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Fatal Human Plague — Arizona and Colorado, 1996

In 1996, five cases of human plague, of which two were fatal, were reported in the United States; both decedents had septicemic plague that was not diagnosed until after they died. This report summarizes the investigation of the two fatal cases and underscores the need for health-care providers in areas with endemic plague to maintain a high level of awareness about the risk for plague in their patients.

Patient 1

On August 2, 1996, an 18-year-old resident of Flagstaff, Arizona, was taken to a local outpatient clinic because of a 2-day history of fever, pain in his left groin, and diarrhea. On examination, he was afebrile, had a pulse rate of 126 beats per minute, respiration rate of 20 breaths per minute, and blood pressure of 130/81 mm Hg. Left groin swelling and tenderness were noted. A groin muscle strain was diagnosed and attributed to a fall 2 days earlier. He was treated with nonsteroidal anti-inflammatory agents, instructed about using a liquid diet, and released. On August 3, the patient reported feeling weak, had difficulty breathing, and collapsed while taking a shower. Emergency medical assistance was called, and the patient experienced cardiac arrest while emergency medical technicians were on site. He was transported to a hospital emergency department (ED) and pronounced dead shortly after arrival.

On August 8, cultures of blood samples obtained in the ED were presumptively positive for *Yersinia pestis* by fluorescent antibody staining and confirmed by specific bacteriophage lysis at the laboratory of the Arizona State Department of Health. Additional isolates from postmortem brain, liver, lung, and vitreous fluid cultures were confirmed as *Y. pestis* at CDC. An epidemiologic investigation by public health officials indicated that the patient most likely became infected on July 27 as the result of bites by *Y. pestis*-infected fleas while walking through a prairie dog (*Cynomys gunnisoni*) colony in Navajo County. High antibody titers to the fraction 1 (F1) antigen of *Y. pestis* were detected in two of four pet dogs living in houses near the prairie dog colony. Dog owners were advised about the risk for plague and instructed to restrain their pets and to periodically dust them with insecticide. Prairie dog burrows within one half mile of the residences were dusted with insecticide to control flea populations.

Fatal Human Plague — Continued

Patient 2

On August 17, 1996, a 16-year-old resident of western Colorado had onset of pain followed by numbness in her left arm and left axillary pain. During August 18–19, she had chills, fever, and several episodes of vomiting. On August 19, she was evaluated at a local hospital ED. Findings included a temperature of 97.4 F (36.3 C), pulse rate of 100 beats per minute, respiration rate of 16 breaths per minute, and blood pressure of 103/59 mm Hg; a chest radiograph was interpreted as within normal limits. She was discharged with a diagnosis of possible brachial plexus injury related to a fall from a trampoline on August 14. She was prescribed analgesics, and an appointment with a neurologist was scheduled.

On August 21, she was found semiconscious at home and taken to the same hospital. She was confused and complained of neck pain and generalized soreness. Findings on examination included a temperature of 102.5 F (39.2 C), pulse rate of 170 beats per minute, respiration rate of 50 breaths per minute, and blood pressure of 130/70 mm Hg. Within an hour of arrival at the hospital, she experienced respiratory arrest and was intubated. Numerous gram-positive diplococci were detected in a blood smear, and a chest radiograph revealed bilateral pulmonary edema. She was administered 2 g ceftriaxone intravenously and transferred to a referral hospital with diagnoses of septicemia, disseminated intravascular coagulation, adult respiratory distress syndrome, and possible meningitis. A gram stain of sputum revealed rare white blood cells and no bacteria; she was treated for gram-positive sepsis. However, her condition rapidly deteriorated, and she died later that day.

On August 23, blood and spinal fluid cultures obtained on August 21 grew an unidentified gram-negative rod and *Streptococcus pneumoniae*. On August 26, *Yersinia pseudotuberculosis* was initially identified in cultures of blood and respiratory aspirate using a rapid microbiologic identification device. This blood culture isolate subsequently was presumptively identified as *Y. pestis* at the Utah Division of Laboratory Services and confirmed as *Y. pestis* at CDC.

