Group B *Streptococcus* Surveillance Report 2007

Oregon Active Bacterial Core Surveillance (ABCs)
Office of Disease Prevention & Epidemiology
Oregon Department of Human Services
Updated: July 2008

Background

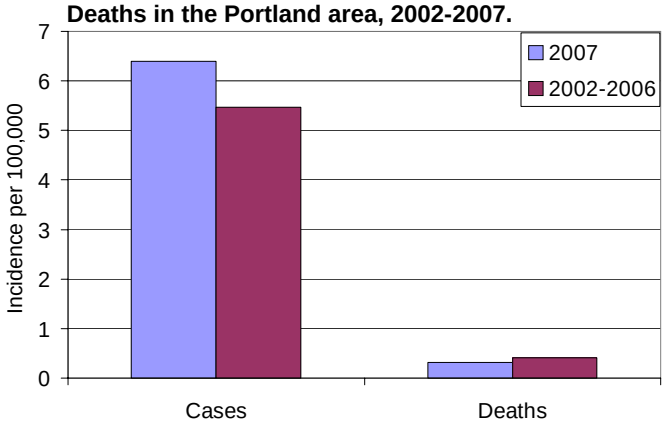
Active Bacterial Core surveillance (ABCs) is a core component of the Emerging Infections Program (EIP) Network sponsored by the Centers for Disease Control and Prevention (CDC). The purpose of ABCs is to determine the incidence and epidemiologic characteristics of invasive disease due to *Haemophilus influenzae*, *Neisseria meningitidis*, group A *Streptococcus* (GAS), group B *Streptococcus* (GBS), and methicillin-resistant *Staphylococcus aureus* (MRSA). The entire EIP Network represents 35.6 million persons in 10 surveillance areas around the United States. More information on the EIP/ABCs Network is found at: http://www.cdc.gov/ncidod/dbmd/abcs.

In Oregon, the surveillance area for invasive GBS disease comprises the Tri-County (Clackamas, Multnomah, and Washington) Portland metropolitan area with a 2006 estimated population of 1,596,370. More information on the Oregon ABCs program is found at: http://oregon.gov/DHS/ph/acd/abc.shtml.

Methods

Invasive disease 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, from where they are forwarded 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 conditions, and illness outcome. Early-onset disease is defined as invasive GBS disease occurring in infants less than seven (<7) days of age; late-onset disease is defined as invasive GBS disease occurring in infants from 7 to 89 days of age.

Figure 1: Incidence of Invasive GBS Cases and Deaths in the Portland area, 2002-2007.



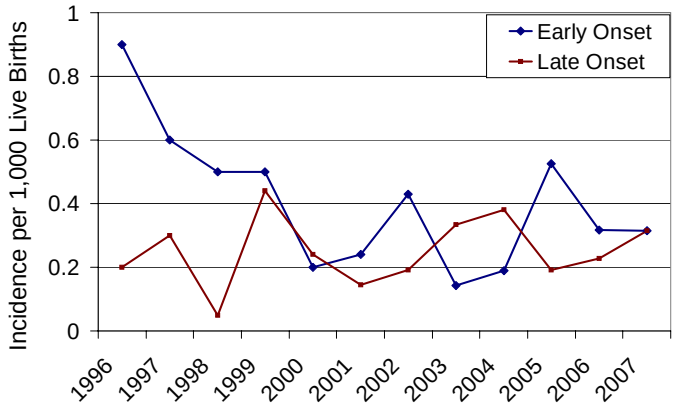
Surveillance Results

Descriptive Epidemiology

In 2007, 102 cases of invasive GBS disease were reported in the Tri-County Portland area, corresponding to an incidence rate of 6.4/100,000 persons (Figure 1). This is 17% higher than the average annual incidence rate in the Portland area from 2002–2006 (5.5/100,000), but 4% lower than the 2007 national projection of invasive disease (6.7/100,000).[1] Of these cases, there were five deaths, for an annual mortality rate due to invasive GBS disease of 0.31/100,000 (Figure 1). This rate is less than that reported from 2002–2006 in the Portland area (0.41/100,000) and the 2007 national projections (0.48/100,000).[1] The 2007 case fatality rate for invasive GBS disease in the Portland area was 5%, lower than the Portland area from 2002–2006 and the entire ABCs network in 2007 (7% for both).[1] Of 102 cases where sex was known, 59% were male; of 58 cases where race was

known, 98% were white and 2% were black; and of 21 cases where ethnicity was known, 48% were Hispanic or Latino.

