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Approval of a New Rapid Test for HIV Antibody

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ON NOVEMBER 7, 2002, THE FOOD AND Drug Administration announced approval of the OraQuick Rapid HIV-1 Antibody Test (OraSure Technologies, Inc., Bethlehem, Pennsylvania) for use by trained personnel as a point-of-care test to aid in the diagnosis of infection with human immunodeficiency virus type 1 (HIV-1). OraQuick is a simple, rapid test that can detect antibodies to HIV in fingerstick whole blood specimens and provide results in ≤20 minutes. The test has been categorized as moderate complexity under the Clinical Laboratory Improvement Amendments of 1988 (CLIA). A second FDA-approved moderate-complexity rapid HIV test, Single Use Diagnostic System for HIV-1(Abbott-Murex Inc., Norcross, Georgia), remains available in the United States for use with serum or plasma specimens.

Use of a rapid test that allows same-day results can substantially increase the number of persons who receive their test results, which improves the delivery of counseling and treatment services.[1] On the basis of data submitted by the manufacturer for test approval, the sensitivity* of OraQuick in the clinical studies performed was 99.6% (95% confidence interval [CI] = 98.5%-99.9%), and specificity was 100% (95% CI = 99.7%-100%), comparable to those of FDA-approved enzyme immunoassays in widespread use. Because HIV prevalence is low in most U.S. testing settings, the negative predictive value† of screening with a single rapid test is high. Therefore, a negative rapid HIV test does not require further testing, and negative results with counseling can be provided at the initial visit. Retesting is recommended for those persons with a recent (within 3 months) history of known or possible exposure to HIV because there might have been insufficient time for detectable antibodies to develop.[2] As with any HIV screening test, all reactive (preliminary positive) rapid test results should be confirmed by supplemental testing by either a Western blot or immunofluorescence assay.[3] The confirmatory tests can be performed on serum specimens obtained by phlebotomy, dried blot spots obtained on filter paper, or oral fluid specimens collected with the OraSure collection device.

Persons whose rapid-test results are reactive should be counseled about their likelihood of being infected with HIV and precautions to prevent HIV transmission, but they should return for definitive test results before medical referrals or partner counseling is initiated.[3] A simple message to convey this information could be a statement that "Your preliminary test result was positive, but we won't know for sure if you are HIV-infected until we get the results from your confirmatory test. In the meantime, you should take precautions to avoid possibly transmitting the virus."

The Public Health Service recommends that rapid HIV tests should be used and preliminary positive test results provided when tested persons might benefit.[1] Decisions about whether to use rapid tests should be based on considerations of return rates for standard test results and urgency of the need for test results (i.e., when necessary to make decisions about postexposure or perinatal prophylaxis).[1,4,5] The use of rapid tests will facilitate the acceptance of HIV testing and improve receipt of results in other health-care settings in which HIV testing is recommended, such as hospitals and acute-care clinics, where persons who are unaware of their HIV status might seek health-care services.[6] Additional information and guidance on the use of rapid HIV tests are available from CDC at http://www.cdc.gov/hiv/testing.htm.

Sites wanting to perform this new HIV-1 rapid test that are not already certified to perform moderate-complexity laboratory tests under CLIA must enroll in the CLIA program, administered by the Centers for Medicare and Medicaid Services. The application and state agency contact information are available at http://www.cms.hhs.gov/clia. Information about enrollment and the requirements for moderate-complexity testing are available at http://www.phppo.cdc.gov/clia/default.asp.

CLIA moderate-complexity requirements provide minimum standards for personnel, quality control, proficiency testing, and quality assurance. In addition, some states have specific requirements that might apply to laboratory testing in general or to HIV testing specifically.

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*Sensitivity is the probability that the test result will be reactive if the specimen is a true positive; specificity is the probability that the test result will be nonreactive if the specimen is a true negative.
†The predictive value of a screening test is the probability that the test accurately predicts the true infection status of the person tested.

Increases in Fluoroquinolone-Resistant *Neisseria gonorrhoeae*—Hawaii and California, 2001

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2 figures omitted

NEISSERIA GONORRHOEAE IS A MAJOR cause of pelvic inflammatory disease, ectopic pregnancy, and infertility, and it can facilitate human immunodeficiency virus (HIV) transmission.[1] Gonorrhea is the second most frequently reported communicable disease in the United States, with 361,705 reported cases in 2001.[2] During the 1980s, gonococcal resistance to penicillin and tetracycline became widespread; as a result, CDC recommended using cephalosporins as first-line treatment for gonorrhea. Since 1993, CDC also has recommended using fluoroquinolones (i.e., ciprofloxacin, ofloxacin, or levofloxacin) for gonorrhea treatment. Fluoroquinolone therapy is used widely because it is a relatively inexpensive, oral, and single-dose therapy. However, fluoroquinolone-resistant *N. gonorrhoeae* (QRNG)* is being identified more frequently.[3] This report summarizes investigations of increases in QRNG in Hawaii and California in 2001 and provides data to support the recommendation that cephalosporins (i.e., ceftriaxone or cefixime) be used instead of fluoroquinolones as first-line treatment for gonorrhea acquired in these two states. The increases in QRNG highlight the importance of monitoring gonococcal resistance throughout the United States to guide local treatment decisions.

