

{Emergence of New, More Virulent Strain of *Clostridium Difficile*-Associated Diarrhea in Low Risk Populations}

Background

Clostridium difficile is the leading cause of diarrhea in health-care settings, frequently resulting in increased hospital stays and costs. It is an anaerobic, toxin and spore-forming bacillus commonly found in the environment. Outbreaks of *Clostridium difficile*-associated diarrhea (CDAD) have occurred in healthcare and other institutional settings where many patients have risk factors of broad-spectrum antibiotic use and multiple co-morbidities. CDAD typically presents with diarrhea, abdominal pain and fever and is usually confirmed by a positive *C. difficile* lab result or pseudomembranous colitis (PMC) seen on endoscopy.

Emergence of a New Clinical Picture

Since 2000, reports of unusually severe CDAD have occurred in the United Kingdom and North America in patients not typically considered at risk for CDAD (young, healthy, without antibiotic use). The clinical course is pronounced by extreme leukocytosis, azotemia, PMC, toxic megacolon, and sepsis, resulting in higher rates of colectomy, intensive care and death. Analysis of the recent presentation revealed new *C. difficile* strains containing binary toxins thought to cause the increased virulence. Specialized toxin testing can be performed on *C. difficile* isolates.

The Oklahoma State Department of Health (OSDH) has investigated five reports of unusually severe CDAD and found the usual risk factors missing in three. Two of the cases were postpartum women, both of whom died. Tissues from one of these patients were obtained for further testing at the Centers for Disease Control and Prevention (CDC). The OSDH is requesting reports of severe *C. difficile* without traditional risk factors so tests can be conducted to determine if one of the highly virulent strains has reached our state.

Treatment

Treatment guidelines for *C. difficile* infections are identical for low virulent and high virulent infections and were published by the Society for Healthcare Epidemiology of America (SHEA) in 1995.¹ Recommendations are to withdraw the inciting antibiotics, avoid antimotility medications, and treat with oral metronidazole for 10-14 days. Oral vancomycin may be used but should be reserved for metronidazole failures or more severe disease.² Patience is needed during the first week of treatment as symptoms resolve slowly, although some improvement should be seen in 24 to 48 hours. Switching treatment is advised only if the patient worsens (i.e., develops ileus, continued leukocytosis, etc.). Intravenous or intracolonic vancomycin should be considered for severe disease.

Recurrence of CDAD is likely due to the persistence of spores, which are not killed by antibiotics. To allow return of normal bowel flora, tapering of antibiotics over six weeks, or adding other drugs such as cholestyramine, rifampin, and probiotics have been suggested,³ however, the efficacy is unknown.

Infection Control

Transmission is fecal-oral and occurs in the hospital via fomites or the contaminated hands of healthcare workers. Consistent meticulous adherence to infection control is crucial to prevent transmission. Guidelines include hand hygiene with soap and water, appropriate use of personal protective equipment, contact isolation, and proper patient placement. Last and most important is staff education to reinforce these concepts.

C. difficile spores survive in the environment up to six months, so measures such as use of disposable or dedicated equipment and sufficient cleaning and disinfection are vital. 1:10 bleach solutions are recommended as no approved Environmental Protection Agency product inactivates *C. difficile* spores.

Prevention

Prevention hinges on the judicious use of antibiotics and a high suspicion of CDAD. Use of formularies and policies to limit antimicrobial use is recommended. Patient education may be the most difficult hurdle in controlling antibiotic use in outpatient settings.

A *C. difficile* vaccine is in development with Phase I trials showing a ≥ 10 -fold increase in IgG levels. Further studies are needed to prove the efficacy of the vaccine in preventing infection.

