



MMWRTM

Morbidity and Mortality Weekly Report

Weekly

September 29, 2006 / Vol. 55 / No. 38

Sports-Related Injuries Among High School Athletes — United States, 2005–06 School Year

Participation in high school sports helps promote a physically active lifestyle. High school sports participation has grown from an estimated 4 million participants during the 1971–72 school year to an estimated 7.2 million in 2005–06 (1). However, despite the documented health benefits of increased physical activity (e.g., weight management, improved self-esteem, and increased strength, endurance, and flexibility) (2,3), those who participate in athletics are at risk for sports-related injuries (4,5). High school athletes account for an estimated 2 million injuries, 500,000 doctor visits, and 30,000 hospitalizations annually (6). To date, the study of these injuries has been limited by inability to calculate injury rates, compare results among groups, and generalize findings from small, nonrepresentative samples. During the 2005–06 school year, researchers at a children's hospital in Ohio used an Internet-based data-collection tool to pilot an injury surveillance system among athletes from a representative national sample of U.S. high schools. This report summarizes the findings of that study, which indicated that participation in high school sports resulted in an estimated 1.4 million injuries at a rate of 2.4 injuries per 1,000 athlete exposures (i.e., practices or competitions). Surveillance of exposure-based injury rates in a nationally representative sample of high school athletes and analysis of injury patterns can help guide activities aimed at reducing these injuries.

The High School Sports-Related Injury Surveillance Study was sponsored by a CDC grant and conducted during the 2005–06 school year by the Center for Injury Research and Policy at Columbus Children's Hospital in Columbus, Ohio. One hundred U.S. high schools nationally representative of geographic location and school size were selected randomly from among schools that agreed to participate.*

*A total of 4,120 eligible ATCs (i.e., affiliated with the National Athletic Trainers' Association (NATA) and high schools and with contact information available) were contacted. Of those, ATCs representing 425 schools agreed to participate. Those schools were placed into eight sampling strata (created by four geographic strata based on U.S. census areas and two size strata based on large [$\geq 1,000$ students] or small [$< 1,000$ students] schools). Twelve schools were drawn randomly from four strata and 13 schools from the other four strata to make an even 100 participating schools.

Certified athletic trainers (ATCs) affiliated with the National Athletic Trainers' Association (NATA) at each participating school reported injury incidence and athletic exposure data for student athletes participating in nine sports: baseball, football, and wrestling (for boys); softball and volleyball (for girls); and basketball and soccer (for boys and girls). Data were reported weekly via an Internet-based surveillance system. Injuries were defined as those 1) resulting from participation in an organized high school athletic practice or competition, 2) requiring medical attention from an ATC or a physician, and 3) restricting the athlete's participation for 1 or more days beyond the day of injury. An athlete exposure was defined as one athlete participating in one practice or competition during which the athlete was exposed to the possibility of athletic injury. Injury rates were calculated as the ratio of the number of injuries in a particular category (e.g., sport or practice versus competition) to the number of athlete exposures in that category. To calculate national estimates of the number of injuries, each reported injury was assigned a sample weight based on the inverse of the probability of the school's selection into the study (based on the total number of high schools

INSIDE

- 1040 [Chikungunya Fever Diagnosed Among International Travelers — United States, 2005–2006](#)
- 1042 [Importance of Culture Confirmation of Shiga Toxin-producing *Escherichia coli* Infection as Illustrated by Outbreaks of Gastroenteritis — New York and North Carolina, 2005](#)
- 1045 [Ongoing Multistate Outbreak of *Escherichia coli* serotype O157:H7 Infections Associated with Consumption of Fresh Spinach — United States, September 2006](#)
- 1047 [Notice to Readers](#)
- 1047 [QuickStats](#)

The *MMWR* series of publications is published by the Coordinating Center for Health Information and Service, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30333.

Suggested Citation: Centers for Disease Control and Prevention. [Article title]. *MMWR* 2006;55:[inclusive page numbers].

Centers for Disease Control and Prevention

Julie L. Gerberding, MD, MPH
Director

Tanja Popovic, MD, PhD
(Acting) Chief Science Officer

James W. Stephens, PhD
(Acting) Associate Director for Science

Steven L. Solomon, MD
Director, Coordinating Center for Health Information and Service

Jay M. Bernhardt, PhD, MPH
Director, National Center for Health Marketing

Judith R. Aguilar
(Acting) Director, Division of Health Information Dissemination (Proposed)

Editorial and Production Staff

Eric E. Mast, MD, MPH
(Acting) Editor, MMWR Series

Suzanne M. Hewitt, MPA
Managing Editor, MMWR Series

Douglas W. Weatherwax
(Acting) Lead Technical Writer-Editor

Catherine H. Bricker, MS
Jude C. Rutledge
Writers-Editors

Beverly J. Holland
Lead Visual Information Specialist

Lynda G. Cupell
Malbea A. LaPete
Visual Information Specialists

Quang M. Doan, MBA
Erica R. Shaver
Information Technology Specialists

Editorial Board

William L. Roper, MD, MPH, Chapel Hill, NC, Chairman

Virginia A. Caine, MD, Indianapolis, IN

David W. Fleming, MD, Seattle, WA

William E. Halperin, MD, DrPH, MPH, Newark, NJ

Margaret A. Hamburg, MD, Washington, DC

King K. Holmes, MD, PhD, Seattle, WA

Deborah Holtzman, PhD, Atlanta, GA

John K. Iglehart, Bethesda, MD

Dennis G. Maki, MD, Madison, WI

Sue Mallonee, MPH, Oklahoma City, OK

Stanley A. Plotkin, MD, Doylestown, PA

Patricia Quinlisk, MD, MPH, Des Moines, IA

Patrick L. Remington, MD, MPH, Madison, WI

Barbara K. Rimer, DrPH, Chapel Hill, NC

John V. Rullan, MD, MPH, San Juan, PR

Anne Schuchat, MD, Atlanta, GA

Dixie E. Snider, MD, MPH, Atlanta, GA

John W. Ward, MD, Atlanta, GA

in each of the eight sampling strata). If a school dropped out of the surveillance study, a replacement school from the same sampling stratum was enrolled.

An estimated 1,442,533 injuries occurred among U.S. high school student athletes participating in practices or competitions for the nine sports studied. The overall (i.e., practice and competition) injury rate in all sports combined was 2.44 injuries per 1,000 athlete exposures (Table). Football had the highest injury rate (4.36 injuries per 1,000 athlete exposures) followed by wrestling (2.50), boys' (2.43) and girls' (2.36) soccer, and girls' basketball (2.01). Boys' basketball, volleyball, baseball, and softball each had injury rates of less than 2.0 injuries per 1,000 athlete exposures. In each sport, the injury rate was higher in competition than practice settings. Although boys' soccer had slightly higher injury rates than girls' soccer, and girls' basketball had slightly higher injury rates than boys' basketball, no statistically significant differences ($p>0.05$) by sex were observed for soccer and basketball.

In each of the nine sports, approximately 80% of the reported injuries were new injuries as opposed to recurrences or complications from previous injuries. Types of injuries varied between practice and competition; for example, concussions and fractures occurred more commonly in competition than practice (Figure 1). The total numbers of injuries were similar for practice and competition; however, because fewer competitions are conducted than practices, the injury rates in competition were higher. Severity of injury, as measured by days lost from play, varied by sport. Overall, approximately half of the injuries reported resulted in <7 days lost; football, girls' basketball, and wrestling had greater proportions of injuries resulting in ≥ 7 days lost (Figure 2). No deaths were reported.

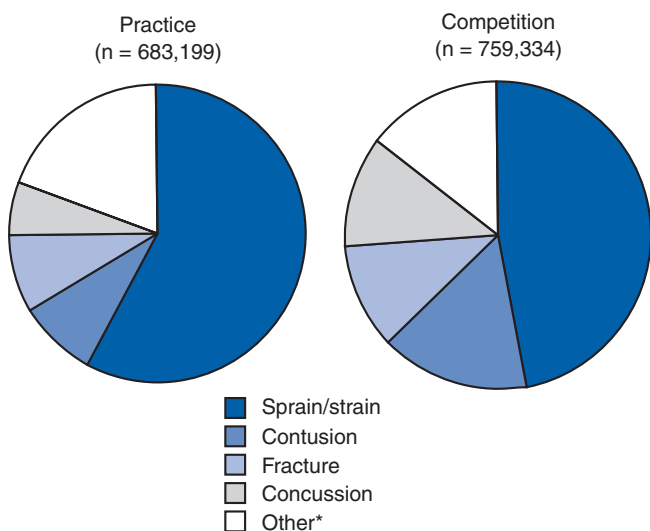
Reported by: RD Comstock, PhD, Center for Injury Research and Policy, Columbus Children's Hospital, and Ohio State Univ; C Knox, MA, E Yard, MPH, Center for Injury Research and Policy, Columbus Children's Hospital, Columbus, Ohio. J Gilchrist, MD, Div of Unintentional Injury Prevention, National Center for Injury Prevention, CDC.

TABLE. Sport-specific injury rates* in practice, competition, and overall — High School Sports-Related Injury Surveillance Study, United States, 2005–06 school year

Sport	Rate		
	Practice	Competition	Overall
Boys' football	2.54	12.09	4.36
Boys' wrestling	2.04	3.93	2.50
Boys' soccer	1.58	4.22	2.43
Girls' soccer	1.10	5.21	2.36
Girls' basketball	1.37	3.60	2.01
Boys' basketball	1.46	2.98	1.89
Girls' volleyball	1.48	1.92	1.64
Boys' baseball	0.87	1.77	1.19
Girls' softball	0.79	1.78	1.13
Total	1.69	4.63	2.44

* Per 1,000 athlete exposures (i.e., practices or competitions).

FIGURE 1. Proportion of injuries in practice and competition, by diagnosis — High School Sports-Related Injury Surveillance Study, United States, 2005–06 school year

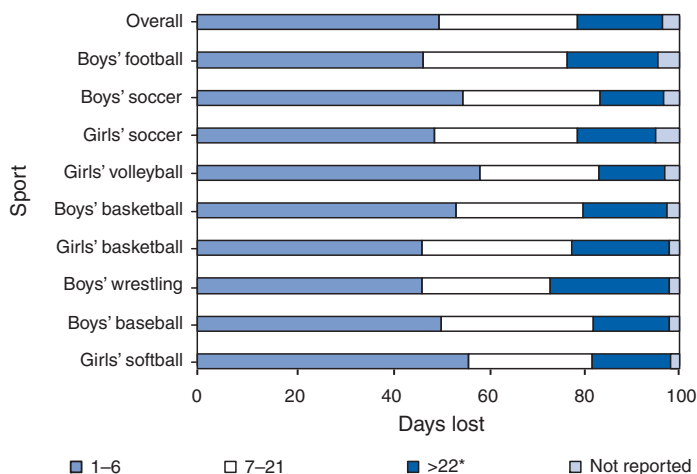


* Includes other injuries (e.g., lacerations or dislocations) and reportable health-related events (e.g., heat illness, skin infections, or asthma attacks).

