VIRGINIA
EPIDEMIOLOGY BULLETIN

Robert B. Stroube, M.D., M.P.H., Health Commissioner
Carl W. Armstrong, M.D., State Epidemiologist
Christopher Novak, M.D., M.P.H., Editor
Vickie L. O'Dell, Layout Editor

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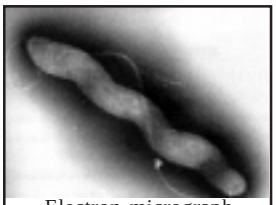
Campylobacter Infections: Clinical and Epidemiologic Perspectives

Introduction

In the United States, infections with *Campylobacter* are the most common bacterial diarrheal illness of humans, causing disease two to seven times more often than infections by *Salmonella*, *Shigella*, or *Escherichia coli* O157:H7.[1,2] This article reviews the clinical characteristics of these important pathogens, and examines the epidemiology of campylobacteriosis in Virginia.


Electron micrograph of *C. jejuni*

Clinical Symptoms

Asymptomatic *Campylobacter* infection can occur in approximately 25% of exposed adults. Among clinical cases, symptoms usually develop one to seven days after exposure to the organism, with the incubation period inversely related to the dose ingested.[3] The two types of illnesses associated with *Campylobacter* infections in humans are enteric (intestinal) and systemic (extraintestinal).[1]

Intestinal manifestations, most often caused by *C. jejuni*, typically result in diarrhea. Initially the diarrhea is watery, but stools may become grossly bloody as the illness progresses. Vomiting, abdominal cramping, fever (up to 40°C), headache, malaise and myalgias may also occur.[1] These symptoms are often indistinguishable from other inflammatory diarrheal illnesses caused by *Shigella*, *Salmonella*, *Yersinia* and *E. coli*.[1,2] Illness may occasionally be confused with early inflammatory bowel disease.[2] Since abdominal pain from campylobacteriosis can be severe, and may localize to the right lower abdomen, it can be misdiagnosed as appendicitis.[1]

Intestinal campylobacteriosis generally resolves after 7-10 days, but may last longer.[1,3] The majority of cases are self-limited, require no hospitalization, and lead to short-term immunity from re-infection. However, chronic or relapsing diarrheal illness has been described.[1] Patients may excrete the organism in their feces for an average of two to three weeks; three months after infection, continued excretion is rare.[1]

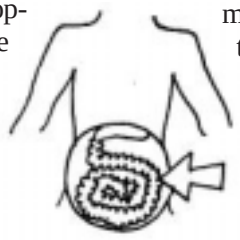
Rarely, long-term sequelae of *Campylobacter* infections occur.[4] Some people may develop a reactive arthritis, with pain and incapacitation lasting for months or becoming chronic.[5] *C. jejuni* is also recognized as the most common infectious agent associated with the development of Guillain-Barré syndrome (GBS). It is estimated that one in every 1,000 reported cases of *Campylobacter* infection leads to GBS, and that campylobacteriosis causes 20-50% of GBS cases in the United States.[1,6] Toxic megacolon may also be a complication of *Campylobacter* infection.[1]

Extraintestinal infections are uncommon, but may include bacteremia, meningitis, endocarditis, cholecystitis, septic arthritis, pancreatitis, cystitis and osteomyelitis.[2]

Pathogenesis

Members of the genus *Campylobacter* are small, motile, non-spore-forming, comma-shaped, Gram-negative rods.[1] Most (99%) cases of human disease result from *C. jejuni* infection.[4] However, infection by "atypical" *Campylobacter* species can occur, especially in immunocompromised persons.[1]

A very small number of *Campylobacter* organisms (as few as 500) can cause illness in humans (note: just one drop of chicken juice may contain 500 infectious organisms).[1,2] While the mechanism of pathenogenesis for *Campylobacter* is unknown, motility by polar flagella is thought to contribute to the organism's ability to colonize and infect the intestinal mucosa. Multiplication of organisms in the intestine, especially in the jejunum, ileum, and colon leads to cell damage and an inflammatory response; the organism appears to be locally invasive.[1,3] The inflammatory process may facilitate the translocation of the organism, leading to proliferation in the appendix, mesenteric lymph nodes, and gallbladder. In addition, *C. fetus* has a surface protein that prevents C3b binding and pro-



In This Issue:

tects these organisms from the normal bactericidal actions of serum.[1]