Hawaii

In 2001, the Hawaii State Laboratory performed gonorrhea culture and antimicrobial susceptibility tests on specimens from 265 (44%) of 605 reported gonorrhea cases. Patients seeking care at the public sexually transmitted disease (STD) clinic accounted for 44% (117 of 265) of these isolates. Overall, QRNG accounted for 20% (53 of 265) of gonococcal isolates tested, compared with 11% in 2000 and 10% in 1999. In 2001, 36% (19 of 53) of QRNG infections were among STD clinic patients.

Medical and interview records of the 117 STD clinic patients with positive gonococcal cultures diagnosed during January-December 2001 were reviewed to identify risk factors for QRNG; 19 (16%) had QRNG isolates. QRNG prevalence was higher for men who had sex exclusively with women than for men who had sex with men (MSM) (11 [20%] of 55 versus one [3%] of 29; p=0.05). Persons with a history of recent travel to Asia or a sex partner with such a history were not significantly more likely to have QRNG (four [36%] of 11) than persons without such a history (14 [14%] of 102; p=0.07). Unlike in Hawaii in 1999,[4] QRNG prevalence was not significantly higher among Asians/Pacific Islanders than among non-Asians/Pacific Islanders (10 [19%] of 54 versus nine [14%] of 63; p=0.54).

Since 2000, the Hawaii Department of Health (HDH) has recommended that clinicians avoid using fluoroquinolones to treat gonorrhea. Because of the 25% increase in reported gonorrhea morbidity (from 39.9 cases per 100,000 population in 2000 to 49.9 in 2001), adherence to this recommendation is particularly important. In February 2002, HDH informed all clinicians of the increases in gonorrhea and QRNG and organized STD training for an expanded network of clinicians and workers in community-based organizations. Preliminary analysis of gonococcal susceptibility results for 147 patients during January-June 2002 suggests that QRNG prevalence remains >14%.

California

San Francisco, Long Beach, Orange County, and San Diego are participants in the Gonococcal Isolate Surveillance Project (GISP), a CDC-sponsored sentinel surveillance system that monitors antimicrobial resistance in *N. gonorrhoeae* through antimicrobial susceptibility testing of male urethral gonococcal isolates obtained from patients at public STD clinics in 26 U.S. cities. During 1990-2000, <1% of isolates tested annually from each GISP site in California were QRNG, except for Orange County, where 5.6% (six of 107) of GISP isolates were QRNG in 2000. In 2001, susceptibility testing was expanded beyond the GISP sample to include all gonococcal isolates from Orange County and San Diego STD clinic patients, including those from women and nonurethral sites. Susceptibility testing also was performed on all gonococcal isolates obtained from patients at a large southern California health maintenance organization (HMO) during February-April 2001. In 2001, QRNG was identified in 2.5% (33 of 1,311) of patients with tested isolates. Among STD clinic patients with gonorrhea, 3.4% (10 of 297) in San Francisco, 3.0% (three of 99) in Long Beach, 3.3% (seven of 212) in Orange County, and 2.4% (eight of 330) in San Diego had QRNG. Among HMO patients with gonorrhea, 1.3% (five of 373) had QRNG. The 1,311 patients with tested isolates accounted for 5.6% of all reported gonorrhea cases in California in 2001. Among 29 men infected with QRNG in 2001 whose sexual orientation was known, 20 (69%) were MSM. Among MSM with QRNG, 19 had a median of three recent (within 2-6 months) sex partners (range: one–40); 10 heterosexual men and women with QRNG had a median of 1.5 recent sex partners (range: one–eight), indicating the potential for more rapid spread among MSM. Although 12 (43%) of 28 QRNG patients interviewed in 2001 reported recent travel to Asia, the Pacific Islands, or Hawaii by themselves or a sex partner, 57% denied such travel, suggesting endemic spread of QRNG within California.

Medical records were reviewed for all 469 gonorrhea patients whose isolates

were tested for susceptibility and who were seen in San Francisco, Long Beach, Orange County, or San Diego STD clinics during July 1–December 31, 2001. QRNG was identified in 23 (4.9%) of the 469 patients tested. QRNG was more common among Asians/Pacific Islanders than among non-Asians/Pacific Islanders (four [16.7%] of 24 versus 19 [4.4%] of 427; p=0.03). QRNG prevalence was similar among MSM (5.2% [15 of 289]), heterosexual men (4.7% [seven of 149]), and women (3.6% [one of 28]). However, geographic variation was noted in California: in San Diego, QRNG was more common among MSM than among heterosexual men and women (6.6% [seven of 106] versus zero of 65; p=0.03), and in San Francisco, QRNG was more common among heterosexuals than among MSM (11.4% [five of 44] versus 1.1% [one of 93]; p=0.01).

In response to the increasing prevalence of QRNG, in May 2002 the California Department of Health Services advised clinicians to avoid using fluoroquinolones for treatment of gonorrhea. Preliminary data collected during January-June 2002 indicate that the prevalence of QRNG infection among STD clinic patients with tested gonococcal isolates in GISP sites in California has increased, exceeding 9% during this period.