Editorial Note: During the 2005–06 school year, an estimated 4.2 million students in the United States participated in the nine high school sports studied (1). This report is the first to use data from an Internet-based injury surveillance study to provide national estimates of injuries among these student athletes. The pilot study determined that an estimated 1.4 million injuries, more than 80% of which were new, occurred among participants in the nine high school sports during the 2005–06 school year. Injury prevention takes on added importance because history of an injury (e.g., sprain) is often a risk factor for future injury.

Two existing injury surveillance systems illustrate the potential usefulness of continuing this pilot study as an ongoing system at the high school level to identify areas for targeted interventions. The National Collegiate Athletic Association Injury Surveillance System conducts surveillance of injuries among collegiate athletes (7), and the National Center for Catastrophic Sports Injury Research collects data on catastrophic injuries (e.g., brain or spinal cord injuries) (8). For approximately 20 years, the data collected by these two surveillance systems have been used by medical committees, rules committees, and researchers to reduce injury rates by driving the development, implementation, and evaluation of injury prevention programs. Recent examples of data-driven changes in policies and practices include 1) educational campaigns to reduce heat-related injuries in football and other sports, 2) elimination of racing starts in shallow pools to reduce catastrophic swimming injuries, and 3) recommendation of eye protection in sports involving sticks (e.g., field hockey).

FIGURE 2. Proportion of injuries, by sport and number of days lost — High School Sports-Related Injury Surveillance Study, United States, 2005–06 school year



* Includes athletes who returned to their teams after ≥ 22 days and athletes who were out for the remainder of the season as a result of their injuries.

Previous sports-related injury studies have used various age groups and definitions for injury and exposure. One conducted among college athletes used a definition for injury that was similar to that used for this study (7). Although injury rates in that study of collegiate sports were two to six times higher compared with high school sports, patterns of injury were similar. Collegiate football had the highest rate of injury, and collegiate injury rates were higher in competition than practice (7). At the high school level, a surveillance study conducted during 1995–1997 in a representative sample of U.S. high schools used a slightly broader definition of injury and collected data on varsity athletes only (6). That study also observed higher injury rates in football compared with other sports and in competition versus practice. However, in each sport except volleyball, injury rates from 1995–1997 were at least two times higher than the injury rates observed in this report. Reasons for this disparity are not fully understood; some of the disparity might be attributable to differences in study methods, and some might have resulted from new injury prevention measures (e.g., rules changes or safety-gear improvements) and improvements in diagnosis and treatment (e.g., greater attention to minor injuries to reduce the rate of more serious injuries) since the earlier study was conducted.

The findings in this report are subject to at least three limitations. First, only injuries that came to the attention of ATCs were included; injuries treated in a physician's office, emergency department, or urgent-care facility and not reported to an ATC were not captured. Second, only U.S. high schools whose athletes had access to care from an ATC affiliated with NATA were eligible. Reliable estimates of the number of U.S.

high schools whose athletes have access to an ATC are not available. Finally, only injuries sustained during participation in nine sports were included. Although an estimated 4.2 million U.S. high school students participated in these nine sports during the 2005–06 school year, more than 30 sports were offered by U.S. high schools (1).

Although the health benefits of a physically active lifestyle, including sports participation, are well known, the risks for sports-related injury and effective prevention strategies are less well established. General recommendations for reducing the risk of injury among high school athletes (e.g., ensure adequate hydration and use of appropriate protective equipment in practices and competitions) and sport-specific recommendations (e.g., block and tackle with the head up to reduce the risk for neck injuries in football) are offered by NATA (9). Additionally, CDC addresses prevention and management of concussion in all sports with a free tool kit for coaches, *Heads Up: Concussion in High School Sports* (10). This pilot study demonstrates that participation and injury data can be collected to calculate exposure-based injury rates. Calculation of rates enables comparison of injuries among age groups, sports, and years. The results support the feasibility and value of targeting research and prevention strategies to those students most at risk for sports-related injuries.

References

1. National Federation of State High School Associations (NFHS). 2005–2006 High School Athletics Participation Survey. Indianapolis, IN: NFHS; 2006. Available at <http://www.nfhs.org/sports.aspx>.
2. World Health Organization. Move for health: benefits of physical activity. Geneva, Switzerland: World Health Organization; 2006. Available at http://www.who.int/moveforhealth/advocacy/information_sheets/benefits/en/index.html.
3. CDC. Guidelines for school and community programs to promote lifelong physical activity among young people. MMWR 1997;46 (No. RR-6).
4. Gotsch K, Annett JL, Holmgren P, Gilchrist J. Nonfatal sports- and recreation-related injuries treated in emergency departments—United States, July 2000–June 2001. MMWR 2002;51:736–40.
5. Conn JM, Annett JL, Gilchrist J. Sports and recreation-related injury episodes in the U.S. population, 1997–1999. *Inj Prev* 2003;9:117–23.
6. Powell JW, Barber-Foss KD. Injury patterns in selected high school sports: a review of the 1995–1997 seasons. *J Athl Train* 1999;34:277–84.
7. National Collegiate Athletic Association (NCAA) Injury Surveillance System. Available at http://www1.ncaa.org/membership/ed_outreach/health-safety/iss/index.html. Indianapolis, IN: NCAA; 2006.
8. University of North Carolina at Chapel Hill. National Center for Catastrophic Sport Injury Research. Chapel Hill, NC: University of North Carolina at Chapel Hill; 2006. Available at <http://www.unc.edu/depts/nccsi>.
9. National Athletic Trainers' Association (NATA). Minimizing the risk of injury in high school athletics: guidelines from the National Athletic Trainers' Association. Dallas, TX: NATA; 2002. Available at <http://www.nata.org/publications/brochures/minimizingtherisks.htm>.
10. CDC. Heads up: concussion in high school sports. Atlanta, GA: US Department of Health and Human Services, CDC; 2005. Available at http://www.cdc.gov/ncipc/tbi/coaches_tool_kit.htm.

Chikungunya Fever Diagnosed Among International Travelers — United States, 2005–2006

Chikungunya virus (CHIKV) is an alphavirus indigenous to tropical Africa and Asia, where it is transmitted to humans by the bite of infected mosquitoes, usually of the genus *Aedes* (1). Chikungunya (CHIK) fever, the disease caused by CHIKV, was first recognized in epidemic form in East Africa during 1952–1953. The word “chikungunya” is thought to derive from description in local dialect of the contorted posture of patients afflicted with the severe joint pain associated with this disease. Because CHIK fever epidemics are sustained by human-mosquito-human transmission, the epidemic cycle is similar to those of dengue and urban yellow fever. Large outbreaks of CHIK fever have been reported recently on several islands in the Indian Ocean and in India (2–6). In 2006, CHIK fever cases also have been reported in travelers returning from known outbreak areas to Europe, Canada, the Caribbean (Martinique), and South America (French Guyana) (2,3,5–7). During 2005–2006, 12 cases of CHIK fever were diagnosed serologically and virologically at CDC in travelers who arrived in the United States from areas known to be epidemic or endemic for CHIK fever. This report describes four of these cases and provides guidance to health-care providers. Clinicians should be alert for additional cases among travelers, and public health officials should be alert to evidence of local transmission of chikungunya virus (CHIKV), introduced through infection of local mosquitoes by a person with viremia.

Case Reports

Minnesota. On May 12, 2005, an adult male resident of Minnesota returned from a 3-month trip to Somalia and Kenya. He had onset of illness hours after arrival in the United States, including fever, headache, malaise, and joint pain mainly in a shoulder and a knee. Serum obtained on May 13 was tested at CDC and determined to be equivocal for CHIKV RNA by reverse-transcription polymerase chain reaction (PCR), consistent with low-level viremia. A recent CHIKV infection was confirmed by demonstration of IgM antibody in this acute-phase serum specimen and neutralizing antibody in convalescent-phase serum (collected 214 days after illness onset). Arthralgias resolved after several weeks.

Louisiana. On January 15, 2006, an adult female resident of India had onset of an illness characterized by fever, joint pain (in the knees, wrists, hands, and feet), and muscle pain (in the thighs and neck). In March 2006, she traveled to Louisiana, where she sought medical attention for persistent joint pain. At CDC, tests of a single serum sample collected on March 30 (74 days after illness onset) were positive for IgM

and neutralizing antibodies to CHIKV. The patient was subsequently lost to follow-up.

Maryland. An adult female resident of Maryland visited the island of Réunion in the Indian Ocean from October 2005 through mid-March 2006. On February 18, 2006, during an ongoing CHIK fever outbreak on the island, she had onset of fever, joint pain (in the hands and feet), and rash. A local physician clinically diagnosed CHIK fever, but no laboratory tests were conducted. After returning to the United States, the patient sought medical attention for persistent joint pain. At CDC, tests of a single serum sample collected on March 22 (32 days after illness onset) were equivocal for IgM and positive for neutralizing antibody to CHIKV, consistent with a recent CHIKV infection in which IgM antibody was waning. At 5 months after onset, the patient had persistent joint pain (in the hands and feet).

Colorado. An adult male resident of Colorado visited Zimbabwe during April 17–May 29, 2006. On April 29, he had onset of illness with fever, chills, joint pain (in the wrists and ankles), and neck stiffness; a rash appeared a few days later. All symptoms resolved within 2 weeks, except for joint pain, which persisted for approximately 1 month. At CDC, tests of a single serum sample collected on June 12 (44 days after illness onset) were positive for IgM and neutralizing antibody to CHIKV.

Reported by: E Warner, Denver, Colorado. J Garcia-Diaz, MD, Ochsner Clinic Foundation, New Orleans; G Balsamo, DVM, Louisiana Dept of Health and Hospitals. S Shranatan, DO, Johns Hopkins Community Physicians at Hager Park, Hagerstown; A Bergmann, MS, Maryland Dept of Health and Mental Hygiene. L Blauwet, MD, M Sobail, MD, L Baddour, MD, Mayo Clinic College of Medicine, Rochester, Minnesota. C Reed, MD, H Baggett, MD, Div of Global Migration and Quarantine, National Center for Preparedness, Detection, and Control of Infectious Diseases (proposed); G Campbell, MD, T Smith, MD, A Powers, PhD, N Hayes, MD, A Noga, J Lehman, Div of Vector-Borne Infectious Diseases, National Center for Zoonotic, Vector-Borne, and Enteric Diseases (proposed), CDC.

Editorial Note: Most CHIKV infections are symptomatic (8). In clinical infections, the incubation period typically is 2–4 days. Illness is characterized by sudden onset of fever, headache, malaise, arthralgias or arthritis, myalgias, and low back pain. Skin rash occurs in approximately half of cases (9). Joint symptoms can be severe and involve small and large joints. Although CHIK fever typically lasts 3–7 days and full recovery is the usual outcome, certain patients experience persistent joint symptoms for weeks or months and occasionally years after illness onset (1). Serious complications (e.g., neuroinvasive disease) are rare, and fatal cases have not been documented conclusively. Transplacental CHIKV transmission and severe congenital CHIKV disease have been described

(10). CHIKV infection is believed to confer life-long immunity (1). Because no specific drug therapy is available, treatment of CHIK fever is supportive. No licensed CHIKV vaccine exists. Therefore, prevention recommendations for travelers to tropical Asia and Africa should emphasize mosquito repellent and avoidance measures. Additional information is available at <http://www.cdc.gov/ncidod/dvbid/chikungunya/chickvfact.htm>.