Diagnosis

Because of their characteristic microscopic morphology, *Campylobacter* spp. may be detected by direct Gram stain examination of stool. Red blood cells and neutrophils have been reported to be present in the stool of approximately 75% of patients with *Campylobacter* enteritis. The diagnosis is confirmed by isolating *Campylobacter* from a fecal specimen, or less frequently, from a blood culture. However, because of its growth requirements (a microaerophilic atmosphere), special media are needed to successfully culture *C. jejuni*.[1]

The preferred type of specimen submitted for enteric examination is feces collected in preservative (e.g., Cary-Blair) but other specimens such as rectal swabs and biopsies may be accepted by laboratories for testing. Preserved stool specimens should be shipped at room temperature (protected from extreme heat or cold) to a laboratory within 24 hours of collection or as quickly as possible, not to exceed 10 days. Storing specimens in deep, airtight containers minimizes exposure to oxygen and drying. Raw specimens, when requested, should be shipped refrigerated or frozen, taking care not to thaw and refreeze. If a stool specimen cannot be collected, a rectal swab placed in preservative should be obtained. Individual laboratories can provide detailed guidance on specimen handling procedures.[5]

In addition, polymerase chain reaction (PCR) techniques have been developed for rapid detection, culture confirmation and typing of *C. jejuni* strains. Although serology testing is not used for routine diagnosis, it has been used in epidemiologic investigations since it may be more sensitive than culture for the diagnosis of recent *C. jejuni* infection.[1]

Treatment

Although most people with *Campylobacter* infection recover fully, the disease can be severe, especially in those who are immunocompromised. Replacement of fluids and electrolytes is the mainstay of treatment.[2] Antimotility agents (e.g., loperamide) should not be used.[1] Antibiotics are occasionally used to treat severe cases, including those with:

- High fever;
- Bloody diarrhea;
- Excessive bowel movements (i.e., >8 stools per day);
- Worsening symptoms;
- Pregnancy;
- Immunocompromised state (e.g., HIV infection);
- Failure of symptoms to lessen; or,
- Persistence of symptoms for longer than 1 week.[1,2]

The decision to use antibiotics must be made carefully since the most common cause of bloody diarrhea is not *Campylobacter* but *E. coli* O157:H7 infection and the administration of antibiotics to children with *E. coli* O157:H7 infection may increase the risk of developing hemolytic uremic syndrome (HUS). Therefore, persons with bloody diarrhea at risk for HUS should generally not be treated with antibiotics unless *E. coli* O157:H7 infection has been ruled out.[2]

Antibiotics such as macrolides or fluoroquinolones may also be used to shorten the fecal shedding phase, which may be important for food handlers, children in day care and healthcare workers.

Extraintestinal infections require additional antimicrobial considerations, and toxic megacolon requires surgical consultation.

Epidemiology

National Patterns

Many animals, including swine, cattle, sheep, rodents, dogs and cats, carry *Campylobacter* in their intestines. Birds, particularly poultry, are commonly asymptomatic carriers. Although *C. jejuni* cannot withstand drying or freezing temperatures, the organism can survive in milk or other foods or in water kept at 4°C for several weeks.

Campylobacteriosis usually occurs in single, sporadic cases.[4] Transmission to humans often occurs through the ingestion of contaminated food or water, or by direct contact with fecal material from infected animals. Most cases (50-70%) are associated with handling raw poultry or eating raw or undercooked poultry meat.[1] Other sources have included raw milk and dairy products, raw clams, raw hamburger, untreated water and even municipal water supplies—these favor passage of the organism through the gastric acid barrier.[1,7] As a result, the risk of campylobacteriosis in rural populations may be increased five-fold because of the consumption of raw milk, and *Campylobacter* is an important cause of acute diarrheal illness suffered by travelers.[1,3] *Campylobacter* infections may also be sexually transmitted (e.g., among men who have sex with men).[3] However, transmission to healthcare workers from patients or specimens appears to be uncommon.[3] When outbreaks of *Campylobacter* infections have occurred, they typically have been associated with drinking unpasteurized milk or contaminated water.[3,4]