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CDC Editorial Note: These data demonstrate that in 2001, QRNG prevalence increased in Hawaii and in California, where the epidemiology of QRNG varies within the state. In California, antimicrobial susceptibility data are available for a smaller proportion of reported gonorrhea cases than in Hawaii (6% versus 44%). Demographic data suggest that this low proportion might limit the generalizability of California's findings: patients with susceptibility-tested isolates in California were more likely to be male, older, and white, and to have their condition diagnosed in STD clinics than were other gonorrhea patients. However, the data from California indicate that QRNG has reached the continental United States, increasing the risk for its spread. Sporadic cases of QRNG have been identified in other states through GISP and non-GISP reporting, but no sustained increase in QRNG >1% has been identified in any other state.[3] Increases in QRNG in California and Hawaii highlight the ongoing need for monitoring antimicrobial susceptibilities of gonococcal isolates throughout the United States.

CDC recommends that fluoroquinolones not be used to treat gonococcal infections acquired in Asia, where QRNG prevalence exceeds 40%[5]; in the Pacific Islands, including Hawaii; in California; and in other areas with increased prevalence of fluoroquinolone resistance.[6] The recommended treatment options for persons who might have acquired infection in those areas are cefixime,[7] ceftriaxone, or spectinomycin. To select appropriate gonorrhea treatment in areas outside Hawaii and California, clinicians should ask suspected gonorrhea patients about their recent travel history and that of their sex partners.[8]

Treatment of gonorrhea with fluoroquinolones can continue in areas where the prevalence of resistance is <1%.[9] In areas where resistance is ≥1%, health departments making local treatment recommendations for gonorrhea also should consider other local factors such as the overall prevalence of gonorrhea, the availability of antimicrobial susceptibility data, and the cost of various diagnostic and treatment options.[10] Fluoroquinolones remain an important gonorrhea treatment option in the United States because they are inexpensive and easy to administer. In addition, their use might decrease use of cephalosporins and delay the development of cephalosporin resistance.

As part of effective gonorrhea control, state health departments should monitor local gonococcal antimicrobial susceptibility prevalence routinely to assist in developing local treatment recommendations. Symptomatic treatment failures are not a reliable indicator of emerging antimicrobial resistance because gonococcal infections, especially in women, are frequently asymptomatic. In 2001, a survey of STD project areas found that nonculture gonococcal tests were used widely and that approximately half of project areas had antimicrobial susceptibility data.[3] Because nonculture tests cannot provide antimicrobial susceptibility results, local gonococcal culture capacity should be maintained. The antimicrobial susceptibility testing panel should, at a minimum, include a fluoroquinolone, cefixime, ceftriaxone, spectinomycin, azithromycin, and any other drugs in local use for gonorrhea treatment.

In cases of persistent gonococcal infection after treatment, clinicians should consider performing culture and antimicrobial susceptibility testing. In areas where fluoroquinolones are used for treating gonorrhea and small numbers of patients with QRNG are identified, health departments should notify and treat partners of patients with known QRNG to minimize the spread of resistance. Through their state and local health departments, clinicians and laboratorians should report treatment failures or resistant gonococcal isolates to CDC, telephone 404-639-8373; isolates may be submitted to CDC's Neisseria Reference Laboratory for confirmation testing, telephone 404-639-3470.

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*Defined as *N. gonorrhoeae* resistant to ciprofloxacin (minimal inhibitory concentration [MIC] ≥1.0 µg/mL by agar dilution or disk diffusion zone size ≤27 mm) or ofloxacin (MIC ≥2.0 µg/mL or disk diffusion zone size ≤24 mm) by the National Committee on Clinical Laboratory Standards.

Discontinuation of Cefixime Tablets—United States

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IN JULY 2002, WYETH PHARMACEUTICALS (Collegeville, Pennsylvania) discontinued manufacturing cefixime (Suprax®) in the United States. In October 2002, the company ceased marketing cefixime tablets (200 mg and 400 mg) because of depletion of company inventory. Wyeth's patent for cefixime expired on November 10, 2002. No other pharmaceutical company manufactures or sells cefixime tablets in the United States. Wyeth will continue to sell cefixime suspension (100 mg/5 ml) until March 31, 2003, or until company inventory is depleted, whichever is sooner.

Cefixime is the only CDC-recommended oral antimicrobial agent to which *Neisseria gonorrhoeae* has not developed significant resistance.[1] Uncomplicated *N. gonorrhoeae* infections may be treated with single-dose regimens of cefixime 400 mg orally, ceftriaxone 125 mg intramuscularly, or an oral fluoroquinolone (ciprofloxacin 500 mg, levofloxacin 250 mg, or ofloxacin 400 mg). However, fluoroquinolones should not be used for treatment of gonorrhea if the infection was acquired in Asia, the Pacific Islands (including Hawaii), or California because the prevalence of fluoroquinolone-resistant *N. gonorrhoeae* is high in those areas.[1,2]

In the absence of cefixime, the primary recommended treatment option for gonorrhea in Hawaii and California is ceftriaxone. Also, in the absence of cefixime, ceftriaxone is the only CDC-recommended gonorrhea treatment option for young children and pregnant women throughout the United States. Fluoroquinolones can continue to be used for treating gonorrhea in areas of the United States with low prevalence of fluoroquinolone-resistant *N. gonorrhoeae*, but antimicrobial susceptibility monitoring should routinely be performed.[2] Other oral antimicrobial agents, such as cefpodoxime, cefuroxime axetil, and azithromycin, are not recommended by CDC for the treatment of gonorrhea. Additional information on the use of oral antimicrobials in treating *N. gonorrhoeae* infections will be available from CDC at http://www.cdc.gov/std.