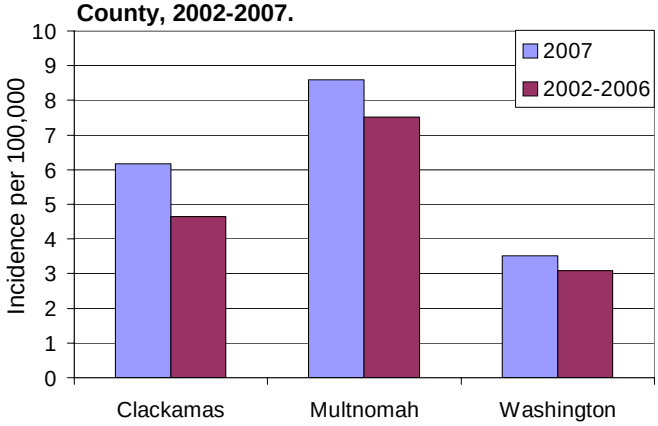
Figure 2: Incidence of Early- and Late-Onset Invasive GBS Disease, 2001-2006.



After a 78% decrease from 1996-2000, the incidence of early-onset GBS disease has stayed largely stable, outside of annual fluctuations. The incidence of late-onset disease has been stable throughout the surveillance period. In 2007, the incidence of early-onset disease – seven cases; 0.32 per 1,000 live births – was equal to the previous five-year average, but below the national estimate of disease (0.36/1,000).[1] The 2007 rate of late-onset disease – seven cases; 0.32 per 1,000 live births - was slightly higher than the previous five-year average (0.26/1,000), and 2007 national estimates (0.29/1,000).[1]

The incidence rate of invasive GBS disease in Multnomah County in 2007 (8.6/100,000) was higher than those reported from Clackamas (6.2/100,000) and Washington (3.5/100,000) Counties. While this is similar to the historical pattern (Figure 3), the rates in all three counties were higher than the 2002-2006 averages, with the largest increase, 33%, seen in Clackamas County. In 2007, the mortality rate due to GBS was highest in Washington County (0.39/100,000), followed by Multnomah (0.28/100,000), and Clackamas (0.27/100,000) counties. This is different than the usual pattern, in which mortality is typically highest in Multnomah County, but marks the second consecutive year in which mortality was highest in Washington County.

Figure 3: Incidence of Invasive GBS Disease by County, 2002-2007.



The burden of disease and death due to invasive GBS disease was highest in those ≥65 years of age (34 cases; incidence 21.7 /100,000 persons and 3 deaths; mortality 1.9 / 100,000). Incidence was also high among those under five years of age (15 cases; incidence 13.8/100,000), although no deaths were reported among those in this age group. For cases reported since 2002, fatal outcome from IGBS is positively associated with age (p=0.044).

Table 1: Percent of Invasive GBS cases reporting common clinical syndromes.†

Syndrome	2007	2002-2006
Primary Bacteremia	42	48
Cellulitis	26	23
Pneumonia	8	13
Meningitis	5	2
Other Syndrome‡	20	15

† Some cases report >1 syndrome; not all syndromes shown.
‡ Includes Abscess (not skin), peritonitis, HUS, pericarditis, septic arthritis, osteomyelitis, and endometritis.

Clinical Manifestations

The clinical manifestations of invasive GBS disease are listed in Table 1. The clinical manifestation profile of invasive GBS disease in 2007 was not significantly

different than that seen from cases reported during the previous five years.
For cases reported since 2002, pneumonia and cellulitis were more common with increasing age (p=0.0006 and p<0.0001, respectively), while bacteremia was less common (p=0.0006). After adjusting for age, a fatal outcome was 2.7 times more likely among those presenting with bacteremia (95% Confidence Interval [CI] 1.3, 5.6) and 14 times less likely among those presenting with cellulitis (CI 1.9, 100).

Among early-onset GBS disease reported in 2007, bacteremia was present in 86% of cases, with the remaining case presenting as meningitis. Among late-onset cases, bacteremia was present in 43% of cases, meningitis in 43% of cases, and pneumonia in 14% of cases. This was not significantly different than the presentations of early- and late-onset cases reported since 2002.

Underlying Conditions

Table 2 lists underlying conditions commonly reported or known risks for adult invasive GBS disease from 2002–2007. Overall, almost all (95%) cases reported at least one underlying condition or disease. Alcohol abuse, cirrhosis, and smoking were significantly associated with cases among younger adults, while cardiovascular disease and chronic obstructive pulmonary disease (COPD) were significantly associated with cases among older adults. Among non-early- or late-onset disease cases occurring in those less than 18 years of age, no underlying conditions were reported.

Table 2: Percent of Adult Invasive GBS Cases with Reported Underlying Conditions, 2002-2007.

	18-64	65+	Total	p-value
Alcohol Abuse	**12**	**3**	**7**	**0.0002**
Cancer	19	27	23	0.054
Cardiovascular Disease	**19**	**55**	**36**	**<0.0001**
COPD	**6**	**17**	**11**	**0.0005**
Cirrhosis	**17**	**4**	**11**	**<0.0001**
Diabetes	42	38	40	0.45
Dialysis*	8	4	6	0.078
Immunosuppression	10	11	10	0.76
Smoking	**23**	**6**	**15**	**<0.0001**
None	5	6	5	0.52

*Dialysis was reported less frequently in 2007 (2%) than during the previous five years (7%), p=0.03; all other underlying conditions were reported in similar proportions.

