Group B Streptococcus Surveillance Report 2014

Oregon Active Bacterial Core Surveillance (ABCs)

Center for Public Health Practice

Updated: November 2015

Background

The Active Bacterial Core surveillance (ABCs) program is a core component of the Emerging Infections Program (EIP) Network sponsored by the Centers for Disease Control and Prevention (CDC). The purpose of the ABCs program is to determine the incidence and epidemiologic characteristics of invasive disease due to *Haemophilus influenzae*, *Neisseria meningitidis*, group A streptococcus (GAS), group B streptococcus (GBS), *Streptococcus pneumoniae*, and methicillin-resistant *Staphylococcus aureus* (MRSA). The entire EIP Network for invasive GBS represents almost 32 million persons in 10 surveillance areas around the United States. More information on the EIP/ABCs Network is found at: http://www.cdc.gov/abcs/index.html.

In Oregon, the surveillance area for invasive GBS (*Streptococcus agalactiae*) disease comprises the tri-county (Clackamas, Multnomah, and Washington) Portland metropolitan area, with a 2014 estimated population of 1,717,766.* More information on the Oregon ABCs program is found at: http://public.health.oregon.gov/DiseasesConditions/CommunicableDisease/EmergingInfections/Pages/ActiveBacterialCoreSurveillance.aspx.

Methods

Invasive GBS disease (IGBS) is defined as the isolation of GBS from a normally sterile body site in a tri-county resident. Tri-county hospital laboratories submit GBS isolates to the Oregon State Public Health Laboratory, which forwards them to CDC for serotyping and susceptibility testing. Additional cases are identified through regular laboratory record reviews. Health record reviews of each case allow standardized reports of demographic characteristics, clinical syndrome manifestations, underlying illnesses or conditions, and illness outcome.

Surveillance Results

Descriptive Epidemiology

In 2014, 142 cases of invasive GBS disease were reported in the tri-county Portland area, corresponding to an incidence rate of 8.3/100,000 persons (Figure 1). This is 10% higher than the average annual incidence rate in the Portland area from 2009-2013 (7.5/100,000) and 6 percent lower than the most recent national projection of invasive disease (8.8/100,000).[1] Of these cases, there were nine deaths, for an annual mortality rate due to invasive GBS disease of 0.52/100,000 (Figure 1). This rate is 27% higher than the figure reported from 2009-2013 in the Portland area (0.41/100,000) and equivalent to the most recent national projections (0.52/100,000).[1]

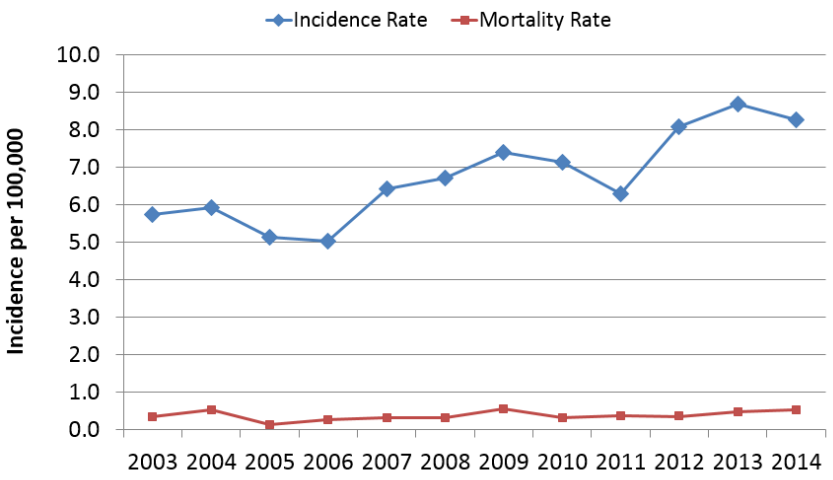
The 2014 case fatality rate for invasive GBS disease in the Portland area was 6 percent, similar to the rates in the Portland area from 2009-2013 (5%) and the entire ABCs network in 2010 (6%).[1]

Of 142 cases where sex was known, 56 percent were male; of 127cases where race was known, 90 percent were white, 4 percent were

* Source: Portland State University Population Research Center (http://www.pdx.edu/prc/)

