III. Disease Summaries
A. Coccioidiomycosis

Discussion of Surveillance Data

Coccioidiomycosis at the southwest regional level has been a nationally notifiable disease since 1995, which included a requirement for laboratory confirmation. In 1997, Arizona instituted mandatory laboratory reporting, which contributed to an increase in the number of reported cases, in addition to improving the timeliness and completeness of reporting. It is important to note that the increase in the incidence of coccioidiomycosis has continued to rise and therefore does not appear to be simply associated with increased reporting (Figure 2).

Reported cases of coccioidiomycosis have been increasing since 1997. To date, the highest number of cases reported in Arizona was in 2004 when a total of 3,654 cases of coccioidiomycosis were reported in Arizona (62.7 cases per 100,000 population), which represents a 281% increase since 1997.

![Figure 2. Rates of reported coccioidiomycosis, Arizona, 1993-2004.](image)

Most infections are sub-clinical or self-limited, and clinical manifestations range from influenza-like illness to severe pneumonia and, more rarely, extra-pulmonary disseminated disease. It is important to note, however, that hospitalizations associated with a diagnosis of coccioidiomycosis have substantially increased from 1998, indicating an increase in the number of cases that present with severe disease. Healthcare providers in Arizona may want to consider coccioidiomycosis in the differential diagnosis of patients with influenza-like illness given that the peak activity of influenza and coccioidiomycosis coincides.¹

Disease incidence in Arizona appears to peak in the winter from November to February and it is during these months that the increase in disease has risen consistently each year. This winter peak in Arizona varies from Southern California, where, in an earlier study, infection rates from

coccidioidomycosis were higher in late summer/early fall. However, in 2004, coccidioidomycosis cases appeared to peak earlier than usual with the highest number of cases reported in August. It is unknown whether this early season was due to unusual weather conditions, including increased amounts of rainfall, or awareness campaigns initiated in August.

A 2002 study of valley fever in Arizona indicated that the increase in coccidioidomycosis infections in the winter were associated with environmental factors and conditions, including the cumulative amount of rain in the preceding 7 months, the average temperature in the last 3 months, presence of dust in the last month, and the amount of rain in the last 2 months in proportion to the last 7 months. An increased likelihood of a seasonal outbreak occurring in the winter of a particular year seems to be preceded by a long period of drought, especially if it was in conjunction with hot and dusty conditions. Notably, this model accurately explained the absence of a peak in the winter of 2000-2001 (Figure 3).

Interestingly, the incidence rate in 2004 was slightly higher among 10-14 year olds than in previous years. In addition, the incidence rate among 55-59 year olds in 2004 was the highest observed in Arizona since coccidioidomycosis became reportable (Figure 4). However, the reasons for this increase are unknown. Overall incidence rates continue to be highest in

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persons aged ≥55 years, who are at highest risk of developing symptoms and therefore most likely to be diagnosed. Overall, males have a higher incidence rate than females, possibly due to occupational exposure and duration of outdoor activities. Incidence rates by county demonstrated the highest rate in Maricopa (76.6 per 100,000), followed by Pima (71.0 per 100,000), then Pinal (69.6 per 100,000); these same counties also had the highest number of cases in 2004: Maricopa with 2,701, Pima with 661, and Pinal with 152 (Figure 5).

Figure 4. Rates of reported coccidioidomycosis by age and year, Arizona, 1999-2004.

Figure 5. Rates of reported coccidioidomycosis, Arizona, 2004
B. Hepatitis B

In 2004, 289 cases (5.0/100,000) of acute hepatitis B and 1,147 cases (19.7/100,000) of chronic hepatitis B were reported in Arizona. While acute hepatitis B has been reportable in Arizona for many years, chronic hepatitis B has been tracked in the state only since 1998. Vaccination continues to be the most effective way to prevent hepatitis B infection. While hepatitis B vaccines have been available in the U.S. since 1981, they did not come into wide use until 1991 when a more comprehensive vaccination strategy was introduced to include routine vaccination of infants, vaccination of adolescents and adults at high risk of infection, and prenatal testing of pregnant women for hepatitis B surface antigen. Infection within the first year of life is a significant risk factor for chronic viral carriage, and thus much of the U.S. prevention program has focused on infants. Recent rates of perinatal infections have been low; no cases were identified in Arizona in 2004. Education to infected persons about transmission of the hepatitis B virus may also help to contain further spread.

