

# Streptococcus pneumoniae Surveillance Report 2007

Oregon Active Bacterial Core Surveillance (ABCs)

Office of Disease Prevention & Epidemiology

Oregon Department of Human Services

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## 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 pneumococcal disease (IPD) comprises the Tri-County (Clackamas, Multnomah, and Washington) Portland metropolitan area with a 2007 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

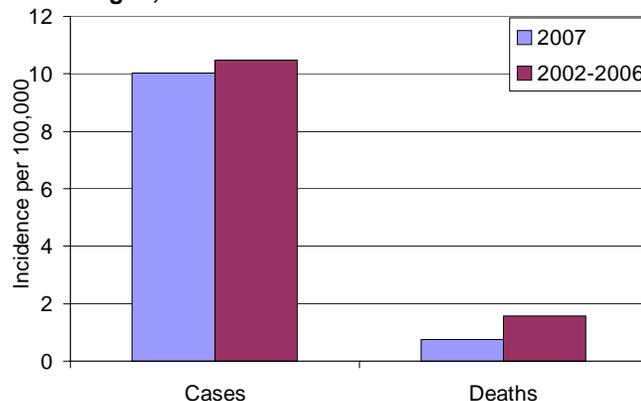
IPD is defined as the isolation of *S. pneumoniae* from a normally sterile body site in a Tri-County resident. Tri-County hospital laboratories submit *S. pneumoniae* isolates to the Oregon State Public Health Laboratory, which forwards them to a CDC-collaborative laboratory for serotyping and antimicrobial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

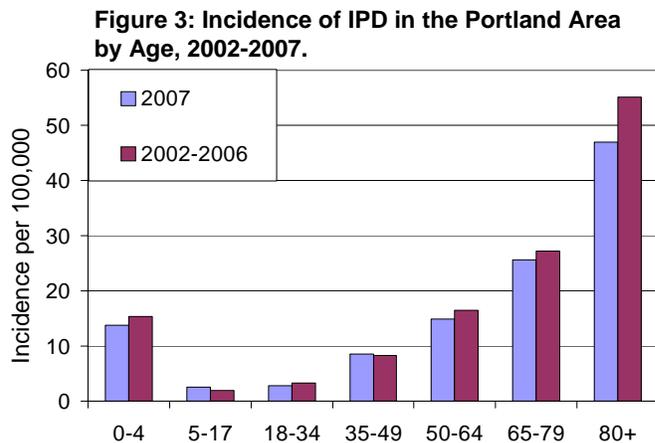
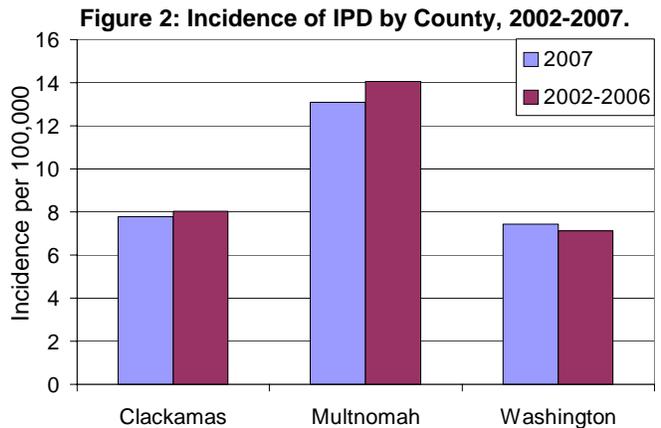
In 2007, 160 cases of IPD were reported in the Tri-County Portland area, corresponding to an incidence rate of 10.0/100,000 persons (Figure 1). This is 4% lower than the average annual incidence rate in the Portland area from 2002–2006 (10.5/100,000) and 29% lower than the 2007 national projection of invasive disease (14.0/100,000).<sup>1</sup> Of these cases, there were 12 deaths, for an annual mortality rate due to IPD of 0.75/100,000 (Figure 1). This is 52% lower than the 2002–2006 Portland area average annual mortality rate (1.6/100,000) and 50% lower than the 2007 national projections (1.5/100,000).<sup>1</sup> The mean and median ages of 2007 IPD cases were 52 and 56 years, respectively, while those of IPD deaths were 69 and 73 years, respectively. The 2007 case fatality rate for IPD in the Portland area was 7.5%, half that reported in the Portland area from 2002–2006 and less than the 11% reported from the entire ABCs network in 2007.<sup>1</sup> Over half (53%) of the cases were male; of 86 cases where race was known, 84% were white, 10% were black, and 6% were another race; and of 27 cases where ethnicity was known, 22% were

Figure 1: Incidence of IPD Cases and Deaths in Oregon, 2002-2007.



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 2007 (13/100,000) was higher than that reported from either Clackamas (8/100,000) or Washington (7/100,000) Counties. This is similar to the historical pattern throughout the Portland area from 2002-2006. In 2007, mortality due to IPD was similar across all three counties, with Clackamas County the highest at 0.81/100,000, followed by Washington (0.78/100,000) and Multnomah (0.70/100,000) Counties. Compared with the previous 5-year average, IPD mortality in 2007 was 68% lower in Multnomah, 25% lower in Washington, and 24% lower in Clackamas County.