*prepared by Becky Coffman RN, MPH, CIC, Epidemiologist, CDD

Additional resources available upon request

¹ Gerding DN et al. *Clostridium difficile*-associated diarrhea and colitis. Infect Control Hosp Epidemiol 1995;16(8):459-477. http://www.shea-online.org/Assets/files/position_papers/Cldiff95.PDF

² Centers for Disease Control and Prevention. Recommendations for preventing the spread of vancomycin resistance: recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC). MMWR 1995;44(No. RR-12) www.cdc.gov/mmwr/rr/vol44/rr12.htm

³ Sunenshine RH and McDonald LC. *Clostridium difficile*-associated disease: New challenges from an established pathogen. Cleveland Clinic Journal of Medicine, Vol.73, No.2., February 2006, page 187-197. http://www.ccm.org/PDFFILES/Sunenshine2_06.pdf

{A Cluster of TB Cases Identified Through Social Network Analysis-Pottawatomie County, August 2004}

Tuberculosis (TB), an airborne infectious respiratory disease, is one of the most deadly and common major infectious diseases globally, infecting two billion people, or approximately one-third of the world's population. The incidence of TB in the United States has decreased by 44% during the years of 1993-2003. During this same time period, the incidence of disease has declined by 21% in Oklahoma. Even with this decline, TB continues to pose a substantial social, public health and economic burden.¹

As the incidence of TB drops, the risk for delayed diagnosis increases as practitioners have less experience in diagnosis and treating TB. The declining incidence of TB has changed the disease's epidemiology, including high morbidity rates in isolated pockets of the population (i.e., homeless, substance abusers, and recent immigrants).

When someone with TB is identified, it is important to find all persons potentially exposed to active TB disease. The traditional method of finding persons exposed to TB is the concentric circle approach. The person with active TB disease identifies all his/her contacts and the investigator divides the patient's environment into high, medium, and low priority circles (i.e., household, work/school, social/leisure) to prioritize testing of contacts for TB.²

Another approach to TB contact tracing is social network analysis. Social network analysis is the mapping of relationships between individuals and places. Social networking is beneficial in situations where the index case may only know their contacts by street names or are reluctant to name contacts in cases of homelessness, substance abuse or illegal activity.³

Another tool in contact investigations is TB genotyping, a method to ascertain the specific genetic code of the organism. Identifying specimens with the same genotype may help us link TB cases that were missed during traditional contact investigations. All three of these public health tools can identify contacts exposed to TB and prevent further transmission.

In August 2004, while delivering medication to an active TB case, a public health nurse encountered another known TB case at the residence, a local gathering place for recreational drug use. In the traditional concentric circle approach, neither case had named the other as a contact. At this same time, a local physician referred another case to the county health department who admitted to frequently going to the same house. This person did not name either of the two previous cases as contacts. In response to this information the OSDH and the local public health nurse investigated this cluster of TB cases further.

The traditional method of contact investigation using the concentric circle approach was used with all cases. After identifying the house as a common location among the cases, social network analysis was used to identify additional contacts connected to the house. Sputum specimens positive for TB were sent for genotyping.

In our investigation, a laboratory confirmed case of TB was defined as a person with isolation of *Mycobacterium tuberculosis* in sputum, body tissue or fluid culture. A clinical case of tuberculosis was defined as a person with a positive tuberculin skin test and other signs and symptoms compatible with tuberculosis (i.e., an abnormal chest x-ray that improves with treatment specific for TB). Latent Tuberculosis infection (LTBI) was defined as a person with a reactive tuberculin skin test and no clinical, bacteriologic, or radiographic evidence of active TB disease.¹

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The accompanying graphic represents the relationships or social networks of both laboratory confirmed and clinical cases identified in this outbreak. Overall we identified 9 laboratory confirmed cases and 6 clinical cases. Investigation of these 15 cases revealed a total of 28 persons with LTBI. Of the laboratory cases that were able to be genotyped, 88% (n=7) were identified as the same genotype; 1 laboratory confirmed case had a different genotype.

Of cases, 60% (n=9) were male and 40% (n=6) were female. Sixty percent (n=9) identified themselves as African-American, 33% (n=5) as Native American and one person identified herself as both Native American and African-American. Fifty-three percent (n=8) were 18 or older and classified as adults; 47% (n=7) were classified as children. Of adults, 88% (n=7) had a history of incarceration and all were incarcerated at the same county jail, although at different times. One hundred percent of adults admitted to a history of drug abuse. The concentric circle approach identified 7 cases and 21 persons with LTBI. Social network analysis yielded an additional 7 cases and 7 persons with LTBI. One case was identified through genotyping data and is suspected to have contact to the common house.

Social network analysis was crucial to the understanding and identification of the cluster. A traditional concentric circle contact investigation by itself would have yielded an incomplete understanding of the cluster epidemiology and demonstrates that earlier identification of the social network may have prevented transmission of TB infection and disease development.