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Primary and Secondary Syphilis—United States, 2000-2001

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2 figures, 2 tables omitted

IN OCTOBER 1999, CDC, IN COLLABORATION with other federal partners, initiated the National Plan to Eliminate Syphilis in the United States. Syphilis elimination is defined as the absence of sustained transmission (i.e., no transmission after 90 days of the report of an imported index case). The national goals for syphilis elimination are to reduce the annual number of primary and secondary (P&S) syphilis cases to <1,000 cases (rate: 0.4 per 100,000 population) and to increase the number of syphilis-free counties to 90% by 2005.[1] To characterize the epidemiology of syphilis in the United States, CDC analyzed national notifiable disease surveillance data for 2000-2001. This report summarizes the results of that analysis, which indicate that the number of reported cases of P&S syphilis increased slightly in 2001. This increase occurred only among men; the number of P&S syphilis cases continued to decline among women and among non-Hispanic blacks. The available data indicate that syphilis cases occurring among men who have sex with men (MSM) contributed to the increase in cases. The data suggest that, although efforts to reduce syphilis among women and non-Hispanic blacks appear effective and should continue, efforts to prevent and treat syphilis among MSM need to be improved.

Data for syphilis cases reported to state health departments and the District of Columbia during 2000-2001 were sent weekly to CDC. These data included information about each patient's county of residence, sex, stage of disease, racial/ ethnic group, and age

group. Data on reported cases of P&S syphilis were analyzed for this report because these cases represented incidence (i.e., newly acquired infections within the evaluated time) better than reported cases of latent infection, which were acquired months or years before diagnosis. P&S syphilis rates were calculated by using population denominators from the U.S. Bureau of the Census; the 2001 rates and numbers of cases were compared with 2000 data.[2]

After declining every year since 1990, the number of reported cases of P&S syphilis increased slightly in 2001. In 2000, the rate of P&S syphilis in the United States declined to 2.1 cases per 100,000 population, the lowest rate since reporting began in 1941.[2] In 2001, the rate of P&S syphilis increased slightly, to 2.2, the first annual rate increase since 1990, and 6,103 cases of P&S syphilis were reported, a 2.1% increase in reported cases compared with 2000.

In 2001, rates of P&S syphilis were 114.3% higher for men than for women. During 2000-2001, the rate increased 15.4% among men and decreased 17.6% among women; the male-to-female P&S syphilis case ratio increased 50% (from 1.4:1 to 2.1:1). Increases in male-to-female case ratios occurred among all racial/ethnic groups.

In 2001, the rate of P&S syphilis among non-Hispanic blacks was 15.7 times the rate reported among non-Hispanic whites. Non-Hispanic blacks accounted for 62.5% of cases in 2001 and 70.9% in 2000. During 2000-2001, the rate among non-Hispanic blacks declined 9.8%, reflecting a 3.5% decrease in the number of cases among men (from 2,371 to 2,289) and an 18.1% decrease among women (from 1,864 to 1,523). The rate among non-Hispanic whites increased 40.0%; cases among men increased 63.0% (from 698 to 1,138), and cases among women decreased 35.3% (from 385 to 249). The rate among Hispanics increased 31.0%; cases among men increased 50.1% (from 405 to 608), and cases among women decreased 9.3% (from 162 to 147). The rate among Asians/Pacific Islanders increased 66.7%; cases among men increased 79.3% (from 29 to 52), and cases among women decreased from eight to four. The rate among American Indians/Alaska Natives increased 75.0%; cases increased among men (from 26 to 49) and women (from 26 to 41).

By region,* the South had the highest rate, accounting for 56.2% of cases occurring in 2001 and 62.0% in 2000. During 2000-2001, rates decreased 8.1% in the South and 10.0% in the Midwest but increased 40.0% in the West and 57.1% in the Northeast. Rates decreased in 16 states, remained the same in nine states, and increased in 25 states and the District of Columbia.

In 2001, no cases of P&S syphilis were reported in 2,516 (80.2%) of 3,139 U.S. counties, and 2,533 (80.7%) counties reported rates less than or equal to the national health objective for 2010 of 0.2 cases per 100,000 persons (objective no. 25-3).[3] In 2001, 20 counties and one city accounted for 50.6% of all reported P&S syphilis cases in the United States. During 2000-2001, the overall rate for 63 of the largest cities in the U.S. with >200,000 population increased 9.1%, from 4.4 per 100,000 persons to 4.8.

Reported by: State and local health depts. JD Heffelfinger, MD, HS Weinstock, MD, SM Berman, MD, EB Swint, MS, Div of Sexually Transmitted Disease Prevention, National Center for HIV, STD, and TB Prevention, CDC.

CDC Editorial Note: The pattern of syphilis infection in the United States has changed during recent years. Although the South continues to have the highest rate of P&S syphilis, disease was less concentrated in this region. Racial/ethnic disparities in syphilis rates are decreasing because of declining rates among non-Hispanic blacks and increasing rates among non-Hispanic whites.

