

Streptococcus pneumoniae Surveillance Report 2008

Oregon Active Bacterial Core Surveillance (ABCs)

Office of Disease Prevention & Epidemiology

Oregon Department of Human Services

Updated: March 2010



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 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 represents over 38 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 pneumococcal disease (IPD) comprises the tri-county (Clackamas, Multnomah, and Washington) Portland metropolitan area with a 2008 estimated population of 1,614,465. More information on the Oregon ABCs program is found at: <http://oregon.gov/DHS/ph/acd/abc.shtml>.

Methods

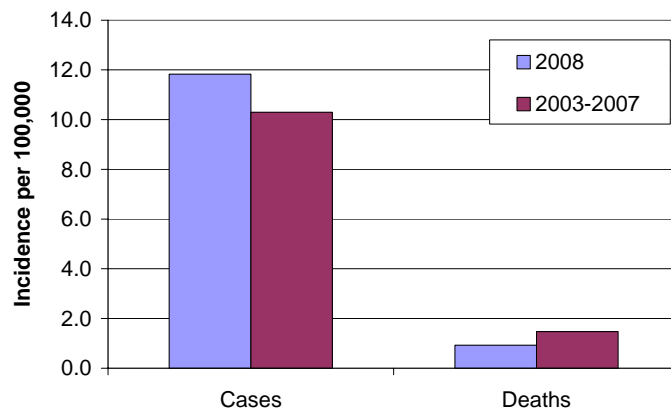
IPD is defined as the isolation of *S. pneumoniae* from a normally sterile body site in a tri-county resident. Tri-county hospital laboratories submit *S. pneumoniae* isolates to the Oregon State Public Health Laboratory, which forwards them to a CDC-collaborative laboratory for serotyping and antimicrobial 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.

Surveillance Results

Descriptive Epidemiology

In 2008, 191 cases of IPD were reported in the tri-county Portland area, corresponding to an incidence rate of 11.8/100,000 persons (Figure 1). This is almost 15 percent higher than the average annual incidence rate in the Portland area from 2003–2007 (10.3/100,000), but 19 percent lower than the 2008 national projection of invasive disease (14.5/100,000).¹ Among these cases there were 15 deaths, for an annual mortality rate due to IPD of 0.9/100,000 (Figure 1). This is 37 percent lower than the 2003–2007

Figure 1: Incidence of IPD Cases and Deaths in Oregon, 2003-2008.

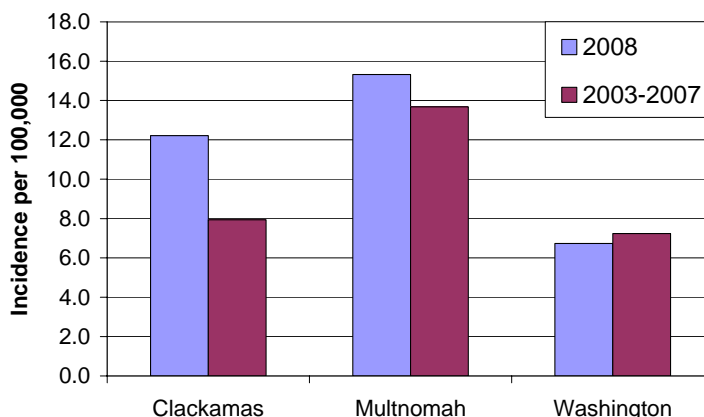


Portland area average annual mortality rate (1.5/100,000) and 36 percent lower than the 2008 national projections (1.4/100,000).¹ The mean and median ages of 2008 IPD cases were 54 and 57 years, respectively, while those of IPD deaths were 67 and 62 years, respectively. The 2008 case fatality rate for IPD in the Portland area was 7.9 percent, almost half that reported in the Portland area from 2003–2007 and less than the 10 percent reported from the entire ABCs

network in 2008.¹ Over half (55%) of the cases were male; of 81 cases where race was known, 81 percent were white, 12 percent were black, and 7 percent were another race; and of 58 cases where ethnicity was known, 12 percent were Hispanic or Latino. These data should be interpreted with caution, however, given that race and ethnicity were not obtained on a majority of the cases.

