2017

Vaccine-Preventable Disease Surveillance in Kentucky

Katherine Jay

University of Kentucky, katherine.jay@uky.edu
STUDENT AGREEMENT:

I represent that my thesis or dissertation and abstract are my original work. Proper attribution has been given to all outside sources. I understand that I am solely responsible for obtaining any needed copyright permissions. I have obtained and attached hereto needed written permission statement(s) from the owner(s) of each third-party copyrighted matter to be included in my work, allowing electronic distribution (if such use is not permitted by the fair use doctrine).

I hereby grant to The University of Kentucky and its agents the non-exclusive license to archive and make accessible my work in whole or in part in all forms of media, now or hereafter known. I agree that the document mentioned above may be made available immediately for worldwide access unless a preapproved embargo applies.

I retain all other ownership rights to the copyright of my work. I also retain the right to use in future works (such as articles or books) all or part of my work. I understand that I am free to register the copyright to my work.

REVIEW, APPROVAL AND ACCEPTANCE

The document mentioned above has been reviewed and accepted by the student’s advisor, on behalf of the advisory committee, and by the Director of Graduate Studies (DGS), on behalf of the program; we verify that this is the final, approved version of the student’s dissertation including all changes required by the advisory committee. The undersigned agree to abide by the statements above.

Katherine Jay, Student

Steven Browning, PhD, MSPH, Major Professor

Corrine Williams, ScD, MS, Director of Graduate Studies
Vaccine-Preventable Disease Surveillance in Kentucky

Capstone Project Paper

A paper submitted in partial fulfillment of the requirements for the degree of Master of Public Health in the University of Kentucky College of Public Health

By

Katherine Jay
Lexington, Kentucky
May 24, 2017

_____________________________________
Steven R. Browning, PhD, MSPH, Chair

_____________________________________
Lorie Wayne Chesnut, DrPH, MPH

_____________________________________
Kathleen Winter, PhD, MPH
Contents

Abstract.........................................................................................................................4
Introduction....................................................................................................................5
  Vaccine-Preventable Disease Surveillance.................................................................5
  Hepatitis B..................................................................................................................8
  Pertussis .....................................................................................................................9
Methods.......................................................................................................................9
Report............................................................................................................................12
  Diphtheria................................................................................................................12
  Haemophilus influenzae..........................................................................................15
  Hepatitis A...............................................................................................................19
  Hepatitis B, Acute....................................................................................................22
  Hepatitis B, Chronic...............................................................................................27
  Measles.....................................................................................................................30
  Meningococcal disease............................................................................................34
  Mumps.......................................................................................................................37
  Pertussis...................................................................................................................40
  Poliovirus infection..................................................................................................47
  Rubella.......................................................................................................................49
  Tetanus......................................................................................................................55
  Varicella....................................................................................................................57
Discussion.....................................................................................................................60
  Hepatitis B, Acute....................................................................................................60
Pertussis........................................................................................................62
Surveillance in Kentucky..................................................................................63
References.........................................................................................................66
Acknowledgments............................................................................................70
Biographical Sketch........................................................................................71
Abstract

Background: Vaccine-preventable disease (VPD) surveillance is important component to the success of vaccines. This report examines the levels of 13 of these VPDs in the state of Kentucky from 2005 to 2015.

Methods: All reported cases in the state of Kentucky for the years 2005 to 2015 of 13 vaccine-preventable diseases were obtained from the Disease Surveillance Module (DSM) and the National Electronic Disease Surveillance System (NEDSS). Where enough cases were reported, descriptive statistics were prepared. For acute hepatitis B and pertussis, more thorough analysis was performed.

Results: Rates of acute hepatitis B in Kentucky from 2005 to 2015 were higher than the rates seen overall in the United States. Rates of pertussis over the same period were more similar to national trends.

Conclusion: Changes to VPD surveillance in Kentucky would allow for a more thorough and informative report. An increased focus on collection of demographic data as well as risk factors would greatly improve future reports.
Introduction

Vaccine-Preventable Disease Surveillance

Vaccine-preventable diseases (VPDs) have long been an essential focus of public health in the United States. Prior to the development and wide-spread use of vaccines, many diseases that are rarely seen today had high morbidity and mortality. For example, in 1921 more than 15,000 Americans died from diphtheria. An effective vaccine was developed for diphtheria in the 1920s and today, only one case has been reported to the Centers for Disease Control and Prevention (CDC) since 2004. Vaccine-preventable diseases also have costs outside of their morbidity and mortality. These include societal and economic costs as well as missing school and work, visiting the doctor, and hospitalizations. While many vaccines have been very successful in controlling or even eliminating certain diseases, continuing to monitor the levels of these illnesses remains necessary to their continued control.

While many vaccines have seen great success in controlling vaccine-preventable diseases, immunization and the study of these diseases remains an important focus of public health. Some diseases, while at low levels in the United States, have not truly been eliminated and are still found elsewhere in the world. This means that these diseases can still be imported into the United States through travel. Measles transmission has been considered to be interrupted in the United States since a record low number of cases was reported in 2004. However, cases of measles continue to be seen in the U.S. after being imported from countries where the disease is still endemic. This underscores the importance of maintaining high levels of vaccination, even when numbers of cases are
low. When populations or groups of people are under-immunized, it opens the door for outbreaks of these preventable diseases. It also puts people who are unable to be vaccinated at increased risk of encountering a vaccine-preventable disease.¹ Despite all this, vaccine coverage has not always been high and vaccination in the United States has not always been a requirement, even when effective vaccines were available.

A national survey conducted in 1941 examined attitudes of the American people towards immunizations to gauge readiness for compulsory immunizations for the public.⁶ Immunizations were already required for those in the armed forces, and it was recognized that immunizing the public would help protect them from the increased possibility of the spread of communicable diseases during wartime.⁶ The results of this survey showed that only half of the population in need of vaccination were willing to be immunized. The results also showed that the public was not well informed about vaccinations overall.⁶ It was not until a surge in the number of polio cases between the early 1940s and 1952 that attitudes really began to change.⁷

Vaccine-preventable disease surveillance grew as national programs for immunization were developed. One of the early examples of surveillance of VPDs came after the inactivated poliomyelitis vaccine was licensed in 1955.² The Poliomyelitis Surveillance Unit was established by the then-CDC in response to cases of paralytic poliomyelitis following vaccination, just days after the first case was recognized. Special officers for polio reporting were designated and were responsible for reporting cases of poliomyelitis in vaccinated individuals, as well as cases of family members and close contacts. The data were collected by the Poliomyelitis Surveillance Unit and disseminated in surveillance reports, with the first report being released only three days
after the surveillance was started. This swift surveillance allowed for the recognition that the problem was associated only with vaccine produced by a single manufacturer. Not only did this realization remove dangerous vaccines from the market, it also allowed continuous vaccination with the safely produced vaccines. This was an early case of modern surveillance, where data were collected and rapidly distributed after analysis, instead of just being archived.\textsuperscript{8}

Since the beginning of this new type of surveillance, technology has streamlined and increased the effectiveness of the process. Reporting of cases was originally done over the phone. Computer data systems, which were developed in the 1980’s, allowed reporting to be done electronically. Data were able to be reported through the National Electronic Telecommunications System for Surveillance (NETSS) starting in 1989. Beginning in 2000, federal funding was given to states to plan for and implement “integrated electronic systems for disease surveillance” which later became the National Electronic Disease Surveillance System (NEDSS), the system that is used to report diseases to the National Notifiable Disease Surveillance System (NNDSS) today. This system allows for easier and faster access to information related to case notifications, which includes both epidemiological and demographic information.\textsuperscript{8}

Notifiable diseases are reported to state and local health departments by healthcare providers, laboratories, and public health personnel. Since disease reporting requirements are mandated by the state, diseases that are reportable differ from state to state. For selected diseases, state health departments send notifications of cases to the CDC through NEDSS and the NNDSS.\textsuperscript{8} Because of the difference in reporting
requirements between states, it is important to look at vaccine-preventable diseases at the state level and not just nationally.

Hepatitis B

Groups at the highest risk of hepatitis B are those where contact with blood from infected persons is frequent or more likely. In the United States, one of the most important modes of transmission is perinatal because the likelihood of transmission from infected mother to child at birth is very high without appropriate post-exposure prophylaxis. Sexual contact with an infected person is another important mode of transmission.²

Risk factors associated with infection are unprotected sex with an infected partner, multiple partners, men who have sex with men, history of other STDs, and injection drug use. Household contact with an infected person and exposure to hepatitis B virus infected blood or body fluids in the healthcare setting are also risk factors.²,⁹

Overall, incidence in the United States of acute hepatitis B has remained stable at around 1 case per 100,000 persons from 2006 to 2014.¹⁰-¹² Over this same time period, Kentucky has seen an increase in the rate of hepatitis B for every year except 2014. The highest rate was seen in 2013 with 4.9 cases per 100,000.¹⁰,¹² A study looking at HBV infection in three similar states (Kentucky, West Virginia, and Tennessee) saw an increase in incident cases of HBV among non-Hispanic whites with injection drug use as a common risk factor from 2009 to 2013, while rates remained stable in the United States overall.¹¹
**Pertussis**

Infants under one year of age are at the greatest risk of hospitalization and death from pertussis, especially those who are too young to be vaccinated.\(^2\) This age group has the highest rate of pertussis in the United States overall, as well as in Kentucky.\(^{13,14}\)

United States pertussis incidence peaks every 3 to 5 years as the number of individuals in the population who are susceptible to pertussis increases. Immunity conferred by the acellular pertussis vaccine wanes more quickly than the previously used whole-cell vaccine. This is contributing to the changing epidemiology of pertussis, with an increasing number of cases being seen in previously vaccinated adolescents.\(^{15}\)

The purpose of this project is to generate a comprehensive report that describes the epidemiology of 13 VPDs in the state of Kentucky from 2005 to 2015. The diseases to be included in the report are diphtheria, *Haemophilus influenzae* disease, hepatitis A, acute hepatitis B, chronic hepatitis B, measles, meningococcal disease, mumps, pertussis, poliovirus infection, rubella, tetanus, and varicella. Special attention will be paid to acute hepatitis B and pertussis due to the large volume of cases seen over the specified time period. This report will allow for the examination of past trends in these preventable diseases and will be important to examining how these trends change going forward.

