Group B streptococci (GBS) cause severe invasive disease in both neonates and adults. Understanding the epidemiology of GBS provides information that can include determining disease prevalence rates in defined populations and geographic regions, documenting the success of GBS screening programs, and understanding antimicrobial susceptibility patterns. In Alberta, only neonatal invasive GBS (iGBS) disease is notifiable to health authorities. We performed a surveillance study of iGBS in Alberta, Canada, from 2003 to 2013. Over the 11-year period, the disease incidence rate increased from a low of 3.92 cases/100,000 population to a high of 5.99 cases/100,000 population. The capsular polysaccharide serotypes (CPSs) found were CPS III (20.3%), CPS V (19.1%), CPS Ia (18.9%), CPS Ib (12.7%), CPS II (11.1%), CPS IV (6.3%), and nontypeable GBS (9.4%). Rates of early-onset disease (0 to 7 days) increased from 0.15 cases/1,000 live births (in 2003) to 0.34 cases/1,000 live births (in 2013). Rates of late-onset disease (>7 to 90 days) also rose, from 0.15 cases/1,000 live births (in 2003) to 0.39 cases/1,000 live births (in 2013). Alberta also experienced an increase in CPS IV isolates, from 2 cases (in 2003) to 24 cases (in 2013), of which the majority were hvgA positive (86.6%). The predominant sequence type (ST) in 2013 was ST459. Erythromycin resistance rose from 23.6% to 43.9% (in 2013). Clindamycin resistance also increased, from 12.2% to 32.5%. In summary, Alberta, Canada, has experienced an increase in GBS disease; the increase includes both neonatal and adult disease. CPS IV cases also notably increased during the surveillance period, as did resistance to erythromycin and clindamycin.

From a global perspective, GBS can also be typed via multilocus sequence typing (MLST) (12). A major hypervirulent MLST clone currently circulating globally is clonal complex 17 (CC17) (9, 13–16). This lineage is strongly associated with the majority of GBS LOD infections, which are characterized by meningitis in infants after the first week of life (13). CC17 clones possess a hypervirulent GBS adhesin gene, hvgA, which is usually present in CPS III strains (9, 13–16). There have been reports of CPS IV isolates that contain CC17-specific hvgA (9); therefore, the PCR amplification of hvgA that had been used to assign GBS strains to CC17 (16) was found to be inconclusive (9). However, PCR amplification could be used as an initial screening step to assign strains to CC17, with confirmation via MLST being required.

Monitoring the ever-changing epidemiology of invasive GBS disease in a large population provides important information regarding disease rates, polysaccharide capsule changes, sequence type (ST) variations, and antimicrobial susceptibility trends. This documentation is critical, as it provides data that can guide clinicians in treating patients with iGBS and help public health officials understand changes in iGBS disease trends. This information takes on added value as more attention is directed to GBS vaccine development, especially for protection against neonatal iGBS disease. The objective of this report is to describe the epidemiology of iGBS disease in the Alberta population, with respect to CPS distribution and GBS antimicrobial susceptibility, from 2003 to 2013.

**Citation**


**Editor:** K. C. Carroll, The Johns Hopkins University School of Medicine

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TABLE 1 Total numbers of iGBS isolates in 2003 to 2013 by year and CPS

<table>
<thead>
<tr>
<th>Year</th>
<th>CPS Ia</th>
<th>CPS Ib</th>
<th>CPS II</th>
<th>CPS III</th>
<th>CPS IV</th>
<th>CPS V</th>
<th>CPS VI</th>
<th>CPS VII</th>
<th>CPS VIII</th>
<th>CPS IX</th>
<th>NT*</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td>2003</td>
<td>23</td>
<td>13</td>
<td>14</td>
<td>16</td>
<td>2</td>
<td>41</td>
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<td>0</td>
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<td>2004</td>
<td>8</td>
<td>11</td>
<td>12</td>
<td>25</td>
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<td>21</td>
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<td>21</td>
<td>2</td>
<td>28</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>15</td>
<td>109</td>
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<tr>
<td>2006</td>
<td>22</td>
<td>8</td>
<td>21</td>
<td>18</td>
<td>1</td>
<td>33</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>12</td>
<td>115</td>
</tr>
<tr>
<td>2007</td>
<td>22</td>
<td>17</td>
<td>12</td>
<td>26</td>
<td>3</td>
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<td>0</td>
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<td>0</td>
<td>0</td>
<td>1</td>
<td>3</td>
<td>8</td>
<td>181</td>
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<tr>
<td>2012</td>
<td>35</td>
<td>27</td>
<td>22</td>
<td>47</td>
<td>24</td>
<td>31</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>19</td>
<td>222</td>
</tr>
<tr>
<td>2013</td>
<td>46</td>
<td>42</td>
<td>21</td>
<td>52</td>
<td>24</td>
<td>23</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>23</td>
<td>234</td>
</tr>
</tbody>
</table>