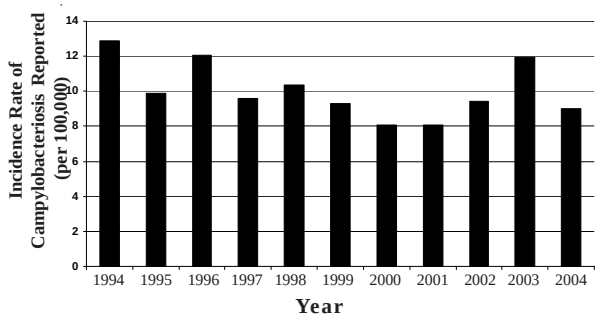
Although it is estimated that campylobacteriosis affects over two million persons every year in the US, the case fatality rate is low (approximately 120 deaths per year, mainly in infants, the elderly, and patients with underlying illnesses).[1]

C. jejuni infections occur year round; however, cases occur much more frequently in the summer months (May-July). Males appear to be more likely to be infected.[2] While infection can occur in all age groups, the age distribution of *Campylobacter* infections is unique among bacterial enteric pathogens in that two age-peaks occur: infants and young adults (20-29 years of age).[1,3]

Campylobacteriosis in Virginia

Campylobacteriosis is a reportable disease in Virginia (State Board of Health's *Regulations for Disease Reporting and Control:* 12 VAC 5-90-80). For this article, a review of *Campylobacter* infections reported to local health departments by healthcare providers and directors of healthcare facilities and laboratories in Virginia from 1994 through 2004 was conducted. A descriptive analysis of cases was performed using *Epi Info 6* and *Microsoft Excel (2002)*. Population estimates for each year in Virginia were obtained from the U.S. Census Bureau (note: since population

Figure 1. Incidence Rate of Campylobacteriosis Reported in Virginia, by Year, 1994 - 2004



data are not yet available for 2004, 2003 population data were used as a proxy).

From 1994-2004, a total of 7,636 cases of campylobacteriosis were reported in Virginia. Only one death was reported (in 2004) as a result of campylobacteriosis during the study period. Although 2003 had an increased number of cases compared to other recent years, the number of cases appears to be within the historical range (Figure 1) and no particular cause for an increase was identified (e.g., no large outbreaks were detected). The overall crude incidence rate of *Campylobacter* infections was 10.0/100,000 persons, a level that is lower than the national incidence of 15/100,000.[4] For 2002-2004, the majority of *Campylobacter* isolates (61.9%) were *C. jejuni*; however, in over 36% of isolates the species was unspecified (*C. jejuni* made up almost 97% of *Campylobacter* isolates for which the species was reported) (Figure 2). Among the five regions in Virginia, from 1994-2004 the Northwest region had the highest incidence (16.4/100,000 persons); the Eastern region had the lowest incidence (6.8/100,000 persons) (Figure 3).

Campylobacteriosis occurred predominantly in males (54.3%) in Virginia. For the 60.8% of cases where race was specified, the largest proportion of cases occurred among whites (86.2%); 9.4% of cases occurred among blacks, and 4.4% of cases occurred among other racial/ethnic groups. The greatest risk of disease occurred in persons less than one year of age (27.5/100,000 persons); in Virginia, persons 10-19 years of age had the lowest incidence of infection (5.5/100,000 persons) (Figure 4).

The occurrence of *Campylobacter* infection followed a seasonal trend. In Virginia over the eleven-year period, a peak in the numbers of cases reported occurred during the summer months (May through August) (Figure 5.)

Figure 2. *Campylobacter* Isolates in Virginia, 2002 - 2004

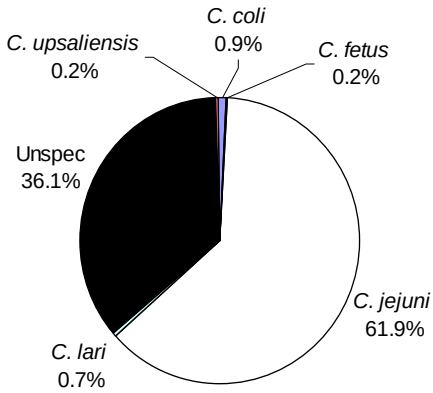
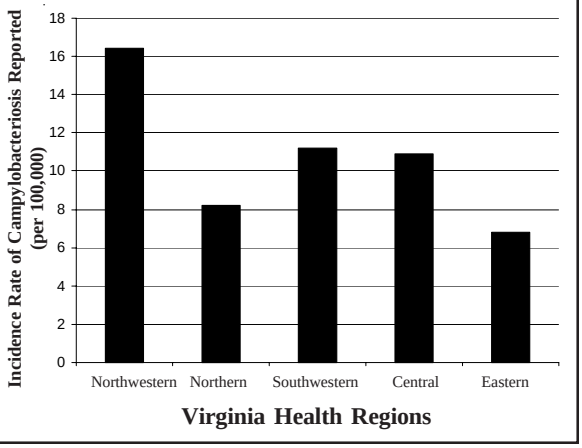