**Methods**

The data for this report were obtained from the Kentucky Department for Public Health and came from both the Disease Surveillance Module (DSM) and the National Electronic Disease Surveillance System (NEDSS). The DSM was used for disease surveillance and reporting in Kentucky prior to the adoption of NEDSS.\(^{16}\) NEDSS is an
online framework that supports the National Notifiable Disease Surveillance System (NNDSS). NEDSS allows for electronic data exchanges as well as case notifications. NEDSS also helps with the reporting of diseases by connecting the healthcare system to health departments, and those health departments to the CDC.17

The de-identified data contained all reported cases in the state of Kentucky for the years 2005 to 2015 of 13 different vaccine-preventable diseases. The diseases included in the data were diphtheria, invasive *Haemophilus influenzae* disease, hepatitis A, acute hepatitis B infection, chronic hepatitis B infection, measles, meningococcal disease, mumps, pertussis, poliovirus infection, rubella, tetanus, and varicella. The data from 2005 to 2012 came from the DSM, and the data from 2011 to 2015 came from NEDSS, with the overlapping years containing data from both sources. The two sets were merged to create a single dataset with all cases from 2005 to 2015.

The data contained many missing values, which were accounted for in different ways depending on the variable. Any records missing a confirmed disease condition were removed. For cases where age was missing, an age was calculated by using the date of birth and the Morbidity and Mortality Weekly Report (MMWR) Year. MMWR Year is a variable that indicates what year the case was reported. Sixty-four records were missing both age and date of birth and were removed because an age was unable to be calculated, and age was a necessary variable for the report. For cases with missing state, the state was assumed to be Kentucky per instructions from the KDPH.

Each record contained multiple variables relating to when the case happened or was reported. These include MMWR Year, Reported Year, and Case Confirm Date. In
most cases where all three of these variables were non-missing, they all had the same year and were therefore interchangeable to determine the year for the report. For consistency, MMWR Year was used when available. If MMWR Year was missing, then Reported Year was used. One record was missing both MMWR Year and Reported Year, so Case Confirm Date was used.

Race and ethnicity were missing for a large number of records. These values were coded as missing, but cases were not excluded based on missing race or ethnicity because of the large number of missing values in these variables.

Case Determination is a variable that was used to determine whether a reported case was actually a case of the disease. Twenty-two records were excluded due to missing case determination.

The data contained many duplicate records. Since data was de-identified and name could not be used to confirm duplicates, duplicate entries that matched on three separate variables were considered the same case and collapsed into one entry. The three variables used were Date of Birth, County, and Date Diagnosed. If Date Diagnosed was missing, then Case Confirm Date, or MMWR Week and MMWR Year were used.

For rate calculations, population estimates were obtained from the National Center for Health Statistics (NCHS).
Diphtheria

Diphtheria is caused by *Corynebacterium diphtheria*, an aerobic gram-positive bacterium. The bacteria itself must be infected with a specific virus that contains genetic information to produce toxin. Only these toxigenic strains of the bacteria cause severe disease.

Diphtheria infection is classified based on infection site. Toxigenic diphtheria bacilli in any mucous membrane of a susceptible individual will produce a toxin that inhibits the synthesis of proteins in cells. This toxin destroys tissue at the site of infection and causes the formation of a pseudomembrane. The toxin can also be absorbed into the bloodstream and spread throughout the body, leading to complications like myocarditis, neuritis, and death. The most common infection sites are pharyngeal and tonsillar, where the pseudomembrane can cause respiratory obstruction.

Diphtheria is usually spread person-to-person via the respiratory tract, although transmission can also occur through contact with discharge from skin lesions of an infected person. Diphtheria has an incubation period of 2 to 5 days with a range of 1 to 10 days. The case fatality rate is 5 to 10%. For certain age groups (less than 5 years of age and greater than 40 years of age) the case fatality can be as high as 20%.

Diphtheria toxoid was developed in the early 1920s and began being widely used in the 1930s. The toxoid is available in combination as pediatric diphtheria-tetanus toxoid (DT) or adult tetanus-diphtheria (Td). It is also combined with tetanus toxoid and the
pertussis vaccine as DTaP and Tdap. The usual vaccine schedule for children is 3 or 4 doses and a booster, as well as boosters every 10 years. Doses should be given at 2, 4, 6, and 15 to 18 months of age with a booster at ages 4 to 6 years.

Epidemiology

Prior to development and widespread use of the diphtheria toxoid, diphtheria was a major cause of morbidity and mortality. In the United States in 1921, there were more than 200,000 cases reported, with 5% to 10% of cases being fatal. Numbers of cases and deaths began to decrease gradually after the toxoid was developed. When the diphtheria toxoid began to be used more widely in the 1940s, numbers of cases decreased more rapidly. Diphtheria is now very rare in the United States. From 1998 to 2004, only seven cases were reported. The toxigenic bacterium that causes diphtheria still circulates in parts of the country where the disease was previously endemic, which highlights the importance of continued vaccination.

Surveillance Case Definition (2001)

Probable: In the absence of a more likely diagnosis, an upper respiratory tract illness with an adherent membrane of the nose, pharynx, tonsils, or larynx; and absence of laboratory confirmation; and lack of epidemiologic linkage to a laboratory-confirmed case of diphtheria.

Confirmed: An upper respiratory tract illness with an adherent membrane of the nose, pharynx, tonsils, or larynx; and any of the following: isolation of Corynebacterium diphtheriae from the nose or throat; or histopathologic diagnosis of diphtheria; or epidemiologic linkage to a laboratory-confirmed case of diphtheria.
Epidemiologic Summary

No cases of diphtheria were reported in Kentucky between 2005 and 2015.

Notes


ii: CDC. Preventing tetanus, diphtheria, and pertussis among adults: use of tetanus toxoid, reduced diphtheria toxoid and acellular pertussis vaccines. Recommendations of the Advisory Committee on Immunization Practices (ACIP) and Recommendation of ACIP, supported by the Healthcare Infection Control Practices Advisory Committee (HICPAC), for Use of Tdap Among Health-Care Personnel. MMWR 2006;55(No. RR-17).
Haemophilus influenzae

Background

*Haemophilus influenzae* bacteria can cause severe infection, particularly in infants. There are six encapsulated strains (types a-f) as well as unencapsulated strains. All encapsulated strains can cause similar invasive disease, but only *Haemophilus influenzae* type b (Hib) is vaccine preventable. The polysaccharide capsule is an important virulence factor, unencapsulated strains rarely cause serious infection. Prior to vaccine development, type B caused 95% of invasive disease.

The most common types of invasive disease caused by *H. influenzae* are meningitis, epiglottitis, pneumonia, arthritis, and cellulitis. It is most likely spread through respiratory droplets.

The first conjugate vaccine for Hib was licensed in the United States in 1987. There are currently three conjugate vaccines currently licensed for use. All infants, including preterm infants, should receive a primary series of the Hib vaccine beginning at 2 months of age. The primary series contains either 2 or 3 doses depending on which vaccine is used, with a booster at 12 to 15 months of age for both vaccines. The vaccine is not recommended for children younger than 6 weeks of age because doses given at this time may reduce the response to doses given later.

Epidemiology

Before effective vaccines were introduced in the 1980s, one in 200 children less than 5 years old developed invasive Hib disease. Incidence of Hib disease has decreased by more than 99% since the licensure of conjugate Hib vaccine.
Surveillance Case Definition (2014)*

Clinical case description

Invasive disease caused by H. influenzae can produce any of several clinical syndromes, including meningitis, bacteremia, epiglottitis, pneumonia, septic arthritis, cellulitis, or purulent pericarditis; endocarditis and osteomyelitis occur less commonly.

Laboratory criteria for diagnosis

- Detection of Haemophilus influenzae type b antigen in cerebrospinal fluid [CSF]
- Detection of Haemophilus influenzae-specific nucleic acid in a specimen obtained from a normally sterile body site (e.g., blood or CSF), using a validated polymerase chain reaction (PCR) assay

OR

- Isolation of H. influenzae from a normally sterile body site (e.g., CSF, blood, joint fluid, pleural fluid, or pericardial fluid)

Case classification

Probable: Meningitis with detection of H. influenzae type b antigen in cerebrospinal fluid [CSF]

Confirmed:
• Isolation of H. influenzae from a normally sterile body site (e.g., cerebrospinal fluid [CSF], blood, joint fluid, pleural fluid, pericardial fluid)

OR

• Detection of Haemophilus influenzae -specific nucleic acid in a specimen obtained from a normally sterile body site (e.g., cerebrospinal fluid [CSF], blood, joint fluid, pleural fluid, pericardial fluid), using a validated polymerase chain reaction (PCR) assay

Epidemiologic Summary

There were a total of 288 reported cases of invasive Haemophilus influenzae disease in the state from 2005 to 2015.