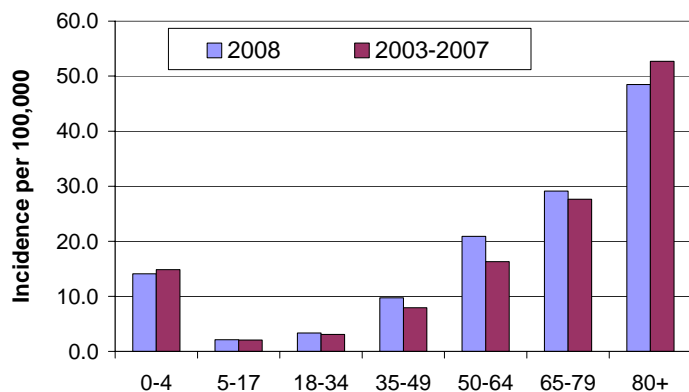
As seen in Figure 2, the incidence rate of IPD in Multnomah county in 2008 (15.3/100,000) was higher than that reported from either Clackamas (12.2/100,000) or Washington (6.7/100,000) counties. Compared with the 2003-2007 average, IPD incidence in 2008 was 54 percent higher in Clackamas, 12 percent higher in Multnomah, and 7 percent lower in Washington counties. In 2008, mortality due to IPD was similar between Clackamas (1.3/100,000) and Multnomah (1.1/100,000) counties, but higher than reports

Figure 2: Incidence of IPD by County, 2003-2008.



from Washington county (0.4/100,000). Compared with the previous 5-year average, IPD mortality in 2008 was 50 percent higher in Clackamas, 45 percent lower in Multnomah, and 65 percent lower in Washington county. These differences are not statistically significant.

Figure 3: Incidence of IPD in the Portland Area by Age, 2003-2008.



In 2008, the burden of disease was highest in those ≥ 80 years of age (24 cases; incidence 48.5/100,000 persons), followed by those 65-79 years of age (34 cases; 29.1/100,000) and those 50-64 years of age (63 cases; 20.9/100,000) (Figure 3). Compared with the previous 5-year average, IPD incidence in 2008 was 5-30 percent higher across all age groups, except for those 0-4 and ≥ 80 years of age.

deaths; 10.1/100,000), followed by those 50-64 years of age (6 deaths; 2.0/100,000). There were no deaths in 2008 among individuals aged 0-34 years and only two deaths due to IPD have been reported throughout the six-year surveillance period in this age group. Increasing age did exhibit a significant, positive association with fatal outcome from IPD ($p=0.027$). Case fatality ranged from 0 percent among those less than 35, to 21 percent in those 80 and over.

In 2008, IPD mortality was also highest in those ≥ 80 years of age (5

Clinical Manifestations

The common clinical syndromes tracked by our IPD surveillance system – bacteremic pneumonia (clinical pneumonia with a positive blood culture), primary bacteremia, and pneumococcal meningitis – are found in Table 1. While the profile of IPD clinical syndromes has been stable over time, the syndromes do vary by age. For instance, with increasing age, bacteremic pneumonia becomes more common while bacteremia, meningitis, and other syndromes all become less common ($p < 0.0001$ for all). Among cases reported since 2003, clinical syndromes were further analyzed by severity of disease. Using a multivariate logistic regression model, we found that age, and not any particular syndrome, was the best predictor of mortality.

Underlying Conditions

Table 2 lists underlying conditions that were found in greater than 5 percent of IPD cases in the Portland area during 2003–2008. Overall, 84 percent of cases had at least one underlying condition, with the presence of any underlying conditions increasing with age ($p < 0.0001$). Among those less than 18 years of age, asthma was the most common condition (7%), with other underlying conditions reported rarely. Among adults, smoking ($p < 0.0001$) and alcohol abuse ($p = 0.0291$) decreased with increasing age, while cardiovascular disease ($p < 0.0001$), cancer ($p < 0.0001$), chronic obstructive pulmonary disease (COPD) ($p < 0.0001$), diabetes ($p < 0.0001$), and cerebrovascular accident (CVA) ($p < 0.0001$) increased.

Conditions affecting the lung – such as COPD, smoking, and asthma – as well as diabetes were significantly positively associated with pneumonia (Table 3) and cancer was positively associated with bacteremia. No other underlying factors were significant predictors of any clinical syndromes of IPD. As for mortality, after controlling for age, fatal outcome was more likely among those reporting alcohol abuse and those with a previous CVA, and less likely among those with diabetes.

Table 1: Clinical Syndromes, 2003-2008.*

Syndrome	2008 (%)	2003-2007 (%)
Bacteremic pneumonia	83	73
Primary bacteremia	7	13
Meningitis	7	9
Other syndrome	4	7

*Not mutually exclusive; some cases may report more than one syndrome.

Table 2: Percent of IPD Cases with Reported Underlying Conditions, 2003-2008.*

	Percent
Smoking	29
Cardiovascular disease	23
Cancer	19
COPD	17
Diabetes	17
Immunosuppression	16
Alcohol abuse	10
Asthma	9
CVA (stroke)	7
None reported	16

*Not mutually exclusive; some cases may report more than one syndrome.