Total 312 (18.9) 213 (12.7) 187 (11.1) 341 (20.3) 106 (6.3) 322 (19.1) 24 (1.4) 0 (0) 3 (0.2) 16 (1.0) 159 (9.4) 1,683 (100)

* NT, nontypeable.

MATERIALS AND METHODS

Epidemiological and demographic information and definitions. Neonatal iGBS disease is notifiable to the provincial public health authorities in Alberta, Canada. This requires all neonatal GBS isolates collected from sterile site specimens in Alberta to be forwarded to the Provincial Laboratory for Public Health (PLPH) for capsular serotyping. All isolates from iGBS disease cases other than neonatal disease are forwarded to the PLPH for capsular serotyping at the discretion of the submitting diagnostic laboratory in Alberta. The time period for the study was 1 January 2003 to 31 December 2013 (an 11-year period). The population of Alberta was 3,134,337 in 2003 and 4,107,762 in 2013 (http://www.ahw.gov.ab.ca/IHDA_Retrieval/ihdaData.do) [accessed 15 June 2015].

EOD and LOD were defined as cases occurring in neonates between 0 and 7 days of age and between 8 and 90 days of age, respectively. Children were defined as being 91 days to 14 years of age, and adults were defined as being ≥15 years of age. iGBS disease was defined as GBS infection isolated from a sterile site, such as soft tissues (necrotic tissues, abscesses, ulcers, wounds, or cellulitis), blood, cerebrospinal fluid (CSF), or pleural, articular, or peritoneal fluid. GBS isolates from urine, sputum, or bronchoalveolar lavage fluid were excluded. In addition to the submission of GBS isolates to the PLPH for serotyping, minimal demographic data, including the date of specimen collection, patient age, anatomical collection site, and patient sex, were collected.

GBS CPS typing. Isolates submitted to the PLPH were confirmed as GBS prior to CPS typing. CPS typing was performed using the Lancefield heat- acid extraction method followed by a double immunodiffusion method, as described previously (17, 18). The immunodiffusion CPS typing assay used for this study was based on reactions with antiserum raised against CPSs Ia, Ib, II, III, IV, V, VI, VII, and VIII. The specific antiserum panel was prepared in rabbits in the laboratory. PCR CPS typing was used for the identification of CPS IX, using the primer pair cpsI-9-F (5'-CTGTAATTTGCAGGATTTGTGATCG) and cpsI-9-R (5'-AATCATCCTTACATTTATCCTCCATT), which amplify target regions specific to CPS IX, as described previously (19).

PCR amplification of hvgA. A previously described PCR assay that targets a 210-bp region of the hvgA gene was used to identify hvgA-positive GBS isolates (14). The hvgA region was amplified by PCR using the primer pair ST-175 (5'-TAAAACTCTTCTTGACCCATTCC and ST-17AS (5'-ATACAAAATCCTGCTGACTCCGG) (16).

MLST assay and assignment to clonal clusters. Multilocus sequence typing (MLST) was carried out as described previously (12). Briefly, PCR was used to amplify internal (500-bp) fragments from seven housekeeping genes (adbP, pheS, atr, glnA, sdhA, gkK, and ikt), which were chosen on the basis of their chromosomal locations and sequence diversity. The seven PCR products were purified and sequenced, and an allele number was assigned to each fragment based on its sequence. Each isolate was assigned a sequence type (ST) based on the allelic profile of the seven amplicons, based on STs (http://pubmlst.org/sagalactiae) for MLST analysis.

Strains were grouped into CCs using eBURST software (20, 21). The default eBURST setting identified groups of related STs using the most stringent (conservative) definition, such that all members assigned to the same group shared identical alleles at six of the seven loci with at least one other member of the group. Singleton STs are STs that did not cluster into a CC.