Notes


Table 1. Reported invasive *Haemophilus influenzae* disease cases by age, race, ethnicity, and sex – Kentucky, 2005-2015

<table>
<thead>
<tr>
<th>Age</th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;12 mos</td>
<td>18</td>
<td>6.25</td>
</tr>
<tr>
<td>1-14 years</td>
<td>34</td>
<td>11.81</td>
</tr>
<tr>
<td>15-24</td>
<td>5</td>
<td>1.74</td>
</tr>
<tr>
<td>25-44</td>
<td>21</td>
<td>7.29</td>
</tr>
<tr>
<td>45-64</td>
<td>55</td>
<td>19.10</td>
</tr>
<tr>
<td>65-74</td>
<td>60</td>
<td>20.83</td>
</tr>
<tr>
<td>75-84</td>
<td>59</td>
<td>20.49</td>
</tr>
<tr>
<td>85+</td>
<td>36</td>
<td>12.50</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Race</th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>American Indian or Alaska Native</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Asian and Pacific Islander</td>
<td>1</td>
<td>0.35</td>
</tr>
<tr>
<td>Black or African American</td>
<td>18</td>
<td>6.25</td>
</tr>
<tr>
<td>Other, Multi</td>
<td>1</td>
<td>0.35</td>
</tr>
<tr>
<td>Unknown</td>
<td>18</td>
<td>6.25</td>
</tr>
<tr>
<td>White</td>
<td>198</td>
<td>68.75</td>
</tr>
<tr>
<td>Missing</td>
<td>52</td>
<td>18.06</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hispanic or Latino</td>
<td>2</td>
<td>0.69</td>
</tr>
<tr>
<td>Not Hispanic or Latino</td>
<td>138</td>
<td>47.92</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
<td>0.69</td>
</tr>
<tr>
<td>Missing</td>
<td>146</td>
<td>50.70</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sex</th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>150</td>
<td>52.08</td>
</tr>
<tr>
<td>Male</td>
<td>129</td>
<td>44.79</td>
</tr>
<tr>
<td>Unknown</td>
<td>9</td>
<td>3.13</td>
</tr>
</tbody>
</table>
Hepatitis A

Background

Hepatitis A is caused by hepatitis A virus (HAV), which is a nonenveloped RNA picornavirus. HAV is spread through fecal-oral transmission and is acquired by mouth usually through ingesting contaminated food or water. Hepatitis A presents similarly to other acute viral hepatitis infections. Symptoms include sudden onset of fever, malaise, anorexia, nausea, abdominal discomfort, dark urine, and jaundice. Children less than six years of age are more likely to be asymptomatic. For older children and adults, the infection usually shows symptoms. Jaundice occurs in 70% of patients in this group. The incubation period of hepatitis A is 28 days, with a range of 15 to 50 days. The hepatitis A vaccine is recommended for all children at 1 year of age as well as individuals at increased risk of infection. This includes individuals traveling to areas where hepatitis A is common, those who are in close contact with an adoptee from a country where hepatitis A is common, men who have sex with men, injection and non injection drug users, individuals with clotting factor disorders, and individuals with occupational risk of exposure. Occupational risk is defined as individuals who work with hepatitis A infected primates or work with the virus in a laboratory setting. No other groups have been shown to have increased risk of infection due to occupational exposures.

Epidemiology

Rates of hepatitis A have been decreasing since 1996 when vaccination was initiated. Rates of hepatitis A have declined by 95% since the vaccine first became available.ii

Surveillance Case Definition (2000)
**Clinical Criteria**

An acute illness with a) discrete onset of symptoms and b) jaundice, elevated serum aminotransferase levels (ALT or AST)

**Laboratory criteria**

Immunoglobulin M (IgM) antibody to hepatitis A virus (anti-HAV) positive

**Case classification**

Confirmed: a case that meets the clinical case definition and is laboratory confirmed or a case that meets the clinical case definition and occurs in a person who has an epidemiologic link with a person who has laboratory-confirmed hepatitis A (i.e., household or sexual contact with an infected person during the 15-50 days before the onset of symptoms).

**Epidemiologic Summary**

There were 242 cases of hepatitis A reported in Kentucky between 2005 and 2015.

**Notes**

i: CDC. Prevention of hepatitis A through active or passive immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 2006;55(No. RR-7)

Table 2. Reported hepatitis A cases by age, race, ethnicity, and sex –Kentucky, 2005-2015

<table>
<thead>
<tr>
<th></th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kentucky</td>
<td>242</td>
<td>-</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-9</td>
<td>30</td>
<td>12.40</td>
</tr>
<tr>
<td>10-19</td>
<td>21</td>
<td>8.68</td>
</tr>
<tr>
<td>20-29</td>
<td>37</td>
<td>15.29</td>
</tr>
<tr>
<td>30-39</td>
<td>26</td>
<td>10.74</td>
</tr>
<tr>
<td>40-49</td>
<td>30</td>
<td>12.40</td>
</tr>
<tr>
<td>50-59</td>
<td>39</td>
<td>16.12</td>
</tr>
<tr>
<td>60+</td>
<td>59</td>
<td>24.38</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>American Indian or Alaska Native</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Asian and Pacific Islander</td>
<td>3</td>
<td>1.24</td>
</tr>
<tr>
<td>Black or African American</td>
<td>7</td>
<td>2.89</td>
</tr>
<tr>
<td>Other, Multi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Unknown</td>
<td>27</td>
<td>11.16</td>
</tr>
<tr>
<td>White</td>
<td>121</td>
<td>50.00</td>
</tr>
<tr>
<td>Missing</td>
<td>84</td>
<td>34.71</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic or Latino</td>
<td>17</td>
<td>7.02</td>
</tr>
<tr>
<td>Not Hispanic or Latino</td>
<td>131</td>
<td>54.13</td>
</tr>
<tr>
<td>Unknown</td>
<td>23</td>
<td>9.50</td>
</tr>
<tr>
<td>Missing</td>
<td>71</td>
<td>29.34</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>121</td>
<td>50.00</td>
</tr>
<tr>
<td>Male</td>
<td>118</td>
<td>48.76</td>
</tr>
<tr>
<td>Unknown</td>
<td>3</td>
<td>1.24</td>
</tr>
</tbody>
</table>
Hepatitis B, Acute

Background

Hepatitis B is caused by hepatitis B virus and is transmitted through contact with blood or other body fluids that are infected. Important routes of transmission, especially in the United States, are perinatal, sexual contact, and injection drug use. Adults are more likely to show symptoms than children and infants, but at least 50% of infections in adults are asymptomatic. If symptoms are present they are not specific to hepatitis B and can include malaise, fever, headache, and muscle pain. Some individuals may progress to chronic infection, which is discussed in the Hepatitis B, Chronic portion of this report. The virus has an incubation period of 45 to 160 days with an average of 120 days. Vaccination is recommended for all children. The vaccine is given in 3 doses, with the first dose given soon after birth before leaving the hospital. This vaccine has an efficacy of 95% in infants and children and 90% in adults. Antibody levels decline with time but remain intact for more than 20 years, so boosters are not recommended.

Epidemiology

Hepatitis B became reportable in the United States in the 1970s. Incidence peaked in the mid 1980s with around 26,000 cases reported annually. Since that time, the number of cases has decreased. Acute hepatitis B incidence decreased by 75% from 1990 to 2005. The largest decrease in this time was 96% and was seen in children and adolescents, which coincides with increased vaccine coverage during this same time period.

Surveillance Case Definition (2000)
Clinical case definition

An acute illness with

- A discrete onset of symptoms, and
- Jaundice or elevated serum aminotransferase levels

Laboratory criteria for diagnosis

- IgM antibody to hepatitis B core antigen (anti-HBc) positive or hepatitis B surface antigen (HBsAg) positive
- IgM anti-HAV negative (if done)

Case classification

Confirmed: A case that meets the clinical case definition and is laboratory confirmed.

Epidemiologic Summary

There were 1,377 cases of acute Hepatitis B reported in Kentucky between 2005 and 2015.