Recommendations

- Include TB in a differential diagnosis for respiratory disease, especially for high risk populations (i.e., history of incarceration, drug abusers, homeless, and foreign-born).
- Consider screening (tuberculin skin test) high risk populations.
- Consider social network analysis in addition to the concentric circle approach when investigating TB cases.
- Continue to use genotyping data to connect active TB cases in a community.

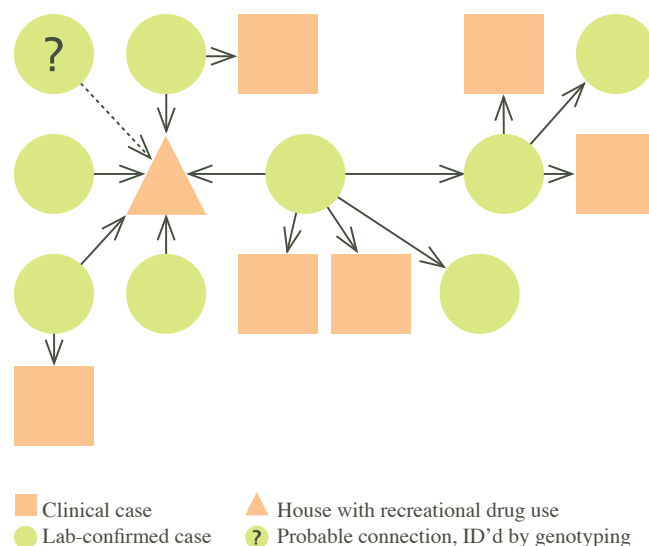
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¹Centers for Disease Control and Prevention. Controlling Tuberculosis in the United States. MMWR 2005;54(No.RR-12)

²Centers for Disease Control and Prevention. Guidelines for the investigation of contacts of person with infectious tuberculosis. MMWR 2005;54:1-47

³Klondahl AS, et al. Networks and tuberculosis: an undetected community outbreak involving public places. Soc Sci & Med 2001;52:681-694

Social Network of Cluster of TB Cases-Pottawatomie County, August 2004



{Summary of West Nile Virus Activity, 2005}

2005 marked the fourth season of West Nile virus transmission in Oklahoma and the seventh year of national tracking since its identification in the New York City area in 1999. After making a steady and progressive continental advance, West Nile (WN) virus infections in animals or people have now been documented in all 48 contiguous states, Canada and Mexico. In 2005, a total of 2,997 human cases of WN disease and 113 associated deaths were reported to the Centers for Disease Control and Prevention (CDC) by 42 states representing a 16% increase from 2004. Nationally, most of the WN activity was seen in California, the southwest, and the central mountain states with the highest number of cases being reported from California (880), Illinois (252), and South Dakota (229).

Consistent with the national trend, Oklahoma also had an increase of WN activity compared to 2004. The state's 2005 incidence of human WN virus disease was 31 cases of WN Fever or WN neuroinvasive disease resulting in a 42% increase over the previous season. These cases were distributed across 23 counties with geographic clustering in the panhandle, north central and south central parts of the state. Counties with the highest incidence rates per 100,000 population included Grant (38.9), Beaver (34.2), Woods (11.0), Noble (8.76), and Haskell (8.48).

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Symptom onsets ranged from July 10 to November 7, 2005. Sixty-one percent of the case patients were male; median age of all patients was 48.5 years (range 17-82). Seventeen of the cases (54.8%) had WN meningoencephalitis or meningitis. There was one WN-associated death in a Beaver County resident following complications from primary encephalitis for an overall case fatality rate of 3.2%. Prominent symptoms reported for all cases included fever (93.5%), muscle weakness (83.3%), vomiting (46.7%), photophobia (44%), rash (41.9%), vertigo (40%), and confusion (36.7%). The number of patients that exhibited a rash (erythematous maculopapular) was disproportionately high compared to 9.5% in 2004. Other common complaints of case patients included a stiff neck, disorientation, tremors, altered mental status, and anorexia. Tracking of WN cases for chronic disabilities is not standardized or available for all cases, but 3 case patients were reported to suffer hearing loss. Thirty-three percent of case patients (5/15) were treated in rehabilitation facilities after an initial hospitalization. Most cases (15/27, 55.6%) reported exposure to mosquitoes within two weeks of symptom onset. Two cases had probable travel exposure in Louisiana; one while performing hurricane relief work.