Antimicrobial susceptibility testing. Antimicrobial susceptibility testing was performed with the disc diffusion method, as described by the National Committee for Clinical Laboratory Standards (NCCLS) (22). The antimicrobial agents assayed were penicillin, erythromycin, clindamycin, vancomycin, and chloramphenicol. All antimicrobial discs were obtained from Oxoid Inc. (Nepean, Canada). Interpretative standards published by the NCCLS were used to categorize the MIC results as susceptible, intermediate, or resistant (23).

Statistical analyses. CPS distributions with respect to age group, gender, and specimen source were compared using the χ² test. Incidence rates, i.e., the frequency with which GBS diseases occurred in a population over a period of time, with 95% confidence intervals (CIs) were calculated for each age group and gender. For categorical data such as the distribution of erythromycin- or clindamycin-nonsusceptible isolates among CPSs and age groups, analysis of variance (ANOVA) was performed. For nonparametric analysis, the Kruskal-Wallis test was used to compare the distribution of hvgA among CPSs. P values of <0.05 were considered statistically significant. Data were analyzed using SPSS version 23 (IBM SPSS Statistics).

RESULTS

CPS distributions and incidence rates. From 2003 to 2013, 1,683 nonduplicate GBS isolates from cases of iGBS disease in Alberta were submitted to the PLPH for CPS typing. Table 1 shows the distribution of GBS isolates presented by year and CPS. The overall isolate submission numbers increased from 123 isolates in 2003 to 234 isolates in 2013. CPS III (20.3% of total), CPS V (19.1%), and CPS Ia (18.9%) were the most predominant types, accounting for each age group and gender. For each gender and specimen source were compared using the χ² test, 0.001). CPS VII was not detected during the 11-year period surveyed. A total of 19 isolates (9.4%) were determined to be non-typeable (Table 1).

The overall incidence of iGBS disease by year increased over the
11 years surveyed, from 3.92 cases/100,000 population in 2003 to 5.99 cases/100,000 population in 2013 (Table 2). The incidence rates for all years combined were 4.59 cases/100,000 population (95% CI, 4.31 to 4.9 cases/100,000 population) and 3.99 cases/100,000 population (95% CI, 3.72 to 4.28 cases/100,000 population) among males and females, respectively, with 895/1,668 cases (53.65%) occurring in male patients. There were 15 cases for which sex was not indicated at the time of submission of the isolate to the PLPH.

A total of 264 cases of iGBS in neonates (≤90 days of age) were identified, of which 134 were categorized as EOD and 130 were categorized as LOD (Table 2). For both EOD and LOD, the numbers of cases showed a gradual increase from 2003 to 2013, with an average incidence for all years combined of 0.26 cases/1,000 live births (95% CI, 0.23 to 0.28 cases/1,000 live births). In 2013, the final year of the survey, the incidence of EOD was 0.34 cases/1,000 births (95% CI, 0.23 to 0.28 cases/1,000 live births). In 2013, the population of neonates was 11.6% of the total population, which contributed to the increase in the number of iGBS cases. The majority of iGBS cases involving persons ≤15 years of age (70% [n = 970]) were diagnosed by blood cultures alone.

CPS IV strains. A total of 106 CPS IV strains were identified during the survey period (Table 1). It was noted that CPS IV was rarely reported in 2003 to 2007; from 2008 to 2013, however, the numbers of CPS IV cases increased from a low of 12 cases (2003 to 2007) to a high of 94 cases (2008 to 2013) (Table 1). This rapid increase in the numbers of CPS IV cases prompted further characterization of these isolates. HvgA is a surface-anchored adhesion molecule that enables persistent colonization by GBS CC17 clones and contributes to meningitis in neonates (19). HvgA has also been observed previously in CPS IV strains (9, 20). We screened for the presence of hvgA-positive GBS strains in our collection of neonatal GBS isolates, as well as CPS IV isolates from all age groups, using a hvgA PCR assay (14). For the neonatal cases, the majority of hvgA-positive isolates reported here were CPS III (64% [n = 32]), followed by CPS IV (18% [n = 9]), CPS Ia (6% [n = 3]), CPS V (6% [n = 3]), CPS Ib (4% [n = 2]), and nontypeable strains (2% [n = 1]) (Kruskal Wallis test, P = 0.001) (Fig. 1). Of the CPS IV isolates from all ages, 86.6% were hvgA positive (91 hvgA-positive isolates/106 CPS IV isolates) (χ² test, P < 0.001) (Fig. 2). In the last year of the study (2013), all 22 of the viable CPS IV GBS isolates were found to be hvgA positive. We further characterized the CPS IV isolates from 2013 using MLST analysis. MLST of the 2013 CPS IV isolates identified 13 ST459 isolates (the most common ST), followed by 2 isolates each of ST2, ST3, and ST671 and 1 isolate each of ST136, ST196, and ST711 (χ² test, P < 0.001). Twenty of 22 isolates clustered in CC1. To determine
whether the increase in CPS IV was due to global expansion of ST459, four isolates were randomly selected from different time points (2003, 2009, 2010, and 2011) and their STs and CCs were identified; the four isolates were typed as ST459 in CC1.

