

# PROFILES OF SELECTED NOTIFIABLE DISEASES

## HEPATITIS A

<i>Number of cases in 2012:</i>	<i>45</i>	<i>Rate in 2012:</i>	<i>0.4</i>
<i>Number of cases in 2011:</i>	<i>34</i>	<i>Rate in 2011:</i>	<i>0.3</i>

\* Rates are based on the U.S. Census 2011 and 2012 estimates and are per 100,000 population.

Hepatitis A is caused by an infection with the hepatitis A virus (HAV).<sup>1</sup> Hepatitis A has an incubation period of approximately 28 days (range 15-50 days). HAV replicates in the liver and is shed in concentrations in the feces from 2 weeks before to 1 week after the onset of clinical illness. HAV infection produces a self-limited disease that does not result in chronic infection or chronic liver disease.

From September 2012 through February 2013, 21 cases (4 suspect and 17 confirmed) of hepatitis A were identified as part of a multi-jurisdictional outbreak (Figure 1). Cases from multiple counties reported meeting in Union County, Ohio either in a parked car or at a residence to inject drugs or share needles.

**Figure 1: Hepatitis A Cases Linked to Outbreak, Ohio, 2011-2012**



Source of disease data: Ohio Disease Reporting System.

The Centers for Disease Control and Prevention (CDC) were contacted for additional support in testing available serum samples from confirmed cases. Seven specimens were sent to CDC for testing; results can be seen in Table 1. All seven specimens shared the genotype IA1, four shared the related sequence SC325 and three shared a unique sequence among one another. The results suggest two clusters were involved in the outbreak: HAOH 2, 22 and 23 are related to each other and are considered a cluster while HAOH 1, 3, 4 and 21 are a separate cluster.

**Table 1: Hepatitis A Specimen Genotyping Results**

Patient ID	HAV RNA	Genotype	VP1-P2B Sequence	Related Sequence ID in Database	Analysis Comments
HAOH 1	Positive	IA1	HAOH 1	SC325	Share VP1-P2B sequence
HAOH 3	Positive	IA1	HAOH 1	SC325	
HAOH 4	Positive	IA1	HAOH 1	SC325	
HAOH 21	Positive	IA1	HAOH 1	SC235	
HAOH 2	Positive	IA1	HAOH 22	Unique	Share VP1-P2B sequence
HAOH 22	Positive	IA1	HAOH 22	Unique	
HAOH 23	Positive	IA1	HAOH 22	Unique	

Source of data: Centers for Disease Control and Prevention.

Several of the cases involved in this outbreak were also co-infected with hepatitis C. Due to the transient nature of intravenous drug users, contacting the cases for follow-up proved very difficult since many did not have voicemail set up on their cellphones, had disconnected cellphones or had moved without leaving forwarding addresses. For the few cases investigators were able to interview, hepatitis B vaccination was discussed if the case was positive for hepatitis A and C. Additionally, hepatitis A vaccination for contacts was encouraged.