During May 2004–May 2006, approximately 300,000 suspected CHIK fever cases were reported on islands in the Indian Ocean, including approximately 264,000 suspected cases on Réunion, a French overseas department (2,3). Other affected areas included Mombasa, Kenya, and the islands of Comoros, Lamu, Madagascar, Mauritius, Mayotte, and the Seychelles. In addition, since early 2006, an estimated 180,000 suspected CHIK fever cases have occurred in the Indian states of Andhra Pradesh, Karnataka, and Maharashtra (4). In recent years, extensive CHIKV activity also has been documented in Southeast Asia (9). In 2006, as of May 11, approximately 340 imported CHIK fever cases were reported in Europe, mainly in France, reflecting the high frequency of travel between Europe and islands in the Indian Ocean (2). To date, no known local mosquito-borne CHIKV transmission has occurred in Europe or other nonindigenous areas.

Aedes aegypti is the primary CHIKV vector in Asia, but *Ae. albopictus* (the Asian tiger mosquito) likely was the primary vector in Réunion (2,3). In Asia, CHIKV epidemics involve a human-mosquito cycle, with humans serving as the sole vertebrate amplifying hosts (1). In Africa, sylvatic cycles involving nonhuman primates and forest-dwelling *Aedes* species (e.g., *Ae. furcifer*) also occur. Most CHIKV epidemics occur during the tropical rainy season and abate during the dry season (1,9). Human CHIKV infections include a transient, high-titered viremia (typically detectable during the first 2 days of illness, ranging up to 6 days after illness onset) that is adequate to infect feeding mosquitoes (1). *Ae. aegypti* and *Ae. albopictus* are abundant peridomestic species and aggressive daytime blood-feeders in all tropical and most subtropical areas of the world, and *Ae. albopictus* now lives in many temperate areas of the eastern and western hemispheres, including Europe and the United States. Therefore, some risk exists that CHIKV might be introduced into previously nonendemic areas by travelers with viremia, leading to local transmission of the virus, especially in tropical or subtropical areas of the United States (e.g., the Gulf Coast and Hawaii) or its territories (e.g., Guam, Puerto Rico, and the U.S. Virgin Islands). Early recognition of local transmission followed by prompt, aggressive vector control and other public health measures might prevent long-term establishment of the virus in new areas. Of the four patients described in this report,

three posed no substantial public health risk because they probably no longer had viremia upon arrival in the United States; although the fourth patient was likely viremic upon arrival in Minnesota in mid-May, transmission to competent local mosquito vectors in that climate was unlikely.

In early illness, the clinical features of CHIK fever can be similar to those of dengue and malaria, especially in patients without joint symptoms. In both dengue and CHIK fever, rash usually is generalized and maculopapular, but petechial rashes occur in certain dengue cases. During 1991–2004, nine confirmed or probable cases of CHIK fever were diagnosed serologically at CDC among travelers to the United States (CDC, unpublished data, 2006). Additional imported but unrecognized cases likely occurred. Clinicians should be aware of possible CHIKV infection in travelers returning from CHIK-fever–endemic or outbreak areas, particularly if an acute febrile illness with arthralgias or arthritis occurs. Suspected cases should be reported promptly to local and state public health officials and to CDC. Mosquito exposure should be strictly avoided (e.g., by staying within a screened environment and using barrier clothing and repellents) during the first week of illness to prevent infection of local mosquitoes.

In the United States, diagnostic tests for CHIKV infection are not available commercially but are available at CDC by special arrangement through state health departments. Laboratory diagnosis depends on antibody-capture IgM ELISA and plaque-reduction neutralization tests of serum. Comparative serologic tests for closely related alphaviruses (e.g., o'nyong-nyong and Sindbis viruses) should be conducted as geographically appropriate, and tests for dengue usually are indicated. Virus isolation attempts and PCR assays are performed selectively. Serologic tests should be performed on both acute- and convalescent-phase serum specimens collected at least 2 weeks apart, but clinicians should not delay submission of acute-phase samples pending collection of convalescent-phase samples. To arrange submission of specimens to CDC for diagnostic testing, clinicians should consult their state public health laboratory and CDC's Arboviral Diseases Branch (telephone, 970-221-6400). Specimen shipping and handling instructions are available at <http://www.cdc.gov/ncidod/dvbid/misc/specimen-submission.htm>.

References

- Jupp PG, McIntosh BM. Chikungunya virus disease. In: Monath TP, ed. *The arboviruses: epidemiology and ecology* (vol 2). Boca Raton, Florida: CRC Press; 1988:137–57.
- Depoortere E, Coulombier D, European Centre for Disease Prevention and Control Chikungunya Risk Assessment Group. Chikungunya risk assessment for Europe: recommendations for action. *Euro Surveill* 2006;11:E060511.2. Available at <http://www.eurosurveillance.org/ew/2006/060511.asp#2>.

- Parola P, de Lamballerie X, Jourdan J, et al. Novel chikungunya virus variant in travelers returning from Indian Ocean islands. *Emerg Infect Dis* 2006;12:1493–9. Available at <http://www.cdc.gov/ncidod/EID/vol12no10/pdfs/06-0610.pdf>.
- Ravi V. Re-emergence of chikungunya virus in India. *Indian J Med Microbiol* 2006;24:83–4.
- Ligon BL. Reemergence of an unusual disease: the chikungunya epidemic. *Semin Pediatr Infect Dis* 2006;17:99–104.
- Public Health Agency of Canada. Notices and international reports. Outbreak of chikungunya virus: south west Indian Ocean and India. Available at http://www.phac-aspc.gc.ca/tmp-pmv/2006/chiku060526_e.html.
- Caribbean Epidemiology Centre (CAREC). Chikungunya in Martinique and French Guyana. CAREC surveillance report. Port of Spain, Trinidad & Tobago: Pan American Health Organization; 2006;26:17. Available at <http://www.carec.org/pdf/csrjune2006.pdf>.
- Retuya TJA Jr, Ting DL, Dacula BD, et al. Chikungunya fever outbreak in an agricultural village in Indang, Cavite, Philippines. *Philippine Journal of Microbiology and Infectious Diseases* 1998;27:93–6.
- Laras K, Sukri NC, Larasati RP, et al. Tracking the re-emergence of epidemic chikungunya virus in Indonesia. *Trans R Soc Trop Med Hyg* 2005;99:128–41.
- Robillard PY, Boumahni B, Gerardin P, et al. Vertical maternal fetal transmission of the chikungunya virus. *Presse Med* 2006;35:785–8.

Importance of Culture Confirmation of Shiga Toxin-producing *Escherichia coli* Infection as Illustrated by Outbreaks of Gastroenteritis — New York and North Carolina, 2005

Escherichia coli O157:H7 and other strains of *E. coli* that produce Shiga toxin are collectively known as Shiga toxin-producing *E. coli* (STEC). The current outbreak of STEC O157 infections associated with eating fresh spinach illustrates the importance of obtaining isolates to identify the source of the infections (1). Laboratory methods that do not require bacterial culture of stool specimens to identify STEC are being used increasingly by clinical diagnostic laboratories, sometimes without subsequent confirmation of a strain by isolating it in culture. This report describes findings from outbreaks of gastroenteritis in 2005 in New York and North Carolina in which clinical diagnostic laboratories initially used only non-culture methods to detect Shiga toxin (Stx). The findings highlight the importance of confirmation of Stx-positive stool specimens by bacterial culture for timely and reliable identification of STEC infections, including *E. coli* O157 and non-O157 STEC, to enable implementation of appropriate public health actions. An important part of that identification is determining the serotype of all STEC isolates and the subtype of STEC O157 strains so that outbreaks can be detected and traced back to sources.

New York

During August 28–September 13, 2005, a total of 52 (2.4%) of 2,160 inmates at a state correctional facility reported diarrhea, including 17 (33%) with bloody diarrhea. Nineteen inmates were treated at the prison infirmary; three were hospitalized for an average of 1.8 days. Stool specimens from these three inmates tested positive for Stx by enzyme immunoassay (EIA) at a clinical diagnostic laboratory. Subsequently, stool specimens collected from 21 ill inmates were submitted to the New York State Department of Health (NYSDOH)-Wadsworth Center. Stool specimens were inoculated to *E. coli* enrichment broth and sorbitol MacConkey agar (SMAC), a selective medium used to screen for STEC serotype O157:H7 because this serotype, unlike most *E. coli* (and unlike most STEC), does not ferment sorbitol. Sixteen of the stool enrichment broths, when tested by polymerase chain reaction (PCR), were positive for the Shiga toxin 1 gene (*stx1*) but negative for STEC O157-specific DNA; 13 of the SMAC agar plates demonstrated growth of sorbitol-fermenting *E. coli* colonies that also were positive for *stx1* by PCR and did not agglutinate with commercial latex reagents for STEC serogroups O26, O91, O103, O111, O128, O145, and O157. Isolates from three patients were sent to CDC and determined to be STEC serotype O45:nonmotile (NM) (one patient had both STEC serotype O45:NM and O45:H2). These STEC O45 isolates were indistinguishable by pulsed-field gel electrophoresis (PFGE) using *Xba*I and *Bln*I restriction endonucleases.

The source of the outbreak likely was an ill food worker. Control measures included enhanced surveillance for additional illness and reminders of the need for exclusion of the infected food worker from food service or other jobs with increased risk for transmission until his specimens no longer tested positive for STEC.

North Carolina

On November 10, 2005, the Davidson County Health Department received a report of non-bloody diarrheal illness in an infant aged 6 months who attended a day care center. Diarrhea was reported in four additional day care center attendees and three family members of the index patient.

An enrichment broth from a stool specimen from the index patient tested positive for Stx by EIA at a clinical diagnostic laboratory. After a delay of some days, the laboratory sent the enrichment broth culture of this stool specimen to the North Carolina State Laboratory for Public Health, where neither STEC O157 nor STEC serogroups O26, O45, O103, O121, O111, or O145 were isolated. The enrichment broth was then sent to CDC, where it again tested positive for Stx by EIA, but PCR tests of the enrichment broth at CDC were negative

for *stx1* and *stx2*. The enrichment broth was plated on SMAC, and PCR tests of both a sweep of growth from the plate and of 10 sorbitol-fermenting colonies were negative for *stx1* or *stx2*. Subsequently, the North Carolina State Laboratory for Public Health performed additional testing on stool specimens from five ill persons, including the index case; each tested positive for norovirus by reverse transcription-PCR.

In response to the initial Stx-positive report, public health control measures appropriate for STEC had been instituted, including exclusion of the index case from the day care center pending receipt of two STEC-negative cultures of stool specimens collected at least 24 hours apart. These exclusion measures also had been enforced for the four other ill children in the day care center. When the outbreak was determined to have been caused by norovirus, not STEC, control measures were revised, and the ill children were allowed to return to the day care center after they became asymptomatic.