An environmental investigation by health officials revealed evidence of an earlier extensive prairie dog die-off adjacent to the patient's residence. High antibody titers to the F1 antigen of *Y. pestis* were present in serum specimens from four of five family dogs and one of three family cats. The seropositive cat had a submandibular lesion consistent with a healing abscess. Family members reported that the cat had been recently ill and had been closely cared for by the decedent. Investigators concluded that the decedent was probably exposed to *Y. pestis* by direct contact with infectious material while handling the cat. None of 10 flea pools or 13 rodents (least chipmunk, *Tamias minimus* [four]; deer mouse, *Peromyscus maniculatus* [six]; and house mouse, *Mus musculus* [three]) collected on the property tested positive for *Y. pestis* or for antibody to *Y. pestis*, respectively. Because the diagnosis was established after the standard 7-day maximum plague incubation period had elapsed, antibiotic prophylaxis of family members and medical personnel was not instituted.

Reported by: M Tenborg, B Davis, MS, D Smith, Coconino County Health Dept, Flagstaff; C Levy, MS, B England, MD, State Epidemiologist, Arizona Dept of Health Svcs. B Koehler, Delta County Health Dept, Delta; D Tanda, J Pape, MS, R Hoffman, MD, State Epidemiologist, Colorado Dept of Public Health and Environment. R Fulgham, MS, B Joe, J Cheek, MD, Indian Health Svc. Bacterial Zoonoses Br, Div of Vector-Borne Infectious Diseases, National Center for Infectious Diseases; Div of Applied Public Health Training (proposed), Epidemiology Program Office, CDC.

Fatal Human Plague — Continued

Editorial Note: In the United States, most cases of human plague are reported from New Mexico, Arizona, Colorado, and California (*1,2*). The principal forms of plague are bubonic, septicemic (primary or secondary), and pneumonic (primary or secondary). From 1947 through 1996, a total of 390 cases of plague were reported, resulting in 60 (15.4%) deaths. Of these, bubonic plague accounted for 327 (83.9%) cases and 44 (13.5%) deaths; primary septicemic plague, for 49 (12.6%) cases and 11 (22.4%) deaths; and primary pneumonic plague, for seven (1.8%) cases and four (57.1%) deaths. Seven (1.8%) cases were unclassified, including one (14.3%) death (CDC, unpublished data, 1997). During 1965–1989, a total of 27 persons with plague were treated at the Gallup Indian Medical Center in New Mexico. Of these, classic signs of bubonic plague were present in only 10 (37%); provisional diagnoses in other patients included apparent upper respiratory tract infections, nonspecific febrile syndromes, gastrointestinal or urinary tract infections, or meningitis (*3*). The syndromes in both patients described in this report initially were attributed to injuries and treated with analgesics.

Bubonic plague may not be considered by a physician if swollen, tender lymph nodes are not detected or present on physical examination. Evidence of regional lymphadenitis should prompt a suspicion of plague in a patient who lives in or has recently visited an area with endemic plague. Septicemic plague without obvious lymphadenopathy is more difficult to diagnose because the manifestations are nonspecific (e.g., elevated temperature, chills, abdominal pain, nausea, vomiting, diarrhea, tachycardia, tachypnea, and hypotension) (*4*).

A patient with clinical signs of sepsis and a history of possible plague exposure, particularly during the spring, summer, and fall months, should be aggressively managed as having plague. Even before a specific laboratory diagnosis is obtained, antibiotic therapy should be initiated with streptomycin; alternatives include gentamicin, chloramphenicol, and the tetracyclines. The penicillins and cephalosporins are not effective in treating plague, although these drugs frequently show activity in vitro (*5*).