During 2000-2001, the number of cases of P&S syphilis increased among men, ending the decade-long trend characterized by annual declines in syphilis cases among both men and women. This increase in syphilis cases among men is associated with reports in several cities of syphilis outbreaks among MSM[4-9]; these outbreaks were characterized by high rates of human immunodeficiency virus co-infection and high-risk sexual behavior among subpopulations of MSM. Although syphilis cases reported nationally do not include information on behavior risk, the continuing decline in syphilis rates among women in conjunction with the increasing male-to-female case ratio suggests that the syphilis rate probably is increasing among MSM and decreasing among heterosexual men.

The findings in this report are subject to at least two limitations. First, the quality of surveillance data vary at local and state levels, and syphilis reporting is incomplete. Second, because cases among patients attending public-sector clinics might be more likely to be reported than cases diagnosed in the private sector and persons of minority race/ethnicity might be more likely to attend public clinics, the racial/ ethnic differences in reported rates might be magnified.

The National Syphilis Elimination Plan announced by CDC in 1999 focused initially on reducing syphilis in the South and among minority populations. Rates of syphilis in the South and among non-Hispanic blacks and women have declined every year since 1997. Ensuring continued progress toward syphilis elimination will require that syphilis trends be monitored and that elimination efforts be maintained among these populations. However, the increase in cases among MSM underscores the need to modify the syphilis elimination plan to develop and implement more effective prevention activities among MSM.[7] National efforts are under way to collect information on behavior to permit better monitoring of syphilis trends among MSM and heterosexual persons, study ethnographic and other factors associated with increases in syphilis among MSM, and improve programs to prevent and treat syphilis. To sustain progress toward syphilis elimination, communities must understand local patterns of syphilis transmission and develop effective, targeted intervention strategies that include education, risk reduction, and appropriate screening and treatment of persons at risk for this disease.

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**Northeast* = Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont; *Midwest* = Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, and Wisconsin; *South* = Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia; *West* = Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, and Wyoming.

Probable Variant Creutzfeldt-Jakob Disease in a US Resident—Florida, 2002

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ON APRIL 18, 2002, THE FLORIDA DEPARTMENT of Health and CDC announced the occurrence of a likely case of variant Creutzfeldt-Jakob disease (vCJD) in a Florida resident aged 22 years. This report documents the investigation of this case and underscores the importance of physicians increasing their suspicion for vCJD in patients presenting with clinical features described in this report who have spent time in areas in which bovine spongiform encephalopathy (BSE) is endemic.

In early November 2001, the patient sought medical care for depression and memory loss that adversely affected the patient's work performance. The primary-care physician referred the patient to a psychologist. In early December 2001, the patient received a traffic ticket for failing to yield the right of way. In mid-December 2001, the patient had involuntary muscular movements, gait changes, difficulty dressing, and incontinence. In January 2002, the patient was evaluated in a local emergency department for these symptoms. A computerized tomography scan of the head revealed no abnormalities; a panic attack was diagnosed, and the patient was treated with an antianxiety medication.

In late January 2002, the patient's mother, a resident of the United Kingdom, took the patient to England, where medical evaluations were conducted during the next 3 months. During this period, the patient's memory loss and other neurologic symptoms worsened. The patient experienced falls with minor injuries, had difficulty taking a shower and dressing, and was unable to remember a home telephone number or to make accurate mathematical calculations. The patient subsequently became confused, hallucinated, and had speech abnormalities with lack of content, bradykinesia, and spasticity. The patient was referred to a neurologist, who suspected vCJD and subsequently referred the patient to the National Prion Clinic in the United Kingdom.

Medical evaluations at the National Prion Clinic included an electroencephalogram (EEG), which revealed a normal alpharhythm, and magnetic resonance imaging (MRI) studies, which revealed signal abnormalities in the pulvinar and metathalamus region that were suggestive of vCJD. The patient had a tonsil biopsy, and a Western blot analysis of the biopsy tissue demonstrated the presence of protease-resistant prion protein (PrP-res) with the characteristic pattern of vCJD; an immunohistochemical test for PrP-res also supported a diagnosis of vCJD. Analysis of the prion protein gene detected no mutation and showed methionine homozygosity at codon 129, consistent with all 105 vCJD patients tested in the United Kingdom (R. Will, Western General Hospital, Edinburgh, Scotland, personal communication, 2002).

The patient received experimental treatment with quinacrine for 3 months. As of late September 2002, the patient had become bedridden, experienced considerable weight loss requiring surgical insertion of a feeding tube, and was no longer communicating with family members. On the basis of a case definition developed in the United Kingdom, the patient's illness met criteria for a probable case of vCJD.[1]

The patient was born in the United Kingdom in 1979 and moved to Florida in 1992. The patient never had donated or received blood, plasma, or organs and never had received human growth hormone. There was no family history of CJD. In October 2001, before the onset of the illness, the patient's wisdom teeth were extracted, but there was no history of major surgery.