Notes


ii: CDC. A comprehensive immunization strategy to eliminate transmission of hepatitis B virus infection in the United States. Recommendations of the Advisory Committee on
Table 3. Rate of hepatitis B, acute by year per 100,000

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis B, Acute</td>
<td>1.60</td>
<td>1.68</td>
<td>1.76</td>
<td>2.35</td>
<td>2.15</td>
<td>3.13</td>
<td>3.37</td>
<td>4.77</td>
<td>3.29</td>
<td>3.55</td>
<td></td>
</tr>
</tbody>
</table>

Rates based on population estimates obtained from National Center for Health Statistics (NCHS)\(^{18}\)

Table 4. Reported acute hepatitis B cases by age, race, ethnicity, and sex –Kentucky, 2005-2015

<table>
<thead>
<tr>
<th></th>
<th>Cases</th>
<th>Rate per 100,000</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kentucky</td>
<td>1377</td>
<td>2.89</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;19</td>
<td>7</td>
<td>0.06</td>
<td>ref</td>
<td>-</td>
</tr>
<tr>
<td>20-29</td>
<td>195</td>
<td>3.04</td>
<td>54.12</td>
<td>(25.46, 115.04)</td>
</tr>
<tr>
<td>30-39</td>
<td>481</td>
<td>7.76</td>
<td>138.25</td>
<td>(65.55, 291.55)</td>
</tr>
<tr>
<td>40-49</td>
<td>406</td>
<td>6.04</td>
<td>107.58</td>
<td>(50.96, 227.11)</td>
</tr>
<tr>
<td>&gt;50</td>
<td>288</td>
<td>1.82</td>
<td>32.45</td>
<td>(15.33, 68.69)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>American Indian or Alaska Native</td>
<td>4</td>
<td>2.89</td>
<td>1.48</td>
<td>(0.56, 3.96)</td>
</tr>
<tr>
<td>Asian and Pacific Islander</td>
<td>3</td>
<td>0.46</td>
<td>0.24</td>
<td>(0.08, 0.74)</td>
</tr>
<tr>
<td>Black or African American</td>
<td>54</td>
<td>1.35</td>
<td>0.69</td>
<td>(0.52, 0.91)</td>
</tr>
<tr>
<td>White</td>
<td>834</td>
<td>1.95</td>
<td>ref</td>
<td>-</td>
</tr>
<tr>
<td>Other, Multi</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Unknown</td>
<td>237</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Missing</td>
<td>242</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic or Latino</td>
<td>12</td>
<td>0.85</td>
<td>0.50</td>
<td>(0.29, 0.89)</td>
</tr>
<tr>
<td>Not Hispanic or Latino</td>
<td>777</td>
<td>1.68</td>
<td>ref</td>
<td>-</td>
</tr>
<tr>
<td>Unknown</td>
<td>199</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Missing</td>
<td>389</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>590</td>
<td>2.44</td>
<td>ref</td>
<td>-</td>
</tr>
<tr>
<td>Male</td>
<td>771</td>
<td>3.29</td>
<td>1.35</td>
<td>(1.21, 1.50)</td>
</tr>
<tr>
<td>Unknown</td>
<td>15</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Missing</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Rates based on population estimates obtained from National Center for Health Statistics (NCHS)\(^{18}\)
**Table 5.** Rate of hepatitis B, acute by area development district per 100,000

<table>
<thead>
<tr>
<th>Area development district</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barren River</td>
<td>1.73</td>
</tr>
<tr>
<td>Big Sandy</td>
<td>12.21</td>
</tr>
<tr>
<td>Bluegrass</td>
<td>1.96</td>
</tr>
<tr>
<td>Buffalo Trace</td>
<td>4.20</td>
</tr>
<tr>
<td>Cumberland Valley</td>
<td>7.15</td>
</tr>
<tr>
<td>Fivco</td>
<td>4.32</td>
</tr>
<tr>
<td>Gateway</td>
<td>4.35</td>
</tr>
<tr>
<td>Green River</td>
<td>0.85</td>
</tr>
<tr>
<td>Kentucky River</td>
<td>5.12</td>
</tr>
<tr>
<td>KIPDA</td>
<td>1.60</td>
</tr>
<tr>
<td>Lake Cumberland</td>
<td>2.07</td>
</tr>
<tr>
<td>Lincoln Trail</td>
<td>1.33</td>
</tr>
<tr>
<td>Northern Kentucky</td>
<td>4.71</td>
</tr>
<tr>
<td>Pennyrile</td>
<td>1.92</td>
</tr>
<tr>
<td>Purchase</td>
<td>1.07</td>
</tr>
</tbody>
</table>

Rates based on population estimates obtained from National Center for Health Statistics (NCHS)\(^{18}\)
Hepatitis B, Chronic

Background

Acute infection with hepatitis B virus (HBV) can progress to a chronic infection. Chronic hepatitis B is responsible for most of the morbidity and mortality associated with HBV. The proportion of patients who will progress to chronic disease varies depending on factors such as age and immune status. Of infants who acquire HBV from their mothers or during childhood, 90% become chronically infected. Of those infected between 1 and 5 years, 30-50% become chronically infected and 5% of adults become chronically infected. Chronic infection may not show any symptoms but can still infect others. HBV related illnesses caused by chronic infection include chronic hepatitis, cirrhosis, liver failure, and hepatocellular carcinoma. In more than 25% of carriers, chronic active hepatitis develops which can often result in cirrhosis.

Surveillance Case Definition (2007)

Clinical description

Persons with chronic HBV infection may have no evidence of liver disease or may have a spectrum of disease ranging from chronic hepatitis to cirrhosis or liver cancer. Persons with chronic infection may be asymptomatic.

Laboratory criteria for diagnosis

- IgM anti-HBc negative AND a positive result on one of the following tests:
  - HBsAg, HBeAg, or HBV DNA OR
- HBsAg positive or HBV DNA positive or HBeAg positive two times at least 6 months apart (Any combination of these tests performed 6 months apart is
acceptable.)

Case classification

**Confirmed:** a case that meets either laboratory criterion for diagnosis

**Probable:** a case with a single HBsAg-positive or HBV DNA-positive or HBeAg-positive laboratory result when no IgM anti-HBc results are available

Comment: Multiple laboratory tests indicative of chronic HBV infection may be performed simultaneously on the same patient specimen as part of a “hepatitis panel.” Testing performed in this manner may lead to seemingly discordant results, e.g., HBsAg negative AND HBV DNA positive. For purposes of this case definition, any positive result among the three laboratory tests mentioned above is acceptable, regardless of other testing results. Negative HBeAg results and HBV DNA levels below positive cutoff level do not confirm the absence of HBV infection.

*Epidemiologic Summary*

There were 109 cases of chronic hepatitis B reported in Kentucky between 2011 and 2015.
<table>
<thead>
<tr>
<th>Age</th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-9</td>
<td>2</td>
<td>1.83</td>
</tr>
<tr>
<td>10-19</td>
<td>2</td>
<td>1.83</td>
</tr>
<tr>
<td>20-29</td>
<td>25</td>
<td>22.94</td>
</tr>
<tr>
<td>30-39</td>
<td>33</td>
<td>30.28</td>
</tr>
<tr>
<td>40-49</td>
<td>19</td>
<td>17.43</td>
</tr>
<tr>
<td>50-59</td>
<td>19</td>
<td>17.43</td>
</tr>
<tr>
<td>60+</td>
<td>9</td>
<td>8.26</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Race</th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>American Indian or Alaska Native</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Asian and Pacific Islander</td>
<td>3</td>
<td>2.75</td>
</tr>
<tr>
<td>Black or African American</td>
<td>3</td>
<td>2.75</td>
</tr>
<tr>
<td>Other, Multi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Unknown</td>
<td>61</td>
<td>55.96</td>
</tr>
<tr>
<td>White</td>
<td>34</td>
<td>31.19</td>
</tr>
<tr>
<td>Missing</td>
<td>8</td>
<td>7.34</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hispanic or Latino</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Not Hispanic or Latino</td>
<td>31</td>
<td>28.44</td>
</tr>
<tr>
<td>Unknown</td>
<td>62</td>
<td>56.88</td>
</tr>
<tr>
<td>Missing</td>
<td>16</td>
<td>14.68</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sex</th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>65</td>
<td>59.63</td>
</tr>
<tr>
<td>Male</td>
<td>44</td>
<td>40.37</td>
</tr>
</tbody>
</table>
Measles

Background

Measles is a highly communicable viral illness. The secondary attack rate, or frequency of cases among susceptible contacts of an infected individual, is nearly 90%. Measles is transmitted through respiratory droplets from person to person. The incubation period is 10 to 12 days from exposure to the first sign of illness and 14 days from exposure to the onset of rash, with a range of 7 to 21 days. The symptoms include an increasing fever, cough, and runny nose, followed by a rash that begins on the face and neck.

The measles vaccine currently available in the United States is combined with the vaccines for mumps and rubella (MMR) or combined with the vaccines for mumps, rubella, and varicella (MMRV). Two doses of the vaccine containing the measles vaccine are recommended for all children 12 months of age or older. The two doses should be separated by at least 4 weeks, although the second dose is routinely given at age 4 to 6. The vaccine series confers lifelong immunity and has an efficacy of 95% when given at 12 months and 98% when given at 15 months.

Epidemiology

Before the development and licensing of a measles vaccine in 1963, infection during childhood was nearly universal. At that time there were an average of 400,000 cases reported each year, although the actual number of cases was estimated to be much higher at 3 to 4 million cases per year.\(^1\) Since 1963, there has been a dramatic 99% decrease in measles incidence. There was a resurgence of measles from 1989 to 1991. During this
time there were more than 55,000 reported cases and 123 deaths. The main cause of this increase in cases was due to low vaccination coverage.\textsuperscript{i}

Following the resurgence, efforts to vaccinate children of preschool age were increased which caused a decrease in reported cases. Available data shows that measles transmission in the United States has been interrupted and the majority of cases are either imported from other countries or linked to imported cases.\textsuperscript{i} The year 2008 saw the largest total measles cases in the country since 1996. This increase was not due to a higher number of cases being imported, but was due to increased transmission after the virus was imported among children who were able to be vaccinated but whose parents chose not to vaccinate them.\textsuperscript{ii} In 2011, 16 outbreaks and 220 measles cases were reported. The majority of these were imported cases in unvaccinated individuals. Outbreaks were also seen in 2013, 2014, and 2015.\textsuperscript{iii}

*Surveillance Case Definition (2012)*\textsuperscript{viii}

*Clinical description:*

An acute illness characterized by:

- generalized, maculopapular rash lasting $\geq 3$ days; and
- temperature $\geq 101^\circ F$ or $38.3^\circ C$; and
- cough, coryza, or conjunctivitis

*Probable:* In the absence of a more likely diagnosis, an illness that meets the clinical description with

- no epidemiologic linkage to a laboratory-confirmed measles case; and
- noncontributory or no measles laboratory testing.
Confirmed: An acute febrile rash illness† with:

• isolation of measles virus‡ from a clinical specimen; or

• detection of measles virus-specific nucleic acid from a clinical specimen using by polymerase chain reaction; or

• IgG seroconversion‡ or a significant rise in measles immunoglobulin G antibody‡ using any evaluated and validated method; or

• a positive serologic test for measles immunoglobulin M antibody‡ §; or

• direct epidemiologic linkage to a case confirmed by one of the methods above.