In suspected cases of plague, several samples of blood should be collected for culture during a 45-minute period before initiation of antibiotic treatment, unless such a delay is contraindicated by the patient's condition. The direct immunofluorescence test for the rapid presumptive identification of *Y. pestis* F1 antigen should be applied to appropriate clinical materials (e.g., lymph node aspirates, culture isolates, or blood films), and if pneumonic plague is suspected, tracheal washes or sputum smears. Rapid microbiologic identification devices may not include adequate *Y. pestis* profiles in their database and, therefore, may misidentify *Y. pestis* as *Y. pseudotuberculosis* (*6*). Acute- and convalescent-phase serum specimens should be obtained to detect antibodies to the *Y. pestis* F1 antigen by using passive hemagglutination assay or enzyme-linked immunosorbent assay methods. Patients with suspected *Y. pestis* infections should be reported immediately to local or state health departments to enable prompt initiation of appropriate public health control and prevention activities. In the United States, testing of clinical specimens and isolates from suspected plague patients should be coordinated through state health departments and sent to CDC's Diagnostic and Reference Laboratory Section, Division of Vector-Borne Infectious Diseases, National Center for Infectious Diseases (telephone [970] 221-6400), for confirmation of *Y. pestis* (*7*).

Fatal Human Plague — Continued

Control measures to prevent human plague include surveillance for plague in rodents and rodent predators as well as public education (*8,9*). When epizootic plague is detected, local health-care providers and the public should be alerted about possible risks. Warnings can be posted at identified epizootic foci (e.g., campgrounds and trailheads), and rodent flea-control measures should be considered. Public education efforts should focus on promoting personal protection measures, including 1) avoiding areas with known epizootic plague; 2) avoiding sick or dead animals; 3) using repellents, insecticides, and protective clothing during potential exposures to rodent fleas; and 4) using gloves when handling animals killed by trapping or hunting. Persons residing in areas with wild rodent plague should keep their dogs and cats free of fleas and restrict pets from wandering. Because plague in cats is especially contagious, persons caring for sick cats should take precautions to avoid exposure to potentially infectious exudates or secretions. Sources of rodent food (e.g., garbage and animal food) and harborage (e.g., brush piles and junk heaps) should be eliminated in the peridomestic environment.

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Transmission of HIV Possibly Associated with Exposure of Mucous Membrane to Contaminated Blood

In February 1996, transmission of human immunodeficiency virus (HIV) by an unknown route involving an HIV-infected man and his previously uninfected female sex partner was reported to CDC. This report summarizes the epidemiologic investigation of this transmission, which suggests that the woman was infected through mucous membrane exposure to contaminated blood.*

In 1992, after obtaining informed consent from the HIV-infected man and his uninfected female sex partner, they were enrolled in a study in which couples with one

*Single copies of this report will be available until July 11, 1998, from the CDC National AIDS Clearinghouse, P.O. Box 6003, Rockville, MD 20849-6003; telephone (800) 458-5231 or (301) 217-0023.

Transmission of HIV — Continued

HIV-infected partner and one non-HIV-infected partner were extensively counseled, administered questionnaires, and tested periodically for HIV infection. Blood drawn from the woman on July 19, 1994, was HIV-negative by both enzyme immunoassay (EIA) and polymerase chain reaction (PCR). However, serum specimens obtained from the woman on July 24, 1995, and September 11, 1995, were positive by both EIA and immunofluorescent assay. During the interval from the month before her last HIV-negative test (June 1994) to the month of her first HIV-positive test (July 1995), the woman denied known risk exposures for HIV (i.e., other sex partners; noninjecting- or injecting-drug use; sexually transmitted diseases; blood transfusion; artificial insemination; occupational exposure to HIV; or acupuncture, tattoos, body piercing, or other percutaneous injections).

The sources of information obtained separately from each partner by two independent interviewers during this investigation and by interview records obtained during the study before the couple was aware of the HIV transmission were consistent about the couple's sex practices during June 1994–July 1995. During this period, the woman and her partner reported having vaginal intercourse an average of six times per month but never during menses. The couple reported always using latex condoms (for men) during sex, most times with the spermicide nonoxynol-9. The couple denied having had anal sex during this period. Although the couple reported a condom breakage that occurred in January 1994, both independently denied awareness of condom breakage or slippage during June 1994–July 1995 and believed that the condom remained in place each time while the penis was withdrawn. The couple engaged in "deep kissing" (open-mouth to open-mouth) several times per month. The man indicated that his gums frequently bled after he brushed and flossed his teeth and that the couple generally engaged in sexual intercourse and "deep kissing" at night after he brushed his teeth. Occasional instances of oral sex between the couple reportedly did not involve the exchange of semen or blood. In addition, the woman recalled using the man's toothbrush and razor, both without visible blood, on one occasion each, but she was unable to specify whether these events occurred during the putative infection period of June 1994–July 1995.