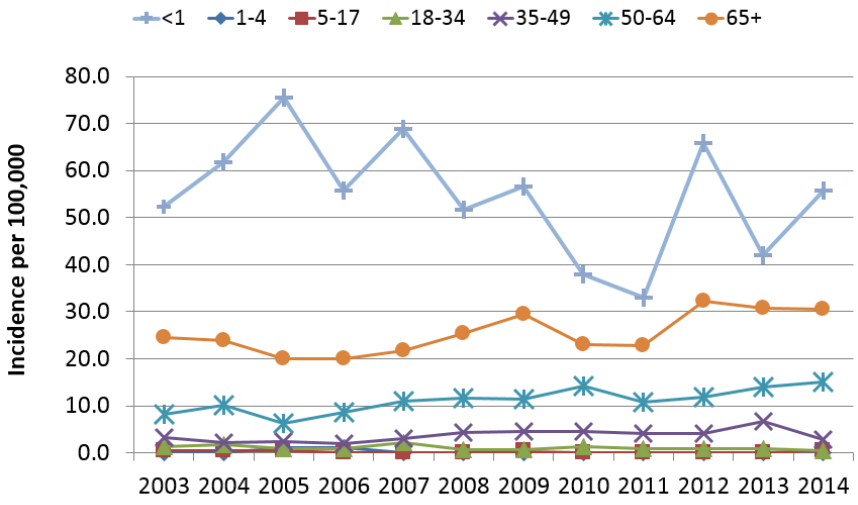
Asian/Pacific Islander, 5percent were black, and 1 percent were American Indian/Alaska Native; and of 131 cases where ethnicity was known, 6 percent were Hispanic or Latino.

Figure 1: Incidence and Mortality Rates of IGBS Cases in Tri-county Area



The incidence rate of invasive GBS disease in Clackamas county in 2014 (11/100,000) was higher than those reported from Multnomah (9/100,000) and Washington (5/100,000) counties. The rates in Clackamas county was slightly higher than their respective 2010-2013 averages (7.6/100,000), while the rates in Multnomah and Washington counties remained stable. In 2014, the six deaths due to GBS occurred in Multnomah county (.78/100,000); two deaths in Washington county (0.36/100,000); and one death in Clackamas county (0.25/100,000).

Figure 2: Incidence of IGBS Cases in Tri-county Area by Age

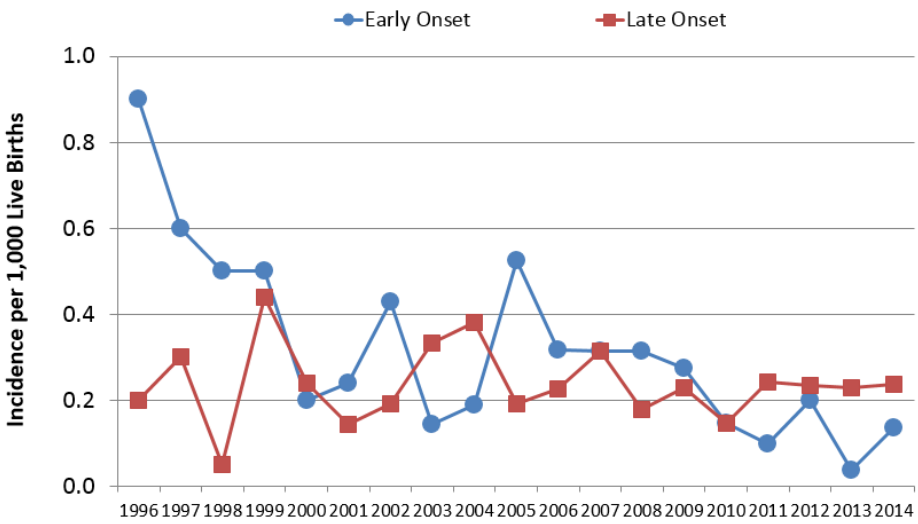


In 2014, the burden of disease due to invasive GBS disease was highest in those <1 year of age (12 cases; incidence 55.8/100,000) (Figure 2). Incidence was also high among those ≥65 years of age (67 cases; incidence 30.4/100,000) and among those between the ages of 50 and 64 (49 cases; incidence 15.1/100,000).

Among the 9 deaths reported, the burden of deaths due to invasive GBS disease was highest in those <1 year of age (1 death; mortality 4.6/100,000). Seven deaths were reported among those ≥65 years of age (mortality 3.2/100,000). One death was reported among the 50-64 age group (mortality 0.31/100,000) For cases reported since 2003, fatal outcome from IGBS has been associated with age (p=0.0172).

Among infants, there are two main types of GBS disease. Early-onset disease is defined as invasive GBS disease occurring in infants less than seven days of age; late-onset disease is defined as invasive GBS disease occurring in infants from 7 to 89 days of age.