Figure 3. Incidence Rate of Campylobacterosis Reported in Virginia, by Region, 1994 - 2004



Outbreaks of campylobacteriosis in Virginia have been noted occasionally. In 2002 an outbreak of campylobacteriosis among attendees of a medical technologist's conference in Southwestern Virginia was reported. Seventeen of 41 (41.5%) conference attendees who were interviewed met the case definition (including two with positive cultures that had matching molecular genetic patterns). Two people required hospitalization, but there were no deaths. Although the mode of transmission was not confirmed for this outbreak, consumption of potato salad served at the conference was strongly associated (relative risk = 3.7) with developing illness.

Public Health Efforts

Prevention

Simple food handling practices can reduce the risk of *Campylobacter* infection. These include:

- treating raw meats as if they were contaminated;

Figure 4. Incidence Rate of Campylobacterosis Reported in Virginia, by Age Group, 1994 - 2004

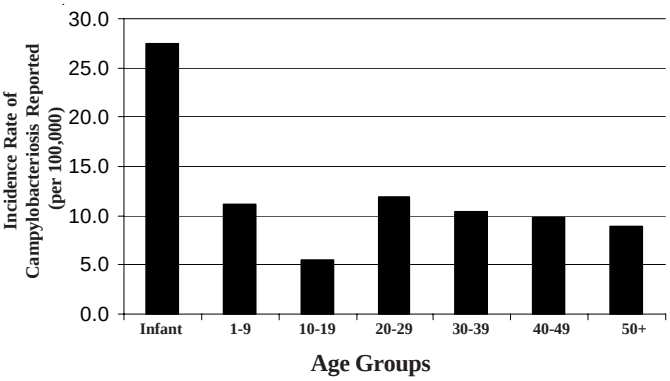
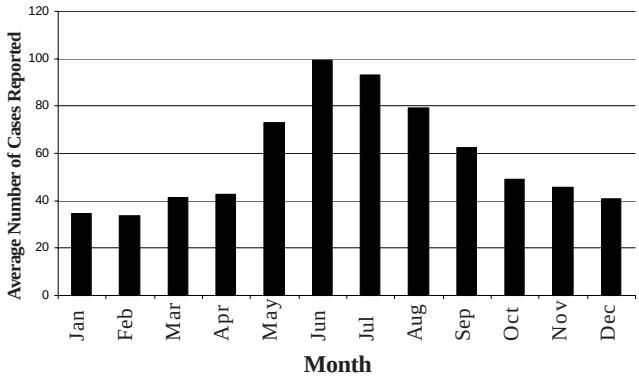


Figure 5. Average Number of Cases of Campylobacterosis Reported in Virginia, by Month, 1994 - 2004

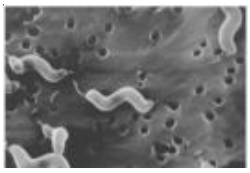


- appropriate cleaning, refrigeration and cooking of foods;
- avoiding the consumption of raw eggs, raw dairy products or untreated surface water;
- keeping sick animals (e.g., puppies and kittens) away from young children; and
- proper handwashing before and after food preparation, and after toileting, diapering children or handling animals.[4]

Prevention of many outbreaks of *C. jejuni* infection could be accomplished by avoiding the consumption of unpasteurized milk; this should be emphasized to pregnant women, the elderly, immunocompromised persons, or other persons in whom *C. jejuni* infection may have serious consequences. Travelers to developing countries and campers should be cautioned against drinking untreated water. However, the routine use of antibiotic prophylaxis to prevent *Campylobacter* infections is not recommended.[2] Finally, infected healthcare workers should not provide direct patient care or prepare food while they have diarrhea or are shedding *Campylobacter* organisms in the stool.[3]

Reporting Cases

The Virginia Board of Health's *Regulations for Disease Reporting and Control* (12 VAC 5-90-80 and 12 VAC 5-90-90) require healthcare providers who diagnose or suspect campylobacteriosis, and clinical laboratories that identify *Campylobacter*, to report their findings to their local health department. When an outbreak is suspected (i.e., two or more cases of *Campylobacter* enteritis having a common exposure within one incubation period of each other), cases must be reported to the local health department by the most rapid means available.