The man had been HIV-infected since 1988 as the result of injecting-drug use, and he reported longstanding poor dentition and occasional sores in his mouth. On August 29, 1994, the man had a normal platelet count and a CD4+ T-lymphocyte count of 110 cells/μL. On September 6, 1994, he sought medical care at a clinic because of a cough, stress, and intermittent weight loss; small vesicles were noted in his throat. At a follow-up visit in April 1995, canker sores, halitosis, and gingivitis were noted. In May 1995, at his first dental visit since 1988, gingivitis and oral hairy leukoplakia were diagnosed. The man had never received antiretroviral medications or prophylaxis against *Pneumocystis carinii* pneumonia although they had been recommended to him.

Because of a 4-month history of increasing dental sensitivity to hot and cold, on August 8, 1994, the woman underwent a dental evaluation followed by endodontic therapy (a "root canal"). Her dental records noted poor condition of gums, 2-mm to 6-mm pockets (indicating periodontitis), poor personal dental hygiene practices, and a recommendation for periodontal therapy. No complications or excessive bleeding from the endodontic therapy were reported by the woman or noted by the dentist. The dentist had been tested for HIV in May 1996 and was negative by EIA.

Transmission of HIV — Continued

On August 26, 1994, the woman had onset of a syndrome of 7–10 days' duration characterized by fever of 102 F (39 C), headache, swollen lymph nodes, sore neck and back, and muscle aches in her legs. On September 2, she sought medical care from her primary-care physician, who noted erythema and inflammation of the gingiva. The physician diagnosed a viral process with concomitant gum infection and prescribed erythromycin for treatment. The woman reported no other clinically important illness from June 1994 to July 1995.

Blood samples were obtained from both HIV-infected partners in April 1996. A nested PCR was used to amplify proviral HIV DNA sequences from peripheral blood mononuclear cells (PBMCs), and viral RNA sequences from serum were amplified using a nested reverse transcriptase PCR. Analysis of a 345-nucleotide segment of the C2V3 region of the *env* gene revealed a 4% nucleotide difference between the man and woman's PBMC proviral sequences and a 9% difference between the viral strains in the man and woman's serum. Sequence analysis of the complete p17 region of the *gag* gene from the PBMC proviral DNA from each partner indicated only a 1.6% nucleotide difference between the proviral sequences of the man and woman. Phylogenetic analysis of the C2V3 sequences grouped all HIV strains from the couple's PBMCs and serum as a monophyletic clade distinct from sequences from other HIV-infected persons in the United States, with a bootstrap support of 87% (*1*). These laboratory results indicate a high degree of relatedness between the viruses infecting the man and woman, supporting the conclusion that HIV was transmitted from one to the other. Testing of stored PBMCs obtained from each partner in 1995 produced similar results.

Reported by: N Padian, PhD, S Glass, Univ of California at San Francisco. HIV Laboratory Investigations Br, Div of AIDS, STD, and TB Laboratory Research, National Center for Infectious Diseases; Epidemiology Br, Div of HIV/AIDS Prevention, National Center for HIV, STD, and TB Prevention, CDC.

Editorial Note: The findings in this report suggest that the woman probably became infected with HIV during June 1994–July 1995, possibly during the weeks before the onset of her symptoms on August 26, 1994; these symptoms were consistent with acute retroviral syndrome (*2*). In addition, during June 1994–July 1995, the man's CD4+ T-lymphocyte count was low, which may be associated with increased infectivity and risk for transmission (*3*). Results of the DNA sequencing and phylogenetic analysis support the epidemiologic findings that the woman's infection was acquired from her infected male partner.