Reported by: R Atkinson, PhD, G Johnson, T Root, T Halse, D Wroblewski, MS, New York State Dept of Health. M Davies, MD, A Byrd, L Long, MSA, Davidson County Health Dept, North Carolina. L Demma, PhD, F Angulo, DVM, PhD, C Bopp, MS, P Gerner-Smith, MD, PhD, N Strockbine, PhD, K Greene, B Swaminathan, PhD, P Griffin, MD, Div of Foodborne, Bacterial, and Mycotic Diseases, National Center for Zoonotic, Vector-Borne, and Enteric Diseases (proposed); J Schaffzin, MD, PhD, B Goode, MPH, EIS officers, CDC.

Editorial Note: The New York outbreak demonstrates that Stx testing of stool specimens from patients with diarrhea by clinical diagnostic laboratories can facilitate detection of outbreaks of non-O157 STEC. However, as the North Carolina outbreak demonstrates, occasional false-positive results from the Stx EIA test can result in inappropriate and unnecessary public health action. These two outbreaks illustrate the importance of culture confirmation of Stx EIA-positive specimens.

Although STEC O45 is an important cause of sporadic non-O157 STEC infections in the United States, the cases in New York represent the first outbreak of STEC O45 infections ever identified in the United States (2). During 1983–2002, public health laboratories submitted 940 non-O157 STEC isolates to CDC, of which 7% were identified as O45, making it the fifth most commonly isolated non-O157 STEC serogroup during that period (2).

The outbreak in North Carolina illustrates the importance both of rapid culturing of all Stx-positive broths specifically for STEC O157 and of rapid culture confirmation of Stx-positive specimens. The initial Stx-positive result prompted public health actions that, in retrospect, placed an unnecessary burden on patients, day care center staff, and public health officials. Had a culture for *E. coli* O157 been performed simultaneously with the EIA or after the EIA was determined

positive, the negative result might have prompted investigation for norovirus sooner. Once the EIA result was determined falsely positive, and the true etiology of the outbreak was determined, control measures appropriate for norovirus were instituted. This is not the first time that an outbreak of norovirus infections was mistakenly attributed to STEC (3).

In 2000, non-O157 STEC infections became nationally notifiable. However, few non-O157 STEC infections are detected because most clinical diagnostic laboratories do not test stool specimens routinely for these organisms (4,5). No selective agar medium exists for isolation of non-O157 STEC. SMAC and other sorbitol-containing selective media such as cefixime-tellurite SMAC (CT-SMAC), Rainbow Agar O157, and CHROMagar O157 enhance isolation of STEC O157 because strains of this serotype typically do not ferment sorbitol or produce beta-D-glucuronidase. However, most non-O157 STEC strains ferment sorbitol and are phenotypically indistinguishable from other *E. coli* strains present in the normal intestinal flora. Non-O157 STEC infections can be diagnosed by use of EIA, PCR, or cell culture methods to detect free Stx or the *stx1* or *stx2* genes in stool or enrichment broths. EIA testing of broth cultures, rather than the stool specimens themselves, is recommended because the amount of free fecal Stx in stools often is low (6). Alternatively, production of Stx or the presence of Stx gene sequences can be demonstrated by selecting colonies from plating media and testing them by EIA or PCR. The development of commercial Stx EIA kits has allowed clinical diagnostic laboratories to easily screen stool specimens for STEC independent of serotype. If the index of clinical suspicion for STEC O157 is high, the stool specimen should be tested simultaneously by Stx EIA and by bacterial culture on a sorbitol-containing medium such as SMAC (7). Virulence factors strongly associated with the development of hemolytic uremic syndrome (HUS) are almost always present in STEC O157, but less frequently in non-O157 STEC (8). The majority of clinical diagnostic laboratories cannot determine the virulence profile of STEC but can identify an STEC O157 infection. Therefore, early diagnosis of at least STEC O157 is important to identify patients at highest risk for HUS. Treatment with parenteral-volume expansion early in the course of STEC O157 infection can decrease renal injury and improve patient outcome (9).

Clinical diagnostic laboratories should strongly consider including STEC O157 in their routine bacterial enteric panel (with *Salmonella*, *Shigella*, and *Campylobacter* spp.). If bacterial culture for STEC O157 is not performed in parallel with EIA, Stx-positive broths should be inoculated to a selective isolation medium, such as SMAC agar, and any resulting sorbitol-negative colonies should be tested with O157 antiserum or latex reagent. All confirmed and presumptive STEC

O157 isolates and Stx-positive broths that do not yield STEC O157 should be forwarded to a public health laboratory as soon as possible for confirmatory testing and further genetic characterization. STEC O157 isolates should be confirmed, characterized, and tested by PFGE, and the pattern promptly entered into the PulseNet database. At the public health laboratory, the broth should be subcultured to selective agar and a representative sample of sorbitol positive and negative colonies tested by Stx EIA or PCR for *stx1* and *stx2* genes. Non-O157 STEC isolates can be tested using commercial antisera for the most common non-O157 STEC serogroups (O26, O45, O103, O111, O121, and O145) and should be sent to the CDC *E. coli* Reference Laboratory for complete serotyping and further genetic characterization, including PFGE.

To facilitate investigation of possible outbreaks, clinicians should inform health departments about clusters of patients with bloody diarrhea or HUS, and clinical diagnostic laboratories should follow recommended procedures for identification of STEC (Box). Screening stool specimens by clinical diagnostic laboratories for Stx using EIA, subsequent bacterial culture of Stx-positive specimens using SMAC, and forwarding enrichment broths from Stx-positive specimens that do not yield STEC O157 to state or local public health laboratories, are crucial steps for public health surveillance of STEC infections. With this coordinated approach, accurate laboratory data can be combined with epidemiologic information to ensure prompt diagnosis and treatment of STEC O157 infections, improved diagnostic accuracy, and improved detection of outbreaks caused by non-O157 STEC.

Acknowledgments

The findings in this report are based, in part, on contributions by M Caldwell, MD, A Evans, S Ireland, M DeFabio, Dutchess County Dept of Health, Poughkeepsie; L Klopff, MEd, C Metzler, New York State Dept of Correctional Svcs; N Dumas, T Quinlan, L Armstrong, J Edwards, K Musser, PhD, R Limberger, PhD, New York State Dept of Health-Wadsworth Center; R Gallo, B Devine, New York State Dept of Health. B Jenkins, C Hartley, L Wolf, North Carolina State Laboratory of Public Health. S Rolando, MHS, Assoc of Public Health Laboratories, Silver Springs, Maryland. KG Holt, DVM, Food Safety and Inspection Svc, US Dept of Agriculture.

References

1. CDC. Ongoing multistate outbreak of *Escherichia coli* serotype O157:H7 infections associated with consumption of fresh spinach—United States, September 2006. MMWR 2006;55:1045–6.
2. Brooks JT, Sowers EG, Wells JG, et al. Non-O157 Shiga toxin-producing *Escherichia coli* infections in the United States, 1983–2002. J Infect Dis 2005;192:1422–9.
3. CDC. University outbreak of calicivirus infection mistakenly attributed to Shiga toxin-producing *Escherichia coli* O157:H7—Virginia, 2000. MMWR 2001;50:489–91.

BOX. Recommendations for laboratory identification of Shiga toxin-producing *Escherichia coli* (STEC)

- Health-care providers should notify clinical diagnostic laboratories when STEC O157 infection is suspected (e.g., because of bloody diarrhea or hemolytic uremic syndrome) so that appropriate testing methods can be applied.
- Clinical diagnostic laboratories should strongly consider including STEC O157 in their routine bacterial enteric panel (with *Salmonella*, *Shigella*, and *Campylobacter*).
- The best way to identify all STEC infections is to screen all stool samples submitted for routine enteric bacterial testing for Shiga toxins (Stxs) using enzyme immunoassay (EIA) or polymerase chain reaction. Ideally, the clinical diagnostic laboratory should culture simultaneously for STEC O157 (e.g., on sorbitol MacConkey agar). Simultaneous culture facilitates rapid diagnosis and treatment of patients with STEC O157 infection and rapid subtyping by public health laboratories; such rapid action is most important when the index of clinical suspicion for STEC O157 is high.
- Clinical diagnostic laboratories that use an Stx EIA but do not perform simultaneous culture for STEC O157 should culture all Stx-positive broths for STEC O157 as soon as possible and rapidly forward these isolates to a state or local public health laboratory for confirmation and subtyping.
- When an Stx-positive broth does not yield STEC O157, the broth culture should be quickly forwarded to the state or local public health laboratory for identification of non-O157 STEC.
- State and local public health laboratories should confirm the presence of Stx in broths sent from clinical laboratories and should attempt to obtain an STEC isolate. All non-O157 STEC isolates should be sent by public health laboratories to CDC for confirmation and further characterization.

4. Klein EJ, Stapp JR, Clausen CR, et al. Shiga toxin-producing *Escherichia coli* in children with diarrhea: a prospective point-of-care study. *J Pediatr* 2002;141:172–7.
5. Voetsch AC, Van Gilder TJ, Angulo FJ, et al. FoodNet estimate of the burden of illness caused by nontyphoidal *Salmonella* infections in the United States. *Clin Infect Dis* 2004;38(Suppl 3):S127–34.
6. Cornick NA, Jelacic S, Ciol MA, et al. *Escherichia coli* O157:H7 infections: discordance between filterable fecal Shiga toxin and disease outcome. *J Infect Dis* 2002;186:57–63.
7. Cohen MB. Shiga toxin-producing *E. coli*: two tests are better than one. *J Pediatr* 2002;141:155–6.
8. Eklund M, Leino K, Siitonen A. Clinical *Escherichia coli* strains carrying stx genes: stx variants and stx-positive virulence profiles. *J Clin Microbiol* 2002;40:4585–93.
9. Ake JA, Jelacic S, Ciol MA, et al. Relative nephroprotection during *Escherichia coli* O157:H7 infections: association with intravenous volume expansion. *Pediatrics* 2005;115:e673–80.

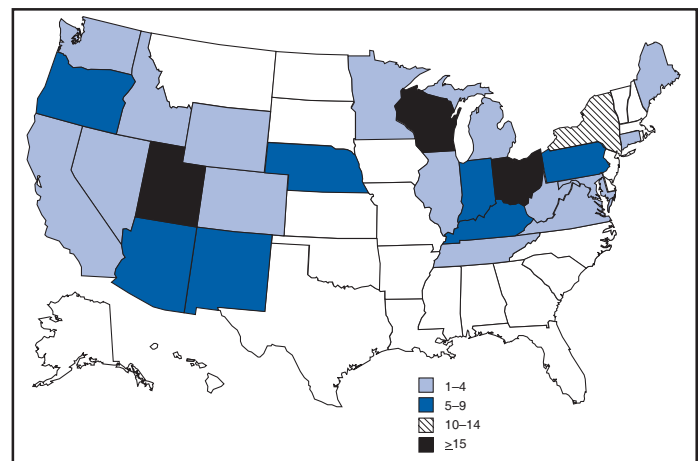
Ongoing Multistate Outbreak of *Escherichia coli* serotype O157:H7 Infections Associated with Consumption of Fresh Spinach — United States, September 2006

On September 26, this report was posted as an MMWR Dispatch on the MMWR website (<http://www.cdc.gov/mmwr>).