Figure 3: Incidence of Early- and Late-Onset IGBS Disease



After a 78 percent decrease from 1996-2000, the incidence of early-onset GBS disease has stayed largely stable, outside of annual fluctuations (Figure 3). The incidence of late-onset disease has been relatively stable throughout the surveillance period. In 2014, the incidence of early-onset disease – four cases; 0.14 per 1,000 live births – was 11 percent lower than the previous five-year average (0.15/1,000), and 46 percent lower than the national estimate of disease (0.25/1,000).[1] The 2014 rate of late-onset disease – seven cases; 0.24 per 1,000 live births – was 9 percent higher than the previous five-year average (0.22/1,000) and 15 percent lower than the national estimate (0.28/1,000).[1]

Clinical Manifestations

The clinical manifestations of invasive GBS disease are listed in Table 1. Meningitis was more commonly seen in cases in 2014 than compared to the previous five years (p=0.02). The remainder of the clinical manifestation profile of invasive GBS disease in 2014 was not statistically significantly different than that seen from cases reported during the previous five years.

Table 1: Percent of IGBS Cases† Reporting Common Clinical Syndromes

Syndrome	2014 (n=142)	2009-2013 (n=620)
Primary bacteremia	41	42
Cellulitis	18	25
Pneumonia	9	10
Meningitis	8	3
Other††	23	20

† Some cases report more than 1 syndrome.
†† Other syndrome includes abscess (not skin), endometritis, endocarditis, HUS (hemolytic uremic syndrome), osteomyelitis, pericarditis, peritonitis, septic arthritis, septic shock.

For cases reported since 2006, pneumonia and cellulitis were more common with increasing age (p<0.0001and p<0.0001, respectively), while bacteremia and meningitis were less common (p<0.02 and p<0.0001, respectively). After adjusting for age, a fatal outcome was 2.1 times more likely among those presenting with bacteremia (95% confidence interval [CI] 1.1, 3.9) and almost five times less likely among those presenting with meningitis (CI 1.2, 17.8). Among four infants with

early-onset GBS disease reported in 2014, the clinical syndromes present were bacteremia and meningitis. Among seven late-onset cases, three presented with bacteremia and four with meningitis.

Underlying Conditions

Ninety-four percent of adults with IGBS reported at least one underlying condition or behavioral risk factor for GBS disease. Alcohol abuse, and smoking were significantly associated with cases among younger adults, while cardiovascular disease, diabetes, and chronic obstructive pulmonary disease (COPD) were significantly associated with cases among older adults (Table 2). COPD, and alcohol were significantly associated with fatal outcome among adults (p=0.0285 and p=0.0009, respectively) after adjusting for age.

Table 2: Distribution of Underlying Conditions by Age Reported Among Adult IGBS Cases, 2006-2014

Underlying Condition	18-64 (n=170) N (%)	65+ (n=776) N (%)	Total (n=946) N (%)	p-value
Alcohol Abuse	10 (6)	19 (2)	29 (3)	p=0.0187
Cancer	9 (5)	57 (7)	66 (7)	p=0.342_
Cardiovascular Disease	12 (7)	323 (42)	335 (35)	p<0.0001
COPD	4 (2)	99 (13)	103 (11)	p<0.0001
Cirrhosis	9 (5)	61 (8)	70 (7)	p=0.247
Diabetes	63 (37)	373 (48)	436 (46)	p=0.0091
Dialysis	8 (5)	24 (3)	32 (3)	p=0.292
Immunosuppression	16 (9)	76 (10)	92 (10)	p=0.879
Smoking	39 (30)	96 (12)	135 (14)	p=0.004
None	16 (10)	39 (5)	55 (6)	p=0.027

Serotype Analysis

For all isolates tested (91%) since 2006, serotype IA was the most common cause of all cases (26%) followed by serotype V (23%). Table 3 displays the serotype distribution of isolates tested between 2006 and 2014.

Table 3: Serotype Distribution of Isolates Tested by IGBS Disease Type, 2006-2014

Serotype	Total (n=961) N (%)	Early-onset (n=40) N (%)	Late-onset (n=45) N (%)	All Other (n=876) N (%)
IA	**247 (26)**	**11 (27)**	13 (29)	**223 (25)**
IB	122 (13)	1 (2)	5 (11)	116 (13)
II	135 (14)	7 (17)	0	128 (15)
III	146 (15)	**15 (37)**	**24 (53)**	107 (12)
IV	72 (7)	0	1 (2)	71 (8)
V	211 (23)	5 (12)	2 (4)	204 (23)
VI	5 (0.5)	1 (2)	0	4 (0.4)
VII	2 (0.2)	0	0	2 (0.2)
Nontypeable	18 (2)	0	0	18 (2)

