

SURVEILLANCE SUMMARY

HEPATITIS A

**Ohio Department of Health
Infectious Disease Surveillance
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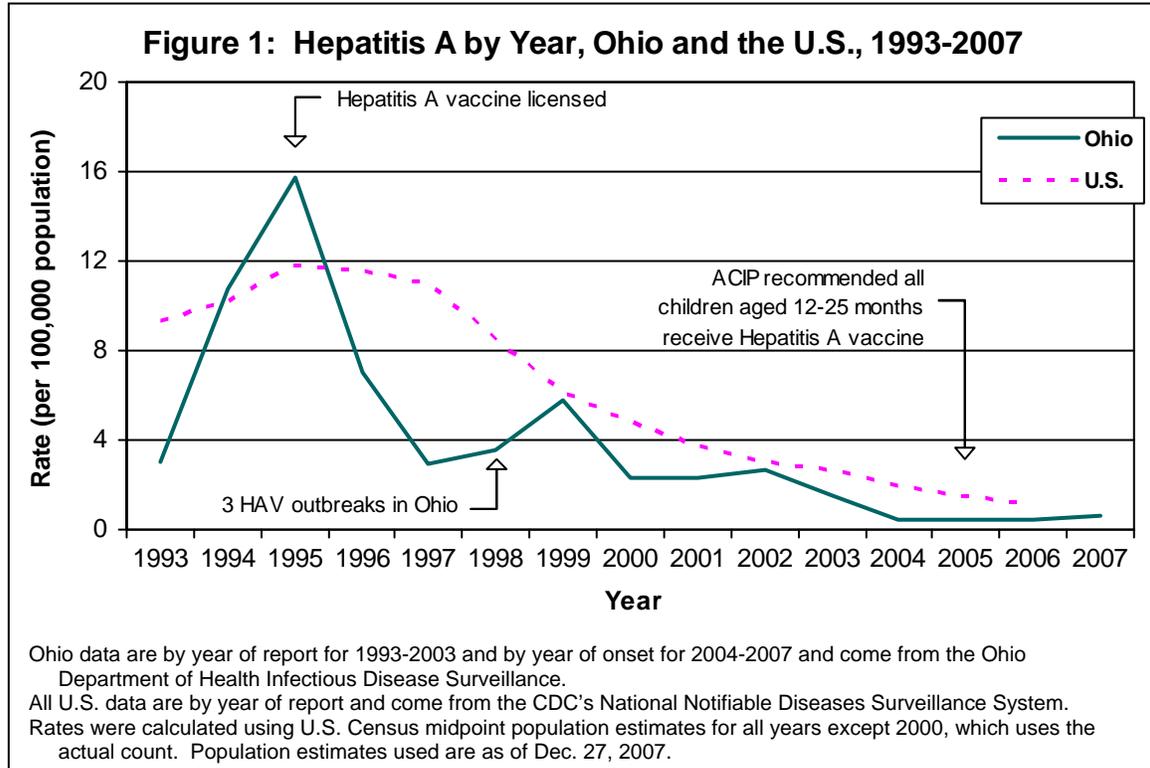
Hepatitis A Surveillance

Hepatitis A is an infection of the liver caused by a virus, the hepatitis A virus (HAV). Approximately one-third of United States citizens have evidence of prior hepatitis A infection, and 100 deaths are attributed to hepatitis A every year.¹

To assess current surveillance trends of Ohio hepatitis A incidence, reported cases with onset of disease from 2002 to 2007 were analyzed. These cases were reported to the Ohio Department of Health (ODH) Infectious Disease Surveillance program via the Ohio Disease Reporting System (ODRS). Ohio trends were compared to national trends when available. These come from the Centers for Disease Control and Prevention's (CDC) National Notifiable Disease Surveillance System.

Burden of Hepatitis A

In the past 15 years, the rate of hepatitis A both nationally and in Ohio has dramatically decreased (see Figure 1). In 1995, two vaccines to prevent hepatitis A infection were licensed in the United States.¹ Following this, rates of acute hepatitis A infection in both the country and the state began a downward trend, seeming to plateau from 2004 on in Ohio. To further reduce the incidence of hepatitis A infection, the Advisory Committee on Immunization Practices (ACIP) recommended in 2005 all children aged 12-25 months be routinely vaccinated against hepatitis A.¹ Ohio adopted this recommendation for routine vaccination in 2007. Except for in 1995, Ohio's rate was at or below the national rate for hepatitis A. In 1998, three outbreaks of hepatitis A were reported in Ohio involving a total of 210 people, which may explain some of the increase in incidence beginning in 1998 and continuing in 1999.



Although the incidence of hepatitis A has substantially decreased in recent years, eliminating this disease is still important because of the substantial economic burden it bears. For each case of hepatitis A identified, an average of 11 close contacts are identified and prophylaxed by a local health department.¹ An adult who becomes ill loses an average of 27 work days.¹ Indirect and direct costs due to hepatitis A are estimated at \$1,817-\$2,459 per adult case and \$433-\$1,492 per pediatric case.¹ Using these estimates, the total cost of hepatitis A in Ohio over the past six years ranged from nearly \$1.1 million to nearly \$1.6 (data not shown).

Causative Agent

Hepatitis A is caused by HAV, which is a member of the Picornaviridae family.² It is a small, non-enveloped, single-stranded RNA virus and is related to the poliovirus.^{1,2} HAV is a very resilient organism. It remains stable in the environment for at least two weeks in fecal matter; the virus cannot be completely inactivated by heat, pasteurization or acid; and it can resist deterioration by chlorine, especially when encased in organic matter.² Only bleach and certain disinfectants are effective at reducing HAV from surfaces.²

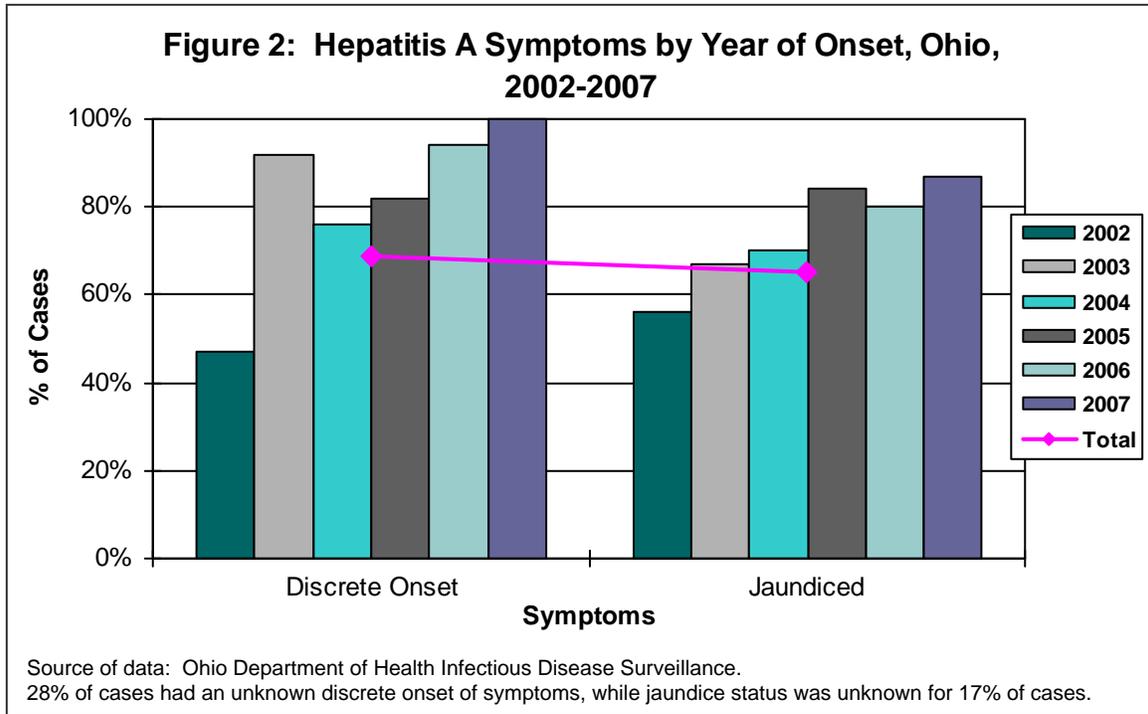
Transmission

Hepatitis A is spread person to person through the fecal-oral route or by ingestion of contaminated food or water.¹ Although transmission through water is not common, there have been a few incidents associated with drinking or swimming in contaminated water.³ Rarely, hepatitis A has been transmitted through blood transfusion.¹ The clinical severity of disease neither depends on the route of infection nor the dose of virus ingested.² HAV is naturally found in humans, but can be sustained in chimps and other non-human primates in a laboratory setting.¹