On September 13, 2006, CDC officials were alerted by epidemiologists in Wisconsin and Oregon that fresh spinach was the suspected source of small clusters of *Escherichia coli* serotype O157:H7 infections in those states. On the same day, New Mexico epidemiologists contacted Wisconsin and Oregon epidemiologists about a cluster of *E. coli* O157:H7 infections in New Mexico associated with fresh spinach consumption. Wisconsin public health officials had first reported a cluster of *E. coli* O157:H7 infections to CDC on September 8. On September 12, CDC PulseNet had confirmed that the *E. coli* O157:H7 strains from infected patients in Wisconsin had matching pulsed-field gel electrophoresis (PFGE) patterns and identified the same pattern in patient isolates from other states. This report describes the joint investigation and outbreak-control measures undertaken by state public health officials, CDC, and the Food and Drug Administration (FDA). This investigation and additional case finding are ongoing.

As of September 26, a total of 183 persons infected with the outbreak strain of *E. coli* O157:H7 had been reported to CDC from 26 states (Figure 1). Among the ill persons, 95 (52%) were hospitalized, 29 (16%) had hemolytic uremic syndrome (HUS), and one person died. The deaths of two other patients possibly related to this outbreak are under

FIGURE 1. Number of confirmed cases (N = 183)* of *Escherichia coli* serotype O157:H7 infection, by state — United States, September 2006



* Confirmed cases reported as of 1:00 p.m. EDT on September 26, 2006.

investigation. Eighty-five percent of patients reported illness onset from August 19 to September 5 (Figure 2). Fresh spinach was identified as the source of the outbreak. One hundred twenty-three of 130 patients (95%) reported consuming uncooked fresh spinach during the 10 days before illness onset. In addition, *E. coli* O157:H7 with a PFGE pattern matching the outbreak strain has been isolated from three open packages of fresh spinach consumed by patients (one from New Mexico, one from Utah, and one from Pennsylvania).

On September 14, FDA advised consumers by press release and press conference to not eat bagged fresh spinach. On September 15, a California company that bags spinach under several brand names announced a voluntary recall of all fresh spinach-containing products. On September 16, FDA expanded its warning and advised consumers to not eat fresh spinach or fresh spinach-containing products. On September 21, FDA informed consumers that only spinach grown in three California counties (Monterey, San Benito, and Santa Clara) was implicated in the outbreak.

A confirmed case is defined as a culture-confirmed *E. coli* O157:H7 infection in a person residing in the United States, with illness onset from August 1 to the present (or, if date of onset is unknown, *E. coli* O157:H7 isolated from August 15 to the present) and a PFGE pattern identified by the *Xba*I restriction enzyme that matches the pattern of the outbreak strain. August 1 was selected as the earliest illness onset date in the case definition to ensure that the earliest cases in the outbreak were identified and investigated. However, the first six confirmed cases (with illness onsets during August 2–15) were in persons who did not report fresh spinach consumption during the week before illness onset. The first date that

illness onset was reported by a person who recently consumed fresh spinach was August 19.

Infections with this outbreak strain of *E. coli* O157:H7 (one of 3,520 unique *E. coli* O157:H7 strains reported to CDC PulseNet since 1996) have been reported sporadically to CDC PulseNet since 2003 (an average of 21 cases per year during 2003–2005). This finding suggests the occasional presence of this strain in the environment and food supply; however, it has not been associated with a recognized outbreak in the past.

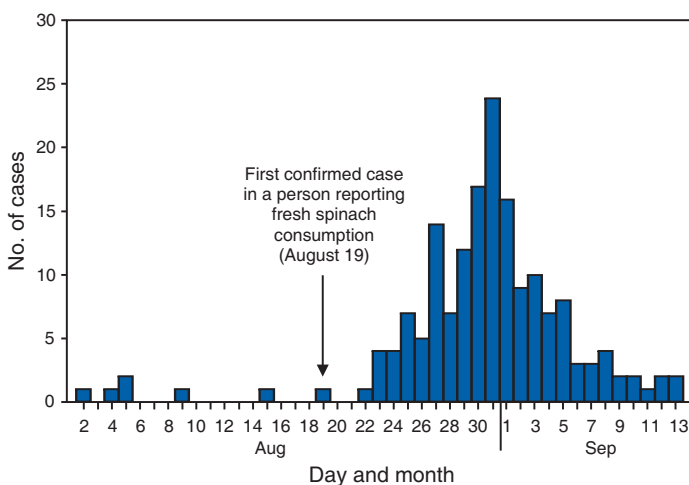
The time from illness onset to confirmation that a case of *E. coli* O157:H7 is part of an outbreak is typically 2–3 weeks, including the time required for an infected person to seek medical care and for health-care providers and public health officials to obtain a culture, transfer the bacterial culture to a public health laboratory, perform PFGE testing, and submit the PFGE pattern into the national database at CDC. In this outbreak, the average time from illness onset to PFGE pattern submission to the national database at CDC has been 15 days; additional information is available at <http://www.cdc.gov/foodborne/ecolispinach/reportingtimeline.htm>.

Parallel laboratory and epidemiologic investigations were crucial in identifying the source of this outbreak. Timely PFGE testing by state public health laboratories, PFGE pattern submission by states to CDC PulseNet, and analysis of PFGE patterns in the CDC PulseNet national database resulted in rapid detection of the outbreak. Concurrent collection of case exposure information by epidemiologists in affected states and sharing of exposure information among states and CDC led to rapid identification of the suspected food source and public health action. Continued rapid diagnosis, culture, PFGE analysis, and reporting to CDC of *E. coli* O157:H7 infections are needed to aid this investigation and to detect and investigate *E. coli* O157:H7 outbreaks in the future.

New information regarding the current *E. coli* O157:H7 outbreak will be available regularly. The most current information is available online at <http://www.cdc.gov/foodborne/ecolispinach>; this website contains information updated daily on the number of cases and affected states in addition to general information regarding *E. coli* O157:H7, resources for clinicians, and activities by CDC and other agencies. The FDA website, at <http://www.fda.gov/oc/opacom/hottopics/spinach.html>, contains advice for consumers on the current outbreak and food-safety guidelines. CDC's public inquiry line (telephone, 1-800-CDC-INFO) also can provide information on the current outbreak to both the public and health-care workers. Information about the current *E. coli* O157:H7 outbreak is also available by RSS (Really Simple Syndication); a subscription to the *E. coli* O157:H7 outbreak RSS information can be obtained at <http://www.bt.cdc.gov/rss>.

Reported by: State and local health departments. *E. coli* O157:H7 investigation team, CDC.

FIGURE 2. Number of confirmed cases (n = 171)* of *Escherichia coli* serotype O157:H7 infection, by date of illness onset — United States, August–September 2006



* Confirmed cases with known dates of illness onset reported as of 1:00 p.m. EDT on September 26, 2006.

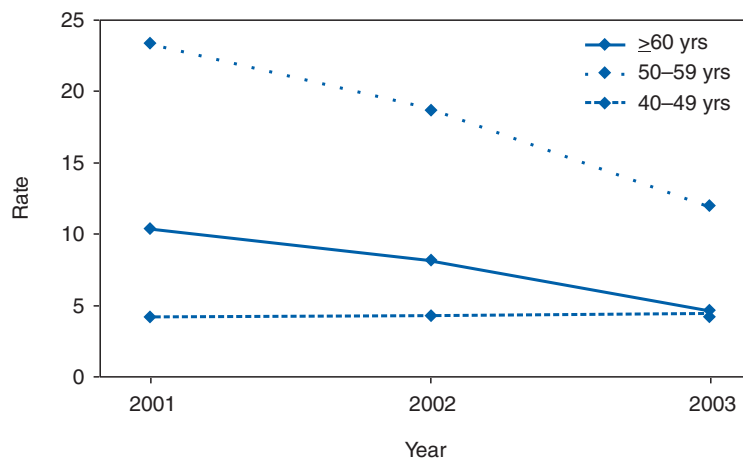
*Notice to Readers***The CDC Experience Application Deadline —
December 4, 2006**

The CDC Experience is a 1-year fellowship in applied epidemiology that is tailored for rising third- and fourth-year medical students and aims to develop a pool of physicians with a population health perspective. Eight competitively selected fellows spend 10–12 months at CDC in Atlanta, Georgia, where they conduct epidemiologic analyses in areas of public health that interest them. The fellowship environment provides multiple opportunities to enhance skills in research and analytic thinking, written and oral scientific presentations, and the practices of preventive medicine and public health.

Applicants do not need experience in public health to apply for this program. Through this training, fellows will acquire practical tools useful for approaching population-based health problems, whether in an entire community or among their own community of patients. Graduates of The CDC Experience have an appreciation of the role of epidemiology in medicine and health and are able to apply their knowledge and skills to enhance their clinical acumen and help improve the quality of the U.S. health-care system.

Information on applying for The CDC Experience is available at <http://www.cdcfoundation.org/thecdcexperience>. Applications for The CDC Experience fellowship class of 2006–07 must be postmarked by December 4, 2006. Questions can be addressed to Catherine Piper, program coordinator, at e-mail, cpiper@cdc.gov.

QuickStats

FROM THE NATIONAL CENTER FOR HEALTH STATISTICS**Annual Rate of Visits* to Office-Based Physicians and Hospital Outpatient Departments During Which Combination Estrogen-Progestin Hormone Therapy Was Prescribed for Women Aged ≥ 40 years, by Age Group — United States, 2001–2003**

* Per 100 women in age group.

From 2001 to 2003, the overall rate of visits to physicians during which combination estrogen-progestin hormone therapy was prescribed decreased by 44%. The decline was greatest among women aged ≥ 50 years. In July 2002, the National Institutes of Health terminated a clinical trial of combined hormone therapy (a component of the Women's Health Initiative) after investigators determined that the associated health risks outweighed the benefits.

SOURCE: Hing E, Brett K. Changes in U.S. prescribing patterns of menopausal hormone replacement therapy, 2001–2003. *Obstet Gynecol* 2006;108:33–40.