† Temperature does not need to reach ≥101°F/38.3°C and rash does not need to last ≥3 days.

‡ Not explained by MMR vaccination during the previous 6–45 days.

§ Not otherwise ruled out by other confirmatory testing or more specific measles testing in a public health laboratory.

Epidemiologic Summary

There were two reported cases of measles in Kentucky between 2005 and 2015.

Notes


Meningococcal disease

Background

Meningococcal disease is caused by the bacterium *Neisseria meningitidis*. Invasive infection with this bacterium usually presents as meningitis and/or meningococcal sepsis. *N. meningitidis* has a polysaccharide capsule and is classified based on the structure of this capsule. There are thirteen serogroups, but nearly all invasive disease is caused by either A, B, C, W, or Y. The bacteria are transmitted by respiratory droplet or direct contact. It has an incubation period of 3 to 4 days with a range of 2 to 10 days. Up to 10% of adolescents and adults are asymptomatic transient carriers of the bacteria, usually of strains that are not pathogenic.

The first vaccine was licensed in the United States in 1974. The current vaccine was licensed in 1981 and is quadrivalent, covering serogroups A, C, W, and Y. The vaccine is recommended at age 11 or 12 with a booster at age 16.

Epidemiology

Incidence of meningococcal disease peaked in the late 1990s. Routine use of the meningococcal vaccine was recommended in 2005. Since this recommendation, incidence has decreased among all age groups. Incidence of disease caused by the serogroup not included in the vaccine (B), has also declined.

Surveillance Case Definition (2005)

*Confirmed case:* A confirmed case of meningococcal disease is defined by isolation of *N. meningitidis* from a normally sterile site (e.g., blood or cerebrospinal fluid [CSF]) from a person with clinically compatible illness.
Probable case: A probable case of meningococcal disease is defined by detection of N. meningitidis DNA by polymerase chain reaction or polysaccharide antigen in CSF (e.g., by latex agglutination or immunohistochemistry), or the presence of clinical purpura fulminans in the absence of diagnostic culture from a person with clinically compatible disease.

Epidemiologic Summary

There were 94 cases of meningococcal disease reported in Kentucky between 2005 and 2015.

Notes

Table 7. Reported invasive meningococcal disease by age, race, ethnicity, and sex – Kentucky, 2005-2015

<table>
<thead>
<tr>
<th></th>
<th>Cases</th>
<th>Percent of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kentucky</td>
<td>94</td>
<td>-</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>11</td>
<td>11.70</td>
</tr>
<tr>
<td>1-4</td>
<td>18</td>
<td>19.15</td>
</tr>
<tr>
<td>5-10</td>
<td>9</td>
<td>9.57</td>
</tr>
<tr>
<td>11-18</td>
<td>6</td>
<td>6.38</td>
</tr>
<tr>
<td>19-24</td>
<td>8</td>
<td>8.51</td>
</tr>
<tr>
<td>25-44</td>
<td>11</td>
<td>11.70</td>
</tr>
<tr>
<td>45-64</td>
<td>13</td>
<td>13.83</td>
</tr>
<tr>
<td>65+</td>
<td>18</td>
<td>19.15</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>American Indian or Alaska Native</td>
<td>1</td>
<td>1.06</td>
</tr>
<tr>
<td>Asian and Pacific Islander</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Black or African American</td>
<td>10</td>
<td>10.64</td>
</tr>
<tr>
<td>Other, Multi</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
<td>2.13</td>
</tr>
<tr>
<td>White</td>
<td>48</td>
<td>51.06</td>
</tr>
<tr>
<td>Missing</td>
<td>33</td>
<td>35.11</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic or Latino</td>
<td>4</td>
<td>4.26</td>
</tr>
<tr>
<td>Not Hispanic or Latino</td>
<td>56</td>
<td>59.57</td>
</tr>
<tr>
<td>Unknown</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Missing</td>
<td>34</td>
<td>36.17</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>49</td>
<td>52.13</td>
</tr>
<tr>
<td>Male</td>
<td>43</td>
<td>45.74</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
<td>2.13</td>
</tr>
</tbody>
</table>
Mumps

*Background*

Mumps is caused by an RNA virus and is spread through respiratory droplets. The characteristic symptom of mumps is salivary gland inflammation, called parotitis. Rates of parotitis among all age groups ranges from 31% to 65%, although this can vary based on age and immunity. The incubation period is 12 to 25 days with parotitis usually developing 16 to 18 days after exposure to the virus. Mumps can cause very serious complications such as orchitis, oophoritis, aseptic meningitis, pancreatitis, and deafness.

An inactivated mumps vaccine was first developed in 1948. The vaccine currently used was licensed in 1967 and was recommended for routine use beginning in 1977. The mumps vaccine is given as part of the combined measles, mumps, rubella (MMR) vaccine. The first dose is recommended when the child is at least 12 months old. The second dose is given between ages 4 and 6, before entering kindergarten or first grade. The two doses have an effectiveness of 88% and confer lifelong immunity.

*Epidemiology*

There were 185,691 cases of mumps reported in the United States in 1967. After the mumps vaccine was licensed in 1967, there was a rapid decrease in reported cases. The number of mumps cases further declined when two doses of the vaccine were recommended, which decreased the rate of vaccine failure. There were fewer than 270 cases reported each year by the early 2000s. There was a large increase in cases in both 2006 (6,584 cases reported) and 2009-2010 (3,503 and 505 cases reported in two separate
outbreaks) due to outbreaks, but these outbreaks were contained due to high vaccine coverage. ii

Surveillance Case Definition (2011)§

Suspect:

• parotitis, acute salivary gland swelling, orchitis, or oophoritis unexplained by another more likely diagnosis,

or

• a positive lab result with no mumps clinical symptoms (with or without epidemiological linkage to a confirmed or probable case).

Probable:

• Acute parotitis or other salivary gland swelling lasting at least 2 days, or orchitis or oophoritis unexplained by another more likely diagnosis, in:

  o a person with a positive test for serum anti-mumps IgM antibody, or
  o a person with epidemiologic linkage to another probable or confirmed case or linkage to a group/community defined by public health during an outbreak of mumps.

Confirmed:

• A positive mumps laboratory confirmation for mumps virus with RT-PCR or culture in a patient with an acute illness characterized by any of the following:

  ◦ Acute parotitis or other salivary gland swelling, lasting at least 2 days
  ◦ Aseptic meningitis
- Encephalitis
- Hearing loss
- Orchitis
- Oophoritis
- Mastitis
- Pancreatitis

**Epidemiologic Summary**

There were 6 cases of mumps reported in Kentucky between 2005 and 2015.

**Notes**


Pertussis

Background

Pertussis, also called whooping cough, is caused by the bacterium *Bordetella pertussis*. Pertussis has an incubation period of 7 to 10 days with a range of 4 to 21 days. Transmission occurs through contact with respiratory droplets of an infected individual. The illness presents with bursts of coughing which make it difficult to breath. The coughing attack is followed by a whooping sound when the individual attempts to inhale. Older individuals and children with partial protection from vaccination may become infected with the bacterium but may have a milder course of the disease than young children and infants. Although the disease may be milder in older individuals, they are still able to transmit the disease to susceptible individuals, such as infants who are too young to be immunized. Young infants are at the highest risk for complications from pertussis. The most common complication is secondary bacterial pneumonia. Pertussis is highly communicable. Among susceptible household contacts, pertussis has a secondary attack rate of 80%.

The pertussis vaccine is included in DTaP combined with diphtheria and tetanus toxoids. The primary series of this vaccine has four doses. The first dose is recommended beginning at 6 weeks of age to two months, with the following two doses given in 4 to 8 week intervals. The final dose is given 6 to 12 months after the third dose. A fifth booster dose is given before entering school if a child received all four doses of the primary series before turning 4. There is also an adult vaccine containing pertussis, diphtheria, and tetanus toxoids called Tdap. A single dose of this vaccine is recommended for ages 11 through 18 years and 19 through 64 years. All pregnant women should receive a dose
between 27 and 36 weeks gestation during each pregnancy. Individuals who are going to be in contact with infants too young to be vaccinated (less than 6 weeks of age) should receive a single dose of Tdap if they have not previously.

Epidemiology

Pertussis was a common cause of childhood morbidity and mortality before the vaccine was licensed. An average of 200,000 cases were reported each year from 1943 to 1943. After the vaccine became routinely recommended in the 1940s, the number of reported cases decreased in the following decades. In 1976, a low of 1,010 cases were reported. Incidence has been gradually increasing since the 1980s, although levels remain well below those seen in the pre-vaccine era.