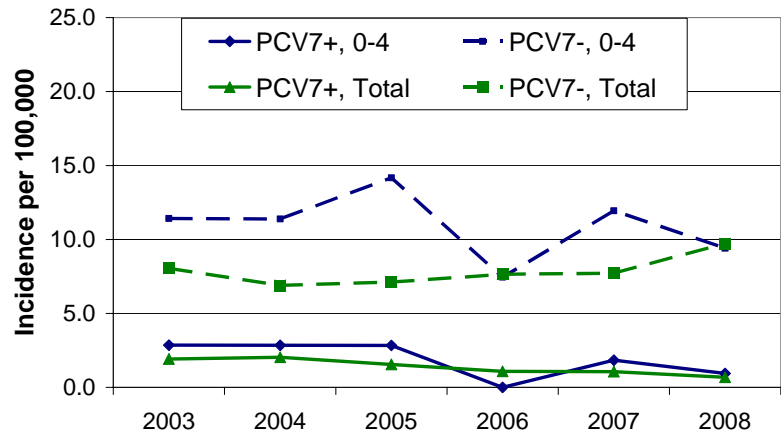
Table 3: Age-Adjusted Significant Associations between Underlying Conditions and Syndrome or Death, 2003-2008.

	aOR	95% CI
Pneumonia		
COPD	3.1	(1.6, 5.8)
Smoking	2.6	(1.8, 3.9)
Asthma	2.0	(1.1, 3.7)
Alcohol abuse	1.9	(1.0, 3.4)
Diabetes	1.8	(1.1, 3.0)
Bacteremia		
Cancer	2.0	(1.2, 3.4)
Fatal Outcome		
Alcohol abuse	2.2	(1.3, 4.0)
CVA	2.0	(1.1, 3.6)
Diabetes	0.4	(0.2, 0.8)

Serotype Analysis

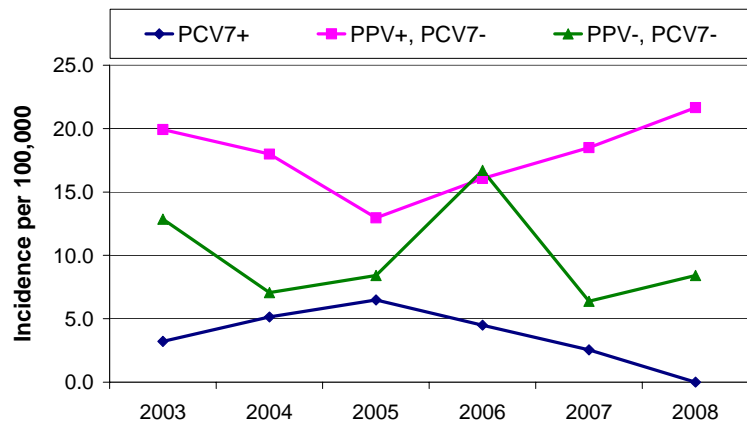
Figure 4 depicts the incidence of IPD due to strains of *S. pneumoniae* that are (PCV7+) and are not (PCV7-) included in the 7-valent pneumococcal conjugate vaccine (PCV7) among those 0-4 years of age and the overall population. From 2003-2008, the incidence of PCV7+ strains decreased 67 percent among those 0-4 years of age. Overall, IPD incidence due to PCV7+ strains decreased 65 percent over this time period. Although some year-to-year variation is seen, the overall incidence of IPD due to PCV7- strains has been relatively stable from 2003-2007, with a slight increase in 2008.

Figure 4: Incidence of PCV7+ and PCV7- Strains in Those 0-4 and Overall, 2003-2008.



The incidence of PCV7+ strains among those 65 or older has also decreased over the past six years (Figure 5). In 2008, there were no cases of IPD due to PCV7+ strains in individuals 65 or older. However, the roughly stable IPD occurrence within this age group can be largely attributed to a sustained increase in incidence due to the 16 strains included in pneumococcal polysaccharide vaccine (PPV), but not PCV7 (PPV+/PCV7-). IPD incidence due to PPV- strains decreased 35 percent over the past six years.

Figure 5: Incidence of PPV+ and PPV- Strains in Those 65+, 2003-2008.



Since 2003, one non-vaccine serotype, 19A, has increased 71 percent in incidence, from 0.7 to 1.2 cases per 100,000 in 2008. Serotype 19A continues to show an increasing trend as a proportion of IPD cases, from 7 percent in 2003 to 10 percent in 2008, albeit nonsignificant. From 2003-2008, serotype 19A decreased with increasing age ($p < 0.0001$), but was not significantly associated with any particular clinical syndrome or fatal outcome due to IPD. A new 13-valent pneumococcal conjugate vaccine (PCV13), which includes serotype 19A, was licensed by the U.S. Food and Drug Administration (FDA) in February 2010.