Clinical Presentation

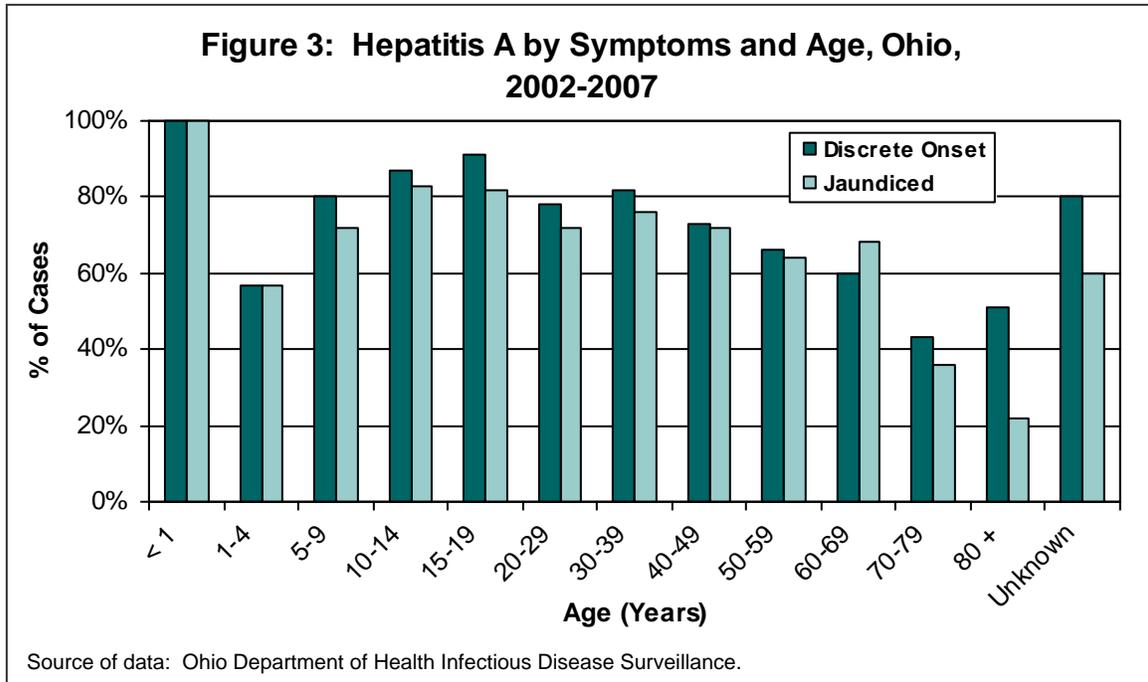
Clinically, hepatitis A ranges from no symptoms to mild illness to severe disease. The most common symptoms include a sudden, discrete onset of fatigue, abdominal pain, loss of appetite, nausea, diarrhea, fever and jaundice.⁴

Figure 2 depicts the percent of Ohio hepatitis A cases that reported experiencing a discrete onset of symptoms and jaundice by year. Because a discrete onset of symptoms is required by the surveillance case definition of hepatitis A, 100 percent of cases counted from 2002-2007 should have been reported with this information. Even though not all counted cases were reported with the discrete onset of acute symptoms, increasingly more cases were reported as experiencing these symptoms over time. In 2007, 100 percent of cases reported an abrupt onset of acute symptoms, up from 47 percent in 2002. Jaundice is a part of the surveillance case definition but is not required on its own, so not all counted cases would be expected to have this, although the majority of hepatitis A cases experience jaundice. Again, increasingly more cases were reported with jaundice over the six years assessed. In 2002, 56 percent of cases reported jaundice, which rose to 87 percent in 2007. Over the six year period, 72 percent of cases reported an abrupt onset of symptoms, and 67 percent reported jaundice. For 28 percent of cases, it was unknown whether there was the characteristic discrete onset of symptoms, the majority of which were in 2002. Jaundice status was unknown for 17 percent of cases during 2002-2007.



The severity and probability of hepatitis A symptoms increase with age.⁵ An estimated 90 percent of children less than 5 years infected with HAV are asymptomatic.³ Symptomatic children under 6 years of age generally experience mild, nonspecific symptoms such as nausea, fever, vomiting, malaise, diarrhea or dark urine.² Less than 5 percent of children 3 years and under become jaundiced, while 10 percent of children aged 4-6 become jaundiced.² In contrast, 70 percent of adults and older children report symptomatic infections with jaundice.⁶ Children 6-14 years of age have illness comparable to that of adults.²

In Ohio, 100 percent of infants less than 1 year experienced a discrete onset of symptoms and jaundice, which was higher than any other age group (see Figure 3). This could be due to small numbers, because less than 1 percent of all cases reported during 2002-2007 were infants, or because these data were more likely to be reported for this age group. Slightly more than half (57 percent) of children aged 1-4 years reported symptoms and jaundice. The percentage of children 5-9 and 10-14 years experiencing symptoms was similar to that of adults. Among adults, the percentage of cases reported with a discrete onset and jaundice decreased with increasing age. Adults aged 70 and over had onset of symptoms and jaundice reported most infrequently (47 percent and 29 percent, respectively).



Symptoms usually appear 28 days following exposure to HAV, but can surface as early as 15 days or as late as 50 days.¹ The duration of symptoms typically lasts one to two weeks, but could linger for several months in those with severe disease.⁵ Hepatitis A infection confers lifelong immunity and does not cause chronic liver disease, cirrhosis or liver cancer.^{3,4}

Nationally, 11 percent to 22 percent of cases are hospitalized for hepatitis A each year.¹ Over the past six years, Ohio has seen an increase in the percentage of cases hospitalized for hepatitis A (see Figure 4). In 2002, 24 percent of cases reported being hospitalized. By 2007, this rose to 46 percent, far exceeding national estimates. Hospitalization status was unknown for 12 percent of cases over the six years evaluated. It is unknown whether Ohio's higher rate of hospitalization for hepatitis A is an indication of Ohioans experiencing more severe illness from hepatitis A than the rest of the country or whether the increase could be due to the misinterpretation of being hospitalized with hepatitis A, versus being hospitalized because of hepatitis A.

Hospitalizations for hepatitis A in Ohio from 2002 to 2007 occurred across all age groups, but the greatest number was among persons aged 20-59 years (Figure 5). The fewest hospitalizations occurred among infants and children.

Figure 4: Hepatitis A Cases Hospitalized by Year of Onset, Ohio, 2002-2007

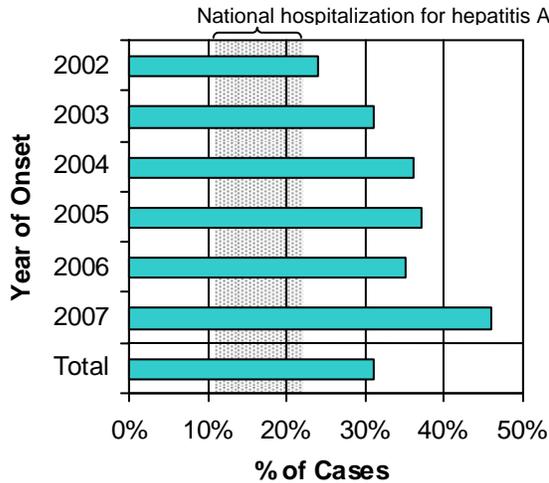
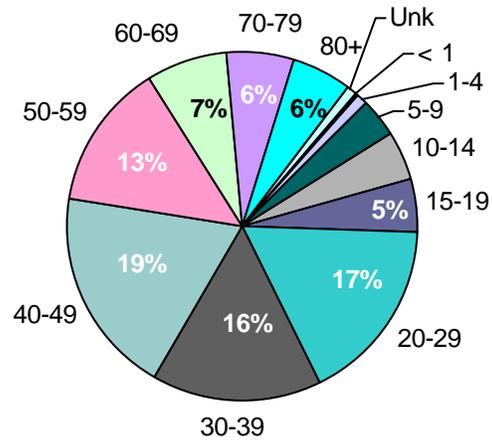


Figure 5: Hepatitis A Hospitalizations by Age, Ohio, 2002-2007



Source of data: Ohio Department of Health Infectious Disease Surveillance.