Surveillance Case Definition (2014)

Clinical case definition

In the absence of a more likely diagnosis a cough illness lasting ≥ 2 weeks with one of the following symptoms:

- Paroxysms of coughing, OR
- Inspiratory “whoop,” OR
- Posttussive vomiting, OR
- Apnea (with or without cyanosis) (FOR INFANTS AGED < 1 YEAR ONLY)

Laboratory criteria for diagnosis

- Isolation of B. pertussis from a clinical specimen
- Positive polymerase chain reaction (PCR) assay for B. pertussis DNA
Epidemiologic Linkage

- Contact with a laboratory-confirmed case of pertussis†*. 

Case classification

Probable:

- Meets the clinical case definition, is not laboratory confirmed, and is not epidemiologically linked to a laboratory-confirmed case OR

- FOR INFANTS AGED < 1 YEAR ONLY:
  - Acute cough illness of any duration with at least one of the following signs or symptoms:
    - Paroxysms of coughing, OR
    - Inspiratory “whoop”, OR
    - Posttussive vomiting, OR
    - Apnea (with or without cyanosis)
  AND
  - Polymerase chain reaction (PCR) positive for pertussis, OR

- FOR INFANTS AGED < 1 YEAR ONLY:
  - Acute cough illness of any duration with at least one of the following signs or symptoms:
    - Paroxysms of coughing, OR
    - Inspiratory “whoop”, OR
    - Posttussive vomiting, OR
    - Apnea (with or without cyanosis)
AND

o Contact with a laboratory-confirmed case of pertussis

Confirmed:

• Acute cough illness of any duration with isolation of B. pertussis from a clinical specimen, OR

• Meets the clinical case definition AND is polymerase chain reaction (PCR) positive for pertussis, OR

• Meets the clinical case definition AND had contact with a case laboratory-confirmed case of pertussis

*Note: An illness meeting the clinical case definition should be classified as “probable” rather than “confirmed” if it occurs in a patient who has contact with an infant aged < 1 year who is polymerase chain reaction (PCR) positive for pertussis and has ≥ 1 sign or symptom and cough duration < 14 days (classified as “probable” case).

†To confirm a case by epidemiologic linkage, the case must be directly linked (i.e., a first-generation contact) to a laboratory-confirmed case by either culture or PCR.

Epidemiologic Summary

There were 2,669 cases of pertussis reported in Kentucky between 2005 and 2015.

Notes

i: CDC. Preventing tetanus, diphtheria, and pertussis among adults: use of tetanus toxoid, reduced diphtheria toxoid and acellular pertussis vaccine: recommendations of the
Table 8. Rate of pertussis by year per 100,000

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pertussis</td>
<td>4.66</td>
<td>1.87</td>
<td>1.20</td>
<td>4.50</td>
<td>7.67</td>
<td>8.16</td>
<td>3.98</td>
<td>7.82</td>
<td>5.82</td>
<td>3.37</td>
<td></td>
</tr>
</tbody>
</table>

Rates based on population estimates obtained from National Center for Health Statistics (NCHS)\textsuperscript{18}

Table 9. Reported pertussis cases by age, race, ethnicity, and sex – Kentucky, 2005-2015

<table>
<thead>
<tr>
<th></th>
<th>Cases</th>
<th>Rate per 100,000</th>
<th>Rate Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kentucky</td>
<td>2669</td>
<td>5.61</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>503</td>
<td>81.70</td>
<td>64.83</td>
<td>(57.12, 73.57)</td>
</tr>
<tr>
<td>1-3</td>
<td>414</td>
<td>22.62</td>
<td>17.95</td>
<td>(15.72, 20.50)</td>
</tr>
<tr>
<td>4-6</td>
<td>216</td>
<td>11.69</td>
<td>9.28</td>
<td>(7.89, 10.91)</td>
</tr>
<tr>
<td>7-9</td>
<td>407</td>
<td>22.05</td>
<td>17.50</td>
<td>(15.31, 19.996)</td>
</tr>
<tr>
<td>10-17</td>
<td>670</td>
<td>13.29</td>
<td>10.55</td>
<td>(9.37, 11.88)</td>
</tr>
<tr>
<td>&gt;18</td>
<td>459</td>
<td>1.26</td>
<td>ref</td>
<td>-</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>American Indian or Alaska Native</td>
<td>1</td>
<td>0.72</td>
<td>0.22</td>
<td>(0.03, 1.53)</td>
</tr>
<tr>
<td>Asian and Pacific Islander</td>
<td>12</td>
<td>1.85</td>
<td>0.55</td>
<td>(0.31, 0.97)</td>
</tr>
<tr>
<td>Black or African American</td>
<td>84</td>
<td>2.09</td>
<td>0.62</td>
<td>(0.50, 0.78)</td>
</tr>
<tr>
<td>White</td>
<td>1436</td>
<td>3.36</td>
<td>ref</td>
<td>-</td>
</tr>
<tr>
<td>Other, Multi</td>
<td>4</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Unknown</td>
<td>534</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Missing</td>
<td>598</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic or Latino</td>
<td>50</td>
<td>3.54</td>
<td>1.25</td>
<td>(0.94, 1.65)</td>
</tr>
<tr>
<td>Not Hispanic or Latino</td>
<td>1310</td>
<td>2.84</td>
<td>ref</td>
<td>-</td>
</tr>
<tr>
<td>Unknown</td>
<td>445</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Missing</td>
<td>864</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>1411</td>
<td>5.83</td>
<td>ref</td>
<td>-</td>
</tr>
<tr>
<td>Male</td>
<td>1199</td>
<td>5.12</td>
<td>0.88</td>
<td>(0.81, 0.95)</td>
</tr>
<tr>
<td>Unknown</td>
<td>53</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Missing</td>
<td>6</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Rates based on population estimates obtained from National Center for Health Statistics (NCHS)\textsuperscript{18}
<table>
<thead>
<tr>
<th>Age</th>
<th>2005</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
<th>2012</th>
<th>2013</th>
<th>2014</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>1.13</td>
<td>1.3</td>
<td>1.5</td>
<td>2.2</td>
<td>2.2</td>
<td>0.75</td>
<td>0.87</td>
<td>0.94</td>
<td>0.95</td>
<td>0.97</td>
</tr>
<tr>
<td>1-3</td>
<td>1.5</td>
<td>1.6</td>
<td>1.6</td>
<td>2.0</td>
<td>2.0</td>
<td>1.9</td>
<td>1.9</td>
<td>1.9</td>
<td>2.0</td>
<td>2.0</td>
</tr>
<tr>
<td>4-6</td>
<td>2.0</td>
<td>2.0</td>
<td>2.0</td>
<td>2.2</td>
<td>2.5</td>
<td>2.3</td>
<td>2.6</td>
<td>2.3</td>
<td>2.5</td>
<td>2.7</td>
</tr>
<tr>
<td>7-9</td>
<td>4.2</td>
<td>4.2</td>
<td>4.2</td>
<td>4.2</td>
<td>4.2</td>
<td>4.2</td>
<td>4.2</td>
<td>4.2</td>
<td>4.2</td>
<td>4.2</td>
</tr>
<tr>
<td>10-20</td>
<td>10.2</td>
<td>10.2</td>
<td>10.2</td>
<td>10.2</td>
<td>10.2</td>
<td>10.2</td>
<td>10.2</td>
<td>10.2</td>
<td>10.2</td>
<td>10.2</td>
</tr>
</tbody>
</table>

TABLE 10. Rate of pertussis by age and year per 100,000
**Poliovirus infection**

*Background*

Poliovirus is an enterovirus with three different serotypes (P1, P2, and P3). For nonparalytic poliomyelitis, the incubation period is 3 to 6 days. In paralytic poliomyelitis, the incubation period for the onset of paralysis is 7 to 21 days. The response to infection with poliovirus varies greatly among individuals and is classified based on the severity of the clinical presentation. A large proportion of infected individuals (up to 72% of infections in children) do not show symptoms but can still shed virus and infect others. Poliovirus is spread through the fecal-oral route as well as the respiratory route. Inactivated polio vaccine (IPV) has been exclusively used in the United States since 2000. The primary series consists of three doses, with the first dose given as early as six weeks of age. The second dose is given at 4 months of age and the third dose is given between 6 and 18 months of age. A final dose is given at four years or older.

*Epidemiology*

Transmission of wild poliovirus was interrupted in the United States in 1979. Inactivated polio vaccine was introduced in 1955 and incidence of poliovirus infection greatly decreased after its introduction. Monovalent oral poliovirus vaccines (MOPV) were introduced for all three types between 1961 and 1962. A trivalent OPV was licensed in 1963 and replaced the use of IPV. Since OPV can cause vaccine-associated paralytic polio, its use was discontinued in the United States in 2000 and replaced with IPV.
Surveillance Case Definition (2010)

Case classification, Paralytic

Probable: Acute onset of a flaccid paralysis of one or more limbs with decreased or absent tendon reflexes in the affected limbs, without other apparent cause, and without sensory or cognitive loss.

Confirmed: Acute onset of a flaccid paralysis of one or more limbs with decreased or absent tendon reflexes in the affected limbs, without other apparent cause, and without sensory or cognitive loss; AND in which the patient

- has a neurologic deficit 60 days after onset of initial symptoms, or
- has died, or
- has unknown follow-up status.

Case classification, Non-paralytic

Confirmed: Any person without symptoms of paralytic poliomyelitis in whom a poliovirus isolate was identified in an appropriate clinical specimen, with confirmatory typing and sequencing performed by the CDC Poliovirus laboratory, as needed.

Epidemiologic Summary

No cases of poliomyelitis were reported in Kentucky between 2005 and 2015.

