In this population-based Canadian survey, the most frequent GBS serotype that caused invasive disease in adults was V, followed by III and Ia. These 3 serotypes make up 62% of the circulating serotypes that cause invasive adult disease in Canada. In patients >65 years old who presented clinically with soft-tissue infections, 40% of the GBS isolates were serotype V. Serotype V was also strongly associated with osteomyelitis and diabetes mellitus, although the significance of this is unclear.

In a prospective population-based survey of invasive GBS disease in Atlanta from June 1992 to June 1993, serotype Ia accounted for >34% of the adult cases, followed by serotypes V (30%), III (20%), Ib (8%), and II (6%) [17]. The major difference in our results is that Ia occurred about half as frequently as in Atlanta. The increase in serotype V in the United States appears to be due to 1 predominate subtype that accounts for most serotype V isolates typed by pulsed-field gel electrophoresis (PFGE) [28]. Whether this is also the case in our collection is under investigation. Only 1 isolate was determined to be serotype VII. No IV, VI, or VIII isolates were found, which is consistent with other North American surveillance studies [17, 18].

More than 13% of our GBS isolates were nontypeable (all from nonpregnant adults). The unexpectedly high rate is in contrast to previous studies that have reported 2%–4% of invasive isolates as nontypeable [17, 18]. Of interest, the NCS, which performs passive surveillance for GBS in Canada on isolates from invasive disease only, found the prevalence of nontypeable GBS serotypes in their collection to be 6% for both 1997 and 1998 (authors’ unpublished data). This contrast may reflect differences in active versus passive surveillance, but other explanations are being sought. We are in the process of analyzing these 12 nontypeable strains by PFGE, to determine if these isolates are due to 1 or multiple strains.

Penicillin remains the treatment of choice for serious streptococcal infections. In our study, all GBS isolates were susceptible to penicillin, ampicillin, and vancomycin. However, erythromycin resistance was 6.7%, and clindamycin resistance was 4.4%. Previous investigators have reported susceptibility rates similar to ours. Fernandez et al. [29] determined the susceptibility profiles of 229 GBS isolates from patients with invasive infections in Houston and found an erythromycin resistance rate of 7.7% and a clindamycin resistance rate of 3.4% [29]. In conclusion, specific adult populations in Canada are at increased risk of acquiring invasive GBS disease. These include pregnant women, persons ≥65 years old, persons with underlying conditions, such as diabetes mellitus and cancer, and those residing in nursing homes or hospitalized. The most prevalent circulating GBS serotype that caused invasive GBS disease in adults in Canada during 1996 was serotype V. This serotype would be an important candidate for inclusion in a polysaccharide vaccine targeted toward the Canadian adult population. The GBS isolates examined in this study were universally susceptible to penicillin, which confirms its continuing effectiveness as the antibiotic of choice for treatment of GBS infections.

Sentinel Health Unit Surveillance System Site Coordinators

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