The 2005 WN season proved to be epidemiologically distinctive in comparison to previous years regarding clinical presentation of rash, seasonality of risk, and blood donor surveillance findings. From 2002 through 2004, human WN cases were first observed in July with peak activity during mid-August. In 2005, the first human case of WN disease was reported in mid-July as expected, but the epidemic curve was prolonged, escalated in August and then peaked in late September with the last case reported during the second week of November (refer to graph on page 5). The prolonged season with peak activity observed five to six weeks later than previous seasons may be attributed to the relatively hot and dry fall that Oklahoma experienced. These ecological factors are favorable to increasing populations of *Culex spp.* mosquitoes.

Another unique feature to the 2005 season was the high proportion of viremic blood donors that developed symptoms of WN disease. Universal blood donor screening for WN viremia became mandatory in July 2003 after transfusion-associated transmission was documented in several cases in 2002. Blood collection facilities perform a nucleic acid amplification test (NAT). All donations from NAT-positive donors are excluded and follow-up testing and interviewing occurs to document WN infection and to determine if WN-associated symptoms develop. In 2005, 17 Oklahoma blood donors tested NAT-positive for WN viremia by routine blood screening.

Strikingly, 53% of those individuals developed symptoms of WN disease in 2005 compared to less than 15% in 2003 and 2004. While human WN activity was not wholly consistent with past seasons, equine activity trends were consistent with surveillance findings in 2004. The first 2005 equine WN case was reported from Comanche County and had illness onset on June 12. Peak activity occurred in late September and early October. Thirty-two horses were diagnosed with WN disease in the state by laboratory confirmation in both 2004 and 2005.

Prevention

WN virus will remain a consistent mosquito-borne threat to Oklahomans in the years to come. Although there is some seasonal variation, four years of surveillance activities have consistently shown that the WN season in Oklahoma begins in May and ends in November with July through October being the period of greatest risk for human illness. Horse owners should be encouraged to include WN vaccination in their routine equine herd health programs. In the absence of a licensed vaccine for human use, personal prevention messages must continue urging routine use of insect repellents that contain an effective ingredient such as DEET, picaridin, or oil of lemon eucalyptus while participating in outdoor activities. The Oklahoma State Department of Health (OSDH) continues to support community mosquito control programs with supplemental funding from the CDC Epidemiologic and Laboratory Capacity grant.

2006 Surveillance and Laboratory Testing

Throughout the mosquito season, Oklahoma physicians are urged to consider the diagnosis of WN in patients with compatible clinical presentations for either WN Fever or WN neuroinvasive disease and to submit specimens to the OSDH Public Health Laboratory (PHL) for testing. Recent medical studies have shown that even patients who have WN Fever may experience lasting and prolonged sequelae. In a study of long-term functional outcomes of WN Fever cases, many patients commonly reported persistent problems such as fatigue (96%), headaches (71%), and concentration problems (53%).¹

In 2006, the OSDH will continue to offer human WN testing free of charge at the PHL. The PHL performs IgM-capture ELISA, the most sensitive screening test for acute infections, on cerebrospinal fluid (CSF) and serum. Polymerase chain reaction (PCR) testing is also available for CSF. Physicians who submit a CSF specimen should also submit a serum

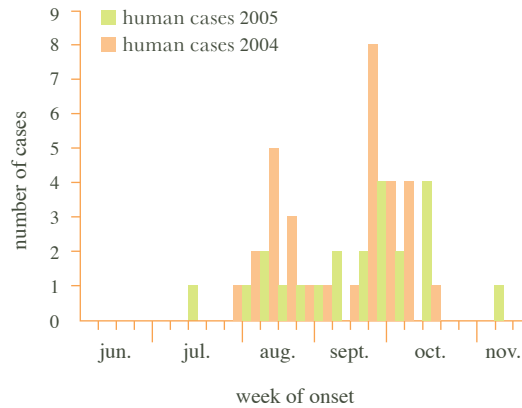
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specimen (for IgM ELISA) from the case patient due to the lower sensitivity of the PCR test. WNV resources and testing information can be accessed on-line at <<www.health.ok.gov/program/cdd/wnv/index.html>>

