Group A *Streptococcus* Surveillance Report 2006

Oregon Active Bacterial Core Surveillance (ABCs)
Office of Disease Prevention & Epidemiology
Oregon Department of Human Services
Updated: February 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 39.5 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 GAS disease comprises the Tri-County (Clackamas, Multnomah, and Washington) Portland metropolitan area with a 2006 estimated population of 1,569,170. More information on the Oregon ABCs program is found at: http://oregon.gov/DHS/ph/acd/abc.shtml.

Methods

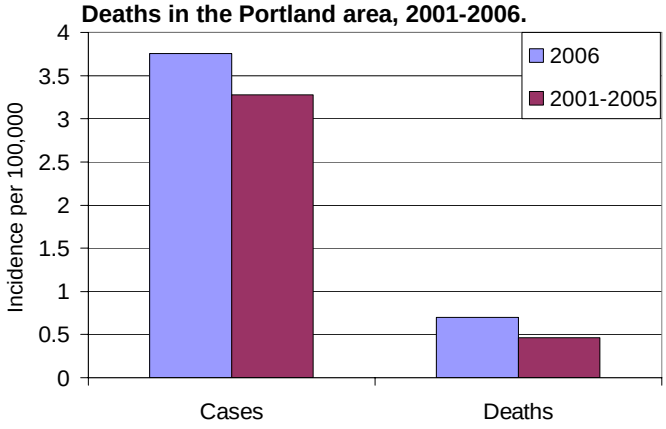
Invasive disease is defined as the isolation of GAS from a normally sterile body site or fluid or from a wound accompanied by necrotizing fasciitis or toxic shock syndrome in a tri-county resident. Tri-county hospital laboratories submit GAS isolates to the Oregon State Public Health Laboratory, from where they are forwarded to CDC for typing. 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.

Surveillance Results

Descriptive Epidemiology

In 2006, 59 IGAS cases were reported in the Tri-County Portland area, for an incidence rate of 3.8/100,000 persons (Figure 1). This is similar to the 2006 national projection of invasive disease (3.8/100,000) but 15% higher than the average annual incidence rate in the Portland area from 2001–2005 (3.3/100,000).[1] With 11 deaths reported, IGAS mortality in 2006 was 0.7/100,000 (Figure 1), 52% higher than the 2001–2005 Portland area and the 2006 national projections (0.5/100,000 for both).[1] The mean and median ages of IGAS cases were 54 and 57 years, respectively (range: 0-94), and those of IGAS deaths were 70 and 75 years, respectively (range: 40-94). The 2006 case fatality rate for IGAS in the Portland area was 19%, compared with 13% for the Portland area from 2001–2005 and the entire ABCs network in 2006 (14%).[1] Over half of cases (53%) were male; of 42 cases where race was known, 33 (79%) where white, 4 (10%) were black, and 5 (12%) were of another race; of 29 cases where ethnicity was known, 2 (7%) were Hispanic/Latino.

Figure 1: Incidence of Invasive GAS Cases and Deaths in the Portland area, 2001-2006.



The incidence of IGAS in 2006 was highest in Clackamas (4.6/100,000), followed by Multnomah (4.3/100,000), and Washington (2.4/100,000) Counties. This is different than the historical pattern, in which Multnomah County typically has the highest occurrence of IGAS. Compared with the previous five year average, the 2006 incidence was 100% higher in Clackamas, 15% higher in Washington, and 8% lower in Multnomah counties. In 2006, IGAS mortality was also highest in Clackamas (1.1/100,000), followed by Washington (0.6/100,000) and Multnomah (0.6/100,000) counties. While mortality has typically been highest in Clackamas county (0.6/100,000), compared with 0.5 and 0.3 per 100,000 in Multnomah and Washington counties, respectively, that in 2006 was 83% higher than average. The high mortality in Clackamas was due to a high case incidence along with a high case fatality rate: 38%, compared with 14% and 11% in Washington and Multnomah counties.

Figure 2: Incidence of Invasive GAS Disease by County, 2001-2006.

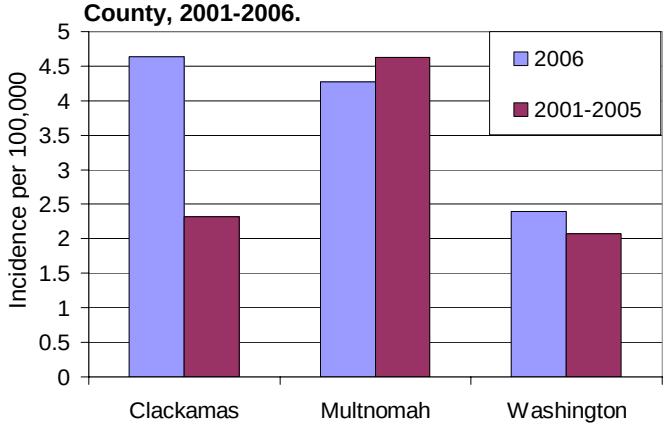
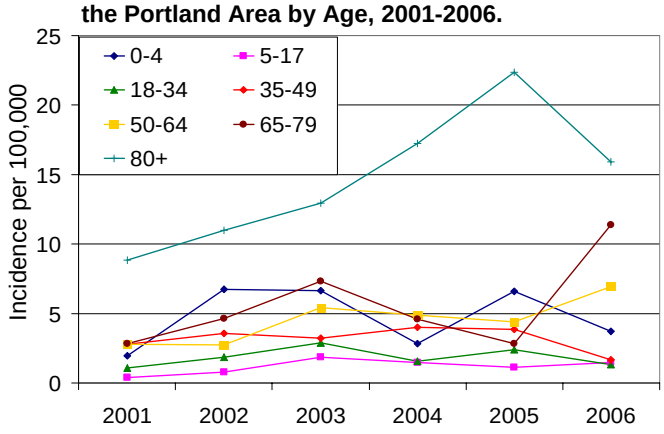