Among adults, fatal outcome was positively associated with alcohol abuse (odds ratio [OR] 3.7; CI 1.2, 11.2, after adjusting for age); bacteremia was associated with dialysis (OR 2.8; CI 1.2, 6.4) and no reported underlying conditions (OR 5.2; CI 1.9, 14.2); and cellulitis was positively associated with diabetes (OR 2.0; CI 1.3, 3.0) and negatively associated with cirrhosis (OR 0.3; CI 0.1, 0.8).

Table 4: Serotype distribution, as a percentage of isolates tested, by invasive GBS disease type, 2002-2007.

Serotype	Early-Onset	Late-Onset	Other
IA	31	31	23
IB	11	0	10
III	28	50	9
V	14	13	31
Other‡	14	3	20
NT	3	3	7

‡Includes serotypes II, IV, VI, and VII

Serotype Analysis

The profile of the 83 isolates from 2007 for which serotype information is available is similar to that for isolates submitted from 2002-2006. For all isolates submitted since 2002, serotype V was the most common cause of all cases (29%), followed by serotype IA (24%) and serotype III (13%) (Table 4). However, serotype profile for early- and late-onset cases was different than that for other cases. While the isolate serotype was not associated with fatal outcome, serotype III was positively associated with bacteremia (OR 2.1; CI 1.1, 3.7) and negatively associated with cellulitis (OR 0.21; CI 0.079, 0.55).

Antibiotic susceptibility
Of 361 invasive GBS isolates tested for susceptibility to common antibiotics since 2002, 100% were susceptible to penicillin, ampicillin, cefuroxime, cefazolin, and vancomycin. Intermediate and full resistance to erythromycin were found among 3 (1%) and 107 (30%) isolates, respectively, and intermediate and full resistance to clindamycin were found among 2 (1%) and 62 (17%) isolates, respectively. The percentages of isolates exhibiting resistance to both antibiotics have not changed since 2002. Serotype V was three times more likely to be resistant to erythromycin (CI 1.8, 5.1) than other or non-typable strains. Fatal outcome from IGBS disease was not associated with erythromycin resistance (p=0.13).

Early-Onset Invasive GBS Prevention Indicators
Two of the seven infants (29%) with early-onset IGBS disease (EO) were born at <37 weeks gestation. All of the women with infants having EO IGBS had received prenatal care, although only five (71%) were screened for GBS prior to admission. Of the five, two (40%) were GBS culture-positive. None of the women had bacteruria during pregnancy, a previous infant with IGBS, or a previous pregnancy with GBS colonization. Two women (29%), both with intrapartum fever, received intrapartum antibiotics. However, this includes only one of the women who had a positive GBS screening culture. While neither woman receiving intrapartum antibiotics had a recorded penicillin allergy, both women were given cefoxitin. 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 are recommended unless the strain is known to be resistant. In the latter case, vancomycin is preferred.

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 guidelines, first released in 1996 and revised in 2002, led to national declines in early-onset GBS disease. That only 71% of women delivering infants with subsequent EO IGBS were screened and that only one of the women with a positive GBS screening culture was administered intrapartum antibiotics represent missed opportunities for EO IGBS prevention. While complete adherence to the guidelines would not prevent all cases of EO IGS disease, occurrence would undoubtedly be higher without prenatal screening and appropriate administration of intrapartum antibiotics. Continued vigilance in surveillance and intervention is required to encourage high adherence to the screening guidelines and to maintain the incidence of early-onset disease below the Healthy People 2010 target rate of 0.5/1,000 live births, which has been seen in Oregon throughout the surveillance period.[1]

The epidemiologic profile of invasive GBS disease in adults in Oregon is consistent with national data and previous studies.[3] In particular, invasive GBS in this population frequently occurs among those with increasing age or with one or more underlying condition. While diabetes is most frequent, other conditions include cancer, cirrhosis, cardiovascular disease, and smoking. Increasing resistance to erythromycin and clindamycin has also been reported nationally.[3] So far in Oregon, however, this increasing resistance does not seem to have translated into broad treatment failures leading to GBS mortality. Continued surveillance for invasive GBS disease among adults will be needed to identify the emergence of additional antibiotic resistance, describe the characteristics of the increases in invasive GBS occurrence, as well as better characterize the disease among a population at an increasing risk.

References:

1. Centers for Disease Control and Prevention. 2008. Active Bacterial Core Surveillance Report, Emerging Infections Program Network, Group B Streptococcus, 2007. Available at http://www.cdc.gov/ncidod/dbmd/abcs/survreports/gbs07.pdf.
2. 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.
3. Farley MM. Group B Streptococcal Disease in Nonpregnant Adults. *Clin Infect Dis*. 2001; 33:556-61.