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¹Watson et al. *Annals of Internal Medicine*, 141:2004

2004 and 2005 Comparison of Human Cases of West Nile Virus by Week of Onset, Oklahoma



Recent Public Health Publication of Interest

A New Report on Tickborne Rickettsial Diseases: A must read for healthcare providers in Oklahoma

It is once again that time of year when the quantity of the tick vectors of tickborne rickettsial diseases (TBRD), including Rocky Mountain spotted fever (RMSF), Ehrlichiosis, and Anaplasmosis are on the rise in Oklahoma. Inevitably, despite the availability of low cost and effective antibiotic therapy, TBRD will continue to cause severe illness and death in otherwise healthy adults and children. The early signs and symptoms of these illnesses are typically nonspecific, and often mimic a benign viral illness, making early diagnosis and treatment a challenge for clinicians. Approximately 60%-75% of patients with RMSF, in areas with a heightened awareness to the disease, receive an alternate diagnosis on their first visit for medical care.

This is significant as antibiotic therapy is most effective early in the clinical course of disease.

The Oklahoma State Department of Health would like to highlight and recommend a recently published report on TBRD. The report titled "Diagnosis and Management of Tickborne Rickettsial Diseases: Rocky Mountain Spotted Fever, Ehrlichiosis, and Anaplasmosis---United States: A Practical Guide for Physicians and Other Health-Care and Public Health Professionals," provides practical information on the diagnosis, treatment, and reporting of TBRD. The goal of the report is to present a framework to assist clinicians in recognizing the symptoms of

TBRD, obtaining appropriate diagnostic tests, and initiating prompt and effective treatment.

The report is published in the CDC's *Morbidity and Mortality Weekly Report* and is available online at:

<http://www.cdc.gov/mmwr/pdf/rr/rr5504.pdf>

A link to the report and additional information regarding tickborne illnesses in Oklahoma are also available online at:

www.health.state.ok.us/program/cdd/tbi.html

ACIP Guidelines for MMR Vaccination

Two doses of MMR vaccine for all children.

-The first dose should be given at 12 months of age in the combination vaccine MMRV (measles, mumps, rubella and varicella).

-The second dose should be given between 4 and 6 years of age in the combination vaccine MMR (measles, mumps, and rubella).

Two doses of MMR vaccine for all health care workers born during or after 1957, unless they have:

-Documentation of physician-diagnosed measles and mumps disease; or

-Laboratory evidence of measles, mumps, and/or rubella immunity.

-Two doses of MMR vaccine for adults born during or after 1957 traveling outside the U.S. In outbreak settings, a second dose of MMR is recommended for adults born during or after 1957.

-Two doses of MMR for students attending post-high school educational institutions.

¹Notifiable Diseases. MMWR 2006;55(14):406.

²Mumps Epidemic-United Kingdom, 2004-2005. MMWR 2006;55:173-5.

³Mumps Outbreak at a Summer Camp-New, 2005. MMWR 2006;55:175-7.

⁴Measles, 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(RR-8):1-57.

{Multi-state Outbreak of Mumps}

Since the mumps vaccine was licensed in 1967, the number of reported cases in the United States has decreased more than 99% from 152,209 cases in 1968 to 298 in 2005.¹ Despite the decrease in mumps incidence in the United States, outbreaks of mumps still occur. Furthermore, mumps is still a common disease in many parts of the world. This article summarizes two recent mumps outbreaks, the 2004-2005 United Kingdom (UK) and 2006 U.S. outbreak, and recommendations for prevention and control.