Although the exact route of transmission in this report cannot be determined, the most likely possibility is that the woman became infected through mucous membrane exposure to the man's saliva that was contaminated by blood from his bleeding gums or exudate from undetected oral lesions. Such exposure may have occurred during "deep kissing"; the woman's inflamed gingival mucosa, as indicated by her dental and medical records, might have been a contributing factor. Exposure to saliva uncontaminated with blood is considered to be a rare mode of HIV transmission for at least five reasons: 1) saliva inhibits HIV-1 infectivity (*4*); 2) HIV is infrequently isolated from saliva (*5*); 3) none of the approximately 500,000 cases of AIDS reported to CDC have been attributed to exposure to saliva; 4) levels of HIV are low in the saliva of HIV-infected persons, even in the presence of periodontal disease (*6*); and 5) transmission of HIV in association with kissing has not been documented in studies of nonsexual

Transmission of HIV — Continued

household contacts of HIV-infected persons (*7*). However, rare bite-related instances of HIV transmission from exposure to saliva contaminated with HIV-infected blood have been reported (*8,9*).

Other exposures of the woman to the man's blood or semen cannot be excluded. Although occasional instances of oral sex did not reportedly involve the exchange of semen or blood between the persons in this report, these routes of transmission cannot definitively be excluded. Sexual exposure through vaginal intercourse is a plausible mechanism of transmission for the case described in this report; however, other studies of couples in which one partner is HIV-infected and the other is not indicate that HIV transmission is rare when heterosexual couples use condoms consistently during vaginal intercourse (*10*). If a condom is not used correctly, it may slip off or break, thereby reducing its effectiveness as a barrier to HIV. However, for this case, both partners could not recall any instances of condom slippage or breakage during the time infection was likely to have occurred. In addition, although the shared use of a toothbrush or razor are theoretically plausible routes of transmission, the woman recalled that each event occurred only once, and she could not specify whether either event occurred during the period when transmission most likely occurred.

The findings of this investigation underscore the multiple routes by which exposure to infectious body fluids can occur among sexually intimate persons. Uninfected persons considering intimate relationships with persons known to be infected with HIV should be educated about the rare possibility of HIV transmission through mucous membrane exposures. Persons choosing to have sex with HIV-infected persons or persons with unknown HIV serostatus should correctly use latex condoms (for men) during each act of intercourse and should avoid any other exposure to potentially infectious body fluids, including blood, semen, or any other body fluid visibly contaminated with blood.

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Reduced Susceptibility of *Staphylococcus aureus* to Vancomycin — Japan, 1996

Staphylococcus aureus is a virulent microorganism responsible for many serious infections among the general population. Since recognition of vancomycin-resistant enterococci (VRE), the emergence of vancomycin resistance in *S. aureus* has been anticipated. This report describes the first documented case of infection caused by *S. aureus* with reduced susceptibility to vancomycin and includes the initial characterization of this isolate (*1*); the case occurred in a pediatric patient in Japan. The emergence of reduced vancomycin susceptibility in *S. aureus* increases the possibility that some strains will become fully resistant and that currently available antimicrobial agents will become ineffective for treating infections caused by such strains.

In May 1996, a 4-month-old boy developed a nosocomial surgical-site infection with methicillin-resistant *S. aureus* (MRSA). He received treatment with vancomycin (45 mg per kg body weight per day) for 29 days, but fever and surgical-site purulent discharge continued, and the C-reactive protein (CRP) remained elevated (4 mg/dL; normal: <1 mg/dL). Treatment was changed to a combination of vancomycin and arbekacin (an aminoglycoside approved for MRSA infection in Japan but not in the United States). After 12 days of this regimen, the purulent discharge subsided, the wound site began to heal, and the CRP declined to 0.9 mg/dL; antimicrobial therapy was discontinued. However, 12 days after antimicrobial therapy was discontinued, fever and surgical-site inflammation recurred, subcutaneous abscesses were detected, and the CRP increased to 3.5 mg/dL. Arbekacin was resumed in combination with ampicillin/sulbactam. After 6 days of this regimen, his fever subsided, and the CRP declined below detectable levels (<0.3 mg/dL). However, during the next several days, the CRP fluctuated between <0.3 mg/dL and 1.0 mg/dL, consistent with persistent infection. After debridement of the subcutaneous abscesses and therapy with arbekacin and ampicillin/sulbactam for an additional 17 days, the patient improved, and his CRP remained below detectable levels; his antimicrobial therapy was discontinued, and he was discharged from the hospital.