Antibiotic Susceptibility

In 2008, susceptibility testing was performed on 88 percent of the isolates (Table 4). At least 95 percent of the tested isolates were susceptible to amoxicillin, cefotaxime, ceftriaxone, cefuroxime, chloramphenicol, clindamycin, linezolid, meropenem, tetracycline, and vancomycin. Ninety-three percent of tested isolates were susceptible to erythromycin, 88 percent to trimethoprim sulfamethoxazole (TMP/SULFA), and 87 percent to penicillin.

Table 4: Antibiotic Susceptibility of IPD Isolates, 2008.

	Susceptible (%)	Intermediate resistance (%)	Full Resistance (%)
Chloramphenicol	100		
Linezolid	100		
Vancomycin	100		
Ceftriaxone	98	1	1
Amoxicillin	97	1	2
Cefotaxime	97	1	2
Clindamycin	96		4
Tetracycline	96		4
Cefuroxime	95		5
Meropenem	95	2	3
Erythromycin	93		7
Trimethoprim-sulfamethoxazole	88	7	5
Penicillin	87	8	5

Discussion

The results of IPD surveillance in Oregon through ABCs are largely consistent with those seen nationally. After the February 2000 licensure of PCV7, the incidence of IPD decreased dramatically. In 1998, two years prior to the introduction of the vaccine, the incidence rate was 56.8 per 100,000 among children under the age of five. In 2002, two years post-vaccine use, the incidence rate had decreased 70 percent to 17.3 per 100,000. While the year-to-year decreases were largest immediately following this event, our data indicate that IPD incidence, particularly those cases due to PCV7+ serotypes, remains low. Additionally, while PCV7 is most effective within the target population², the benefits of decreased incidence of PCV7-covered serotypes extend to other ages.³ With the decrease in these serotypes, the overall rates of IPD in 2008 (14 per 100,000 in those less than five and 35 per 100,000 in those 65 or older) remained below the Healthy People 2010 Objectives of 46 per 100,000 and 42 per 100,000, respectively.¹

The profile of IPD in Oregon is also consistent with previous studies, which have demonstrated that all of the underlying conditions reported frequently among cases are recognized risk factors for invasive disease.⁴⁻⁶

While rates of PCV7-type IPD have declined dramatically following PCV7 introduction, rates of IPD caused by some serotypes not included in PCV7, especially 19A, have increased since PCV7 introduction. Fortunately, as was previously mentioned, a new 13-valent pneumococcal conjugate vaccine (PCV13), which includes serotype 19A, was recently licensed by the FDA. The Oregon ABCs program will be participating in a post-licensure evaluation of PCV13 effectiveness to assess how well the vaccine works among children who receive the vaccine as part of routine immunization schedules. With the impending introduction of PCV13, the epidemiology of IPD will undoubtedly continue to evolve. We will be closely monitoring trends in Oregon.

References

1. Centers for Disease Control and Prevention. 2009. Active Bacterial Core Surveillance Report, Emerging Infections Program Network, *Streptococcus pneumoniae*, 2008. Available via the Internet: <http://www.cdc.gov/abcs/reports-findings/survreports/spneu08.pdf>.
2. Advisory Committee on Immunization Practices. Preventing pneumococcal invasive disease among infants and young children: recommendations of the Advisory Committee on Immunization Practices (ACIP) *MMWR Recomm Rep*. 2000;49(RR-9)1-35.
3. Lexau CA, Lynfield R, Danila R, et al. Changing Epidemiology of Invasive Pneumococcal Disease Among Older Adults in the Era of Pediatric Pneumococcal Conjugate Vaccine. *JAMA*. 2005;294(16):2043-51.
4. Lipsky BA, Boyko EJ, Inui TS, Koepsell TD. Risk factors for acquiring pneumococcal infections. *Arch Int Med*. 1986; 146(11):2179-85.
5. Levine OS, Farley M, Harrison LH et al. Risk factors for invasive pneumococcal disease in children: a population-based case-control study in North America. *Pediatrics*. 1999; 103: E28.
6. Talbot TR, Hartert TV, Mitchel E. Asthma as a Risk Factor for Invasive Pneumococcal Disease. *N Engl J Med*. 2005;352:2082-90.
7. Singleton RJ, Hennessy TW, Bulkow LR, Hammitt LL, et al. Invasive pneumococcal disease caused by nonvaccine serotypes among Alaska Native children with high levels of 7-valent pneumococcal conjugate vaccine coverage. *JAMA* 2007;297:1784-92.
8. Hicks LA, Harrison LH, Flannery B, et al. Incidence of disease due to non-pneumococcal conjugate vaccine (PCV7) serotypes in the United States during the era of widespread PCV7 vaccination, 1998-2004. *J Infect Dis* 2007; 196:1346-54.
9. Flannery B, Heffernan RT, Harrison LH, et al. Changes in invasive pneumococcal disease among HIV-infected adults in the era of childhood pneumococcal immunization. *Ann Intern Med* 2006; 144:1-9.