Approximately 15 percent of those infected with HAV experience relapsing symptoms over a six to nine-month period of time, but this usually resolves completely.^{3,4} Among adults with hepatitis A, 0.15 percent to 0.5 percent may develop fulminant disease, of which half die.⁷ Fulminant hepatitis is characterized by severe jaundice, encephalopathy, coagulopathy and liver failure within two months of onset of symptoms.³

Nationally, the case fatality rate for hepatitis A is low at 0.1 percent to 0.3 percent across all ages, but can reach 1.8 percent for adults over 50 years of age.⁵ From 2002 to 2007, there were two deaths reported in Ohio cases that were attributed to hepatitis A, making Ohio's case fatality rate 0.3 percent over six years (data not shown). Both deaths were in individuals greater than 60 years old.

There is no treatment for people with hepatitis A other than supportive care, maintaining a proper diet and avoiding alcohol.⁷

Diagnosis

Because hepatitis can be caused by several different agents with similar clinical features, hepatitis A cannot be diagnosed based on signs and symptoms alone.¹ Individuals infected with hepatitis A will have detectable IgM antibody levels to HAV in their serum for up to six months after exposure to the virus.¹ Detection of this IgM antibody, IgM anti-HAV, in a symptomatic person along with evidence of liver dysfunction (increased liver enzyme levels or jaundice) is considered a confirmed case of acute hepatitis A, according to the surveillance case definition.² A positive test for total anti-HAV (both IgG and IgM anti-HAV antibodies) is not enough to diagnose hepatitis A because it does not distinguish past from present infections.²

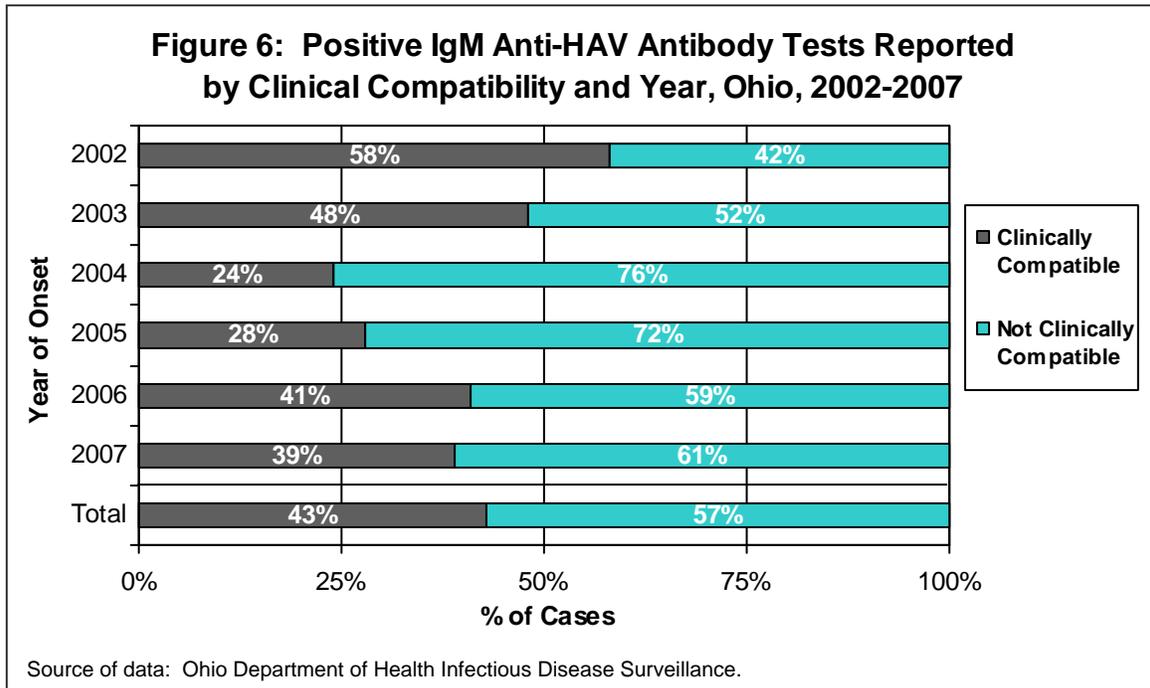
The clinical component in the case definition of hepatitis A is necessary because many asymptomatic individuals test positive for IgM anti-HAV. Hepatitis A test results analyzed from six United States counties in 2003 showed 62 percent of persons testing positive for IgM anti-HAV did not have illness compatible with hepatitis A.⁸ Those asymptomatic, lab-positive individuals

were more likely to be older and female than their symptomatic, lab-positive counterparts.⁸ Positive IgM anti-HAV tests in those without symptoms of hepatitis A may indicate:

- An asymptomatic infection of acute hepatitis A,
- Previous exposure to HAV with continued elevation of the IgM antibody or
- A false positive test.⁸

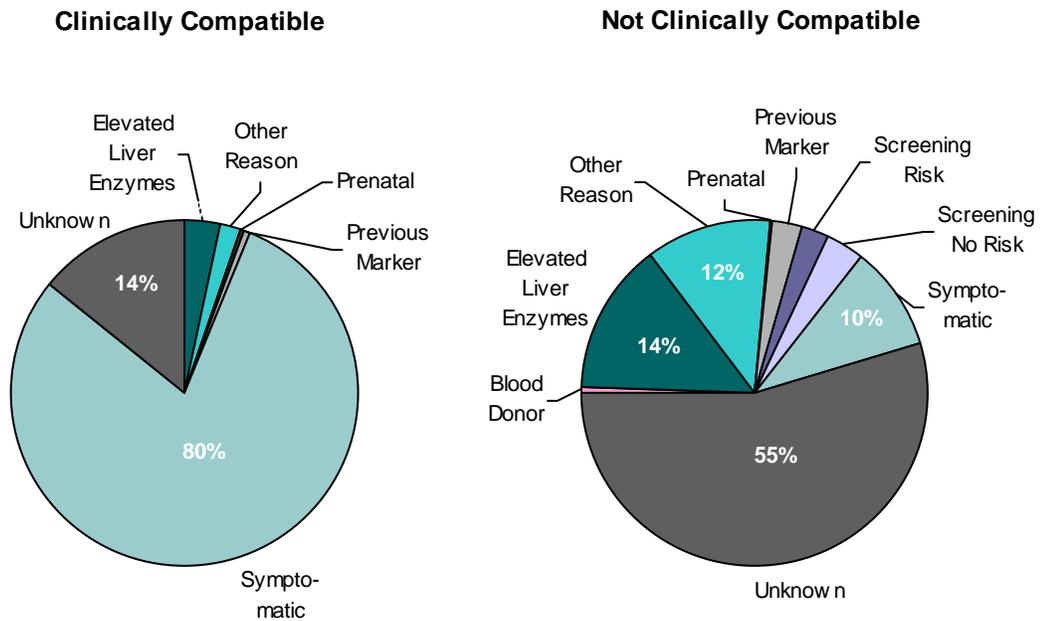
Based on these findings, the testing of individuals without symptoms of acute hepatitis who have a low risk for acquiring hepatitis A is not recommended.⁸

In Ohio, the majority (57 percent) of positive IgM anti-HAV tests reported during 2002-2007 were in individuals without clinically compatible acute disease (Figure 6). With the exception of 2002, most positive tests were in asymptomatic Ohioans in all years evaluated. Like national trends, the asymptomatic, IgM anti-HAV positive Ohioans were more likely to be female (50 percent versus 38 percent) and older (68 percent versus 28 percent were 50 years of age and older) than their symptomatic, IgM anti-HAV positive counterparts (data not shown).