TABLE I. Provisional cases of infrequently reported notifiable diseases (<1,000 cases reported during the preceding year) — United States, week ending September 23, 2006 (38th Week)*

Disease	Current week	Cum 2006	5-year weekly average†	Total cases reported for previous years					States reporting cases during current week (No.)
				2005	2004	2003	2002	2001	
Anthrax	—	1	0	—	—	—	2	23	
Botulism:									
foodborne	1	4	0	19	16	20	28	39	AK (1)
infant	2	61	2	90	87	76	69	97	OH (1), WA (1)
other (wound & unspecified)	1	41	1	33	30	33	21	19	CA (1)
Brucellosis	—	72	2	122	114	104	125	136	
Chancroid	—	23	1	17	30	54	67	38	
Cholera	—	6	0	8	5	2	2	3	
Cyclosporiasis§	1	89	2	734	171	75	156	147	PA (1)
Diphtheria	—	—	—	—	—	1	1	2	
Domestic arboviral diseases§¶:									
California serogroup	—	25	8	80	112	108	164	128	
eastern equine	—	4	0	21	6	14	10	9	
Powassan	—	1	—	1	1	—	1	N	
St. Louis	—	2	2	13	12	41	28	79	
western equine	—	—	—	—	—	—	—	—	
Ehrlichiosis§:									
human granulocytic	25	271	11	790	537	362	511	261	NY (1), MN (23), CA (1)
human monocytic	7	258	9	522	338	321	216	142	NY (1), MN (6)
human (other & unspecified)	1	117	1	122	59	44	23	6	MO (1)
<i>Haemophilus influenzae</i> ,**									
invasive disease (age <5 yrs):									
serotype b	—	6	0	9	19	32	34	—	
nonserotype b	2	65	2	135	135	117	144	—	OH (1), NC (1)
unknown serotype	1	152	2	217	177	227	153	—	GA (1)
Hansen disease§	3	49	1	88	105	95	96	79	CA (3)
Hantavirus pulmonary syndrome§	—	24	0	29	24	26	19	8	
Hemolytic uremic syndrome, postdiarrheal§	11	158	5	221	200	178	216	202	NY (3), OH (4), MN (1), AZ (1), OR (1), CA (1)
Hepatitis C viral, acute	4	550	33	771	713	1,102	1,835	3,976	MN (2), MO (2)
HIV infection, pediatric (age <13 yrs)§,††	—	52	4	380	436	504	420	543	
Influenza-associated pediatric mortality§,§§,¶¶	1	42	0	49	—	N	N	N	CA (1)
Listeriosis	8	447	19	892	753	696	665	613	NY (2), MO (1), GA (3), FL (1), AL (1)
Measles	—***	43	1	66	37	56	44	116	
Meningococcal disease,††† invasive:									
A, C, Y, & W-135	—	162	3	297	—	—	—	—	
serogroup B	1	107	1	157	—	—	—	—	FL (1)
other serogroup	—	14	0	27	—	—	—	—	
Mumps	27	5,712	4	314	258	231	270	266	NY (2), MO (1), KS (5), NC (9), FL (1), OK (4), AZ (2), OR (1), CA (2)
Plague	—	12	0	8	3	1	2	2	
Poliomyelitis, paralytic	—	—	0	1	—	—	—	—	
Psittacosis§	—	17	0	19	12	12	18	25	
Q fever§	—	104	2	139	70	71	61	26	
Rabies, human	—	1	0	2	7	2	3	1	
Rubella	—	6	0	11	10	7	18	23	
Rubella, congenital syndrome	—	1	—	1	—	1	1	3	
SARS-CoV§,§§	—	—	—	—	—	8	N	N	
Smallpox§	—	—	—	—	—	—	—	—	
Streptococcal toxic-shock syndrome§	1	78	1	129	132	161	118	77	NC (1)
<i>Streptococcus pneumoniae</i> ,§									
invasive disease (age <5 yrs)	7	755	8	1,257	1,162	845	513	498	OH (2), MN (1), OK (3), AZ (1)
Syphilis, congenital (age <1 yr)	2	184	9	361	353	413	412	441	TX (2)
Tetanus	—	17	0	27	34	20	25	37	
Toxic-shock syndrome (other than streptococcal)§	—	67	2	96	95	133	109	127	
Trichinellosis	—	11	0	19	5	6	14	22	
Tularemia§	—	61	3	154	134	129	90	129	
Typhoid fever	2	196	10	324	322	356	321	368	MN (1), CA (1)
Vancomycin-intermediate <i>Staphylococcus aureus</i> §	—	2	0	2	—	N	N	N	
Vancomycin-resistant <i>Staphylococcus aureus</i> §	—	—	—	3	1	N	N	N	
Yellow fever	—	—	—	—	—	—	1	—	

—: No reported cases. N: Not notifiable. Cum: Cumulative year-to-date counts.

* Incidence data for reporting years 2005 and 2006 are provisional, whereas data for 2001, 2002, 2003, and 2004 are finalized.

† Calculated by summing the incidence counts for the current week, the two weeks preceding the current week, and the two weeks following the current week, for a total of 5 preceding years. Additional information is available at <http://www.cdc.gov/epo/dphsi/phs/files/5yearweeklyaverage.pdf>.

§ Not notifiable in all states.

¶ Includes both neuroinvasive and non-neuroinvasive. Updated weekly from reports to the Division of Vector-Borne Infectious Diseases, National Center for Zoonotic, Vector-Borne, and Enteric Diseases (proposed) (ArboNET Surveillance).

** Data for *H. influenzae* (all ages, all serotypes) are available in Table II.

†† Updated monthly from reports to the Division of HIV/AIDS Prevention, National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention (proposed). Implementation of HIV reporting influences the number of cases reported. Data for HIV/AIDS are available in Table IV quarterly.

§§ Updated weekly from reports to the Influenza Division, National Center for Immunization and Respiratory Diseases (proposed).

¶¶ A total of 47 cases were reported since the beginning of the 2005-06 flu season (October 2, 2005 [week 40]).

*** No measles cases were reported for the current week.

††† Data for meningococcal disease (all serogroups and unknown serogroups) are available in Table II.

TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending September 23, 2006, and September 24, 2005 (38th Week)*

Reporting area	Giardiasis					Gonorrhea					Haemophilus influenzae, invasive All ages, all serotypes				
	Current week	Previous 52 weeks		Cum 2006	Cum 2005	Current week	Previous 52 weeks		Cum 2006	Cum 2005	Current week	Previous 52 weeks		Cum 2006	Cum 2005
		Med	Max				Med	Max				Med	Max		
United States	227	312	1,029	11,343	13,559	3,912	6,494	14,136	236,035	239,539	14	38	142	1,490	1,709
New England	15	24	75	904	1,237	123	104	288	3,963	4,332	—	3	19	124	129
Connecticut	6	0	37	214	262	67	40	241	1,563	1,875	—	0	9	37	38
Maine†	2	2	13	118	158	5	2	6	92	100	—	0	4	17	8
Massachusetts	—	10	25	357	553	40	46	86	1,775	1,870	—	1	7	52	63
New Hampshire	1	0	9	23	47	—	4	9	142	123	—	0	2	6	7
Rhode Island	6	0	25	78	86	11	8	19	345	324	—	0	7	4	7
Vermont†	—	3	9	114	131	—	1	3	46	40	—	0	2	8	6
Mid. Atlantic	44	53	254	1,927	2,439	480	608	1,014	22,131	24,465	3	7	30	288	319
New Jersey	—	7	15	206	327	35	103	143	3,589	4,160	—	2	4	45	61
New York (Upstate)	32	24	227	834	832	186	123	455	4,552	4,875	1	2	27	98	95
New York City	2	8	32	348	657	105	161	357	5,983	7,296	1	1	4	32	58
Pennsylvania	10	15	29	539	623	154	212	393	8,007	8,134	1	3	8	113	105
E.N. Central	24	49	110	1,715	2,437	795	1,284	7,047	46,748	47,768	3	5	14	214	299
Illinois	—	10	25	317	577	151	376	709	13,978	14,366	—	1	6	47	101
Indiana	N	0	0	N	N	150	165	237	6,418	5,935	1	1	11	64	54
Michigan	5	13	24	459	593	441	252	5,880	10,443	8,029	—	0	3	17	18
Ohio	19	16	32	577	556	35	331	648	11,130	15,262	2	1	6	63	93
Wisconsin	—	10	40	362	711	18	130	172	4,779	4,176	—	0	4	23	33
W.N. Central	15	28	260	1,304	1,487	65	362	436	13,141	13,658	2	2	15	100	85
Iowa	5	5	14	207	201	—	34	46	1,199	1,157	—	0	1	1	—
Kansas	1	4	11	148	145	38	46	124	1,519	1,917	—	0	3	14	9
Minnesota	—	2	238	477	610	—	62	105	1,972	2,512	2	0	9	51	37
Missouri	9	9	32	334	334	—	189	251	7,100	6,889	—	0	6	24	26
Nebraska†	—	2	8	74	96	23	23	56	1,003	851	—	0	2	6	12
North Dakota	—	0	7	11	11	—	2	7	69	72	—	0	3	4	1
South Dakota	—	1	7	53	90	4	6	15	279	260	—	0	0	—	—
S. Atlantic	33	49	95	1,723	1,983	1,054	1,503	2,334	57,588	56,502	6	10	26	397	403
Delaware	—	1	4	30	43	34	26	44	1,075	619	—	0	1	1	—
District of Columbia	—	1	5	50	40	—	34	61	1,138	1,521	—	0	1	3	7
Florida	21	18	39	752	697	365	433	553	16,788	14,519	1	3	9	129	99
Georgia	12	10	44	368	528	12	305	1,014	10,217	10,679	3	2	12	79	87
Maryland†	—	4	11	141	146	125	128	186	4,810	5,062	—	1	5	50	53
North Carolina	N	0	0	N	N	123	283	766	12,193	11,428	2	0	9	46	65
South Carolina†	—	1	7	65	86	141	128	748	5,839	5,898	—	1	3	25	26
Virginia†	—	7	50	300	411	236	130	288	4,840	6,272	—	1	8	48	43
West Virginia	—	0	5	17	32	18	17	42	688	504	—	0	4	16	23
E.S. Central	4	8	40	318	301	252	572	859	21,520	20,096	—	2	7	78	91
Alabama†	4	4	29	165	135	20	183	310	6,901	6,518	—	0	5	20	17
Kentucky	N	0	0	N	N	5	55	132	2,288	2,248	—	0	1	4	10
Mississippi	—	0	0	—	—	—	141	435	5,222	5,106	—	0	1	3	—
Tennessee†	—	4	12	153	166	227	187	236	7,109	6,224	—	1	4	51	64
W.S. Central	5	6	31	185	227	568	878	1,430	33,585	33,152	—	1	15	47	93
Arkansas	3	2	6	82	63	78	78	142	2,956	3,318	—	0	2	7	7
Louisiana	1	0	4	13	45	—	158	354	5,941	7,164	—	0	2	4	32
Oklahoma	1	2	24	90	119	139	78	764	3,252	3,295	—	1	14	34	49
Texas†	N	0	0	N	N	351	548	820	21,436	19,375	—	0	2	2	5
Mountain	25	30	55	1,103	1,050	147	216	552	7,872	9,933	—	4	8	153	174
Arizona	2	3	36	112	98	131	90	201	3,236	3,603	—	1	7	72	89
Colorado	—	9	33	371	374	—	46	90	1,462	2,333	—	1	4	41	35
Idaho†	—	3	11	116	100	1	2	10	113	77	—	0	1	3	4
Montana	6	2	11	76	54	4	3	20	145	113	—	0	0	—	—
Nevada†	—	1	6	38	77	—	24	194	985	2,089	—	0	1	—	13
New Mexico†	2	1	6	44	60	—	30	64	1,242	1,165	—	0	4	19	20
Utah	15	7	19	319	269	10	17	24	603	499	—	0	4	15	7
Wyoming	—	1	4	27	18	1	2	6	86	54	—	0	2	3	6
Pacific	62	59	202	2,164	2,398	428	808	963	29,487	29,633	—	2	20	89	116
Alaska	6	1	7	56	78	13	11	23	429	424	—	0	19	9	25
California	33	43	105	1,551	1,706	337	662	830	24,362	24,671	—	0	9	21	47
Hawaii	—	1	3	37	49	—	18	29	667	750	—	0	1	13	8
Oregon†	6	7	15	286	315	37	28	58	979	1,110	—	1	6	44	36
Washington	17	6	90	234	250	41	75	142	3,050	2,678	—	0	4	2	—
American Samoa	U	0	0	U	U	U	0	2	U	U	U	0	0	U	U
C.N.M.I.	U	0	0	U	U	U	0	0	U	U	U	0	0	U	U
Guam	—	0	0	—	11	—	1	15	—	71	—	0	2	—	6
Puerto Rico	—	2	20	52	199	—	5	16	188	281	1	0	1	1	3
U.S. Virgin Islands	—	0	0	—	—	—	0	5	30	45	—	0	0	—	—

C.N.M.I.: Commonwealth of Northern Mariana Islands.