Conclusions

Campylobacter infections represent a significant preventable disease burden in the United States and in Virginia. Although the overall incidence in Virginia was found to be lower than the national level, this may have been a result of underreporting. This is suggested by the higher frequency of salmonellosis cases relative to campylobacteriosis cases reported in Virginia.[8] While there was some variability in the number of cases seen from year to year, the expected seasonal pattern was observed in Virginia, as well as the high risk among young children (the second peak, in adults, was not prominent). As expected, the majority of cases were due to *C. jejuni*; however 'atypical' species have been isolated occasionally.

Overall, consideration of *Campylobacter* as a cause for diarrheal illness is important. While individual cases can be managed clinically, and often resolve completely, timely reporting to the local health department of any suspected or known cases can improve the ability to detect, intervene, and reduce the impact of potential outbreaks early.

Submitted by: Blythe Allen-Dickerson, MD, MPH
Preventive Medicine Resident

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Welcome to the State Epidemiologist

Carl W. Armstrong, M.D.

On March 25, 2005, Dr. Carl W. Armstrong became the new Director of the Office of Epidemiology and State Epidemiologist for the Virginia Department of Health (VDH).

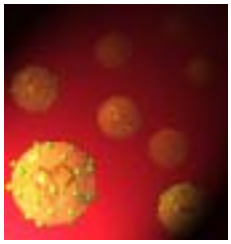
Dr. Armstrong is board certified in internal medicine, infectious diseases and preventive medicine. During his career, he has served as a commissioned officer in the Centers for Disease Control and Prevention's Epidemic Intelligence Service (EIS), as the Director of the Division of Health Hazards Control at VDH, as Health Director of the Piedmont Health District, and as Acting Deputy State Health Commissioner at VDH. Most recently he served as the Vice President and Senior Medical Advisor for the Virginia Hospital and Healthcare Association (VHHA), where his duties included administering the Health Resources and Services Administration (HRSA) Emergency Preparedness and Response grant. In all of these roles Dr. Armstrong is well known for his scholarship, his innovations, and his devotion to quality.

We welcome Dr. Armstrong, and look forward to his further contributions to improving the health of the people of Virginia.

In addition, special thanks go to Dr. Suzanne Jenkins (State Public Health Veterinarian and Director of the Division of Zoonotic and Environmental Epidemiology) for serving as the acting State Epidemiologist during the recruitment of Dr. Armstrong.

Submitted by: Dr. Jim Burns, Deputy State Health Commissioner

May: National Hepatitis Awareness Month
Update on Hepatitis C



The 2002 federal designation of May as National Hepatitis Awareness Month provides an opportunity to address the Virginia Department of Health's (VDH) efforts in combating hepatitis C and to highlight information on hepatitis C that may be of use to healthcare providers.

Hepatitis C

Hepatitis C is a liver infection caused by the hepatitis C virus (HCV). A bloodborne virus, HCV is transmitted when blood or body fluids from an infected person enters the body of a susceptible person; injection drug use is the most common mode of transmission. While there has been remarkable progress in diagnosing and treating HCV since the virus was first identified in 1989, an estimated 25,000 new HCV infections occur each year in the United States. Currently, there are approximately 3 million people in the United States living with chronic HCV, and estimates suggest that around 90,000 of these are Virginia residents. We also know that 10-20% of people with chronic HCV will progress to cirrhosis of the liver, and that 1-5% will develop liver cancer. As a result, HCV is estimated to cause 8,000-10,000 deaths each year in the United States.

Those infected with HCV produce detectable HCV antibodies, usually by twelve weeks after infection. These antibodies remain detectable throughout life. There is one exception to this**:** 6% of HIV/HCV co-infected patients never produce an HCV antibody.

HCV Clearance

Unlike HIV, where all who are infected by that virus become chronically infected, approximately 15-30% of HCV-infected people clear the virus spontaneously and resolve the infection completely. If resolution is to occur, however, it will be by the sixth month after initial infection. In addition, unlike hepatitis B (HBV) and hepatitis A (HAV) where circulating antibodies offer lifelong immunity from subsequent infection, those who clear HCV can be re-infected.

Higher rates of spontaneous clearance of HCV have been seen among five groups**:** females, whites, those of younger age, those *with* evident symptoms during acute infection, and those with *lower* viral replication during acute infection. Research has shown that spontaneous HCV resolution may be more likely when the immune system is able to mount a vigorous attack at the time of infection (which may be one reason for the association between clearance and *more* severe symptoms during the acute phase).