The burden of disease was highest in those  $\geq 80$  years of age (24 cases; incidence 47/100,000 persons), followed by those 65-79 four years of age (27 cases; 26/100,000) and those 50-64 years of age (45 cases; 15/100,000) (Figure 3). Since 2002, the incidence of IPD has shown decreasing trends among those less than 18 years, with incidence in the remaining age groups roughly stable. However, compared with 2006, incidence was 64% higher among those 0-4 years of age and 37% higher among those 5-17 years of age in 2007.

In 2007, IPD mortality was also highest in those  $\geq 80$  years of age (4 deaths; 8/100,000), followed by those 65-79 years of age (3 deaths; 3/100,000). There were no deaths among individuals from 0-34 years of age and no 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 ( $p < 0.024$ ). Case fatality increased from 0% among those less than 35 to 17% in those 80 and over. Mortality due to IPD was 40-60% lower in 2007, compared to the 5-year previous average, across all age groups.

### *Clinical Manifestations*

The common clinical syndromes reported for IPD cases – 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 – syndromes in 2007 were similar in proportion to those reported over the previous five years – they 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).

**Table 1: Percent of IPD Reporting Clinical Syndromes.\***

Syndrome	2007	2002-2006
Bacteremic Pneumonia	74	74
Primary Bacteremia	13	12
Meningitis	9	9
Other Syndrome	6	6

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

**Table 2: Percent of IPD Cases with Reported Underlying Conditions, 2002-2007.\***

	Percent
Smoking	28
Cardiovascular disease	25
Cancer	19
COPD	18
Diabetes	16
Immunosuppression	15
Alcohol abuse	11
Asthma	9
CVA	7
None Reported	16

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

Among cases reported since 2002, clinical syndromes were further analyzed with regard to severity of disease, as measured by fatal outcome. In a multivariate, logistic regression model controlling for age, compared with other invasive syndrome, a fatal outcome was 2.5 times more likely (95% confidence interval [CI] 1.2, 5.0) with meningitis and 2.3 times (95% CI 1.1, 4.9) more likely with bacteremia. No difference in fatal outcome was seen between pneumonia and other invasive syndrome.

### *Underlying Conditions*

Table 2 lists underlying conditions that were reported in greater than 5% of IPD cases in the Portland area during 2002–2007. Overall, 84% of cases had at least one underlying condition, with the presence of any underlying conditions increasing with increasing age ( $p < 0.0001$ ).

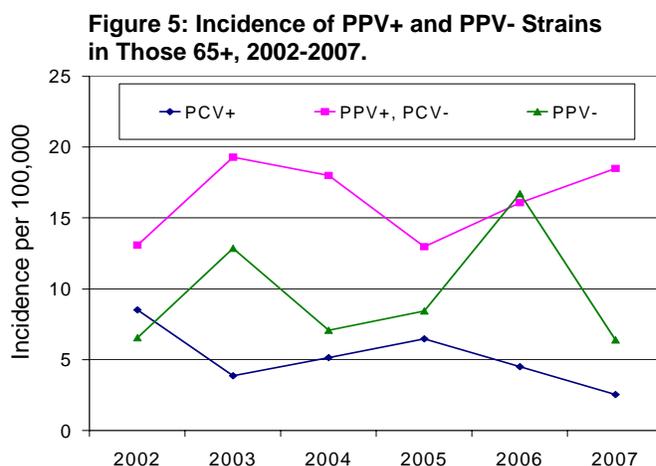
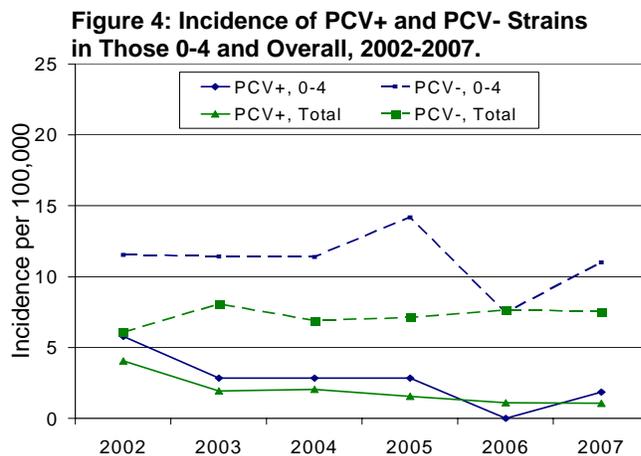
Among those less than 18 years of age, asthma was the most common condition (5%), with other underlying conditions reported rarely and over half (55%) of cases reported with no underlying condition. Among adults, smoking ( $p < 0.0001$ ) and alcohol abuse ( $P < 0.0001$ ) decreased with increasing age, while cardiovascular disease ( $p < 0.0001$ ), cancer ( $p < 0.0001$ ), chronic obstructive pulmonary disease (COPD) ( $p < 0.0001$ ), diabetes ( $p = 0.0002$ ), and cerebrovascular accident (CVA) ( $p < 0.0001$ ) increased.