Currently, classification of hepatitis B into acute and chronic cases in Arizona is largely based on the type of positive test reported. Many reports of hepatitis B are not investigated and classifications are usually based on reported test results. Therefore, cases with positive IgM results are recorded as acute infections, while a positive surface antigen test in the absence of a positive IgM result results in classification as a chronic infection. However, further investigation into symptoms, liver function, and past history of hepatitis B is warranted in order to more accurately classify cases.

Data for hepatitis B cases reflect the date of report to the state health department, not the date of infection or onset of symptoms. Acute hepatitis B rates in Arizona have been increasing slowly throughout the last decade, with a consistently high number of cases reported in the last three years (Figure 6). Meanwhile, the opposite trend was observed in the U.S. rates; the rate for 2003 (2.6/100,000) was approximately half of the 1994 rate (4.8/100,000). Rates of reported chronic hepatitis B in Arizona have been consistently higher than those for acute infections.

Figure 6. Rates of reported hepatitis B by year of report, Arizona, 1994-2004.

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Approximately 50% of acute cases and 30% of chronic cases reported in 2004 were between the ages of 40 and 59 years (Figures 7 & 8). Males account for 57% and 52% of acute and chronic cases, respectively. For acute hepatitis B, rates of reported cases in 2004 are higher among men for most age groups, with peak rates for both sexes in the 40 to 59 year age groups. Interestingly, peak rates of reported chronic hepatitis B by age group differ between the two sexes. Among men, rates are highest in the 45-49 year age group; peak rates for women occur in the 25 to 34 year age groups, with significant declines in older groups. Additionally, women in the 15 to 34 year age group have higher rates than men in the same age group whereas the reverse is true for all other age groups. Whether this represents more testing in women of child-bearing age or a distinct difference in infection or disease between men and women is unknown.

Figure 7. Rates of acute hepatitis B, by age and sex, Arizona, 2004.

Figure 8. Rates of chronic hepatitis B, by age and sex, Arizona, 2004.
While acute hepatitis B appears to have been increasing in Arizona in recent years, more information is needed about whether this increase represents a true rise in symptomatic cases, or rather an increase in requests for testing, changes in the sensitivity of the tests, or other factors. Further investigation of hepatitis B cases regarding risk factors, transmission, and reasons for testing is needed in order to better understand the epidemiology of hepatitis B in Arizona.
C. Influenza and Respiratory Syncytial Virus (RSV)

Influenza and respiratory syncytial virus (RSV) both became reportable by laboratories in October, 2004. While neither was a reportable disease before this change, influenza surveillance has been carried out for many years because of the potential public health impact of the virus. There are several purposes for influenza surveillance, which differ somewhat from those for other communicable diseases. These are to: determine where and when influenza cases are occurring; determine the predominant types and subtypes circulating in Arizona; assess the intensity and impact of activity; and detect emergence of novel influenza viruses or unusual events.

Influenza surveillance in Arizona relies on: sentinel providers, laboratory reports, subtyping of isolates, and hospital emergency department visits or school absenteeism in select counties. Sentinel physicians throughout the state submit weekly reports of influenza-like illness (ILI) to the U.S. Influenza Sentinel Provider Surveillance Network, a collaboration between health care providers, state and local health departments, and the CDC. These reports help to determine the period when influenza-like illnesses account for a larger proportion of patient visits, both statewide and nationally. Viral isolation and subtyping at the Arizona State Laboratory and other select laboratories detect the predominant circulating types and subtypes and identify any novel strains. Laboratory reports provide further indication of relative influenza activity levels. Since the 2003-2004 season, influenza-associated pediatric mortalities have been tracked and investigated.

On October 5, 2004, one of the two manufacturers of U.S. influenza vaccine announced that it would be unable to provide any doses for the 2004-2005 season. This resulted in a vaccine shortage that forced health departments and health care providers around the country to institute new strategies to protect those at highest risk of complications or severe infections. Vaccine was prioritized based on age or high-risk conditions.

Fortunately, the 2004-2005 influenza season was mild. Widespread activity was never declared in Arizona, and activity was regional from late January until mid-March, with activity peaking in February. This pattern is temporally similar to many influenza seasons in Arizona (Figure 9).