Notes

Rubella

Background

Rubella is caused by an RNA virus and has an incubation period of 14 days with a range of 12 to 23 days. It is spread through respiratory droplets. Rubella often presents with mild symptoms, with up to half of infections being subclinical. In children, the first symptom is usually a rash. Rubella infection in pregnant women can have severe consequences. If a woman is infected with rubella during her pregnancy, especially during the first trimester, it can lead to congenital rubella syndrome (CRS). CRS can cause fetal death or premature delivery, as well as a number of birth defects such as deafness, eye defects, cardiac defects, neurological abnormalities, bone alterations, and damage to the liver and spleen. Deafness is the most common manifestation of CRS.

The main goal of rubella vaccination programs in the United States is the prevention of CRS. The rubella vaccine is combined with measles and mumps vaccines as MMR or with measles, mumps, and varicella vaccines as MMRV. The first dose of rubella containing vaccine is recommended for all children at 12 months of age or older. The second dose should be given between ages 4 and 6 before starting kindergarten or first grade. The duration of immunity conferred by this vaccine is lifelong.

Epidemiology

In 1969, the United States saw its largest annual total of reported rubella cases with 57,600 reported cases. The incidence of rubella decreased rapidly after a vaccine was licensed in 1969, with only 12,400 cases reported in 1976. Fewer than 1,000 cases per
year were reported by 1984.\textsuperscript{1} In 2004, rubella was declared to no longer be endemic in the United States.\textsuperscript{2}

*Rubella Surveillance Case Definition (2012)*\textsuperscript{8}

**Suspected:** Any generalized rash illness of acute onset that does not meet the criteria for probable or confirmed rubella or any other illness.

**Probable:** In the absence of a more likely diagnosis, an illness characterized by all of the following:

- acute onset of generalized maculopapular rash; and
- temperature greater than $99.0^\circ\text{F}$ or $37.2^\circ\text{C}$, if measured; and
- arthralgia, arthritis, lymphadenopathy, or conjunctivitis; and
- lack of epidemiologic linkage to a laboratory-confirmed case of rubella; and
- noncontributory or no serologic or virologic testing.

**Confirmed:** A case with or without symptoms who has laboratory evidence of rubella infection confirmed by one or more of the following:

- isolation of rubella virus; or
- detection of rubella-virus specific nucleic acid by polymerase chain reaction; or
- significant rise between acute-and convalescent-phase titers in serum rubella immunoglobulin G antibody level by any standard serologic assay; or
- positive serologic test for rubella immunoglobulin M (IgM) antibody*,†

OR

An illness characterized by all of the following:
• acute onset of generalized maculopapular rash; and
• temperature greater than 99.0° F or 37.2° C; and
• arthralgia, arthritis, lymphadenopathy, or conjunctivitis; and
• epidemiologic linkage to a laboratory-confirmed case of rubella.

* Not explained by MMR vaccination during the previous 6-45 days.

† Not otherwise ruled out by more specific testing in a public health laboratory.

Congenital Rubella Syndrome Surveillance Case Definition (2009)\(^8\)

**Suspected:** An infant who does not meet the criteria for a probable or confirmed case but who has one or more of the following findings:

• cataracts,
• congenital glaucoma,
• congenital heart disease (most commonly patent ductus arterious or peripheral pulmonary artery stenosis),
• hearing impairment,
• pigmentary retinopathy,
• purpura,
• hepatosplenomegaly,
• jaundice,
• microcephaly,
• developmental delay,
• meningoencephalitis, or
• radiolucent bone disease.
Probable: An infant who does not have laboratory confirmation of rubella infection but has at least two of the following, without a more plausible etiology:

• cataracts or congenital glaucoma,

• congenital heart disease (most commonly patent ductus arteriosus or peripheral pulmonary artery stenosis),

• hearing impairment, or

• pigmentary retinopathy;

OR

An infant who does not have laboratory confirmation of rubella infection but has at least one or more of the following, without a more plausible etiology:

• cataracts or congenital glaucoma,

• congenital heart disease (most commonly patent ductus arteriosus or peripheral pulmonary artery stenosis),

• hearing impairment, or

• pigmentary retinopathy;

AND one or more of the following:

• purpura,

• hepatosplenomegaly,

• microcephaly,

• developmental delay,
• meningoencephalitis, or

• radiolucent bone disease.

**Confirmed:** An infant with at least one of the symptoms clinically consistent with congenital rubella syndrome listed above; and laboratory evidence of congenital rubella infection demonstrated by:

• isolation of rubella virus, or

• detection of rubella-specific immunoglobulin M (IgM) antibody, or

• infant rubella antibody level that persists at a higher level and for a longer period of time than expected from passive transfer of maternal antibody (i.e., rubella titer that does not drop at the expected rate of a two-fold decline per month), or

• a specimen that is PCR-positive for rubella virus.

Note: In probable cases, either or both of the eye-related findings (cataracts and congenital glaucoma) count as a single complication. In cases classified as infection only, if any compatible signs or symptoms (e.g., hearing impairment) are identified later, the case is reclassified as confirmed.

*Epidemiologic Summary*

One case of rubella was reported in Kentucky between 2005 and 2015.

*Notes*

i: CDC. Measles, mumps, and rubella—vaccine use and strategies for elimination of measles, rubella, and congenital rubella syndrome and control of mumps.
Recommendations of the Advisory Committee on Immunization Practices (ACIP).

MMWR 1998;47(No. RR-8).
Tetanus

Background

Tetanus is caused by the toxin tetanospasmin produced by the spore-forming bacterium *Clostridium tetani*. The spores are often found in soil and manure. *C. tetani* is transmitted through a contaminated wound. It cannot be spread person to person. The toxin produced binds in the central nervous system causing the characteristic symptoms of muscle spasms and stiffness, with the first sign often being lockjaw. The incubation period can range from 3 to 21 days, with the average being 8 days. Generally, the further the site of the injury is from the central nervous system, the longer the incubation period. Higher chances of death are associated with shorter incubation periods.

Everyone should receive tetanus toxoid, which is available in combination with multiple other vaccines. These include pediatric diphtheria-tetanus toxoid (DT), adult tetanus-diphtheria (Td), and with pertussis vaccine as well as diphtheria toxoid as DTaP or Tdap. Children should receive 4 doses of DTaP by 15 to 18 months of age, with a booster before entering school between the ages of 4 and 6. The next booster, recommended to be Tdap, is given at age 11 or 12. Boosters of Td are then recommended every 10 years to maintain protective antitoxin titers.

Epidemiology

Tetanus toxoid was first produced in 1924 and introduced into routine childhood vaccines in the late 1940s.\(^2\) Reported incidence rates of tetanus declined steadily following the introduction of the vaccine, falling to an all time low of 18 reported cases in 2009.\(^1\)
**Surveillance Case Definition (2009)**

*Tetanus clinical case definition*

In the absence of a more likely diagnosis, an acute illness with muscle spasms or hypertonia and diagnosis of tetanus by a health care provider; or death, with tetanus listed on the death certificate as the cause of death or a significant condition contributing to death.

*Case classification*

Probable: A clinically compatible case, as reported by a healthcare professional.

There is no definition for confirmed tetanus.

*Epidemiologic Summary*

There were two case of tetanus reported in Kentucky between 2005 and 2015.

*Notes*

Varicella

Background

Varicella, or chicken pox, is caused by the DNA virus varicella zoster virus (VZV). This virus can persist after the primary infection in the sensory nerve ganglia. Reactivation of the latent VZV results in herpes zoster, or shingles. The incubation period is 14 to 16 days with a range of 10 to 21 days. Primary infection with VZV presents with a generalized, pruritic, maculo-papulovesicular rash that usually appears first on the head and then the trunk and extremities. Over the course of several days, lesions will develop in several crops and then eventually crust over. The risk of complications from varicella are greater for persons older than 15 years, less than 1 year, those with compromised immune systems, and newborns of women whose rash started between 5 days before delivery to 2 days after delivery. VZV is transmitted person to person through infected secretions from the respiratory tract. Varicella can also be transmitted by direct contact with lesions. It is a highly contagious disease. Herpes zoster occurs mainly in older adults and is uncommon in children. Herpes zoster presents with a painful rash that is unilateral and follows the distribution of a sensory nerve.

The vaccine was licensed in 1995 and there are currently 3 VZV containing vaccines licensed in the United States: a varicella vaccine, a measles-mumps-rubella-varicella (MMRV) combination vaccine, and a herpes zoster vaccine. The varicella vaccine is recommended for all children at 12 through 15 months with a second dose given between 4 and 6 years of age. The herpes zoster vaccine is approved for persons over 50 years, but not recommended for individuals under 60 years of age.
Epidemiology

It is estimated that there were 10,632 hospitalizations due to varicella each year from 1988 to 1995.\textsuperscript{i} There were an average of 105 deaths due to varicella reported each year from 1970 to 1994.\textsuperscript{i} Since the introduction of the vaccine, hospitalizations due to varicella have declined 70% and deaths due to varicella have declined 88%.\textsuperscript{i}

Surveillance Case Definition (2009)\textsuperscript{8}

Varicella Clinical Case Definition

An illness with acute onset of diffuse (generalized) maculopapulovesicular rash without other apparent cause. In vaccinated persons who develop varicella more than 42 days after vaccination (breakthrough disease), the disease is usually mild with fewer than 50 skin lesions and shorter duration of illness. The rash may also be atypical in appearance (maculopapular with few or no vesicles).