Conditions affecting the lung – such as smoking, COPD, 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 with a previous CVA and those reporting alcohol abuse (95% CI 1.5, 4.6), and less likely among those with diabetes.

### Serotype Analysis

Figure 4 depicts the incidence of IPD due to strains of *S. pneumoniae* that are (PCV+) and are not (PCV-) included in the pneumococcal conjugate vaccine (PCV) among those 0-4 years of age and the overall population. From 2002-2007, the incidence of PCV+ strains decreased 68% among those 0-4 years of age. Overall, IPD incidence due to PCV+ strains decreased 73% over this time period. Although some year-to-year variation is seen, the incidence of IPD due to PCV- strains has been stable over the past six years.

The incidence of PCV+ strains among those over 64 also decreased, by 70%, over the past six years. (Figure 5). However, the roughly stable IPD occurrence within this age group was largely due to a 41% increase in incidence due to the 16 strains included in pneumococcal polysaccharide vaccine (PPV), but not PCV (PPV+/PCV-). The occurrence of PPV- strains also seems to be trending upward, although occurrence in 2007 was similar to that seen in 2002.



**Table 3: Age-Adjusted Significant Associations between Underlying Conditions and Syndrome or Death, 2002-2007**

	aOR	95% CI
<b>Pneumonia</b>		
Asthma	2.8	(1.7, 4.5)
COPD	2.4	(1.6, 3.8)
Smoking	2.2	(1.6, 3.1)
Diabetes	1.5	(1.0, 2.1)
<b>Bacteremia</b>		
Cancer	2.9	(2.0, 4.2)
<b>Fatal Outcome</b>		
Alcohol Abuse	2.6	(1.5, 4.6)
CVA	2.1	(1.2, 3.7)
Diabetes	0.4	(0.2, 0.7)
None Reported	0.4	(0.2, 0.9)

Since 2002, one non-vaccine serotype, 19A, has increased significantly in incidence, from 0.1 to 1.0 case per 100,000 in 2007. Although occurrence of serotype 19A is lower in 2007 than in 2006, 19A continues to show a significant increasing trend, as a proportion of IPD cases, from 8% in 2002 to 12% in 2007 ( $p=0.039$ ).

From 2002-2007, type 19A decreased with increasing age ( $p=0.0002$ ), but was not associated with clinical syndrome or fatal outcome due to IPD.

### Antibiotic susceptibility

The reported susceptibilities of isolates to a variety of antibiotics reflected a continued increasing trend that has been seen in recent years. In 2007, >99% of isolates were fully susceptible to amoxicillin, ceftazidime, ceftriaxone, chloramphenicol, clindamycin, linezolid, rifampin, and vancomycin. High susceptibilities to cefuroxime (97%), erythromycin (96%), and meropenem (98%) were also noted. Ninety-one percent of isolates were susceptible to penicillin, as were 84% to TMP/SULFA. The susceptibilities to erythromycin and

penicillin were the highest of any point within the past six years.

## Discussion

The results of IPD Surveillance in Oregon through ABCs are largely consistent with those seen nationally. In particular, after the March 2000 licensure of PCV, the incidence of IPD decreased dramatically. While the year-to-year decreases were largest immediately following this event, our data indicate that IPD incidence, particularly those cases due to PCV+ serotypes, has continued to decrease seven years post-licensure. Additionally, while PCV is most effective within the target population<sup>2</sup>, the benefits of decreased incidence of PCV-covered serotypes extend to other ages.<sup>3</sup> With the decrease in these serotypes, the overall rates of IPD in 2007 (14 per 100,000 in those less than five and 33 per 100,000 in those over 64) remained below the Healthy People 2010 Objectives of 46 per 100,000 and 42 per 100,000, respectively.<sup>1</sup>

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.<sup>4-6</sup>

Despite the continued decline in the burden of IPD overall, 2007 surveillance results are not all welcome news, particularly with regard to the increasing proportion of cases that are due to serotype 19A. While lower in incidence than in 2006, the continued upward trend in serotype 19A occurrence adds to the discussion of potential serotype replacement disease, in which IPD caused by serotypes not included in the conjugate vaccine may slow or reverse gains made against IPD. As serotype 19A has increased as a percentage of cases, especially among those less than five; and as serotype replacement disease has been seen among children, the elderly, and HIV-infected individuals elsewhere,<sup>7-9</sup> it may only be a matter of time before increases in IPD are seen more broadly in Oregon. The next few years should provide more insight into this phenomenon.

## References:

1. Centers for Disease Control and Prevention. 2008. Active Bacterial Core Surveillance Report, Emerging Infections Program Network, *Streptococcus pneumoniae*, 2007-provisional. Available at <http://www.cdc.gov/ncidod/dbmd/abcs/survreports/spneu07.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.