HCV Chronic Infection

If spontaneous HCV resolution **does not** occur by the sixth month, the patient will very likely become a chronic carrier of the hepatitis C virus. So, while results of the enzyme immunosorbent assay (EIA) and recombinant immunoblot assay (RIBA) tests are very useful in detecting and confirming the presence of HCV antibody (exposure), only the polymerase chain reaction (PCR) test can confirm the actual presence of the HCV virus.

HCV and Virginia

Given that as early as 2010 the HCV-attributable death rate in the United States could quadruple to 40,000/year, hepatitis C and its complications will likely remain an important public health issue. Accordingly, VDH's Division of HIV, STD, and Pharmacy Services is augmenting its efforts to proactively monitor and prevent HCV infection in Virginia over the coming years. This will include**:**

- Collaborating with public and private entities in order to provide regional HCV trainings for healthcare professionals within Virginia;
- Developing a new hepatitis webpage with updated links to information about hepatitis A, hepatitis B and hepatitis C;
- Establishing a web page referral link to specialists who treat HCV in Virginia; and
- Promoting a "Know Your Status" educational campaign on HCV in Virginia designed to apprise citizens of their HCV status and thereby slow the rate of new HCV infections.

Additionally, VDH will be incorporating HCV data collection into the Centers for Disease Control and Prevention's (CDC) National Electronic Disease Surveillance System (NEDSS). This tool will allow for more precise estimates of the impact of HCV on Virginia's population.

For further information on National Hepatitis Month, please see www.cdc.gov/hepatitis (Available May 1, 2005).

Division of HIV, STD, and Pharmacy Services Expands Incidence/ Resistance Testing Program

The Division has been collaborating with the Centers for Disease Control and Prevention to develop procedures for obtaining specimens to measure HIV incidence (i.e., new HIV infection) and monitor the transmission of antiretroviral HIV drug resistant virus in treatment naïve individuals in Virginia. A new HIV-1 incidence test (BED EIA) was recently approved by the Food and Drug Administration for the purposes of public health surveillance and will make the implementation of these programs much easier. Collaboration with local health departments has begun, with some having already started collecting data.

Flu Corner

Influenza – U.S. Patterns

Influenza activity in the United States peaked in early February and continued to decline as of the week of April 30, 2005; no states reported widespread influenza activity, one state reported regional influenza activity, two states reported local activity, 32 states (including Virginia) reported sporadic activity, and thirteen states reported no influenza activity. During the week of April 30, 2005, U.S. World Health Organization (WHO) and National Respiratory and Enteric Virus Surveillance System (NREVSS) laboratories in the U.S. reported testing 1,176 specimens for influenza viruses:

- 42 (3.6%) were positive
  - o Of these, nine were influenza A(H3N2) viruses;
  - o Nine were influenza A viruses that were not subtyped; and,
  - o 24 were influenza B viruses.[1]

Nationally, influenza B viruses became more frequently reported than influenza A viruses during the week ending March 26 and have predominated each week since then.

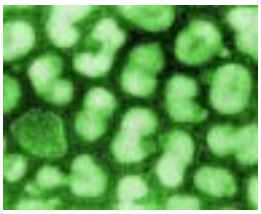
Since October 3, WHO and NREVSS laboratories in the United States have tested a total of 140,682 specimens for influenza viruses and 22,074 (15.7%) were positive. Among the 22,074 influenza viruses:

- 16,887 (76.5%) were influenza A viruses;
  - o Of 5,554 (32.9%) subtyped;
    - ■ 5,538 (99.7%) were influenza A(H3N2);
    - ■ 16 (0.3%) were influenza A(H1) viruses.
- 5,187 (23.5%) were influenza B viruses.[1]

For the week of April 30, 2005, the proportion of patient visits to sentinel providers for influenza-like illness (ILI) was below the national baseline. In addition, 7.2% of all deaths reported by the vital statistics offices of 122 U.S. cities were attributed to pneumonia or influenza. This percentage is below the epidemic threshold of 7.7% for the week. There have been 31 influenza-associated pediatric deaths reported to the CDC this season.[1]

Influenza in Virginia

As of April 30, 2005, influenza activity in Virginia had declined to sporadic (laboratory confirmed cases but no outbreaks of ILI detected and no increase in ILI).[3] As of April 28, 2005, the Division of Consolidated Laboratory Services (DCLS) had reported 148 confirmed cases of influenza (131 type A and 17 type B) by Direct Fluorescent Antibody (DFA) and/or culture.