Varicella Case Classification

Probable: A case that meets the clinical case definition but is not laboratory confirmed nor epidemiologically linked to another probable or confirmed case

Confirmed: A case that is laboratory confirmed or that meets the clinical case definition and is epidemiologically linked to a confirmed or a probable case

Note: Two probable cases that are epidemiologically linked are considered confirmed, even in the absence of laboratory confirmation.

Epidemiologic Summary
There were no cases reported in the state from 2005 to 2015 because varicella was not reportable in the state of Kentucky during these years.

Notes

Discussion

Hepatitis B, Acute

From 2005 to 2015, rates of acute hepatitis B in Kentucky have been very different from the rates seen nationally. Rates have been higher in Kentucky than in the United States for every year except 2005. The rate in the United States has remained stable over this time period, while the rate in Kentucky has increased, with some years being nearly four times the national rate. These differences highlight the importance of state level surveillance because the high rates of this disease in Kentucky would not have been identified from only looking at or calculating national rates.

Over the ten-year period, the highest rate was seen in the 30-39 age group with 7.76 cases per 100,000. This rate is 138.25 times the rate of acute hepatitis B compared to those in the <19 age group. The second highest rate was seen in the 40-49 age group with 6.04 cases per 100,000. This is 107.58 times the rate of acute hepatitis B compared to those in the <19 age group. These high rates of acute hepatitis B in older age groups have been seen in other studies of the disease and could be due to low hepatitis B vaccine coverage and past changes in vaccine recommendations. Hepatitis B vaccine coverage is low among adults and routine hepatitis B vaccination was recommended for infants starting in 1991 and for children ≤18 in 1999. This means that portions of these age groups, such as individuals who were 34 or older in 2015, would be too old to have received these routine vaccinations, putting them at risk of infection.

There were a large proportion of missing and unknown values for race and ethnicity. For race, 35% of cases had a missing or unknown value. For ethnicity, 43% of
cases had a missing or unknown value. There were also small numbers in multiple
categories, which contributed to the difficulty in drawing conclusions. The highest rate
was seen in American Indians/Alaska Natives, with 2.89 cases per 100,000 although this
rate was not significantly different from the reference category, whites. The second
highest rate was seen in whites, with 1.95 cases per 100,000. Not Hispanic/Latino had the
highest rate with 1.68 cases per 100,000, two times the rate of Hispanic/Latino.
Compared to females, males had 1.35 times the rate of acute hepatitis B.

To look at the geographic distribution of rates of hepatitis B in the state area
development districts were used. Big Sandy had the highest rate with 12.21 cases per
100,000, followed by Cumberland Valley with 7.15 cases per 100,000, and Kentucky
River with 5.12 cases per 100,000. The seven area development districts with the highest
rates (Big Sandy, Cumberland Valley, Kentucky River, Northern Kentucky, Gateway,
Fivco, and Buffalo Trace) are all located in the eastern portion of the state, with the
exception of Northern Kentucky. Of these eastern area development districts, the lowest
rate was seen in Buffalo Trace with 4.20 cases per 100,000. This rate is still over twice as
high as the next highest district. Although risk factor data was not available for this
report, this could be related to injection drug use which has been seen in other studies. 11,20
More research needs to be done to further understand this relationship, specifically in
rural areas such as eastern Kentucky.

There is some evidence that hepatitis B peaks in the spring and summer. 21 This
could be important for future study and prevention efforts.
Pertussis

Rates of pertussis in Kentucky fluctuated between 2005 and 2015, hitting a low of 1.20 cases per 100,000 in 2007 and a high of 12.34 cases per 100,000 in 2012. These fluctuations in rates makes sense given that pertussis peaks in the United States in 3 to 5 year cycles.\textsuperscript{15} Over this same time period, rates of pertussis also fluctuated nationally. The lowest and highest rates in the United States were also seen in those same years, with 3.49 cases per 100,000 in 2007 and 15.4 cases per 100,000 in 2012.\textsuperscript{13} This was the highest number of cases of pertussis reported in the United States since 1955.\textsuperscript{22} This large increase could be due to multiple factors. These include increased awareness, increased use of polymerase-chain-reaction (PCR) for diagnosis of pertussis, as well as potential genetic changes in the bacterium that causes pertussis.\textsuperscript{23}

Over the ten-year period, the highest rate was seen in the <1 age group with 81.70 cases per 100,000. This rate is 64.83 times the rate of pertussis compared to those in the >18 age group. The second highest rate was seen in the 1-3 age group with 22.62 cases per 100,000, a rate 17.95 times the rate seen in the >18 age group. This is reasonable because pertussis is the greatest risk to very young people, especially infants under one year.\textsuperscript{2,15} The next highest rate was seen in the 7-9 age group with 22.05 cases per 100,000, a rate 17.50 times the rate of those >18. Differences in immunity conferred by the previously used whole cell vaccine and the currently used acellular pertussis vaccine help explain the increase in the rate of pertussis in older groups. Immunity from the acellular pertussis vaccine wanes more quickly, resulting in adolescents who have been previously vaccinated becoming susceptible to pertussis again.\textsuperscript{15,23} Looking at the rate of pertussis by age and year shows large variability between age groups in each year, as well
as large variability between age groups across time. A similar pattern is seen with the <1 age group consistently having the highest rate of pertussis. Beginning in 2008, the second highest rate is more often seen in an age group other 1-3.

A large portion (42%) of cases had missing or unknown values for race. Whites had the highest rate of pertussis with 3.36 cases per 100,000, followed by blacks/African Americans with 2.09 cases per 100,000. A large portion (49%) of cases also had missing or unknown values for ethnicity. Hispanics/Latinos had higher rates of pertussis with 3.54 cases per 100,000 compared to non Hispanics/Latinos with 2.84 cases per 100,000, but the difference between these rates was not significant. Females had a rate of 5.83 cases per 100,000, which was higher than the 5.12 cases per 100,000 seen in males.

A study of vaccine coverage of children enrolled in kindergarten for the 2015-2016 school year found 93.9% had received 5 doses of DTaP. Median vaccination coverage for DTaP in the country was 94.2%. It has been shown that pertussis peaks in the summer, though the reason behind this seasonality is not well understood.

**Surveillance in Kentucky**

The main issue with VPD surveillance in Kentucky is a lack of completeness, especially relating to demographic variables like race and ethnicity. The state would benefit greatly from an increase in the collection of this demographic data. The large number of missing values for race and ethnicity made it difficult to draw conclusions about which groups truly had the highest rates of these diseases. Having better data would allow for a more complete picture of the distribution of disease in the state, which would in turn allow for better identification of high risk groups. This is especially
important for diseases like hepatitis B, where what is happening in Kentucky is very different than what is happening in the rest of the United States. Since the overall rates in the state vary greatly from the national rates, having a better idea of the demographic distribution in the state would be helpful to compare to the demographic distribution of the disease in the country overall. This could help to identify why rates in Kentucky are higher.

The three different methods of surveillance (passive, active, and sentinel) each offer different advantages and disadvantages. Passive surveillance relies on reports of certain diseases to public health officials from places like hospitals, health care providers, and laboratories. The National Notifiable Disease Surveillance System (NNDSS) is a passive surveillance system, and this is the method most commonly used for VPD surveillance. This type of surveillance can cover large areas, like an entire state or country, but the reports are not always complete. This was a large part of the problem with the data used in this report. Active surveillance can provide a more complete and timely picture of the number of cases, but is more expensive to perform than passive surveillance since it involves going out and looking for cases. It is often in outbreak situations, but is not as plausible for long-term disease surveillance. Sentinel surveillance uses specific sites where data is collected and then generalized to a larger population. While this is valuable in some situations, it does not apply well to situations where actual number of cases and distribution of these cases is important, such as VPD surveillance.

Data capture could potentially be improved with certain design changes to the surveillance system. The use of an electronic system to submit case reports and forms that cannot be submitted without certain critical information included could help with the
high number of missing demographic variables. Cases could also be cross referenced with other sources of data that may have more complete information relating to demographics, such as birth certificates.

Collecting other information such as vaccination status or specific risk factors would also allow for more meaningful conclusions to be drawn. For example, if data about the vaccination status of the pertussis cases were available, it could point to whether the increase in cases in certain years was related to unvaccinated individuals or vaccine failure in those who were previously vaccinated. Collecting data about some key risk factors for hepatitis B, such as injection drug use and number of sexual partners, could help improve the public health response to this disease. The answers to these kinds of questions are very important, especially when it comes to focusing public health efforts to continue to prevent these diseases.
References


5. Roush SW, Murphy TV, Vaccine-Preventable Disease Table Working G. Historical comparisons of morbidity and mortality for vaccine-preventable diseases in the United States. JAMA. 2007;298(18):2155-2163.


Acknowledgments

I would like to thank Dr. Steven Browning for all of his help throughout this process and for taking over as the chair of my committee. I would like to thank Dr. Lorie Chesnut for her support as both my advisor and a committee member, her help and encouragement were invaluable during my time as a student. I would like to thank Dr. Kathleen Winter for her excellent guidance and insight throughout the course of this project. I would like to thank Hollie Sands for providing me the opportunity to do this project. Finally, I would like to thank my family and George for their constant support and positivity.
Biographical Sketch

Katherine Jay attended the University of Kentucky where she earned a Bachelor of Science degree in Biology in May of 2014. She continued her education at the University of Kentucky in August of 2014, where she pursued a Master of Public Health with a concentration in epidemiology.