Among persons with a positive IgM anti-HAV test reported from 2002 to 2007, the reason for initial hepatitis testing in those who were clinically compatible versus those who were not clinically compatible for disease differed considerably (see Figure 7). The majority of clinically compatible cases with a positive IgM anti-HAV test were tested because they were symptomatic (80 percent), while 6 percent of clinically compatible cases reported reasons for testing other than being symptomatic or unknown reasons. In comparison, most of the non-clinically compatible positive IgM anti-HAV cases had an unknown reason for initial testing (55 percent), while only 10 percent reported the reason as being symptomatic. Other reasons for testing were reported for 35 percent of non-clinically compatible persons with positive tests. These included donating blood, evaluating elevated liver enzyme levels, prenatal screening, having a previous marker for hepatitis, screening an asymptomatic person with a risk factor, screening an asymptomatic person without a risk factor and other reasons.

Figure 7: Reasons for Testing of Positive IgM Anti-HAV Persons by Clinical Compatibility, Ohio, 2002-2007



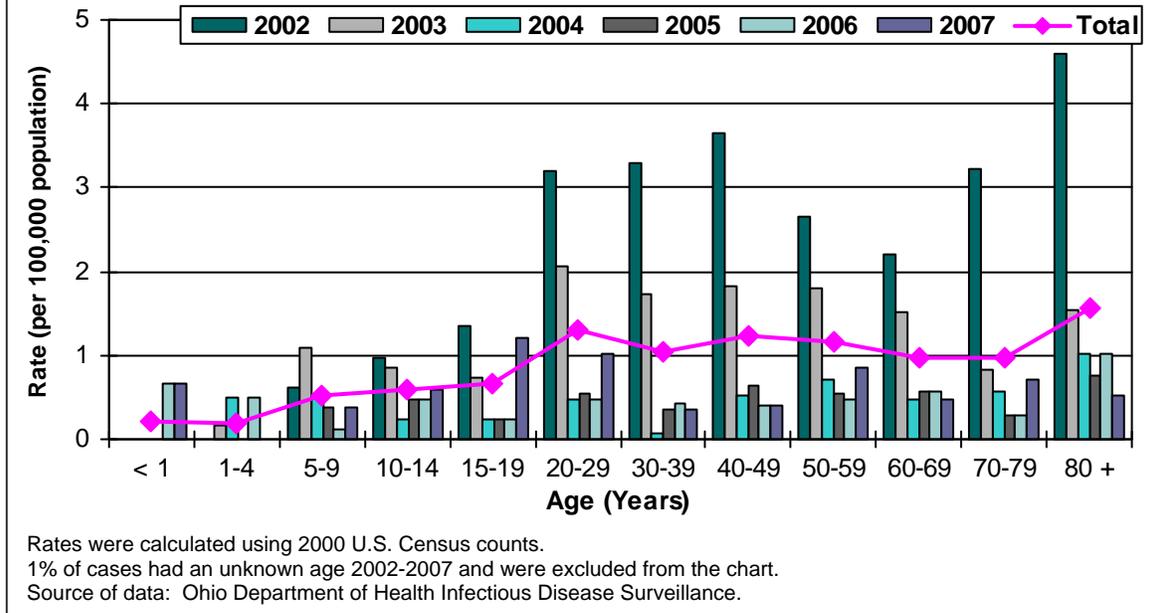
Source of data: Ohio Department of Health Infectious Disease Surveillance.

Demographic Trends

Although children have the highest rates of infection, most are asymptomatic and are not considered cases of hepatitis A for surveillance purposes unless they exhibit the classic symptoms of hepatitis A.⁶ Recently, rates of hepatitis A have increased among adults and decreased among children in the United States.⁹ National rates of hepatitis A by age in 2004 showed low and steady rates (slightly less than two per 100,000) in adults 40 years and older.⁹ This stability has been attributed to a high proportion of individuals in this group with natural immunity due to prior infection.⁹

The highest incidence of acute hepatitis A in Ohio occurred among adults in 2002-2007 (Figure 8). Rates were similar and rather stable for adults aged 20 and over, although adults aged 80 and older had slightly higher rates. The lowest rate of hepatitis A during the six-year period occurred in infants and gradually increased through children and adolescents until it seemed to plateau in adults aged 20 years and older.

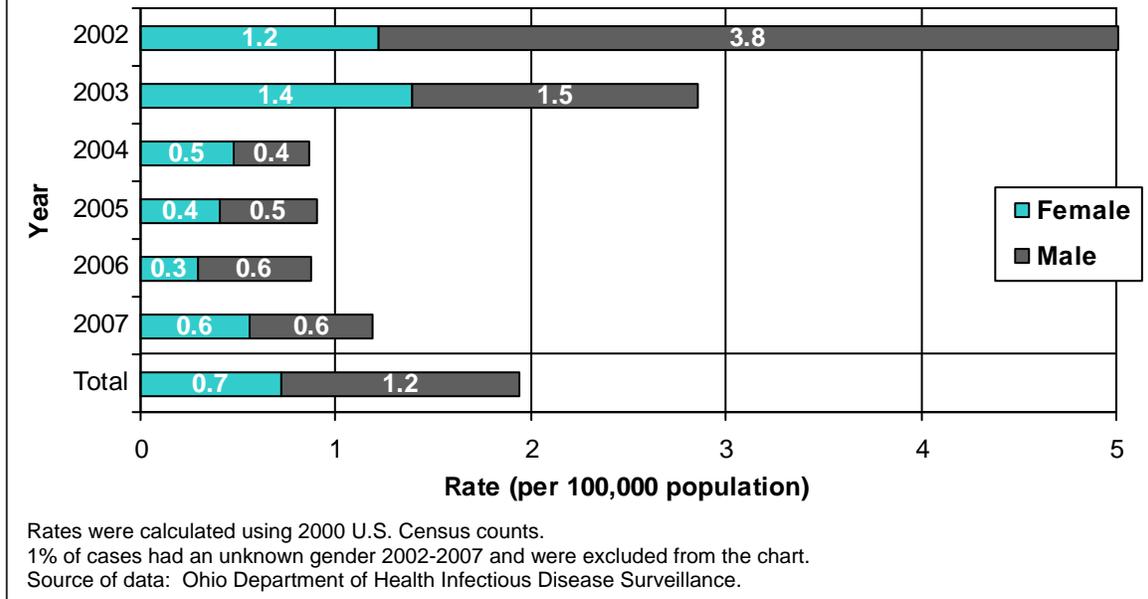
Figure 8: Hepatitis A by Age and Year of Onset, Ohio, 2002-2007



Nationally, from the late 1990s to 2001, rates of hepatitis A in males were almost twice as high as rates in females.⁹ Since 2001, the rate of disease in men has decreased to only slightly above the rate in women.⁹ By 2004, the rate of hepatitis A in the United States for men was 2.1 per 100,000, while the rate for women was 1.8 per 100,000.⁹ The modest increase of hepatitis A among men may be an indication of more men partaking in risk behaviors such as drug use, homosexual activity and international travel.²

In Ohio, rates of hepatitis A were slightly higher in males as compared to females from 2002-2007 at 1.2 per 100,000 and 0.7 per 100,000, respectively (see Figure 9). In 2002, the rate was more than three times higher in males, but the rate of hepatitis A in females was actually a little higher than in males in 2004. The rates were equal between the genders in 2007.