Avian Influenza

Influenza A(H5N1) is a subtype of the type A influenza virus. Wild birds are the natural hosts of the virus, hence, the name "avian influenza" or "bird flu." The H5N1 is very contagious among birds and can be deadly to them. Infected birds shed the virus in saliva, nasal secretions, and feces and the virus spreads among susceptible birds through contact with contaminated excretions. Outbreaks of highly pathogenic H5N1 occurred among poultry in eight countries in Asia during 2003-4. At that time, more than 100 million birds either died from the disease or were culled. In June 2004, new lethal outbreaks of H5N1 infections among poultry were reported by several countries in Asia: Cambodia, China, Indonesia, Malaysia, Thailand and Vietnam. In March 2005, the Democratic People's Republic of Korea (North Korea) officially reported the country's first outbreak of avian influenza in poultry.[4]

The H5N1 virus does not typically infect humans. From January 28, 2004 to April 14, 2005, 88 human cases of avian influenza A(H5N1) have been reported in Vietnam, Thailand, and Cambodia resulting in 51 deaths. Most of these infections have resulted from contact with infected poultry or contaminated surfaces.[4] However, since the H5N1 epizootic in Asia is not expected to diminish significantly in the short term, it is likely that human H5N1 infections will continue to occur.

Although no sustained human-to-human transmission of the H5N1 virus has been identified, and no evidence for genetic reassortment between human and avian influenza virus genes has been found, avian influenza poses an important public health threat. Since there is little preexisting natural immunity to H5N1 infection in humans, if the virus acquires the capacity for efficient and sustained human-to-human transmission an influenza pandemic could result. In addition, genetic sequencing of H5N1 samples shows that the virus possesses resistance to the antiviral medications amantadine and rimantadine. Finally, the availability of an influenza A(H5N1) vaccine is some time off.[4] These factors, combined with the highly pathogenic nature of the virus, could lead to significant human morbidity and mortality.

For the Centers for Disease Control and Prevention's surveillance, diagnostic evaluation, and infection control precautions for avian influenza A (H5N1), visit www.cdc.gov/flu/avian/professional/han081304.htm.

Novel Influenza Viruses Added to List of Federally Quarantinable Diseases

On April 1, 2005, an Amendment to Presidential Executive Order 13295 made influenza caused by novel or re-emergent influenza viruses that have the potential to cause a pandemic a Federally Quarantinable Communicable Disease.[5] The other diseases on the list are: cholera, diphtheria, infectious TB, plague, smallpox, yellow fever, viral hemorrhagic fevers, and Severe Acute Respiratory Syndrome (SARS).

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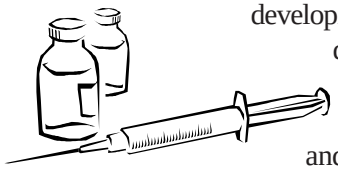
5. Executive Order: Amendment to E.O. 13295 www.whitehouse.gov/news/releases/2005/04/20050401-6.html. Accessed: April 19, 2005.

National Infant Immunization Week (NIIW), April 24 - 30, 2005, is an annual observance established 11 years ago by the Department of Health and Human Services (DHHS) and the Centers for Disease Control and Prevention (CDC) to remind parents, healthcare professionals, and the public that children deserve a healthy start to life by immunizing them against vaccine-preventable diseases.

This year's theme is, "Love Them. Protect Them. Immunize Them." For the second year NIIW is partnering with Vaccination Week in the Americas (VWA) to promote infant immunization in all countries of the Americas. More than 500 events will take place nationwide to celebrate and promote this important issue. Details about NIIW are available at www.cdc.gov/nip/events/niiw/2005/05default.htm.

Vaccines are one of history's most successful and cost effective public health tools for preventing serious disease and death. Diseases that were once commonplace, such as polio, measles, mumps, whooping cough, diphtheria, and rubella, are now only distant memories for most Americans. Today, there are few reminders of the suffering, disabilities, and premature deaths caused by diseases that are now preventable with vaccines.

Immunization coverage among children in the United States is the highest ever recorded for most vaccines. High immunization coverage translates into record or near record low levels of vaccine-preventable disease. In Virginia, 79.8% (±5.7%) of children 19 to 35 months of age are up-to-date on their immunizations with four or more doses of diphtheria, tetanus and acellular pertussis (DTaP) vaccine, three or more doses of poliovirus vaccine, one or more doses of measles, mumps and rubella (MMR) vaccine, three or more doses of haemophilus influenzae type b (Hib) vaccine, three or more doses of hepatitis b (HepB) vaccine, and one or more doses of varicella vaccine. In comparison, nationwide 72.5% (±1.0%) of children 19 to 35 months of age are up-to-date on their immunizations.