During 2004-2005, the UK experienced a nationwide epidemic of 72,757 cases (16,367 in 2004 and 56,390 in 2005); this was a dramatic increase from the 1,556 cases reported in 2003. During 2004, 8,128 (49.7%) cases were laboratory confirmed. Among confirmed cases, the majority occurred among persons aged 15 to 24 years. Only 3.3% had a history of 2 doses of measles, mumps, and rubella (MMR) vaccine, 30.1% had a history of 1 MMR. Mumps vaccination was not added to the UK vaccination schedule until 1988 as part of the combined MMR vaccine; a second MMR dose was added to the recommended immunization schedule for children 3 to 5 years in 1996. Only 2.4% of confirmed cases in 2004 occurred in persons who would have been eligible for 2 doses of MMR.² During July 2005, 31 cases of mumps were identified among attendees of a New York summer camp. The index case was a camp counselor who was a UK resident and had not been vaccinated against mumps.³

The U.S. experienced a mumps epidemic that resulted in more than 2,000 cases among Midwestern states. From January 1-July 12, 2006, 1,938 cases were reported in Iowa. The median age of cases in Iowa is 22 years (range: 1 to 92 years) and 26% are college students. Most (64%) have evidence of at least 1 MMR vaccination and 50% have documentation of 2 MMRs. Mumps cases have also occurred in Kansas (814), Nebraska (360), Illinois (545), and Missouri (145). States that have experienced an increase in reported cases have responded by recommending MMR vaccination for persons using the Advisory Committee on Immunization Practices (ACIP) guidelines and excluding persons with mumps from school, work and other group settings until 5 days after symptom onset.⁴

Mumps is a self-limited viral infection that can result in parotitis. Parotitis may be unilateral or bilateral and any combination of single or multiple salivary glands may be affected. Complications can include meningitis, orchitis, and spontaneous abortion if infection occurs during the first trimester. These two recent outbreaks emphasize the need for clinicians to suspect mumps regardless of patient vaccination status and to perform laboratory testing to confirm the diagnosis. Acute mumps infection can be confirmed by the presence of serum mumps immunoglobulin M (IgM), a four-fold rise in serum mumps immunoglobulin G (IgG) titer between acute and convalescent phase serum specimens or by positive mumps virus culture. Mumps virus can be isolated from a parotid gland duct swab when collected within 9 days of symptom onset. In Oklahoma, mumps virus culture is available through the Oklahoma State Department of Health Public Health Laboratory. Suspected cases of mumps should be reported to the local county health department for investigation.

*prepared by Laurence Burnsed, MPH, Interim Communicable Disease Division Director

{Preventing Meningococcal Disease} Vaccine Recommendations

Neisseria meningitidis is the causative agent for meningococcal disease and has the potential to cause serious illness including septicemia and meningitis. The case-fatality rate ranges from 8% to 15%; approximately 20% of survivors suffer from permanent sequelae including neurologic damage, renal failure or limb amputation.¹ Meningococcal disease must be reported to the Oklahoma State Department of Health (OSDH) immediately upon diagnosis or suspicion (OAC 310: 515) due to the importance of identifying contacts and recommending prophylaxis to potentially exposed persons. Sterile site isolates of *N. meningitidis* must be forwarded to the OSDH Public Health Laboratory (PHL) for confirmation and serogrouping. We describe a recent case of meningococcal disease in a college student due to serogroup B, summarize *N. meningitidis* serogroup data among Oklahoma cases from 1996 through 2005, and describe meningococcal vaccination recommendations.

During March 2006, the OSDH Communicable Disease Division was notified about a suspected case of meningococemia in a 21-year-old, female college student. She presented to a local emergency room with fever, headache, vomiting, and purpura fulminans. Her condition rapidly deteriorated resulting in renal failure and amputation of three fingers. The case was a junior who lived off-campus and had no roommates. She had recently traveled home to visit family and out of the country for spring break with several friends. State and local public health officials identified 20 persons who were close contacts to the case during the seven days prior to illness onset and were recommended to receive prophylaxis. The case's bacterial isolate was sent to the OSDH-PHL and was identified to be serogroup B.

From 1996 to 2005, 318 cases of meningococcal disease were reported to the OSDH, the average annual number of cases was 32 (range 10 to 46 cases). The average annual incidence rate per 100,000 population was 0.81. Of the 318 cases, 224 (70%) isolates were submitted to the OSDH-PHL. Sixty-two percent of the isolates were vaccine preventable serogroups (A, 1%; C, 21%; Y, 35%; and W-135, 5%). Serogroup B accounted for 21% of isolates. Sixteen percent of isolates were non-groupable and 1% was due to other uncommon serogroups.