U: Unavailable. —: No reported cases. N: Not notifiable. Cum: Cumulative year-to-date counts. Med: Median. Max: Maximum.

* Incidence data for reporting years 2005 and 2006 are provisional.

† Contains data reported through the National Electronic Disease Surveillance System (NEDSS).

TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending September 23, 2006, and September 24, 2005 (38th Week)*

Reporting area	Hepatitis (viral, acute), by type										Legionellosis				
	A					B									
	Current week	Previous 52 weeks		Cum 2006	Cum 2005	Current week	Previous 52 weeks		Cum 2006	Cum 2005	Current week	Previous 52 weeks		Cum 2006	Cum 2005
	Med	Max				Med	Max				Med	Max			
United States	39	71	245	2,335	3,020	27	193	597	7,510	3,814	38	42	127	1,492	1,465
New England	2	3	20	140	351	—	1	9	46	112	10	2	12	80	100
Connecticut	2	1	3	33	41	—	0	3	—	37	6	0	8	25	22
Maine†	—	0	2	6	3	—	0	2	13	12	—	0	2	7	5
Massachusetts	—	1	13	51	218	—	0	5	14	37	—	1	6	27	45
New Hampshire	—	0	16	35	74	—	0	2	11	21	—	0	1	1	7
Rhode Island	—	0	4	8	10	—	0	4	8	1	4	0	10	16	16
Vermont†	—	0	2	7	5	—	0	1	—	4	—	0	3	4	5
Mid. Atlantic	6	7	20	229	496	1	118	199	5,002	500	15	13	43	491	498
New Jersey	—	2	7	55	104	—	2	10	75	191	—	1	10	60	90
New York (Upstate)	3	1	14	63	76	—	1	43	47	39	9	5	29	203	124
New York City	—	2	10	64	235	—	113	192	4,767	103	—	1	9	30	76
Pennsylvania	3	1	5	47	81	1	3	9	113	167	6	4	17	198	208
E.N. Central	2	6	12	201	265	4	7	24	267	416	7	9	25	323	303
Illinois	—	1	4	40	96	—	1	6	24	117	—	1	4	21	44
Indiana	1	0	5	21	13	3	0	17	42	28	—	0	6	22	20
Michigan	1	2	8	73	84	—	3	7	101	136	—	2	7	82	85
Ohio	—	1	4	44	39	1	2	10	94	101	7	4	19	165	129
Wisconsin	—	1	5	23	33	—	0	4	6	34	—	0	5	33	25
W.N. Central	—	2	30	93	69	2	4	22	117	203	—	1	15	48	59
Iowa	—	0	2	8	18	—	0	3	13	20	—	0	3	9	4
Kansas	—	0	5	24	13	—	0	2	8	24	—	0	2	3	2
Minnesota	—	0	29	9	3	1	0	13	17	27	—	0	11	11	16
Missouri	—	1	3	33	27	1	2	7	69	106	—	0	3	16	23
Nebraska†	—	0	3	12	8	—	0	1	10	21	—	0	2	5	2
North Dakota	—	0	2	—	—	—	0	0	—	—	—	0	1	—	2
South Dakota	—	0	3	7	—	—	0	1	—	5	—	0	6	4	10
S. Atlantic	16	11	30	395	531	9	23	66	837	1,037	3	8	19	299	290
Delaware	—	0	2	10	5	—	1	4	34	23	—	0	2	8	13
District of Columbia	—	0	2	5	2	—	0	2	5	10	—	0	5	16	8
Florida	11	4	14	157	214	7	8	19	301	357	2	3	9	125	79
Georgia	—	1	7	51	102	1	3	7	124	159	1	0	4	13	23
Maryland†	—	1	6	45	52	—	3	10	120	112	—	1	5	53	86
North Carolina	5	0	20	67	64	—	0	23	116	118	—	0	5	28	23
South Carolina†	—	0	2	15	30	—	2	7	55	121	—	0	1	2	11
Virginia†	—	1	11	40	59	—	1	18	38	110	—	1	7	46	33
West Virginia	—	0	3	5	3	1	0	18	44	27	—	0	3	8	14
E.S. Central	—	2	8	91	211	—	6	14	232	268	—	1	9	56	59
Alabama†	—	0	4	12	39	—	2	8	75	60	—	0	2	7	11
Kentucky	—	0	5	29	22	—	1	5	50	52	—	0	4	17	19
Mississippi	—	0	1	5	16	—	0	3	10	44	—	0	1	1	3
Tennessee†	—	1	5	45	134	—	2	8	97	112	—	1	7	31	26
W.S. Central	1	4	77	132	331	2	14	315	480	433	—	1	32	43	32
Arkansas	—	0	9	33	16	—	1	4	33	51	—	0	3	3	5
Louisiana	—	0	4	12	53	—	0	3	15	61	—	0	2	4	1
Oklahoma	1	0	2	5	4	2	0	17	30	31	—	0	3	1	6
Texas†	—	3	73	82	258	—	12	295	402	290	—	0	26	35	20
Mountain	1	5	18	190	227	—	4	39	123	397	2	2	7	86	74
Arizona	1	2	16	107	114	—	1	23	32	256	2	1	4	32	16
Colorado	—	1	4	32	33	—	1	5	28	41	—	0	2	16	17
Idaho†	—	0	2	9	18	—	0	2	10	10	—	0	2	9	3
Montana	—	0	3	9	7	—	0	7	—	3	—	0	1	5	5
Nevada†	—	0	2	7	17	—	0	4	14	41	—	0	2	3	16
New Mexico†	—	0	3	12	19	—	0	3	15	14	—	0	1	4	3
Utah	—	0	2	11	18	—	0	5	24	30	—	0	1	17	10
Wyoming	—	0	1	3	1	—	0	1	—	2	—	0	0	—	4
Pacific	11	20	163	864	539	9	9	61	406	448	1	2	9	66	50
Alaska	—	0	1	—	4	1	0	1	4	7	—	0	1	—	—
California	10	16	162	783	447	7	7	41	312	299	1	2	9	66	48
Hawaii	—	0	2	8	21	—	0	1	5	6	—	0	1	—	2
Oregon†	—	1	5	37	34	—	1	5	52	81	N	0	0	N	N
Washington	1	1	13	36	33	1	0	18	33	55	—	0	0	—	—
American Samoa	U	0	0	U	1	U	0	0	U	—	U	0	0	U	U
C.N.M.I.	U	0	0	U	U	U	0	0	U	U	U	0	0	U	U
Guam	—	0	0	—	2	—	0	0	—	18	—	0	0	—	—
Puerto Rico	—	0	5	21	56	—	1	8	24	36	—	0	1	1	—
U.S. Virgin Islands	—	0	0	—	—	—	0	0	—	—	—	0	0	—	—

C.N.M.I.: Commonwealth of Northern Mariana Islands.

U: Unavailable. —: No reported cases. N: Not notifiable. Cum: Cumulative year-to-date counts. Med: Median. Max: Maximum.

* Incidence data for reporting years 2005 and 2006 are provisional.

† Contains data reported through the National Electronic Disease Surveillance System (NEDSS).

TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending September 23, 2006, and September 24, 2005 (38th Week)*

Reporting area	Lyme disease					Malaria				
	Current week	Previous 52 weeks		Cum 2006	Cum 2005	Current week	Previous 52 weeks		Cum 2006	Cum 2005
		Med	Max				Med	Max		
United States	441	248	2,153	12,279	17,002	18	24	125	863	1,044
New England	73	37	780	2,073	3,002	—	1	11	44	55
Connecticut	72	10	753	1,475	490	—	0	5	11	11
Maine†	—	2	34	132	205	—	0	1	4	5
Massachusetts	—	2	37	33	2,064	—	0	3	19	31
New Hampshire	1	5	50	371	178	—	0	3	9	5
Rhode Island	—	0	5	1	27	—	0	8	—	2
Vermont†	—	1	9	61	38	—	0	1	1	1
Mid. Atlantic	181	153	1,176	7,118	9,862	8	4	13	151	284
New Jersey	—	25	165	1,578	3,036	—	1	3	28	68
New York (Upstate)	164	74	1,150	3,035	2,859	7	1	11	33	37
New York City	—	1	15	14	330	—	2	8	60	151
Pennsylvania	17	40	217	2,491	3,637	1	1	3	30	28
E.N. Central	1	11	123	1,049	1,568	1	2	7	91	113
Illinois	—	0	2	—	118	—	1	4	38	63
Indiana	—	0	3	16	24	1	0	3	9	4
Michigan	—	1	6	36	45	—	0	2	15	19
Ohio	1	1	6	37	48	—	0	3	22	17
Wisconsin	—	9	118	960	1,333	—	0	3	7	10
W.N. Central	167	7	91	491	596	—	0	32	32	40
Iowa	—	1	8	72	82	—	0	1	1	7
Kansas	—	0	2	4	3	—	0	2	6	5
Minnesota	167	6	88	398	494	—	0	30	14	11
Missouri	—	0	3	8	12	—	0	1	5	16
Nebraska†	—	0	1	8	3	—	0	2	4	1
North Dakota	—	0	3	—	—	—	0	1	1	—
South Dakota	—	0	1	1	2	—	0	1	1	—
S. Atlantic	6	28	103	1,288	1,782	4	6	15	242	227
Delaware	4	8	28	384	554	—	0	1	5	3
District of Columbia	—	0	7	37	8	—	0	2	3	8
Florida	1	1	5	27	31	3	1	6	46	38
Georgia	—	0	1	2	5	1	1	6	66	42
Maryland†	—	14	60	609	948	—	1	5	51	84
North Carolina	—	0	4	23	42	—	0	8	20	23
South Carolina†	—	0	3	8	18	—	0	2	8	7
Virginia†	—	3	25	189	166	—	1	9	41	21
West Virginia	1	0	44	9	10	—	0	2	2	1
E.S. Central	1	0	4	19	30	—	0	3	19	22
Alabama†	—	0	1	5	1	—	0	2	8	4
Kentucky	1	0	2	6	5	—	0	2	3	7
Mississippi	—	0	0	—	—	—	0	1	3	—
Tennessee†	—	0	2	8	24	—	0	2	5	11
W.S. Central	—	0	3	10	66	—	2	31	51	97
Arkansas	—	0	1	—	4	—	0	1	1	5
Louisiana	—	0	0	—	3	—	0	1	1	2
Oklahoma	—	0	0	—	—	—	0	6	7	9
Texas†	—	0	3	10	59	—	1	29	42	81
Mountain	—	0	4	19	19	—	1	9	51	42
Arizona	—	0	4	4	7	—	0	9	17	10
Colorado	—	0	1	4	—	—	0	2	11	20
Idaho†	—	0	1	2	2	—	0	1	1	—
Montana	—	0	0	—	—	—	0	1	2	—
Nevada†	—	0	1	1	3	—	0	1	1	2
New Mexico†	—	0	1	1	2	—	0	1	3	3
Utah	—	0	1	6	2	—	0	2	16	5
Wyoming	—	0	1	1	3	—	0	0	—	2
Pacific	12	4	20	212	77	5	4	13	182	164
Alaska	—	0	1	2	4	—	1	0	4	5
California	12	4	19	200	48	3	4	10	123	122
Hawaii	N	0	0	N	N	—	0	2	4	14
Oregon†	—	0	2	7	17	—	0	1	9	9
Washington	—	0	3	3	8	1	0	5	23	14
American Samoa	U	0	0	U	U	U	0	0	U	U
C.N.M.I.	U	0	0	U	U	U	0	0	U	U
Guam	—	0	0	—	—	—	0	0	—	—
Puerto Rico	N	0	0	N	N	—	0	1	—	3
U.S. Virgin Islands	—	0	0	—	—	—	0	0	—	—