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CDC Editorial Note: Variant CJD was first reported in 1996 in the United Kingdom, where an outbreak of BSE had been occurring among cattle since the early 1980s.[2] Strong laboratory and

epidemiologic evidence indicates that vCJD is linked causally with BSE.[3] Although specific foods that transmit the BSE agent to humans have not been identified, transmission is believed to occur primarily by processed food items that contain infectious bovine tissues such as the brain or spinal cord. As of early October 2002, a total of 138 vCJD cases were reported worldwide, including the case described in this report. Consistent with the conclusion that the agent of BSE is also the agent responsible for vCJD, most vCJD cases (n=128) were reported in the United Kingdom, where most BSE cases in cattle have occurred.[1]

The patient described in this report represents the first probable vCJD case in a U.S. resident. The patient had grown up in the United Kingdom when the BSE outbreak was increasing and when the risk for human exposures to BSE was probably at its peak. Therefore, it is likely that this patient was exposed to the BSE agent one or more times during 1980-1992 before moving to the United States and that the interval between the patient's exposure to BSE and onset of illness was 9-21 years. Such an incubation period would be consistent with known incubation periods for other similar diseases in humans, such as kuru and CJD related to exposures to pituitary-derived human growth hormone.[4]

The patient is unlikely to have transmitted the disease to others because the patient did not have surgical procedures that involved manipulation of known infectious tissues. In addition, the disease is not communicable by usual personal contact. Appropriate infection-control procedures should be followed while performing invasive procedures in patients with vCJD.[5] Although concerns exist about possible trans-mission of vCJD by transfusion of blood, this risk remains theoretical. The patient never had donated blood or organs. In 1999, because of the theoretical possibility of vCJD transmissions from infected blood donors, blood collection agencies in the United States began implementing a donor-deferral policy to exclude donors who might be at increased risk for infection because of a history of ≥6 months (later changed to ≥3 months) residence or travel to the United Kingdom during 1980-1996. In 2001, this donor-deferral policy was expanded to exclude donors who have traveled to other European countries for an extended period of time since 1980.[6]

Compared with the classic form of CJD endemic in the United States,[7] vCJD patients typically have illness onset at an unusually young age (median age: 26 years versus approximately 68 years for classic CJD). All but one of the reported vCJD decedents had illness onset and died before age 55 years, compared with approximately 10% of classic CJD cases.[7,8] Early in the course of the disease, vCJD patients usually have early and persistent psychiatric symptoms, including anxiety, depression, and social withdrawal; persistent painful sensory symptoms with dysesthesia and/or parasthesia also have been reported.[8] Evaluation of the clinical manifestations of the first 100 vCJD patients in the United Kingdom indicated that onset of frank neurologic signs (e.g., gait disturbances, slurring of speech, and tremor) was usually delayed by several months after illness onset. Other neurologic signs (e.g., chorea, dystonia, and myoclonus) frequently developed late in the course of the illness.[8] A prominent, symmetrical pulvinar high signal on T2-weighted and/or proton-density–weighted MRI has been reported in most vCJD patients.[9] In the absence of any other more plausible explanation, patients showing these clinical and radiologic features should be investigated for vCJD. In such patients, a history of travel to a BSE-endemic area increases the clinical suspicion for vCJD. In vCJD, but not other forms of CJD, there is prominent involvement of the lymphoreticular tissues.[10] A tonsil biopsy with demonstration of a characteristic abnormal prion protein by Western blot and immunohistochemistry can help establish a diagnosis of vCJD. The EEG in vCJD patients is typically normal or shows nonspecific abnormalities. All 105 vCJD patients tested in the United Kingdom were homozygous for methionine at the polymorphic codon 129 of the prion protein gene (R. Will, Western General Hospital, Edinburgh, Scotland, personal communication, 2002). The possible benefits of treating classic CJD and vCJD patients with quinacrine are under evaluation.

Physicians should report suspected vCJD cases to their local and state health departments. Because the clinical manifestations and age distribution of vCJD patients can overlap with those of classic CJD patients, a brain autopsy should be conducted in all such cases to distinguish suspected or diagnosed vCJD from classic CJD. A neuropathologic evaluation, in addition to helping to confirm the diagnosis, would help identify other potentially emerging prion diseases in humans. To facilitate neuropathologic studies of suspected or diagnosed prion diseases in humans, CDC, in collaboration with the American Association of Neuropathologists, established the National Prion Disease Pathology Surveillance Center. Physicians are encouraged to use the free services of this pathology center to confirm the diagnosis in suspected vCJD or classic CJD patients. Information about the center is available at http://www.cjdsurveillance.com.

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Update: Fatal and Severe Liver Injuries Associated With Rifampin and Pyrazinamide Treatment for Latent Tuberculosis Infection

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REPORTS OF FATAL AND SEVERE LIVER INjury associated with treatment of latent tuberculosis infection (LTBI) with the drug combination rifampin and pyrazinamide (RZ) prompted CDC to issue revised guidelines for the use of this regimen on August 31, 2001.[1] To determine if these revised guidelines were effective in reducing morbidity and mortality, CDC has continued to collect reports on adverse effects associated with this regimen. This update summarizes the results of this ongoing investigation.

A case of severe liver injury was defined as a hospital admission or death of a patient being treated for LTBI with RZ.[1,2] As of September 25, 2002, a total of 40 cases (eight fatal) were reported, of which 23 (five fatal) have been described.[1,2] Of the 17 cases (three fatal) that have not been described in previous reports, two occurred in patients who started RZ after publication of the revised guidelines. Both patients survived. One patient had contraindications for RZ (i.e., hepatitis and alcoholism). The other did not have contraindications for RZ and received RZ twice a week by directly observed therapy (DOT). According to information collected during DOT visits, the patient did not complain of any symptoms until the last week of the regimen. However, because the patient did not speak English, comprehension might have been a barrier. The patient missed two scheduled clinic appointments; serum aminotransferase and bilirubin levels were measured before treatment, but no biweekly tests were performed while the patient was on RZ, as is recommended in the revised guidelines. Physicians who choose to administer RZ instead of the preferred INH should follow the revised guidelines.