Our success also means that many parents don't understand the importance of childhood immunization and what diseases can be prevented. Most of today's parents have never seen these diseases and the suffering that they can cause and, therefore, are less concerned about the need for immunization compared to other parental priorities. However, these diseases are not diseases of the past. They are still circulating in many parts of the world and are only an airplane ride away.

Parents and healthcare providers must work together to ensure that all children are fully immunized. Physicians are encouraged to talk with parents about the importance of immunization and be willing to answer their questions about vaccine risks and benefits. A few key messages are listed below to assist healthcare providers as they converse with parents.

- Vaccines are among the most successful and cost-effective public health tools available for preventing disease and death. They not only help protect vaccinated individuals from developing potentially serious diseases, they also help protect families and communities by preventing and reducing the spread of infectious agents.
- Infants and young children are particularly vulnerable to infectious diseases; that is why it is critical that they are protected through immunization.
- Immunizations are extremely safe thanks to advancements in medical research and ongoing review by doctors, researchers, and public health officials. Children are far more likely to be harmed by serious infectious diseases than by immunization.

Providers are also encouraged to use every visit as an opportunity to review immunization records and vaccinate when appropriate.

Cases of Selected Notifiable Diseases Reported in Virginia*

Disease	Total Cases Reported, March 2005: State	Regions: NW	N	SW	C	E	Total Cases Reported Statewide, January - March: This Year	Last Year	5 Yr Avg
AIDS	63	12	16	9	12	14	170	108	166
Campylobacteriosis	33	4	14	9	1	5	68	87	74
E. coli O157:H7	1	0	0	0	0	1	1	0	4
Giardiasis	48	8	8	9	14	9	110	69	71
Gonorrhea	793	62	62	111	205	353	2,220	2,275	2,319
Hepatitis, Viral									
A	12	2	7	1	0	2	20	15	28
B, acute	11	1	1	5	1	3	44	35	35
C, acute	6	1	1	2	0	2	6	8	2
HIV Infection	79	7	21	11	21	19	177	187	192
Lead in Children†	23	3	4	5	8	3	67	104	102
Legionellosis	1	0	0	1	0	0	4	4	3
Lyme Disease	12	0	10	0	1	1	14	4	4
Measles	0	0	0	0	0	0	0	0	0
Meningococcal Infection	3	0	0	1	1	1	5	2	10
Mumps	0	0	0	0	0	0	0	1	1
Pertussis	16	1	6	5	2	2	43	28	20
Rabies in Animals	34	8	11	6	3	6	80	111	113
Rocky Mountain Spotted Fever	0	0	0	0	0	0	0	0	0
Rubella	0	0	0	0	0	0	0	0	0
Salmonellosis	75	7	31	9	12	16	147	125	122
Shigellosis	9	0	8	0	1	0	19	22	77
Syphilis, Early§	15	0	9	1	0	5	39	23	54
Tuberculosis	27	1	14	3	3	6	49	30	44

Localities Reporting Animal Rabies This Month: Accomack 1 raccoon; Augusta 1 raccoon; Campbell 1 raccoon; Carroll 1 raccoon; Chesterfield 1 raccoon; Culpeper 1 skunk; Fairfax 1 cat, 3 foxes, 5 raccoons; Floyd 1 raccoon; Halifax 1 raccoon; Henry 1 cat; Isle of Wight 1 skunk; Loudoun 1 raccoon; New Kent 1 raccoon; Northampton 1 raccoon; Patrick 1 raccoon; Prince William 1 raccoon; Rockbridge 1 skunk; Rockingham 2 raccoons, 1 skunk; Shenandoah 2 skunks; Tazewell 1 raccoon; Virginia Beach 1 cat, 2 raccoons.

Toxic Substance-related Illnesses: Adult Lead Exposure 2; Pneumoconiosis 5.

*Data for 2005 are provisional. †Elevated blood lead levels ≥10µg/dL. §Includes primary, secondary, and early latent.

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Office of Epidemiology
P.O. Box 2448
Richmond, Virginia 23218
http://www.vdh.virginia.gov
Telephone: (804) 864-8141

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