![Figure 9. Influenza activity, Arizona, 1997-2005.](image)
Nationally, influenza A(H3N2) predominated while influenza B circulated somewhat later in the season. In Arizona, approximately equal numbers of culture- or PCR-confirmed influenza A and influenza B were reported throughout the season (Figure 10). The Mountain Region (which includes Arizona) consistently had a higher proportion of influenza B than the rest of the country.

**Figure 10. Culture- or PCR-confirmed influenza, by type or subtype, Arizona, 2004-2005.**

The 2004-2005 influenza vaccine contained three components: A/Fujian or A/Wyoming(H3N2); A/New Caledonia(H1N1); and B/Shanghai of the Yamagata lineage. Early in the season, A/Fujian predominated nationally, but in November a new strain was identified, influenza A/California(H3N2), which accounted for the majority of antigenically-typed influenza by the end of the season (Figure 11). An A/California-like strain replaced the A/Fujian-like strain in the 2005-2006 vaccine. Two lineages of influenza B co-circulated in the U.S. Very little A(H1N1) circulated in the country.

**Figure 11. 2004-2005 Influenza season antigenic characterization.**

*Data as of June 24, 2005. Proportion of viruses characterized by CDC may not be representative of circulating viruses due to surveillance for less frequently seen subtypes collected by WHO/SIVC/99 laboratories throughout this season.*

**Courtesy CDC.**

Arizona Department of Health Services
Infectious Disease Epidemiology Section
Laboratory-reporting of influenza in Arizona began in 2004-2005 and thus baseline data are not available. However, this information proved valuable for monitoring the timing of activity in the state (Figure 12) and identifying counties where the virus circulated.

Figure 12. Laboratory-confirmed influenza, Arizona, 2004-2005.

Children less than 15 years of age accounted for 39% of reported influenza cases and the incidence rate was much higher in infants less than one year of age than in other age groups (Figure 13). This probably reflects a bias in diagnostic testing for influenza in the younger age groups. Infants and young children are susceptible to severe influenza and complications and have hospitalization rates comparable to those aged 65 years and over.\(^5\) However, infants may be more likely to visit a health care provider and be tested to distinguish influenza from other respiratory infections.

Respiratory syncytial virus (RSV) is a common respiratory infection, especially among infants and young children. It follows a seasonal pattern similar to influenza, though peaks in the seasons will often occur at different times. Because RSV reporting started late in 2004, there are no past data for comparison. Case-specific information on RSV is not available. The RSV reports during the 2004-2005 season peaked in late January, preceding the influenza peak (Figure 14).
D. Methicillin-resistant *Staphylococcus aureus*

Sterile site isolates of Methicillin-resistant *Staphylococcus aureus* (MRSA) became reportable by laboratories in October 2004. The purpose of this new surveillance is to determine the incidence of invasive disease due to MRSA in Arizona. Historically, MRSA has been associated with outbreaks in the hospital and long term care facility settings. In the past few years, there has been an increasing number of outbreaks identified in community settings. These have included outbreaks associated with athletes and sporting events. Community acquired-MRSA (CA-MRSA) infections are those acquired by persons who have not been hospitalized within the past year, or have not had a medical procedure (such as dialysis, surgery, catheters). These CA-MRSA infections are an emerging problem in Arizona. Patients, health care providers, and facilities are increasingly burdened with infections that do not resolve with normal first line therapy. Subsequent healthcare provider visits and testing are needed to diagnose and successfully treat these patients.

*Staphylococcus aureus* is part of the normal skin flora and is the most common cause of skin infections. It is not possible to differentiate colonization and infection with only the laboratory information; therefore, asking laboratories to send information on all MRSA isolates would not assist in classifying cases and would create a reporting burden for the labs. The majority of CA-MRSA infections are skin and/or soft tissue infections and will not be identified by the new reporting requirement unless the organism spreads to the blood.

National surveillance for MRSA is currently under development at the Centers for Disease Control and Prevention (CDC). It usually takes several months for labs to begin reporting new diseases on a consistent basis. Approximately 125 invasive MRSA isolates have been reported monthly in the first three months of reporting. Additional resources are necessary to ascertain epidemiologic characteristics and risk factors, and to categorize healthcare- versus community-associated MRSA.