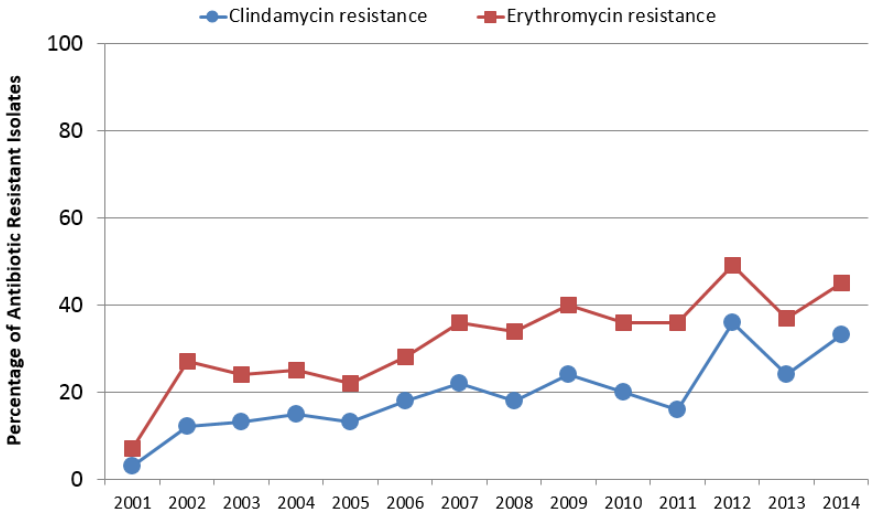
Antibiotic Susceptibility

Of 692 invasive GBS isolates tested (72%) for susceptibility to common antibiotics since 2006, 100 percent were susceptible to ampicillin, cefotaxime, penicillin and vancomycin. Intermediate and full

resistance to erythromycin were found among 10 (1%) and 311 (45%) isolates, respectively, and intermediate and full resistance to clindamycin were found among 7 (1%) and 193 (28%) isolates, respectively. Nine isolates were found to be fully resistant to levofloxacin. Of the early-onset IGBS cases since 2006, 32 percent were resistant to erythromycin and 10 percent were resistant to clindamycin.

Figure 4 displays clindamycin and erythromycin resistance among invasive GBS isolates since 2001. For cases reported since 2006, clindamycin and erythromycin resistance were not statistically significantly associated with age. Fatal outcome was associated with clindamycin resistance (p=0.03).

Figure 4: Clindamycin and Erythromycin Resistance Among IGBS Isolates



Early-Onset Invasive GBS Prevention Indicators

In 2014, all of the two women with infants having EO IGBS had received prenatal care and were screened for GBS prior to admission. Neither woman had a positive culture. Neither of the women had bacteruria during pregnancy, a previous infant with IGBS, or a previous pregnancy with GBS colonization.

Guidelines for prophylaxis recommend penicillin or ampicillin, given that GBS is still fully susceptible to both. For women with documented penicillin allergies who are at low risk for anaphylaxis, cefazolin is recommended; if the risk of anaphylaxis is high, then either clindamycin or erythromycin is recommended unless the strain is known to be resistant. In the latter case, vancomycin is preferred.[2]

Discussion

A major focus of surveillance for invasive GBS disease has been the epidemiologic characterization of early-onset disease to assess the impact of screening and treatment guidelines for pregnant women. The screening guidelines for prevention of IGBS, first released in 1996 and revised in 2002, have led to national declines in early-onset GBS disease.[3] While complete adherence to the guidelines would not prevent all cases of EO IGBS disease, occurrence would undoubtedly be higher without prenatal screening and appropriate administration of intrapartum antibiotics.

The epidemiologic profile of invasive GBS disease in adults in Oregon is consistent with national data and previous studies.[4] Invasive GBS in nonpregnant adults is increasing, particularly in elderly persons and those with significant underlying diseases.[4] In Oregon, alcohol abuse, and smoking were significantly associated with cases among younger adults, while cardiovascular disease, diabetes, and

chronic obstructive pulmonary disease (COPD) were significantly associated with cases among older adults.

Increasing resistance to erythromycin and clindamycin has also been reported nationally.[4] So far in Oregon, however, this increasing resistance does not seem to have translated into broad treatment failures leading to GBS mortality. Continued surveillance of invasive GBS disease among adults will be needed to monitor trends in antibiotic resistance, describe the characteristics of increases in invasive GBS occurrence, and better characterize the disease among this population.

References

1. Centers for Disease Control and Prevention. 2014. Active Bacterial Core Surveillance Report, Emerging Infections Program Network, Group B *Streptococcus*, 2014. Available at http://www.cdc.gov/abcs/reports-findings/survreports/gbs14.htmlAccessed 12 Jul 2015.
2. Centers for Disease Control and Prevention. Prevention of Perinatal Group B Streptococcal Disease – Revised Guidelines from CDC. *MMWR Recomm Rep*. 2002; 51(RR-11)1-22.
3. Centers for Disease Control and Prevention. Early-Onset and Late-Onset Neonatal Group B Streptococcal Disease – United States, 1996-2004. *MMWR*. 2005; 54(47)1205-8.
4. Farley MM. Group B Streptococcal Disease in Nonpregnant Adults. *Clin Infect Dis*. 2001; 33:556-61.