Figure 9: Hepatitis A by Gender and Year of Onset, Ohio, 2002-2007



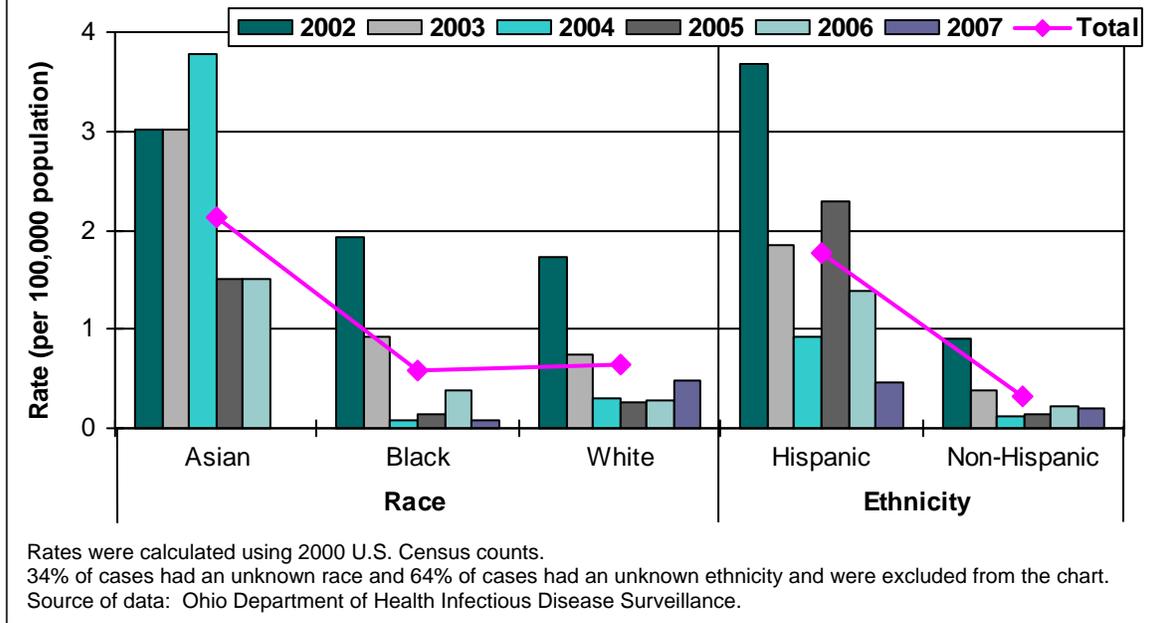
In the United States, American Indians/Alaskan Natives had always experienced significantly higher rates of hepatitis A than any other race until 1995, when widespread vaccination among this group began drastically reducing rates.⁹ Since 2001, rates of hepatitis A among American Indians/Alaskan Natives have been equal to or lower than rates among other races.⁹ Nationally, rates for all race groups decreased during 2001-2004, except for Asian/Pacific Islanders, which experienced a slight increase in 2003-2004 from two to approximately three cases per 100,000.⁹ In 2004, rates in white, black and American Indian/Alaskan Native Americans were similar at around one case per 100,000.⁹

In Ohio, Asians had the highest rates of acute hepatitis A during 2002-2007 at 2.14 cases per 100,000 (Figure 10). This was more than three times the rate for blacks (0.59 per 100,000) and whites (0.64 per 100,000). Like national trends, rates for blacks and whites were very comparable for all years, except in 2007 when the rate among whites was higher at 0.49 per 100,000, compared with blacks at 0.08 per 100,000. This could be a result of steadier rates with larger numbers among white Ohioans. Over the six years analyzed, 34 percent of cases had no race reported, and there were no cases reported in Hawaiian Natives/Pacific Islanders or American Indians/Alaskan Natives.

Rates have always been higher for Hispanics than non-Hispanics in this country; however, the disparity has decreased since 1997.⁹ From 2001-2004, the rate of hepatitis A among Hispanic Americans dramatically decreased from almost 10 cases per 100,000 to fewer than four cases per 100,000.⁹

As seen in Figure 10, the rate of acute hepatitis A in Hispanics was more than five times greater than the rate in non-Hispanics in Ohio (1.77 versus 0.33 per 100,000, respectively). The rate was consistently higher for Hispanics each year during 2002-2007. However, most cases during this period were reported with an unknown ethnicity (64 percent), so the true trends with respect to ethnicity cannot be interpreted with certainty.

Figure 10: Hepatitis A by Race/Ethnicity and Year of Onset, Ohio, 2002-2007



Risk

Persons at increased risk for acquiring acute hepatitis A include:

- Household contacts of HAV-infected people,
- Sexual contacts of infected people,
- People living in areas of increased hepatitis A rates,
- Travelers to hepatitis A-endemic countries,
- Injection and non-injection drug users and
- Men who have sex with men.⁴

The most commonly reported source of hepatitis A infection in the United States during the past decade was contact with an infected person at 14 percent of cases.¹ From 2002-2007 in Ohio, the most commonly reported risk factor for hepatitis was recent international travel (13 percent), and the second-most common risk identified was personal contact with an infected person at 11 percent (see Table 1). This differs from the national figure of 5 percent of cases reporting recent international travel.¹ The men who have sex with men risk was equally reported by cases in the United States as well as Ohio at 10 percent.¹ Less commonly reported risk factors included being a child or employee at a day care facility, having contact with a child or employee at a day care facility, using injection drugs and using non-injection street drugs. Despite these recognized risk factors, 45 percent of cases nationally could not identify a risk factor.¹ In Ohio, 22 percent of cases had no identifiable risk factor, and risk factors were unknown or incomplete for 44 percent of cases in 2002-2007.

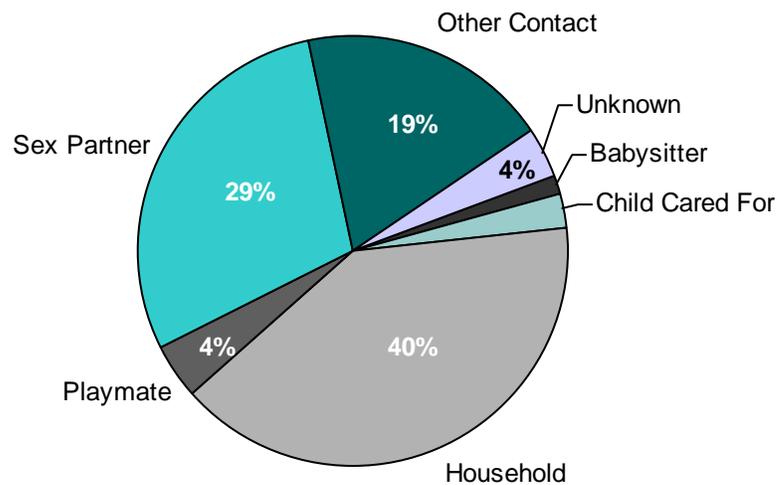
Table 1: Reported Risk Factors Among Hepatitis A Cases, Ohio vs. U.S.

Risk	Ohio	U.S.
Personal contact with a known case	11%	14%
Child/employee at day care	1%	2%
Contact of child/employee at day care	2%	2%
Recent international travel	13%	5%
Injection drug use	1%	6%
Non-injection street drug use	1%	-
Men who have sex with men	10%	10%
No identifiable risks	22%	45%
Unknown/incomplete risk	44%	-

U.S. risk factor data are from 1990-2000.
Ohio data are from 2002-2007 and come from the Ohio Department of Health Infectious Disease Surveillance.

Household contacts were the most commonly reported type of personal contact 2002-2007 (see Figure 11). Household contacts were identified in 40 percent of cases reporting contact with another case of hepatitis A, while 29 percent reported the contact as a sex partner. Nearly one-fifth of cases reporting contacts classified their contact as other, and 4 percent reported their contact as unknown. Other reported types of personal contact to cases of hepatitis A included playmates, babysitters and children cared for.