C.N.M.I.: Commonwealth of Northern Mariana Islands.

U: Unavailable. —: No reported cases.

N: Not notifiable.

Cum: Cumulative year-to-date counts.

Med: Median.

Max: Maximum.

* Incidence data for reporting years 2005 and 2006 are provisional.

† Contains data reported through the National Electronic Disease Surveillance System (NEDSS).

TABLE II. (Continued) Provisional cases of selected notifiable diseases, United States, weeks ending September 23, 2006, and September 24, 2005 (38th Week)*

Reporting area	West Nile virus disease [†]									
	Neuroinvasive					Non-neuroinvasive				
	Current week	Previous 52 weeks		Cum 2006	Cum 2005	Current week	Previous 52 weeks		Cum 2006	Cum 2005
		Med	Max				Med	Max		
United States	—	1	152	956	1,165	1	1	326	1,653	1,594
New England	—	0	2	6	9	—	0	1	2	3
Connecticut	—	0	2	6	4	—	0	1	2	2
Maine [§]	—	0	0	—	—	—	0	0	—	—
Massachusetts	—	0	0	—	4	—	0	1	—	1
New Hampshire	—	0	0	—	—	—	0	0	—	—
Rhode Island	—	0	0	—	1	—	0	0	—	—
Vermont [§]	—	0	0	—	—	—	0	0	—	—
Mid. Atlantic	—	0	8	16	43	—	0	3	5	21
New Jersey	—	0	2	2	3	—	0	2	1	3
New York (Upstate)	—	0	4	—	17	—	0	1	—	4
New York City	—	0	4	7	9	—	0	2	3	3
Pennsylvania	—	0	2	7	14	—	0	1	1	11
E.N. Central	—	0	33	147	240	—	0	17	65	145
Illinois	—	0	19	92	125	—	0	16	47	108
Indiana	—	0	4	11	10	—	0	2	5	11
Michigan	—	0	5	17	51	—	0	1	2	7
Ohio	—	0	10	19	44	—	0	3	4	13
Wisconsin	—	0	2	8	10	—	0	2	7	6
W.N. Central	—	0	28	170	151	—	0	67	320	451
Iowa	—	0	3	13	12	—	0	4	11	22
Kansas	—	0	3	14	9	—	0	3	10	N
Minnesota	—	0	6	26	17	—	0	7	34	25
Missouri	—	0	8	35	16	—	0	3	8	13
Nebraska [§]	—	0	7	30	50	—	0	17	82	126
North Dakota	—	0	4	18	12	—	0	26	108	74
South Dakota	—	0	7	34	35	—	0	21	67	191
S. Atlantic	—	0	3	7	28	—	0	2	3	24
Delaware	—	0	0	—	1	—	0	1	—	—
District of Columbia	—	0	1	—	2	—	0	1	1	1
Florida	—	0	2	3	8	—	0	0	—	11
Georgia	—	0	1	2	7	—	0	2	2	8
Maryland [§]	—	0	1	1	4	—	0	0	—	1
North Carolina	—	0	0	—	2	—	0	0	—	2
South Carolina [§]	—	0	1	—	4	—	0	0	—	—
Virginia [§]	—	0	0	—	—	—	0	1	—	1
West Virginia	—	0	1	1	—	N	0	0	N	N
E.S. Central	—	0	12	78	58	—	0	13	67	30
Alabama [§]	—	0	1	4	5	—	0	2	—	2
Kentucky	—	0	1	2	3	—	0	1	1	—
Mississippi	—	0	9	65	37	—	0	13	65	27
Tennessee [§]	—	0	3	7	13	—	0	1	1	1
W.S. Central	—	1	46	237	224	—	0	23	122	140
Arkansas	—	0	3	13	11	—	0	1	4	15
Louisiana	—	0	13	57	99	—	0	8	46	51
Oklahoma	—	0	6	19	10	—	0	3	9	9
Texas [§]	—	1	28	148	104	—	0	14	63	65
Mountain	—	0	57	239	116	—	0	192	899	219
Arizona	—	0	8	10	32	—	0	6	10	45
Colorado	—	0	9	40	19	—	0	32	159	81
Idaho [§]	—	0	29	94	3	—	0	128	542	10
Montana	—	0	3	10	8	—	0	7	19	17
Nevada [§]	—	0	9	33	11	—	0	13	71	17
New Mexico [§]	—	0	2	1	17	—	0	1	2	13
Utah	—	0	7	40	21	—	0	15	74	30
Wyoming	—	0	5	11	5	—	0	6	22	6
Pacific	—	0	13	56	296	1	0	39	170	561
Alaska	—	0	0	—	—	—	0	0	—	—
California	—	0	13	54	295	1	0	30	150	555
Hawaii	—	0	0	—	—	—	0	0	—	—
Oregon [§]	—	0	1	2	1	—	0	9	19	6
Washington	—	0	0	—	—	—	0	1	1	—
American Samoa	U	0	0	U	U	U	0	0	U	U
C.N.M.I.	U	0	0	U	U	U	0	0	U	U
Guam	—	0	0	—	—	—	0	0	—	—
Puerto Rico	—	0	0	—	—	—	0	0	—	—
U.S. Virgin Islands	—	0	0	—	—	—	0	0	—	—

C.N.M.I.: Commonwealth of Northern Mariana Islands.

U: Unavailable. —: No reported cases.

N: Not notifiable.

Cum: Cumulative year-to-date counts.

Med: Median.

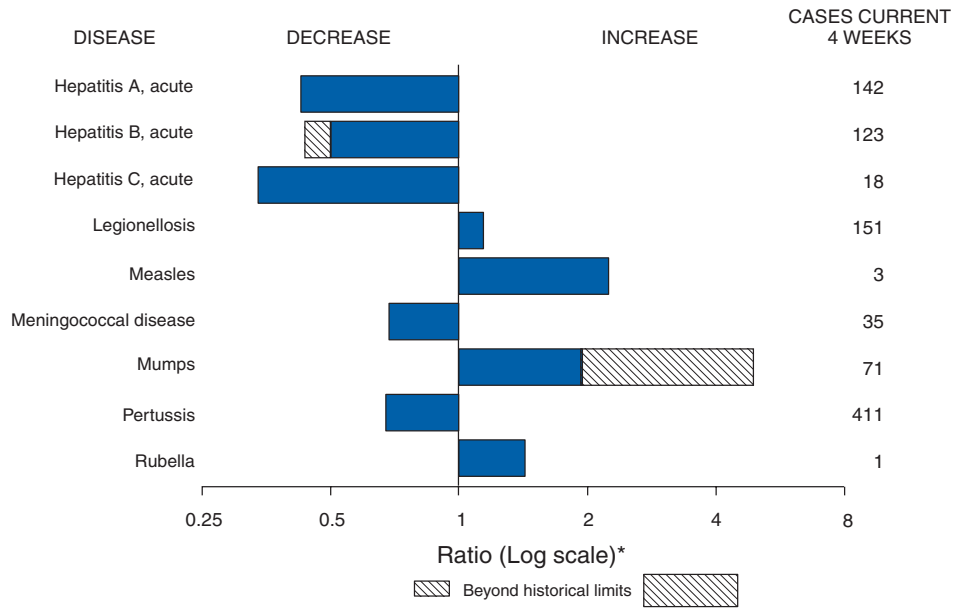
Max: Maximum.

* Incidence data for reporting years 2005 and 2006 are provisional.

† Updated weekly from reports to the Division of Vector-Borne Infectious Diseases, National Center for Zoonotic, Vector-Borne, and Enteric Diseases (proposed) (ArboNET Surveillance).

§ Contains data reported through the National Electronic Disease Surveillance System (NEDSS).

FIGURE I. Selected notifiable disease reports, United States, comparison of provisional 4-week totals September 23, 2006, with historical data



* Ratio of current 4-week total to mean of 15 4-week totals (from previous, comparable, and subsequent 4-week periods for the past 5 years). The point where the hatched area begins is based on the mean and two standard deviations of these 4-week totals.

Notifiable Disease Morbidity and 122 Cities Mortality Data Team
 Patsy A. Hall
 Deborah A. Adams Rosaline Dhara
 Willie J. Anderson Vernitta Love
 Lenee Blanton Pearl C. Sharp

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format. To receive an electronic copy each week, send an e-mail message to listserv@listserv.cdc.gov. The body content should read *SUBscribe mmwr-toc*. Electronic copy also is available from CDC's Internet server at <http://www.cdc.gov/mmwr> or from CDC's file transfer protocol server at <ftp://ftp.cdc.gov/pub/publications/mmwr>. Paper copy subscriptions are available through the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone 202-512-1800.

Data in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the following Friday. Data are compiled in the National Center for Public Health Informatics, Division of Integrated Surveillance Systems and Services. Address all inquiries about the *MMWR* Series, including material to be considered for publication, to Editor, *MMWR* Series, Mailstop E-90, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333 or to www.mmwrq@cdc.gov.

All material in the *MMWR* Series is in the public domain and may be used and reprinted without permission; citation as to source, however, is appreciated.

Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services.

References to non-CDC sites on the Internet are provided as a service to *MMWR* readers and do not constitute or imply endorsement of these organizations or their programs by CDC or the U.S. Department of Health and Human Services. CDC is not responsible for the content of these sites. URL addresses listed in *MMWR* were current as of the date of publication.