Figure 3: Incidence of Invasive GAS Disease in the Portland Area by Age, 2001-2006.



The burden of disease was highest in those ≥80 years of age (8 cases; 16/100,000 persons), followed by those 65-79 years of age (12 cases; 11/100,000) (Figure 3). The incidence among those 18-49 has remained largely stable, within annual variation, over the past six years, while increasing among all other age groups. This increase has been the most dramatic for those 65-79 years of age (304% over the 2001 rate), followed by those 50-64 (151%), and those 80 years and older (80%). The incidence rate among those 80 and older did, however, decrease by 40% from 2005. Four of the 11 deaths in 2006 were among persons in the eldest age group, for a mortality rate of 8.0/100,000 and a case fatality of 50%. Increasing age is significantly related to fatal outcome from IGAS (p<0.0001).

Clinical Manifestations

The clinical syndromes of IGAS reported in 2006 were largely similar to the average profile seen from 2001-2005 (Table 1). In 2006, three cases of necrotizing fasciitis and five cases of toxic shock syndrome were reported. Since 2001, the percentage of cases manifesting as cellulitis and abscess have significantly decreased (p=0.004 and p=0.008, respectively), although reports of abscess have been stable since 2003.

Table 1: Percent of Invasive GAS cases reporting common clinical syndromes.[†]

Syndrome	2006	2001-2005
Cellulitis	34	39
Bacteremia	24	26
Pneumonia	17	14
Septic Arthritis	9	13
Toxic Shock	9	4
Necrotizing Fasciitis	5	6
Abscess	3	7
Meningitis	2	2

[†] Some cases report >1 syndrome; not all syndromes shown.

The clinical syndromes of invasive GAS

disease vary by age. Among cases reported since 2000, meningitis and septic arthritis decrease significantly with increasing age (p=0.035 and p=0.0024, respectively). Additionally, among adults, necrotizing fasciitis, toxic shock, and sterile abscess infection were reported more frequently among those 18–64 years of age (p=0.0024, p=0.018, and p=0.0024, respectively).

Pneumonia and bacteremia cases were significantly more likely - and cellulitis significantly less likely - to be associated with a fatal outcome among IGAS cases in bivariate analysis. After controlling for patient age, pneumonia was no longer significant while a fatal outcome was three times (95% confidence interval [CI] 1.6, 6.3) more likely among those with bacteremia and over eight times less likely among those with cellulitis (CI 3.1, 22.7) than among those with other clinical manifestations.

Table 2: Percent of adult IGAS cases with reported underlying conditions, 2001-2006.†

	18-64	65+
Asthma	12	5
Blunt Trauma	15	14
Cardiovascular Disease	**11**	**54**
COPD	9	14
Diabetes	**13**	**30**
Dialysis	3	5
Immunosuppression	8	12
IDU	**18**	**0**
Obesity	6	5
Penetrating Trauma	11	5
Surgical Wound	5	6
None	**19**	**6**

†Bold type indicates a significant difference; p<0.05.

Underlying Conditions

One child (13%) reported in 2006 had trauma previous to IGAS disease; the remainder had no underlying conditions reported. Among adults, the profile of underlying conditions reported in 2006 was similar to that reported from 2001-2005, with the exception of injection drug use (IDU), which was reported less frequently in 2006 (15% vs. 3%, p=0.0045) (Table 2). Younger adults were more likely to report IDU or no underlying conditions, while older adults were more likely to have cardiovascular disease. All other underlying conditions were reported similarly among all adults. Only immunosuppression was significantly related to fatal outcome among cases reported since 2001 (Odds Ratio [OR] 3.4, CI 1.4, 8.7).

In terms of clinical manifestation, after adjusting for age, pneumonia was associated with immunosuppression (OR 2.8, CI 1.3, 5.9) and chronic obstructive pulmonary disease (COPD) (OR 2.1, CI 1.1, 4.3); septic arthritis and necrotizing fasciitis were associated with blunt trauma OR 2.6, CI 1.2, 5.6 and OR 3.2, CI 1.2, 8.4, respectively; and cellulitis was associated with IVDU (OR 2.4, CI 1.4, 4.1) and penetrating trauma (OR 2.2, CI 1.0, 4.6).