Summary of Revised Guidelines

The 9-month regimen of isoniazid (INH) remains the preferred treatment for patients who have LTBI and indications for treatment.[1,3] Daily RZ for 2 months or twice-weekly RZ for 2 or 3 months should be used with caution, especially in patients taking other medications associated with liver injury and in those with alcoholism, even if alcohol is discontinued during treatment. RZ is not recommended for persons with underlying liver disease or for those who have had INH- associated liver injury. If RZ is prescribed, evaluation of patients should include tests of serum aminotransferase and bilirubin at baseline and at 2, 4, and 6 weeks of treatment. No more than a 2-week supply of RZ (with a pyrazinamide dose of $\leq$20 mg/kg/d and a maximum of 2 gm/d) should be dispensed at a time.

CDC continues to collect data on reports of severe liver injury leading to hospital admission or death in persons receiving any treatment for LTBI. To determine the incidence of and risk factors for this problem, CDC is investigating cohorts of patients who received RZ. Health-care providers should report possible cases to CDC's Division of Tuberculosis Elimination, telephone 404-639-8442.

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Multistate Outbreaks of *Salmonella* Serotype Poona Infections Associated With Eating Cantaloupe From Mexico—United States and Canada, 2000-2002

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THREE MULTISTATE OUTBREAKS OF *SALmonella* serotype Poona infections associated with eating cantaloupe imported from Mexico occurred in the spring of consecutive years during 2000-2002. In each outbreak, the isolates had indistinguishable pulsed-field gel electrophoresis (PFGE) patterns; the PFGE patterns observed in the 2000 and 2002 outbreaks were indistinguishable, but the pattern from 2001 was unique among them. Outbreaks were identified first by the California Department of Health Services (2000 and 2001) and the Washington State Department of Health (2002) and involved residents of 12 states and Canada. This report describes the investigations, which led ultimately to an import alert on cantaloupes from Mexico. To limit the potential for can-

taloupe contamination, the Food and Drug Administration (FDA) continues to work with the Mexican government on a food-safety program for the production, packing, and shipping of fresh cantaloupes.

April-June 2000 Outbreak

A total of 47 confirmed cases of *S.* Poona infections with indistinguishable PFGE patterns were identified from California (26), Washington (10), Nevada (five), New Mexico (three), Oregon (two), and Colorado (one), with illness onset occurring during April 14–June 2. The median age of ill persons was 7 years (range: 1-95 years); 28 (60%) patients were aged <10 years, and nine (19%) were aged >60 years. Twenty-four (51%) patients were male and nine (19%) were hospitalized.

A matched case-control study was conducted; 20 case-patients were matched by age category to 37 community controls. A case was defined as laboratory-confirmed infection with *S.* Poona of the outbreak PFGE pattern in a person with illness onset during April-June. By multivariable modeling, illness was associated only with eating cantaloupe (matched odds ratio [MOR] = 6.7; 95% confidence interval [CI] = 1.3-34.0), with 16 (80%) case-patients versus seven (19%) controls reporting eating cantaloupe. Cantaloupe was purchased either pre-cut or whole.

April-May 2001 Outbreak

In April, an initial cluster of *S.* Poona was identified in California. Isolates had a rare biochemical trait, the inability to produce hydrogen sulfide (H_2S), and PFGE patterns that were indistinguishable. A total of 50 cases of H_2S-negative *S.* Poona infections were identified in residents of California (28), Washington (eight), Nevada (seven), Arizona (six), and Oregon (one). Demographic and illness-history data from the 28 California patients indicated that illness onset occurred during April 6–May 28. The age distribution was bimodal; the 19 children had a median age of 3 years (range: 1-5 years) and the nine adults had a median age of 80 years (range: 39-91 years). Fifteen (54%) patients were female. Ten (36%) patients were bacteremic; one infant girl had *S.* Poona isolated from a urine specimen. Nine (33%) patients were hospitalized, and two patients (a man aged 78 years and a woman aged 91 years) died with *Salmonella* septicemia.

A matched case-control study was conducted; 11 case-patients from California (seven), Nevada (two), Arizona (one), and Washington (one) were matched by age category to 19 community controls. Case-patients had laboratory-confirmed infections of the outbreak strain of H_2S-negative *S.* Poona and illness onset during the first 2 weeks of April. Illness was associated only with eating cantaloupe (MOR = 7.4; 95% CI = 1.0-178.0). Eight (80%) case-patients and six (33%) controls recalled eating cantaloupe. Cantaloupe was purchased either pre-cut or whole.

March-May 2002 Outbreak

A total of 58 cases with *S.* Poona isolates with indistinguishable PFGE patterns were identified in California (21), Washington (nine), Oregon (five), British Columbia (four), Colorado (three), Nevada (three), Manitoba (two), Missouri (two), Ontario (two), Saskatchewan (two), Texas (two), Arkansas (one), Minnesota (one), and Vermont (one). Illness onset occurred during March 30–May 31; the median age of patients was 6 years (range: 4 months–91 years); 32 (55%) were aged <10 years, and 11 (19%) were aged >60 years. A total of 31 (55%) were female. Ten patients were hospitalized.