Figure 11: Types of Personal Contacts Reported by Hepatitis A Cases, Ohio, 2002-2007

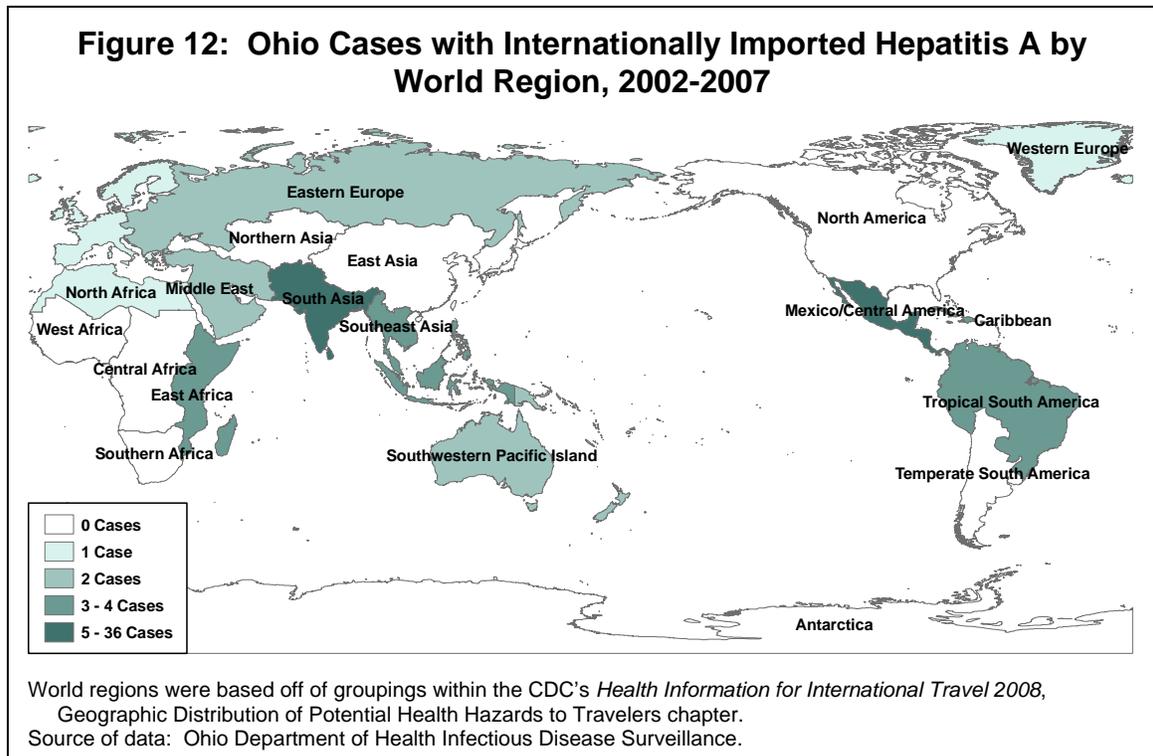


Source of data: Ohio Department of Health Infectious Disease Surveillance.

Because children usually show few to no signs of hepatitis A infection, and they have the highest rates of infection, they play a major role in the transmission of the virus, often serving as a source of infection to others.^{1,6} Therefore, children or employees in day care centers (especially those caring for diapered children) and contacts to children or employees in day care centers can put those individuals at increased risk for disease.³

The most recognized risk factor for hepatitis A in Ohio in recent years has been recent travel to areas with endemic disease (Table 1). Infection of HAV is very common in developing countries, where close to 100 percent of people have been infected by 10 years of age.⁷ Hepatitis A infection is most common in Central and South America, Africa, the Middle East, Asia and the Western Pacific.¹ The risk of acquiring hepatitis A to non-immune travelers visiting resort areas of developing countries is estimated at one per 1,000 per week of exposure, while the risk to non-immune travelers visiting remote areas of developing countries is estimated at five per 1,000 per week of exposure.⁷ There is still some risk of infection when traveling to developed countries as well.⁷

Figure 12 depicts which regions of the world Ohio cases reported recent travel prior to becoming diagnosed with acute hepatitis A. Mexico/Central America and South Asia had the greatest number of cases with internationally acquired hepatitis A (46 percent and 27 percent of cases, respectively). Other areas where Ohio cases reported recent travel prior to symptoms coincide with known endemic areas: Southeast Asia, tropical South America, the Caribbean, North and East Africa, the Middle East, Eastern Europe, the Southwestern Pacific Islands and Western Europe.



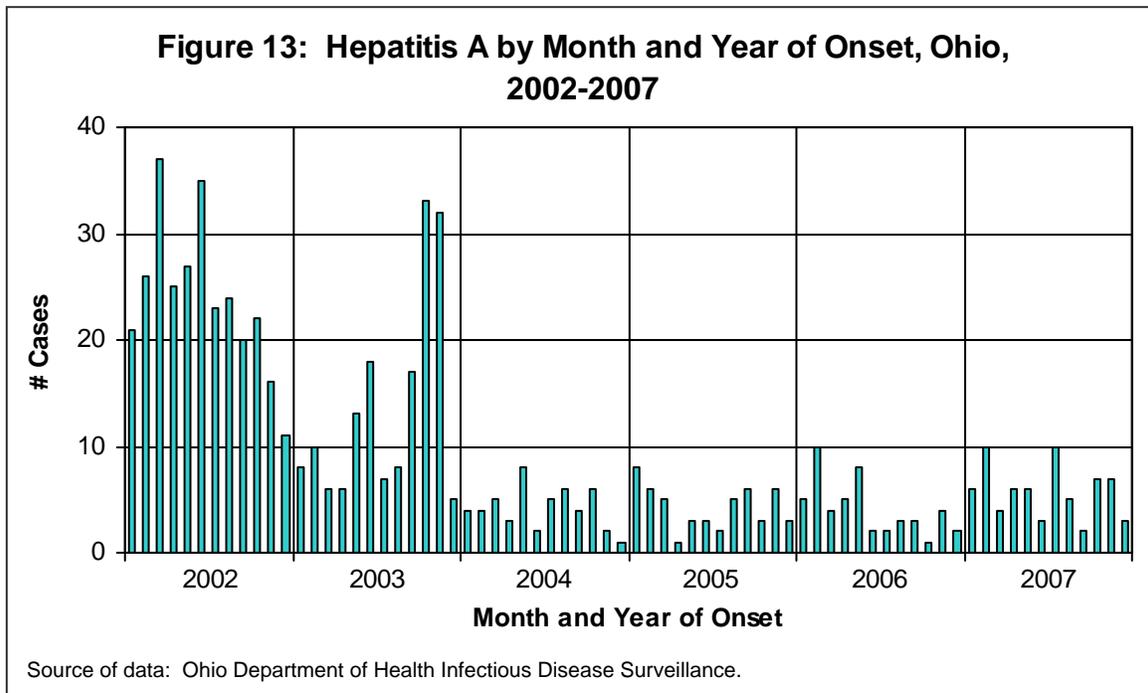
Drug use, whether through injection or not, was not a notable risk factor for Ohioans at 2 percent of cases 2002-2007 (Table 1). However, it is still recognized as a risk factor for hepatitis A. The risk of acquiring acute hepatitis A through injection drug use may be more related to poor personal hygiene and unsanitary conditions common to injection drug users rather than representing a blood-borne risk of hepatitis A transmission.³

In Ohio and the United States, 10 percent of hepatitis A cases occurred in men who have sex with men (Table 1).¹ While this was one of the leading reported risks associated with infection, there has been no indication that HAV is sexually transmitted.³

Seasonal Variation

Before and immediately following World War II, acute hepatitis A incidence in the United States was higher and experienced seasonal fluctuation with the highest rates occurring in the late summer and early fall.² Widespread, national epidemics also occurred every six to 10 years.² Due to improvements in sanitation and personal hygiene, the seasonal and epidemic patterns of hepatitis A have mostly subsided.² Today, there is no noticeable seasonality to hepatitis A, and epidemics no longer occur nationwide but still occur in small, well-defined, high-rate community settings.¹