Emm type Analysis

The surface M protein – a known virulence factor for disease – has been the basis for GAS strain typing for decades. Since 1995, CDC has determined the M protein type through sequencing the DNA of the corresponding gene (*emm*), providing an *emm* type.[2] In 2006, 24 *emm* types were determined for isolates from all 59 cases reported. The five most frequent *emm* types reported in 2006 were 1 (25%), 12 (15%), 83 (8%), 28 (7%), and 3 (5%). Since 2001, 35 *emm* types were determined for 287 isolates (94% of cases). The five most frequent *emm* types seen over this time are presented in Table 4. Type 89, which first emerged in Oregon in 2004 and which comprised one of the five most common *emm* types in 2005, was reported among only one isolate (2%) in 2006.

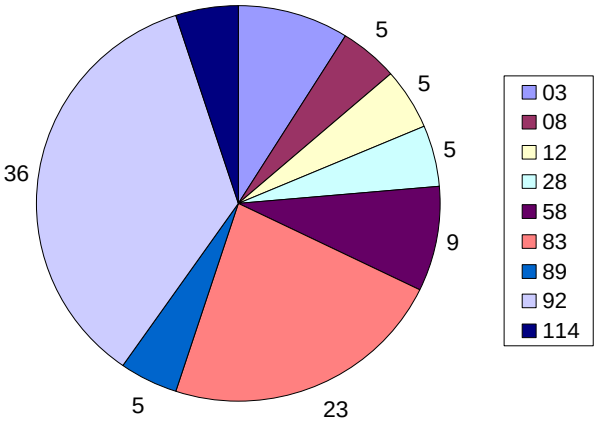
Table 4: Selected Demographic and Clinical Attributes of Invasive GAS Disease, by Isolate *emm* Type, 2001-2006.†

emm type	N (%)	Fatal N (%)	Age 65+ N (%)	Necrotizing Fasciitis N (%)	Pneumonia N (%)
1	64 (21)	13 (20)	12 (19)	7 (11)	18 (28)
3	18 (6)	6 (33)	5 (28)	2 (11)	2 (11)
12	28 (9)	4 (14)	7 (25)	1 (4)	5 (18)
28	23 (8)	4 (17)	14 (61)	1 (4)	1 (4)
92	30 (10)	3 (10)	2 (7)	2 (7)	3 (10)

Antibiotic susceptibility

The antibiotic susceptibility profile of invasive GAS strains has been assessed at several points since the beginning of ABCs. In 2006, of 50 isolates for which antibiotic susceptibility results were assessed, 100% were susceptible to penicillin, ampicillin, cefotaxime, cefazolin, and vancomycin. Eight isolates (16%) exhibited some level of antibiotic resistance: six were resistant to erythromycin alone and two were resistant to erythromycin and clindamycin (the first clindamycin-resistant isolates reported since 1999).

Figure 4: Percentage of erythromycin-resistant isolates, by *emm* type, 2001-2006.



Of 129 cases reported since 2001 for which antibiotic susceptibility information was available, 100% were susceptible to penicillin, clindamycin, ampicillin, cefotaxime, cefuroxime, cefazolin, and vancomycin. Four and 16 percent had intermediate and full resistance, respectively, to erythromycin alone and 2% were fully resistant to erythromycin and clindamycin.

Erythromycin resistance is not associated with any particular clinical manifestation of invasive GAS disease or with a fatal outcome. Figure 4 shows the percentage of erythromycin-resistant isolates by *emm* type. Since 2001, *emm* type 92 has accounted for a disproportionate percentage of the erythromycin-resistant isolates—36%—compared with 10% of all isolates during this time period.

Discussion

Invasive GAS disease disproportionably affects the elderly in Oregon, a pattern seen nationally. Additionally, the profile of invasive disease differs across the age of the population. For instance, children are much more likely to experience GAS meningitis than are adults. Among young adults, invasive disease is more likely to manifest as a sterile abscess or septic arthritis and is more likely to be associated with injection drug use or no reported underlying condition. The elderly are more likely to have systemic disease associated with chronic underlying conditions that may affect immune function. Although it is not possible to assess risk factors for disease through surveillance alone, this association has been well documented.[3]

Monitoring trends in necrotizing fasciitis and toxic shock syndrome as well as potentially-preventable nosocomial infections (such as surgical wound infections) is one of the objectives of IGAS surveillance through the ABCs network. That these manifestations have remained stable over the past six years is welcome news However, the general increase in IGAS disease over this time period among the eldest age groups will require better characterization to determine the true nature of the change in IGAS epidemiology within this population.

References:

1. Centers for Disease Control and Prevention. 2007. Active Bacterial Core Surveillance Report, Emerging Infections Program Network, Group A Streptococcus, 2006. Available at http://www.cdc.gov/ncidod/dbmd/abcs/survreports/gas06.pdf.
2. Beall B, Facklam RR, Thompson T. Sequencing *emm*-specific PCR products for routine and accurate typing of group A streptococci. *J Clin Microbiol* 1996;34:953-8.
3. Factor SH, Levine OS, Schwartz B, et al. Invasive Group A Streptococcal Disease: Risk Factors for Adults. *Emerg Infect Dis* 2003;8:970–7.