Additional information on MRSA infections is available at [http://www.cdc.gov/ncidod/hip/ARESIST/mrsa.htm](http://www.cdc.gov/ncidod/hip/ARESIST/mrsa.htm).
E. Invasive Meningococcal Disease

Meningococcal disease is caused by the bacteria *Neisseria meningitidis* and is the most common cause of bacterial meningitis for toddlers, adolescents and young adults in the U.S. *N. meningitidis* is divided into numerous serogroups based on immunogenicity, but 95% of illness worldwide is caused by five serogroups: A, B, C, Y and W-135. *N. meningitidis* is spread via respiratory and nasal secretions. Case fatality has decreased with antibiotic treatment; however, it remains high at 10%. A quadrivalent polysaccharide vaccine has been available in the U.S. since 1978 and covers serogroups A, C, Y and W-135, but does not provide protection against serogroup B, which is common in the U.S. This vaccine has been used to control outbreaks and is also given to people at increased risk of acquiring meningococcal disease; however, it is not approved for use in children under the age of 2 years.

The reported rate of invasive meningococcal disease in Arizona has been decreasing over the past decade (Figure 15). In 2004, only 15 cases reported statewide, the lowest rate in 10 years. There were no deaths reported in 2004 attributed to meningococcal disease.

Figure 15. Rates of reported invasive meningococcal disease, Arizona, 1994-2004.

![Graph showing rates of reported invasive meningococcal disease, Arizona, 1994-2004.](image)

Rates of meningococcal disease vary by age group (Figure 16). The highest incidence rate occurs in children under one year, followed by children ages 1-4 years. Adolescents also have high rates of infection compared to other age groups. A large percentage of disease in those under one year (49% of those with known serotype in 1994-2004) is caused by serogroup B and thus is not currently vaccine-preventable (Figure 17). Analysis of meningococcal cases indicates that over 50% of infections in Arizona are caused by serogroups represented in the current vaccine. As indicated in Figure 17, a high percentage of Arizona cases, especially among adolescents and adults, are potentially vaccine-preventable.
Figure 16. Rates and cases of reported invasive meningococcal disease, Arizona, 1994-2004.

Figure 17. Serogroup distribution by age group, invasive meningococcal disease, Arizona, 1994-2004.

The serogroup distribution for the years 1994-2004 in Arizona is shown in Figure 18. Nationally, the proportion of meningococcal cases caused by serogroup Y has increased from 2% in 1989-
1991 to 37% in 1997-2002. However, no clear trend in serogroup distribution has been observed in Arizona over the similar time frame shown below. In 2004, serogroups C and Y each accounted for approximately 50% of cases with known serogroup; however, 47% of the reported cases were not serogrouped.

**Figure 18. Meningococcal serogroups, invasive disease, Arizona, 1994-2004.**

The introduction of new immunization recommendations for adolescents and young adults will likely reduce the incidence of meningococcal disease and may result in changing the epidemiology of the disease in Arizona.

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F. Norovirus

Background

Norovirus is a common cause of gastroenteritis both nationally and in Arizona and is associated with acute illness typically of short duration. Symptoms include nausea, vomiting, abdominal pain, headache, watery diarrhea, low-grade fever or any combination of these symptoms. The virus is usually spread via the fecal-oral route; however, airborne transmission through aerosolization of emesis has been documented. Norovirus has been associated with foodborne and waterborne outbreaks, but person-to-person spread is the most common source of cluster. Attack rates of greater than 50% are common during outbreaks and the virus spreads easily in long term care facilities, schools, daycares, and other institutions in which people are in close contact.

While individual cases of norovirus are not reportable in Arizona, outbreaks of gastrointestinal illness are required to be reported and investigated. Since 2002, norovirus surveillance in Arizona has increased with the addition of testing capabilities at the Arizona State Public Health Laboratory (ASPHL). This has enhanced our capacity to respond to gastrointestinal outbreaks and has allowed for confirmation of suspected outbreaks of viral gastroenteritis within two days of specimen collection.

Surveillance Data

Norovirus was suspected or confirmed as the causative organism in over half of the documented outbreaks of gastrointestinal illness in Arizona in 2004. Seventeen outbreaks of norovirus were confirmed in 2004, compared with six in 2003 and none in 2002. Ten gastrointestinal clusters were of unknown etiology; some of these may have been due to norovirus, but no clinical specimens were obtained. Peaks of activity in Arizona in 2004 occurred in late spring and winter (Figure 19).