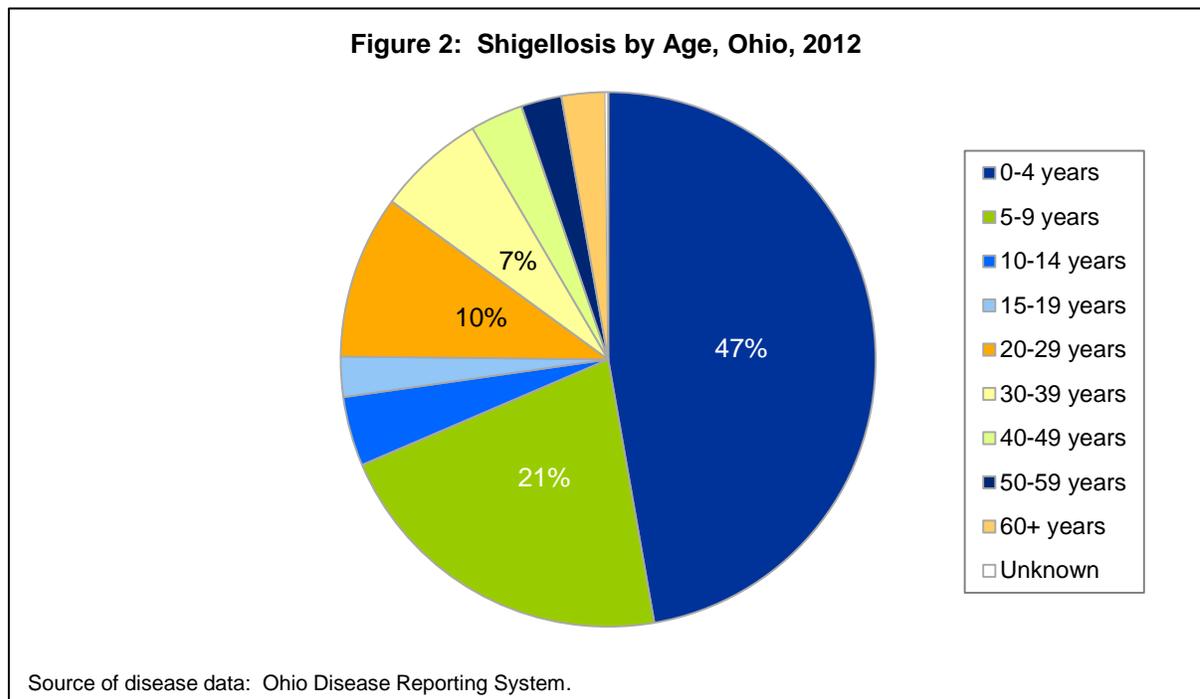
## SHIGELLOSIS

<i>Number of cases in 2012:</i>	1,812	<i>Rate in 2012:</i>	15.7
<i>Number of cases in 2011:</i>	338	<i>Rate in 2011:</i>	2.9

\* Rates are based on the 2011 and 2012 U.S. Census estimates and are per 100,000 population.

Shigellosis is an infectious disease caused by a group of bacteria known as *Shigella*.<sup>2</sup> There are four species of *Shigella*: *Shigella boydii*, *Shigella dysenteriae*, *Shigella flexneri* and *Shigella sonnei*. *Shigella* is spread directly via person-to-person contact by the fecal-oral route. Eating contaminated foods and/or swallowing contaminated water are common vehicles for *Shigella* infection.

In 2012, Central Ohio experienced a community outbreak of *Shigella*. The greatest proportion of cases in 2012 occurred in children aged 0-4 years followed by children aged 5-9 years (see Figure 2).



Outbreaks that began in day care centers quickly spread throughout the Central Ohio community; 1,050 cases were linked to this outbreak in Central Ohio. This accounted for 58 percent of all shigellosis cases reported in 2012. Throughout the state, a total of 1,230 cases of *Shigella* were linked to outbreaks in 2012.

Table 2 demonstrates the proportion of shigellosis cases linked to a known outbreak. In 2011, 28 percent of cases were linked to a known outbreak. In 2012, the number of cases linked to a known outbreak increased to 68 percent. Outbreak-associated cases occurred in all age groups during 2012, especially in children under 9 years of age.

**Table 2: Shigellosis by Age and Outbreak Status, Ohio, 2011-2012**

Age Group	2011				2012			
	Outbreak-Associated		Sporadic		Outbreak-Associated		Sporadic	
0-9 years	84	39%	130	61%	896	72%	346	28%
10-19 years	2	12%	15	88%	70	58%	50	42%
20-29 years	1	3%	37	97%	127	71%	52	29%
30-39 years	2	10%	19	90%	74	63%	44	37%
40-49 years	3	20%	12	80%	27	47%	31	53%
50-59 years	3	15%	17	85%	20	45%	24	55%
60+ years	0	0%	13	100%	13	27%	35	73%
Unknown	0	0%	0	0%	3	100%	0	0%
<b>Total</b>	<b>95</b>	<b>28%</b>	<b>243</b>	<b>72%</b>	<b>1,230</b>	<b>68%</b>	<b>582</b>	<b>32%</b>

Source of disease data: Ohio Disease Reporting System.