A matched case-control study was conducted; 27 case-patients were matched by age category to 54 community controls. A case was defined as *S.* Poona infection with the outbreak PFGE pattern in a person aged ≥2 years with illness onset during March 15–May 3. The only exposure significantly associated with illness was eating cantaloupe; 20 (74%) case-patients recalled eating cantaloupe compared with 11 (20%) controls (MOR = 15.5; 95% CI = 3.3-125.0). Case-patients (50%) were more likely than controls (13%) to eat cantaloupe purchased whole (MOR = 5.8; 95% CI = 1.6-23.3) or to eat cantaloupe in a fruit salad or as a garnish (28% versus 5%) (MOR = 6.5; 95% CI = 1.2-63.0). No other factors were significantly associated with illness.

Traceback and Regulatory Action

FDA, in conjunction with state and provincial food regulatory agencies, conducted traceback investigations of cantaloupe purchased by patients in all three outbreaks. In each instance, point-of-sale sources of cantaloupe were traced back to shippers and then to farms in Mexico. In response to the 2000 and 2001 outbreaks, FDA conducted on-farm investigations in Mexico and concluded that measures were not in place to minimize microbial contamination in the growing, harvesting, packaging, and cooling of cantaloupe. Possible sources of contamination include irrigation of fields with water contaminated with sewage, processing (cleaning and cooling) produce with *Salmonella*-contaminated water, poor hygienic practices of workers who harvest and process the cantaloupe, pests in packing facilities, and inadequate cleaning and sanitizing of equipment that comes in contact with cantaloupe. In association with the 2001 outbreak, FDA detained product imported by the shipper on May 31, and the shipper voluntarily recalled its imported Mexican cantaloupe. The shipper and the implicated farm in Mexico remain on detention. In association with the 2002 outbreak, the importer voluntarily recalled the implicated Mexican cantaloupe, and FDA placed the implicated farms on detention. On October 28, 2002, FDA issued an import alert on cantaloupe from Mexico that detains all products offered for entry at all U.S. ports.

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CDC Editorial Note: *Salmonella* infections have been linked to melons at least since 1990 when *Salmonella* serotype Chester traced to cantaloupe caused 245 illnesses in 30 states.[1] The cantaloupe were imported from either Mexico or Guatemala. In 1991, an outbreak of cantaloupe-associated *S.* Poona infections caused 400 illnesses in 23 states.[2] Illness was associated with eating precut cantaloupe in fruit salads or from salad bars. Although industry sources identified the lower Rio Grande Valley in Texas as the probable source of the implicated cantaloupe, some might have come from Mexico. In response to this outbreak, FDA conducted a microbiologic survey that isolated a variety of *Salmonella* serotypes from approximately 1% of sampled imported cantaloupe and watermelon.[2] In 1997, an outbreak of *Salmonella* serotype Saphra infections affected 25 persons in California. Illness was associated with cantaloupe imported from Mexico.[3] After the 2000 and 2001 *S.* Poona outbreaks, FDA conducted farm investigations in Mexico, issued press releases to warn consumers, placed implicated farms on detention, and conducted sampling surveys of imported cantaloupe. The 1999 and 2000 FDA surveys of imported produce indicated that 5% of cantaloupe sampled (eight of 151) was contaminated with *Salmonella*.[4] A 2001 survey of imported produce indicates that of 29 cantaloupes from Mexico tested, none yielded *Salmonella*, *Shigella*, or *Escherichia coli* O157:H7 (FDA, unpublished data, 2001). The interpretation of the 2001 survey is limited by of the small sample size.

S. Poona is a relatively rare serotype that is responsible for 1% of human *Salmonella* isolates reported in the United States in 2001; however, of the six cantaloupe-associated *Salmonella* outbreaks, four were attributed to infections with *S.* Poona. Typically, human infection with *S.* Poona is associated with reptile exposure.[5,6] The three outbreaks attributed to *S.* Poona-contaminated cantaloupe traced to Mexican farms suggest the possibility of a unique natural reservoir in the Mexican farm environment, possibly from reptiles such as iguanas drawn to feed on melon crops that enter the packing sheds and contaminate the equipment. Subsequently, water used in the washing and cooling process might spread the contamination.

FDA provides information about the decontamination of melons to the retail industry, food-service establishments, and commercial processors of pre-cut melon.[7,8] The use of sodium hypochlorite or other permitted antimicrobials in combination with brushing is recommended. The potential for microbial contamination also might be reduced by using only good-quality fruit that is free from open wounds or defects that might allow bacteria to contaminate the interior of the fruit.[9] Additional research is needed to determine the effectiveness of consumer produce-washing practices. Consumers should be sure that fresh-cut melons are refrigerated or surrounded by ice; leftover cut melons should be discarded if left at room temperature for >2 hours. Additional information for consumers is available at http://www.fda.gov/bbs/topics/answers/2002/ans01167.html.

On October 28, 2002, in response to the three outbreaks during 2000-2002 and analytical results from the sampling of imported Mexican cantaloupe, FDA issued an import alert that detains all cantaloupe from Mexico offered for entry at all U.S. ports. FDA will continue to work with the Mexican government on a food-safety program for the production, packing, and shipping of fresh cantaloupe. The Mexican government is developing a certification program based on sound agricultural and manufacturing practices that would allow FDA to identify farms that have adopted and implemented such a food-safety program.

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