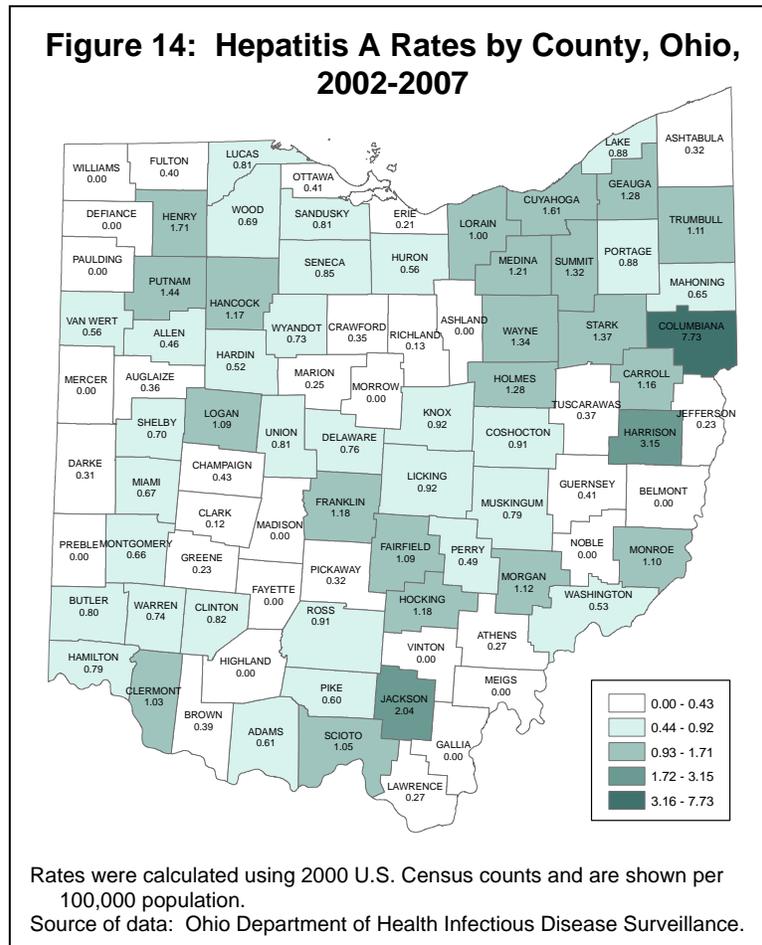
As seen in Figure 13, there really was no substantial seasonal variation to acute hepatitis A in Ohio in 2002-2007. In 2002, the majority of cases had onset of symptoms in the late winter/early spring, which then trailed off during the remainder of the year. During 2003, the majority of cases had symptom onset in early summer and again in late fall, with this latter peak reflecting cases involved in a multi-state outbreak of hepatitis A at that time. From 2004 to 2007, the months of onset fluctuated, with some years showing peaks during the winter, some during the summer and some during the late fall.



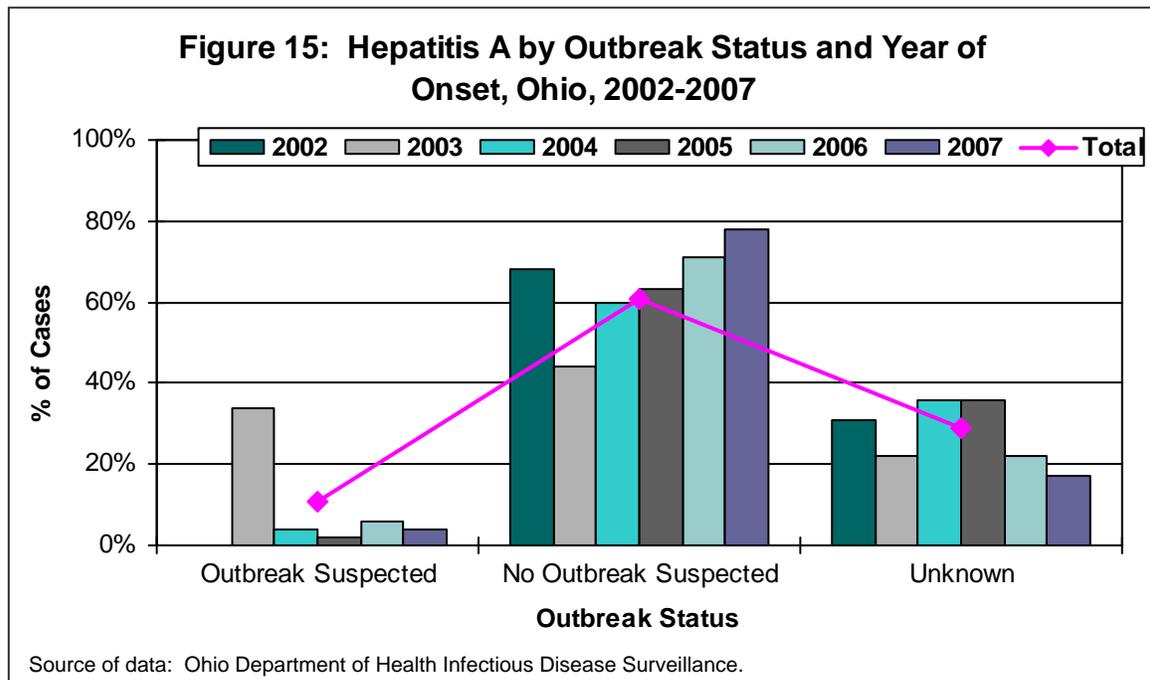
Geographic Distribution

As previously noted, hepatitis A occurs worldwide, most commonly in developing countries.¹⁰ Until recently, half of hepatitis A incidence in the United States was reported in the western-most states, which disproportionately contained 22 percent of the country's population.¹ Since the widespread use of vaccination programs in these areas, incidence across the country has been geographically similar since 2002, although community-wide epidemics still occur in isolated, high-rate communities.¹

When looking at geographic trends in Ohio by county in 2002-2007, areas surrounding urban centers appeared to have slightly higher rates, especially in northeast Ohio (see Figure 14). The county with the highest rate of acute hepatitis A was Columbiana at 7.73 cases per 100,000; however, this county was involved in the 2003 multi-state outbreak of hepatitis A. The rate in 2003 for Columbiana County was 44.61 cases per 100,000, which skewed the overall six-year rate. Harrison and Jackson counties had the next highest rates at 3.15 and 2.04 per 100,000, respectively. All other counties had lower rates, ranging from zero to 1.71 cases per 100,000.



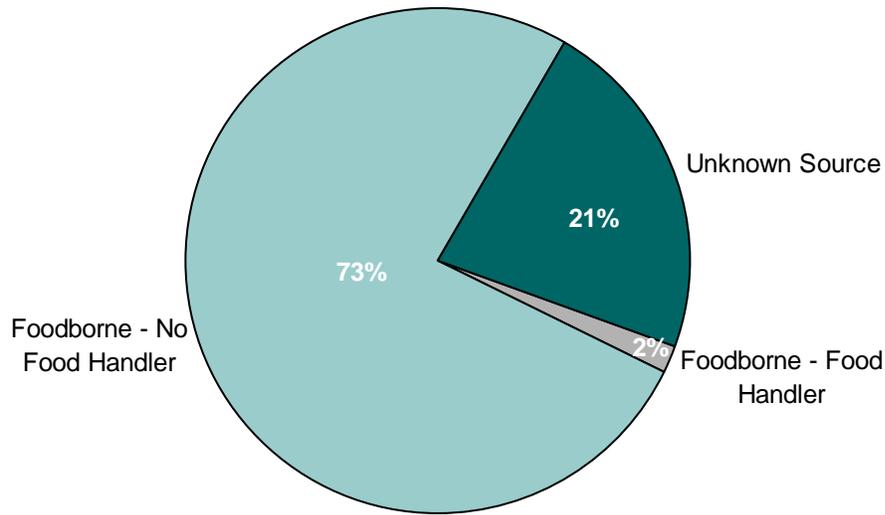
In Ohio, 62 percent of acute hepatitis A cases were not suspected to be part of an outbreak in 2002-2007 (Figure 15). Due to a multi-state outbreak of hepatitis A in 2003, 34 percent of cases with symptom onset in that year were suspected to be involved in an outbreak. However, for other years, less than 6 percent of cases were suspected as part of an outbreak. It was unknown for 28 percent of all cases over the six-year period whether they were associated with an outbreak.