Of the 13 serogroups of meningococcal disease, groups A, B,

C, Y, and W-135 are most frequently associated with human disease. Two vaccines are licensed to prevent meningococcal disease in the U.S., however, both only provide protection against serogroups A, C, Y and W-135. MPSV4, the polysaccharide vaccine (Menomune,TM Aventis Pasteur), is recommended for those at higher risk for development of disease in persons aged 2-10 years old and those greater than 55 years old, and produces short-term immunity lasting 3-5 years.² MCV4, the conjugate vaccine (Menactra,TM Sanofi Pasteur), provides a longer duration of immunity and is licensed for use in persons 11-55 years of age. It is recommended for all adolescents and those groups that have an elevated risk of meningococcal disease including college freshman living in dormitories, microbiologists routinely exposed to isolates of *N. meningitidis*, persons who have anatomic or functional asplenia, persons who have terminal complement component deficiencies, and travelers to areas where meningococcal disease is hyperendemic.²

College freshmen, particularly those that reside in dormitories, are at a modestly increased risk for meningococcal disease relative to other persons of the same age. National surveillance data from the 1998-1999 school year suggested the rate of meningococcal disease among freshmen that lived in a dormitory (5.1/100,000 population) was higher than any other age group in the population other than children < 2 years of age.² Advisory Committee on Immunization Practices recommends medical providers educate incoming or current college freshman, particularly those living in dormitory housing, about meningococcal disease and the benefits of vaccination. In November 2003, Oklahoma legislation (70 O.S. § 3243) became effective requiring educational institutions to provide detailed information on meningococcal disease and the vaccine to students residing in on-campus housing. Students must be vaccinated against meningococcal disease or sign a written waiver declining vaccination.

Clinicians should educate patients about the benefits of the meningococcal vaccination to reduce the risk of disease. Meningococcal vaccination will decrease but not eliminate the risk of disease among persons recommended to receive the vaccination.

*prepared by Jolianne Stone, MPH, Epidemiologist, CDD

¹ American Public Health Association. Bacterial Meningitis. In: Heymann DL, ed. *Control of Communicable Diseases Manual*. 18th Edition. Baltimore, MD: American Public Health Association;2004.

² CDC. Prevention and Control of Meningococcal Disease. MMWR 2005;54 No. (RR07):1-21.

Summary of Selected Notifiable Disease Reports in Oklahoma

diseases/conditions	summer quarter ¹	year to date ²	5 year average ³
AIDS	22	44	103
Campylobacteriosis	81	135	186
Chlamydial infections	1947	5027	5106
Cryptosporidiosis	8	18	11
<i>E. coli</i> O157:H7	3	4	12
Ehrlichiosis	0	0	24
Giardiasis	26	49	44
Gonorrhea	646	1703	2220
<i>H. influenzae</i> (all types)	12	32	33
<i>H. influenzae</i> , type B (kids < 5)	0	0	0
Hepatitis A	1	4	28
Hepatitis B (acute)	5	13	46
Hepatitis C (acute)	0	2	6
HIV infections	36	71	65
Meningococcal invasive	1	8	13
Rabies, animal	23	37	357
Rocky Mountain spotted fever	4	6	84
Salmonellosis	104	159	149
Shigellosis	29	49	355
<i>Streptococcus</i> invasive group A	18	64	47
<i>Streptococcus pneumoniae</i> , invasive	98	312	167
Syphilis (primary)	7	16	10
Syphilis (secondary)	2	11	16
Syphilis (early latent)	21	48	48
Tuberculosis	25	80	72

diseases/conditions	year to date ²	5 year average ³
Brucellosis	0	0
Hemolytic Uremic Syndrome (HUS)	0	1
Legionellosis	1	6
Listeriosis	0	1
Lyme disease	0	1
Malaria	2	3
PAM	0	0
Psittacosis	0	0
Tularemia	1	6
Typhoid fever	0	0
Vibriosis	0	1
Yersiniosis (<i>Yersinia enterocolitica</i>)	1	1

number of animal rabies cases by animal type	year to date ²	percent
Bat	3	8
Cat	3	8
Cow	3	8
Dog	0	0
Goat	0	0
Horse	3	8
Skunk	25	68
Total	37	100

¹ 04.01.06 through 06.30.06

² 01.01.06 through 06.30.06

³ Five year average of year to date data for 2001 through 2005.

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