Through genetic sequencing done at the State Laboratory, a wide variety of norovirus clones have been identified since this technique was implemented in 2003 (Figures 19 & 20). The percentage of sequenced outbreaks continues to increase over time, enhancing our capacity to identifying trends associated with the spread of norovirus clones. Although data are not available for individual cases, sequencing information can be useful in monitoring the epidemiology of infection and suggesting sources of infection. In Arizona, several different clones were identified throughout 2004 and varied depending on the location and source of the cluster. While the majority of norovirus clones seen nationally are of the GII.4 or “Farmington Hills” strain, Arizona data have not indicated a trend towards this clone. However, data represent reported outbreaks only and sequencing data are unavailable for over 50% of outbreaks, limiting our ability to interpret data and identify trends.

**Figure 20. Identified norovirus clones, Arizona, 2004.**

- Farmington Hills: 7%
- GI/634: 4%
- Oxford (B2S16): 11%
- Oxford (B8S5): 11%
- Parris Island: 4%
- Saitama: 7%
- Oxford (B6S6): 4%

**Summary of Norovirus Outbreaks in Arizona, 2004**

Norovirus contributed to more foodborne outbreaks in Arizona during 2004 than any other pathogen. It is believed that three factors contribute to this observation, including the low infective dose, large human reservoir of infection, and the ability to be transmitted by a variety of routes. Norovirus outbreaks in Arizona during 2004 have been documented in long-term care and assisted-living facilities, hospitals, schools, restaurants, cruises, and among members of sports teams.
The largest outbreak of norovirus documented in Arizona during 2004 occurred among students and employees at a Maricopa County elementary school. When the school reported the cluster, it was estimated that over 150 children had been absent due to illness. This outbreak reinforced the need to exclude actively ill students, practice careful and prompt disposal of vomit, and appropriately disinfect all surfaces during norovirus outbreaks in school settings.

There have been several documented cases of norovirus clusters among athletic teams, both nationally and statewide. Members of sports teams have close contact and typically share personal items, allowing for enhanced person-to-person transmission. An investigation of a norovirus outbreak among a football team in Pima County found environmental and person-to-person transmission among players; however, a specific exposure was not identified.
G. Shigellosis

Although Shigella was first identified over 100 years ago, it continues to infect millions of people every year. Shigellosis is endemic throughout the world and there are approximately 164.7 million cases worldwide, of which 163.2 million are in developing countries and 1.5 million in industrialized countries. In 2004, Arizona reported 409 cases of shigellosis, which represents the lowest annual number of cases reported since 1980.

Shigella is a highly contagious bacterium that infects the intestinal tract of humans. The genus Shigella consists of four species: Group A, Shigella dysenteriae; Group B, S. flexneri; Group C, S. boydii; and Group D, S. sonnei. In general, S. dysenteriae, S. flexneri, and S. boydii account for most isolates in developing countries. In industrialized countries, S. sonnei is most common followed by S. flexneri. This trend is consistent within Arizona, where each year the number of cases of S. sonnei outnumbers those for S. flexneri.

Shigellosis has shown a cyclical trend, ranging from 6 to 30 years. Some researchers believe that this cyclical pattern may reflect the development of population-based serotype specific immunity. The cyclical trend of shigellosis is evident when analyzing the number of cases in Arizona by year, as shown in Figure 21. Shigellosis outbreaks were common with a peak of over a thousand cases of shigellosis reported in 1990 (1,995). Although statewide numbers of shigellosis were at decreased levels during 2004, localized clusters were identified in Arizona; however, they involved a small number of people.

Figure 21. Reported Shigella cases, Arizona, 1980–2004.

Additional trends are identified when analyzing Shigella according to serotype. Figure 22 presents the number of shigellosis cases reported in Arizona over the last 12 years by total cases and by the two most commonly reported species, S. sonnei and S. flexneri. As shown, the number of S. flexneri cases has remained relatively constant over the last six years, while
the number of \textit{S. sonnei} cases has fluctuated. In general, \textit{S. sonnei} species are the most common type found in large outbreaks in the United States. Therefore, it appears that recently the number of \textit{S. sonnei} cases is most often responsible for the fluctuating trends in the number of shigellosis cases reported in Arizona.