Most common source outbreaks in the United States have been foodborne and were associated with an HAV-infected food handler.¹¹ Food handlers are not at increased risk for hepatitis A, but they tend to be members of demographic groups that experience an increased incidence of disease, specifically those younger in age and of lower socioeconomic status.¹¹ Outbreaks due to the contamination of food during growing, harvesting, processing or distribution (non-food handler associated) also occur and have involved shellfish and fresh produce such as lettuce, strawberries, green onions and tomatoes.¹¹ Waterborne common source outbreaks are rare in developed countries because water treatment procedures seem to be effective at neutralizing HAV's infectivity.¹¹

Among Ohio cases suspected to be associated with an outbreak, the majority were foodborne, not associated with an infected food handler (Figure 16). Almost all of these non-food handler-associated foodborne outbreaks occurred during the large multi-state outbreak in 2003 associated with green onions. For all years except 2003, nearly all hepatitis A cases suspected to be part of an outbreak had an unknown source.

Figure 16: Hepatitis A Cases Suspected in an Outbreak by Type, Ohio, 2002-2007



No waterborne hepatitis A outbreaks were reported during 2002-2007.
Source of data: Ohio Department of Health Infectious Disease Surveillance.

Prevention

Because there is no specific treatment for hepatitis A, preventing infection is the most appropriate way to control both the morbidity and the expense of this disease.

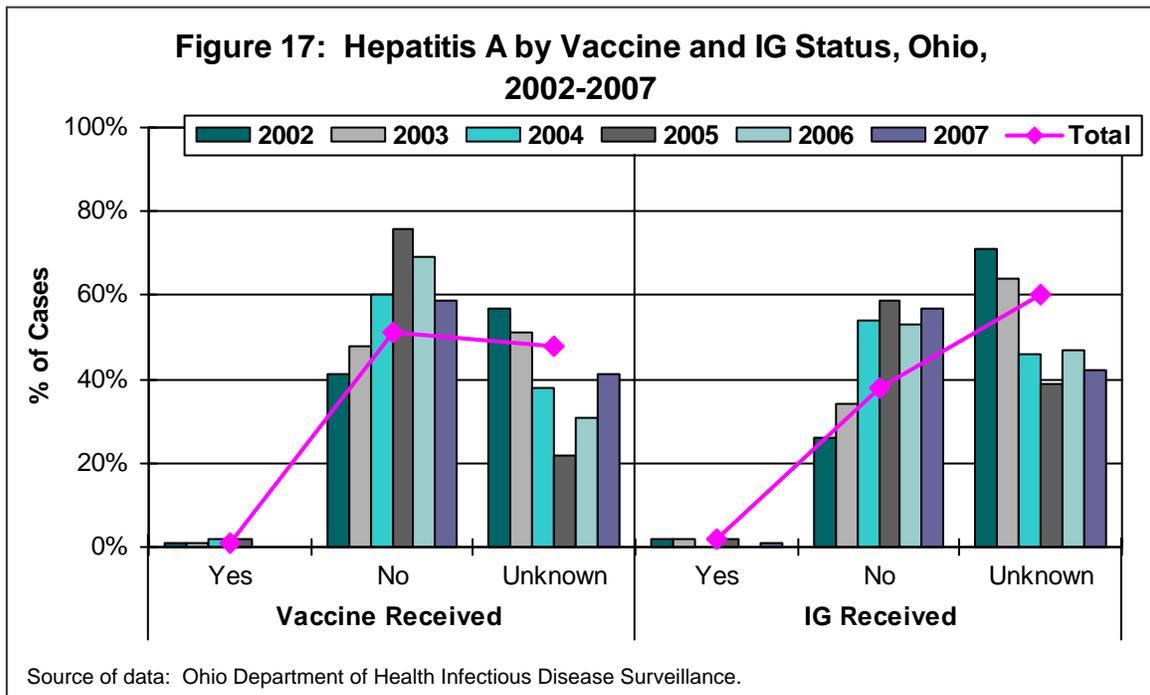
The best method of prevention is through the use of one of the licensed vaccines, HAVRIX® or VAQTA®.¹ Both vaccines provide a very good, protective immune response and are approved for use in children 12 months of age and older.¹ Within four weeks of receiving one dose of either vaccine, 95 percent of adults and 97 percent of children had protective antibody levels to HAV.¹ This rose to 100 percent of adults and children one month after receiving the second dose of either vaccine.¹ The two doses of hepatitis A vaccine should be administered at least six months apart.¹

Upon the licensure of the hepatitis A vaccines in the United States in 1995, ACIP recommended vaccinating people at high risk for acquiring the disease, especially international travelers.¹ However, these recommendations did not decrease the overall incidence of hepatitis A in the country.¹ In 1999, the routine vaccination of children 2 years and older in areas with a high incidence of hepatitis A was recommended.¹ Within these targeted areas, the vaccines significantly lowered the occurrence of hepatitis A.¹ Because this approach was successfully decreasing incidence, all United States children were recommended to receive the first dose of the hepatitis A vaccine at 12-23 months of age in 2005.¹ This recommendation was adopted in Ohio in 2007. Protective antibody levels against HAV provided by the vaccine are estimated to persist for at least 20 years.¹

Another way to prevent hepatitis A among people with known exposures to infected cases is with immune globulin (IG). IG is a concentrated solution of antibodies produced from pooled human plasma.¹ IG is given via injection within two weeks of exposure to the virus, and it is 80 percent to 90 percent effective in preventing disease for three to five months.¹ IG given later than two weeks after exposure may only lessen the severity of disease.¹ IG is recommended for those who have had close personal contact with someone infected with hepatitis A, employees/children

at day cares where hepatitis A has been identified and for persons involved in a common source outbreak of hepatitis A.¹

Considering the effectiveness of these two methods of prevention, few Ohioans with acute hepatitis A reported receiving either the hepatitis A vaccine or IG during 2002-2007 (see Figure 17). Only 1 percent of Ohioans reported receiving the hepatitis A vaccine and 2 percent reported receiving IG. More than half of cases reporting a history of hepatitis A vaccination received the vaccine during the same year of symptom onset (data not shown). While the exact vaccination date is not known, it is possible these cases received the vaccine after exposure to HAV before the vaccine could provide adequate protection. Among Ohio cases reporting IG administration, 8 percent received IG after symptom onset, and 42 percent received IG 15-30 days before symptom onset (data not shown).



Practicing good personal hygiene is another way to prevent hepatitis A, especially through diligent hand washing after using the bathroom, changing a diaper and before preparing and eating foods.⁴ When traveling to endemic areas, either the vaccine or IG is the best method to prevent hepatitis A infection.⁷ Even if travelers have received the vaccine or IG prior to travel, it is still recommended for them to avoid exposure by consuming only beverages that have been boiled, commercially bottled, carbonated or chemically treated, avoiding ice cubes unless made with “safe” water and not eating raw/undercooked foods and salads.⁷ Improved sanitation is another way to prevent the spread of hepatitis A in the community.²

Conclusion

The notable surveillance trends in acute hepatitis incidence detected in Ohio over the past six years included:

- Ohio’s reported hospitalization rate for hepatitis A was higher than the national hospitalization rate and was continuing to increase over the years,
- The majority of positive IgM anti-HAV tests reported were in non-clinically compatible individuals,

- The lowest incidence occurred among infants and gradually increased in children and adolescents until it seemed to plateau in adults aged 20 years and older,
- Men had slightly higher rates of disease than women,
- Asians had the highest rates, while rates among blacks and whites were comparable,
- Hispanics had an incidence five times higher than non-Hispanics,
- The leading risk factors reported were recent international travel, personal contact with a known case and sexual activity among men who have sex with men,
- No notable seasonality was detected,
- Incidence was higher in counties surrounding urban centers, especially in northeast Ohio,
- Few cases were suspected of being involved in an outbreak and
- Because of the effectiveness of the hepatitis A vaccines and IG, very few cases reported receiving these preventive measures prior to becoming symptomatic with hepatitis A.

Hepatitis A was the most commonly reported type of hepatitis in the United States until 2004.¹ Routine hepatitis A vaccination and improvements in hygiene and sanitation have contributed to lowering the incidence in the country and the State of Ohio to all-time lows. Continued efforts toward widespread vaccination could continue to significantly reduce cases and eventually eradicate hepatitis A.¹ Despite the potential for eradication in the United States, hepatitis A is still very much endemic in the rest world, so continued vaccination and vigilance, especially among travelers to endemic countries, will be necessary.

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