**iGBS antimicrobial resistance rates in 2003 to 2013.** Antimicrobial susceptibility assays with penicillin, erythromycin, clindamycin, vancomycin, and chloramphenicol were performed for 98.5% (1,658/1,683 isolates), 98.3% (1,656/1,683 isolates), 98.6% (1,659/1,683 isolates), 98.6% (1,660/1,683 isolates), and 98.5% (1,657/1,683 isolates) of the isolates, respectively, over the 11-year period. All iGBS isolates were susceptible to penicillin, vancomycin, and chloramphenicol. Figure 3 displays a gradual increase in erythromycin nonsusceptibility, from 23.6% in 2003 to 43.8% in 2013. There was no significant difference in the numbers of erythromycin-nonsusceptible iGBS isolates derived from different age groups. The number of erythromycin-nonsusceptible iGBS isolates was significantly greater among CPS IV strains (86/107 isolates [80.4%]) (ANOVA, \(P < 0.001\)) (Table 5).

The proportion of clindamycin-nonsusceptible GBS isolates increased from 12.2% in 2003 to 32.3% in 2013. The majority of clindamycin-nonsusceptible strains were derived from persons 15 to 50 years of age, in comparison with EOD, LOD, children, and persons >50 years of age (ANOVA, \(P < 0.001\)). The clindamycin-nonsusceptible phenotype was most predominant in CPS IV (84/107 isolates [78.5%]; \(P < 0.001\)). Most of the iGBS isolates that were erythromycin resistant (63%) were also clindamycin resistant (94.6% [375 isolates]) (Pearson \(\chi^2\) test, \(P < 0.001\)).

**DISCUSSION**

GBS is one of the most important causal agents of bacterial pneumonia, meningitis, and sepsis among newborns in North America (1, 24, 25). The estimated incidence rate of neonatal GBS disease in Canada and the United States decreased from 1 to 3 cases per 1,000 live births in the early 1990s to 0.35 to 0.5 cases per 1,000 live births with the application of intrapartum chemoprophylactic prevention measures (10, 26–28). Our screening study took place after the implementation of the preventive measures in Canada. Overall, the incidence rate of EOD and LOD in Alberta in 2003 to 2013 was 0.26 cases per 1,000 live births, which is lower than the aforementioned rate in North America. However, there was a 2-fold increase in EOD cases from 2003 to 2013 (from 0.15 to 0.34 cases per 1,000 live births) even with implementation of the pre-

<table>
<thead>
<tr>
<th>Source</th>
<th>EOD</th>
<th>LOD</th>
<th>91 days to 14 yr</th>
<th>15–50 yr</th>
<th>&gt;50 yr</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>107</td>
<td>117</td>
<td>14 (77.8)</td>
<td>264 (62.1)</td>
<td>706 (73.5)</td>
<td>1,208 (72.4)</td>
</tr>
<tr>
<td>Joint/synovial fluid</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>33 (7.8)</td>
<td>105 (11.0)</td>
<td>138 (8.3)</td>
</tr>
<tr>
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<td>0</td>
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<td>65 (6.7)</td>
<td>114 (6.8)</td>
</tr>
<tr>
<td>Peritoneum/dialysate</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>19 (4.4)</td>
<td>16 (1.2)</td>
<td>35 (2.1)</td>
</tr>
<tr>
<td>Placental/cord</td>
<td>11</td>
<td>2</td>
<td>1 (5.6)</td>
<td>3 (0.7)</td>
<td>1 (0.1)</td>
<td>16 (1.0)</td>
</tr>
<tr>
<td>CSF</td>
<td>4</td>
<td>7</td>
<td>1 (5.6)</td>
<td>3 (0.7)</td>
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<td>16 (1.0)</td>
</tr>
<tr>
<td>Pleura</td>
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<td>0</td>
<td>0</td>
<td>2 (0.5)</td>
<td>9 (0.9)</td>
<td>11 (0.7)</td>
</tr>
<tr>
<td>Other</td>
<td>9</td>
<td>3</td>
<td>3 (16.6)</td>
<td>45 (10.6)</td>
<td>59 (6.1)</td>
<td>119 (7.1)</td>
</tr>
<tr>
<td>Total</td>
<td>134</td>
<td>130</td>
<td>18 (1.1)</td>
<td>425 (25.5)</td>
<td>961 (57.6)</td>
<td>1,668</td>
</tr>
</tbody>
</table>