## STREPTOCOCCAL DISEASE, GROUP B, IN NEWBORN

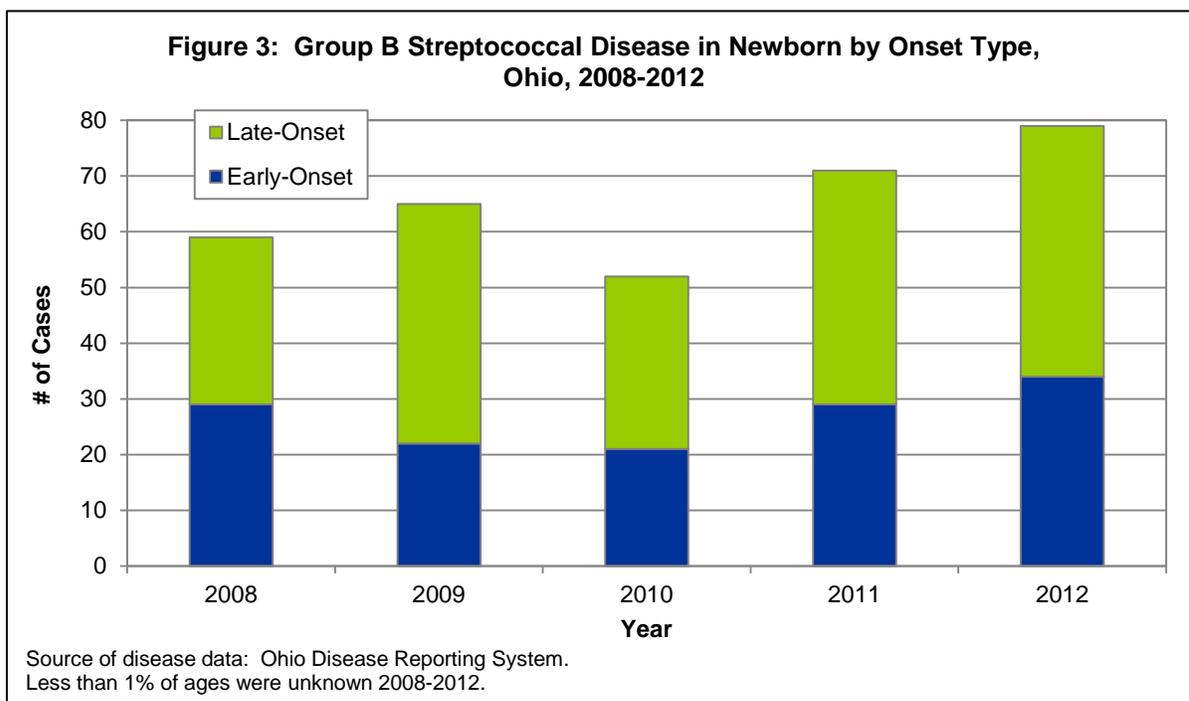
<i>Number of cases in 2012:</i>	79	<i>Rate in 2012:</i>	0.6
<i>Number of cases in 2011:</i>	71	<i>Rate in 2011:</i>	0.6

\* Rates are based on the U.S. Census births reported for Ohio and are per 1,000 population.

Group B streptococci are bacteria commonly found in the digestive tract and birth canal of pregnant women. Group B streptococci can cause systemic and focal infections in infants from birth until three months of age. Disease in young infants is categorized on the basis of chronologic age at onset. Early-onset disease usually occurs within the first 24 hours of life (range 0-6 days). Late-onset disease occurs between seven days and three months.

Guidelines to use intrapartum antimicrobial prophylaxis for women at increased risk of transmitting group B streptococci to their newborns were first issued in 1996. However, it wasn't recommended to screen all pregnant women for group B streptococcal colonization at 35-37 weeks' gestation until 2002.<sup>3</sup> Since these recommendations, the incidence of early-onset group B streptococcal disease in newborns has greatly decreased nationwide.