\textbf{Figure 22. Reported Shigella cases by serotype, Arizona, 1992–2004.}
H. Invasive *Streptococcus pneumoniae*

Surveillance of invasive *Streptococcus pneumoniae* (pneumococcal disease) through laboratory reporting has been ongoing in Arizona since April 1997. In 2004, 670 cases were reported (11.5 cases per 100,000 population), compared to 718 cases reported in 2003 (12.9 cases per 100,000 population) representing a 7% decrease in cases (Figure 23). This decrease in incidence is thought to be due to the continuing use of the pneumococcal conjugate vaccine for children. Other states have reported a greater decrease in disease than is seen in Arizona.

**Figure 23. Rates of reported invasive *Streptococcus pneumoniae* infection, Arizona, 1997-2004.**

Eighty seven children less than five years old were reported with invasive pneumococcal disease in Arizona in 2004 compared to 200 cases in 2000. The significant drop in cases is likely due to the use of the pneumococcal conjugate vaccine in this age group. Forty six (53%) of cases were male. Of the 46 children with known disease outcome, four died.

Resistance to penicillin decreased from a high of 46% in 2000 to 25% in 2004. Thirty-two of the isolates were available at the Arizona State Public Health Laboratory for serotyping. Twenty six (81%) were typable. Only two of the isolates from children were serotypes found in the vaccine, 19F and 6B. Pneumococcal types typically found in Arizona children include: 1, 3, 6, 7F, 9, 10, 10A, 12, 15, 15B, 19, 19A, 22, 23, and 33. The most common isolate identified was 19A in four patients.

Antibiotic resistance and treatment of *Streptococcus pneumoniae* invasive disease has become an emerging world, national and state problem. In the United States, drug-resistance *Streptococcus pneumoniae* (DRSP) has increased substantially in the past fifteen years; DRSP varies regionally and has been reported to be over 30% in some areas of the U.S. In 1999, ADHS began posting antibiograms of ISP on the Infectious Disease Epidemiology Program’s website on a quarterly basis (Figures 24 & 25). Children less than five years of age have higher rates of penicillin non-susceptibility (Figures 26 & 27). In addition, children under age 5 showed an increase in high level resistance in 2000.
**Figure 24.** Antibiotic susceptibility among reported invasive *Streptococcus pneumoniae* isolates, Arizona, 2004.

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Number of Isolates Tested</th>
<th>Susceptible [n (%)]</th>
<th>Intermediate [n (%)]</th>
<th>Resistant [n (%)]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin</td>
<td>356</td>
<td>286 (80)</td>
<td>47 (13)</td>
<td>17 (5)</td>
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<tr>
<td>Ceftriaxone</td>
<td>294</td>
<td>282 (96)</td>
<td>8 (3)</td>
<td>2 (1)</td>
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<tr>
<td>Vancomycin</td>
<td>285</td>
<td>283 (100)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>309</td>
<td>276 (89)</td>
<td>5 (1)</td>
<td>27 (9)</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>66</td>
<td>65 (98)</td>
<td>0 (0)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Levofloxacin</td>
<td>180</td>
<td>175 (97)</td>
<td>5 (3)</td>
<td>0</td>
</tr>
</tbody>
</table>

**Figure 25.** Antibiotic susceptibility among reported invasive *Streptococcus pneumoniae* isolates in children less than 5 years, Arizona, 2004.

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Number of Isolates Tested</th>
<th>Susceptible [n (%)]</th>
<th>Intermediate [n (%)]</th>
<th>Resistant [n (%)]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin</td>
<td>34</td>
<td>23 (67)</td>
<td>6 (17)</td>
<td>5 (14)</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>23</td>
<td>22 (95)</td>
<td>0</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>30</td>
<td>30 (100)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>37</td>
<td>29 (78)</td>
<td>1 (.02)</td>
<td>7 (18)</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>7</td>
<td>7 (100)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Levofloxacin</td>
<td>12</td>
<td>11 (91)</td>
<td>1 (9)</td>
<td>0</td>
</tr>
</tbody>
</table>

**Figure 26.** Penicillin non-susceptibility among reported invasive *Streptococcus pneumoniae* isolates, Arizona, 1998-2004.
Figure 27. Penicillin non-susceptibility among reported invasive *Streptococcus pneumoniae* isolates in children <5 years, Arizona, 1998-2004.

The distribution of cases by county in 2004 is shown in Figure 28.

Figure 28. Rates and number of cases of invasive *Streptococcus pneumoniae*, 2004.