* Fifteen of the 1,683 isolates were of unknown specimen source.

![FIG 1](http://jcm.asm.org/) Distribution of CPSs among hvgA-positive neonatal strains in 2003 to 2013. NT, nontypeable.

![FIG 2](http://jcm.asm.org/) Distribution of hvgA-positive and hvgA-negative CPS IV GBS isolates for all ages in 2003 to 2013.
ventive measures; it is not clear why Alberta experienced this increase. The LOD incidence rate increased from 0.15 to 0.39 cases per 1,000 live births (>2.5-fold change) between 2003 and 2013. Although the intervention guidelines are designed only to prevent EOD iGBS diseases (10), the increases in incidence were documented for both EOD and LOD cases. This is similar to the reported increase in the United States in 2004 to 2005 (10). Thus, surveillance of EOD and LOD rates in the upcoming years is important to assess whether the increase is sustained.

The most common GBS CPSs seen in Alberta over the 11-year surveillance period were CPSs III, V, and Ia. This is not unusual, as CPS III has been documented to be the most common CPS identified among cases of invasive disease and in carriage in studies elsewhere (Toronto, Beijing, Ireland, England, Portugal, and globally) (9, 15, 29–32). CPS III accounted for one-quarter to one-half of the total GBS isolates serotyped in past epidemiological surveys. CPS V ranked a close second in our study in Alberta, Canada, and in the recently reported study from Toronto, Canada, indicating that CPS V is common in Canada (9).

Interestingly, in the meta-analysis study by Edmond et al., CPS Ia was found to be more common than CPS V (29). CPS V was not as common in the 1990s (and earlier) as CPS Ia; however, CPS V became more prevalent in the 2000s, indicating that GBS CPS changes in the population can occur gradually (29, 30, 33, 34). Closer examination by age showed that CPS III was the predominant type among cases of EOD, LOD, and disease in persons up to 14 years of age. The predominance of CPS III as a neonatal pathogen is well documented (8, 13, 29, 30, 35, 36). For our study, CPS III was the most common CPS, followed by CPS Ia, for cases of EOD (28%) and LOD (65%). These findings are similar to the meta-analysis reported by Edmond et al. (29). Those investigators collected data from previous publications on GBS disease in infants up to 3 months of age and found that CPS III accounted for 49% of the cases in that age group, followed by CPS Ia at 23% (together accounting for 72% of iGBS cases). The remaining CPSs reported in that meta-analysis were all under 10% each (29).

It is interesting to note that the incidence of iGBS disease for all ages over the 11-year surveillance period increased 1.5-fold, from 3.92 cases/100,000 population in 2003 to 5.99 cases/100,000 population in 2013 (from 123 cases to 234 cases). The explanation for this increase may be multifactorial. It is possible that this increase is due to elevated rates of iGBS disease and/or increased interest in submission of invasive GBS isolates for CPS typing from diagnostic microbiology laboratories in the province. It is difficult to determine whether elevated rates of iGBS disease are the cause of the increase, as iGBS cases in persons over the age of 91 days (i.e., not EOD or LOD) are not included in the Alberta Health notifiable disease regulations. Therefore, there is no requirement for diagnostic laboratories to forward GBS isolates from cases of iGBS disease to the public health laboratory for analysis for those age groups. Elevated rates of GBS isolate submission first became apparent in 2009 with an incidence rate of 4.15 cases/100,000 population, peaking in 2013 at 5.99 cases/100,000 population. It may be theorized that, if the incidence rate increases were due to increased interest from diagnostic laboratories in sending GBS isolates to the PLPH for CPS typing, then it is likely that the increase numbers should be fairly consistent for all CPSs from year to year. The data from Table 1 for the six most common CPSs suggest that this is not the case. The case numbers for CPSs Ia, Ib, III, and IV showed increases, whereas the case numbers for CPSs II and V remained fairly constant. Whether the increases seen over the 11-year period for CPSs Ia, Ib, III, and V are due simply to increased submission rates or increased numbers of cases of active disease, the data represent important information regarding circulating CPSs causing iGBS disease in Alberta.