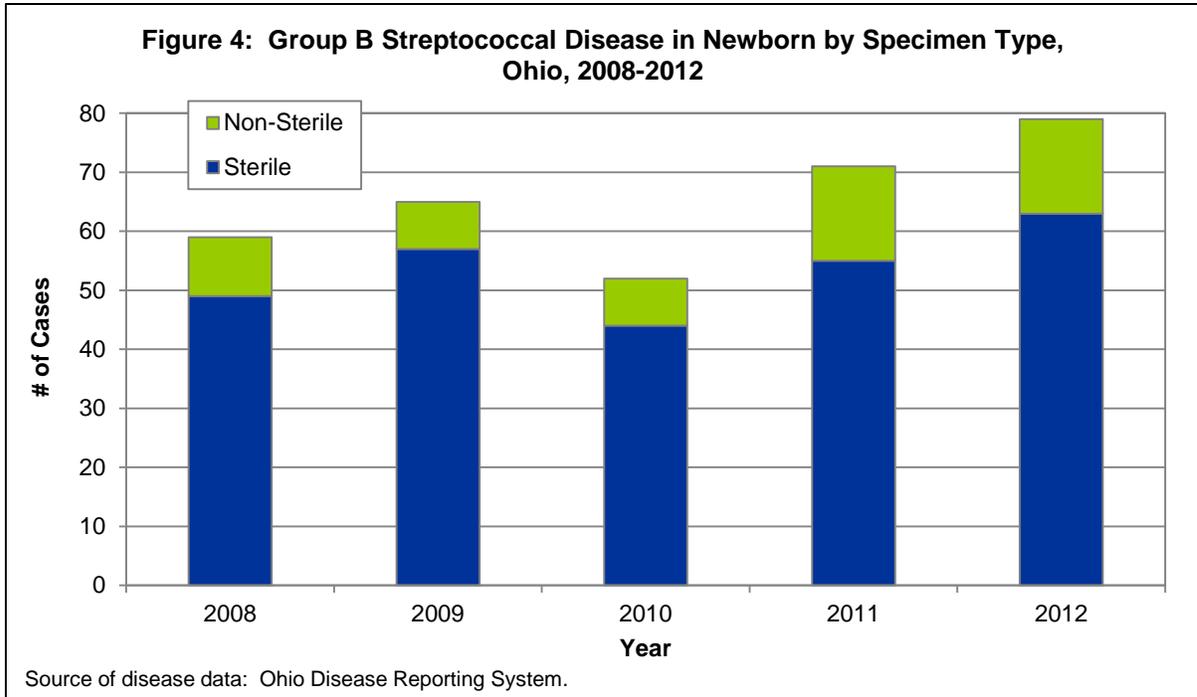
Figure 3 demonstrates the burden of group B streptococcal infections in Ohio newborns over the past five years by onset type. Over the past five years, higher incidence of infection was observed among infants older than 6 days of age than infants 6 days old or less.



Early-onset infections of group B *Streptococcus* may present as signs of systemic infection, respiratory distress, apnea, shock, pneumonia and, less often, meningitis. Late-onset infections commonly manifest as occult bacteremia or meningitis; other focal infections such as osteomyelitis, septic arthritis, adenitis and cellulitis can occur.

Figure 4 demonstrates the number of cases of early-onset and late-onset infections occurring in sterile sites (e.g., blood or cerebrospinal fluid) and non-sterile sites. Over the last five years, 41% of

cases occurred in infants less than 7 days old. Group B *Streptococcus* was isolated from a normally sterile site in 85% of early-onset cases reported over the last five years. Infections in infants less than 7 days old usually occur during the intrapartum period or during delivery. Infections in infants greater than 6 days of age is through person to person contact.