While the increases in CPSs Ia, Ib, II, and V may not be clearly understood, the increases seen for CPS IV are likely due to the introduction of a number of CPS IV strains into the Alberta population. From 2003 to 2013, the identification of CPS IV gradually increased 12-fold (from 2 cases to 24 cases). This increase is likely part of the global increase in CPS IV reported previously for North America, South America, Europe, and Asia (15, 37–41). Recently, a study from Toronto, Canada, by Teatero et al. (9) described a collection of 600 GBS isolates collected from cases of iGBS between 2009 and 2012 in the greater Toronto area; more than 6% (37 isolates) were identified as CPS IV. This is nearly identical to our CPS IV proportion of 6.3%. The Toronto study identified six

![FIG 3 Erythromycin and clindamycin nonsusceptibility among all iGBS isolates in 2003 to 2013.](image-url)
STs, representing three CCs, among the 37 isolates examined (9). The major sequence type identified in the Toronto analysis was ST459 (51%), which is similar to our findings (60% ST459) for the year assayed (2013). Those authors published a follow-up report in 2015 that described increased numbers of cases of CPS IV in Saskatchewan (19% of total) and Manitoba (16% of total), two other provinces in Canada (42); the isolates were collected between January 2010 and May 2014. Those investigators also showed that the vast majority of CPS IV isolates causing adult disease were ST459 (94% in Saskatchewan and 87% in Manitoba) (42). Interestingly, another ST, ST425, accounted for 30% of the isolates in the Saskatchewan and Manitoba surveillance study, but this ST was not identified in our Alberta collection in 2013. This may be due to the small number of CPS IV isolates identified in 2013 for which MLST analysis was performed (22 isolates) or the collection. ST2 is part of CC1, in which only 0.9% of isolates were identified group B streptococcal isolates from cases of invasive disease and submitted them for CPS typing.

ACKNOWLEDGMENTS

We gratefully acknowledge the diagnostic laboratories in Alberta that identified group B streptococcal isolates from cases of invasive disease and submitted them for CPS typing.

REFERENCES


Correction for Alhhazmi et al., Epidemiology of Invasive Group B Streptococcal Disease in Alberta, Canada, from 2003 to 2013

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Division of Diagnostic and Applied Microbiology, Department of Laboratory Medicine and Pathology, University of Alberta, Edmonton, Alberta, Canada; Provincial Laboratory for Public Health (Microbiology), Edmonton, Alberta, Canada


Page 1774, abstract, lines 10 and 11: “of which the majority were \textit{phygA} positive (86.6%)” should read “of which the majority were \textit{phygA} negative (93.4%).”

Page 1776, column 2, “CPS IV strains” paragraph, lines 13 to 17: The sentence beginning “For the neonatal cases” should read “For the neonatal cases, the majority of \textit{phygA}-positive isolates reported here were CPS III (74.4% \([n = 32]\)), followed by CPS IV (11.6% \([n = 5]\)), CPS Ia (7% \([n = 3]\)), CPS Ib (4.7% \([n = 2]\)), and CPS V (2.3% \([n = 1]\)) (\chi^2 test, \(P < 0.001\)) (Fig. 1).”

Page 1776, column 2, “CPS IV strains” paragraph, lines 18 to 21: The sentence beginning “Of the CPS IV isolates from all ages” should read “Of the CPS IV isolates from all ages, 6.6% were \textit{phygA} positive (7 \textit{phygA}-positive isolates/106 CPS IV isolates) (\chi^2 test, \(P < 0.001\)) (Fig. 2).”

Page 1777, column 1: Fig. 1 should appear as shown below.
Page 1777, column 2: Fig. 2 should appear as shown below.

![Bar Chart](https://example.com/bar_chart.png)

Page 1779, column 2, lines 14 to 16: The sentence reading “In addition, the CPS IV GBS strains seen in the province have become dominated by $hvgA$-positive strains” should be deleted.