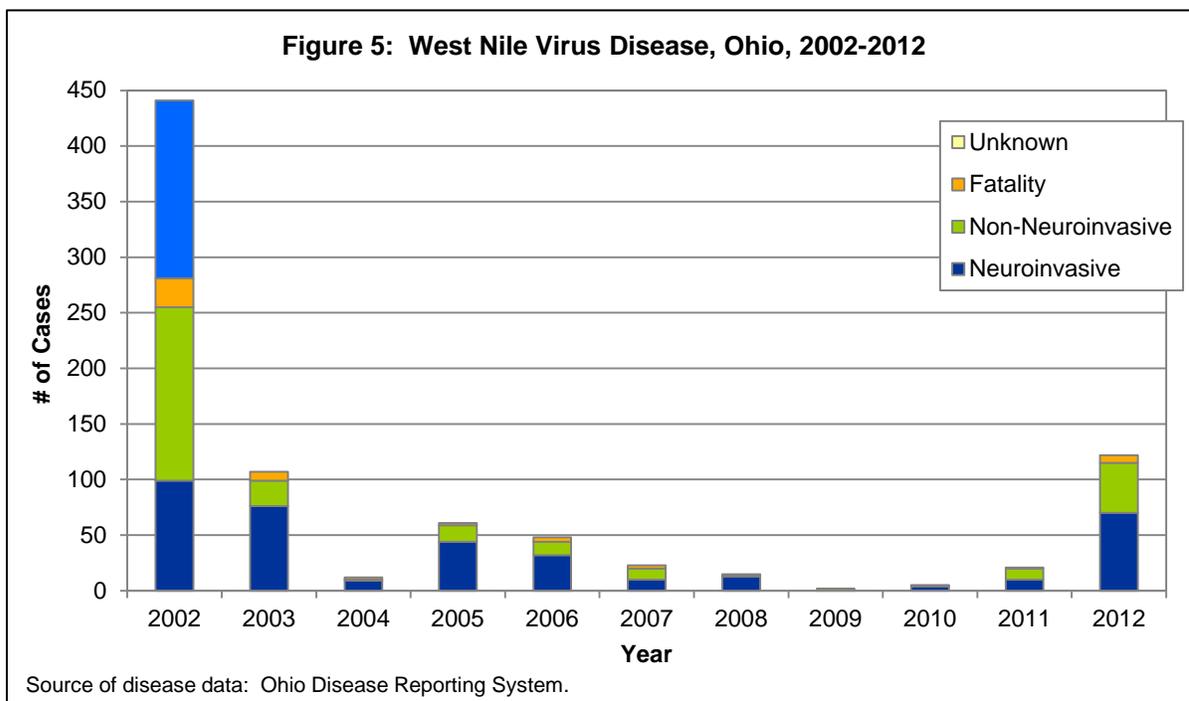
## WEST NILE VIRUS INFECTION

<i>Number of cases in 2012:</i>	122	<i>Rate in 2012:</i>	1.1
<i>Number of cases in 2011:</i>	21	<i>Rate in 2011:</i>	0.2

\* Rates are based on the 2011 and 2012 U.S. Census estimates and are per 100,000 population.

West Nile virus disease is a viral illness transmitted through the bite of infected mosquitoes, particularly *Culex pipiens*. Although rare, West Nile virus can also be transmitted through transfusion or transplantation with infected blood, tissue or organ products. There are two clinical syndromes associated with West Nile virus disease: non-neuroinvasive disease and neuroinvasive disease. Symptoms of non-neuroinvasive disease include a fever, headache, and sometimes nausea, vomiting, a skin rash, and body aches. Neuroinvasive disease is a more serious illness and is characterized by a fever and stiff neck and can include other meningeal symptoms, altered consciousness (disorientation, lethargy, stupor, coma) and signs of neurologic dysfunction (tremors, rigidity, convulsions). Cases occur during the late summer and early fall in Ohio, corresponding to mosquito activity and amplification of the virus in birds.

West Nile virus was introduced into the United States in 1999, and Ohio's first cases were identified in 2002. Since its introduction, the incidence of West Nile virus disease in Ohio dramatically decreased 2003-2004, increased in 2005, and then continued to decrease until 2011-2012 (Figure 5). There was a slight increase in 2011 to 21 cases followed by a significant, nearly six-fold increase in 2012 to 122 cases, the second highest recorded number of cases in a year. During 2002-2012, more cases were of neuroinvasive disease when compared to non-neuroinvasive disease (49 percent vs. 32 percent), and fatalities ranged from 0 to 17 percent of cases. More than 50 deaths have been reported in Ohio West Nile virus cases 2002-2012.



Like 2002-2011, the majority of West Nile virus disease incidence in 2012 occurred in the northern and western parts of Ohio (Figure 6). Nearly 90 percent of cases reported in 2012 occurred in the

Northeast, Northwest, West Central, and Southwest regions of Ohio. More than half of the cases reported in 2012 had neuroinvasive disease (63%), and there were 7 deaths reported.

**Figure 6: West Nile Virus Disease Incidence, Ohio, 2012 Compared to 2002-2011**