The MRSA strain that was isolated from the purulent discharge at the surgical site and from the debridement sample demonstrated a vancomycin minimum inhibitory concentration (MIC) of 8 μg/mL (National Committee for Clinical Laboratory Standards breakpoints: susceptible, ≤4 μg/mL; intermediate, 8–16 μg/mL; and resistant, ≥32 μg/mL) by the broth microdilution method performed in Japan and at CDC (*2*). The organism was negative when tested by polymerase chain reaction for *vanA* and *vanB,* the principal genes responsible for vancomycin resistance in enterococci. The mechanism of decreased susceptibility is still under investigation.

Reported by: K Hiramatsu, MD, Juntendo Univ, Tokyo, Japan. Hospital Infections Program, National Center for Infectious Diseases, CDC.

Editorial Note: *S. aureus* is a gram-positive, coccoid bacteria that causes pneumonia and infections of the bloodstream, skin, soft tissues, and bone; this pathogen frequently causes community-acquired infections and is the most common cause of nosocomial infections. In the pre-antibiotic era, *S. aureus* infections were a common cause of death. Although the availability of penicillin in the 1940s offered an important advance in the treatment of infection, susceptibility of *S. aureus* was short-lived.

Staphylococcus aureus — *Continued*

Resistance was first recognized in 1944 and was caused by production of a penicillinase enzyme capable of deactivating penicillin; by the late 1950s, approximately 50% of strains were resistant to penicillin. These strains were associated with widespread outbreaks before the development of semisynthetic penicillinase-resistant agents, such as methicillin, in 1960; however, resistance to methicillin was reported as early as 1961 in England. In the United States, the proportion of MRSA isolates reported to the National Nosocomial Infections Surveillance system increased from 2% in 1975 to 35% in 1996. In Japan, analysis of approximately 7000 strains isolated from patients in various geographic areas during 1992–1993 (*3*) indicated that 60% of *S. aureus* isolates were resistant to methicillin.

Until the identification of the isolate described in this report, MRSA had been susceptible to vancomycin, a glycopeptide antibiotic introduced clinically in 1958. Initially, vancomycin was used infrequently as an alternative to other agents; however, because of the increase in MRSA and other factors (e.g., increased incidence of prosthetic device-related infections and *Clostridium difficile* colitis), its use has increased since the late 1970s. In the late 1980s, clinically important resistance to vancomycin was identified among enterococci (i.e., VRE) associated with *vanA* or *vanB* genes. Transfer of the *vanA* genes experimentally from enterococci to *S. aureus* (*4*) suggested the potential for *S. aureus* to acquire these genes in vivo, producing clinical resistance. Such resistance could result in serious clinical and public health consequences because no currently licensed alternative to vancomycin is available to treat serious MRSA infections.

Infections caused by less virulent coagulase-negative staphylococci (CNS) with reduced susceptibility to vancomycin have been previously recognized (e.g., *S. haemolyticus* [*5*] and *S. epidermidis* [*6*]). In addition, laboratory studies in which both CNS and *S. aureus* isolates have been exposed to increasing levels of glycopeptides have demonstrated the ability of these agents to select for resistant subpopulations (*7,8*). Given these findings and the spread of VRE, for which the prudent use of vancomycin has been recommended as an important control measure (*9*), the prudent use of all antibiotics, especially vancomycin, is critical for preventing the emergence of resistance among staphylococci in the United States.

The impact of reduced vancomycin susceptibility on clinical outcome may be difficult to assess because serious infections caused by fully susceptible *S. aureus* often require treatment with a combination of aggressive surgical and antimicrobial therapy. Reduced vancomycin susceptibility as described in this report may signal the onset of an increase in the MICs of vancomycin against *S. aureus.* The clinical importance of such reduced susceptibility may become most evident for treatment of infections at sites where achievable drug concentrations are lower than those commonly achieved in the bloodstream (e.g., closed space or central nervous system infections) or in treating infections in the presence of a foreign body. Patients with infections caused by *S. aureus* (i.e., MRSA) with reduced susceptibility to vancomycin and who unequivocally have not responded to appropriate therapy may be candidates for treatment with an investigational drug. CDC and the Food and Drug Administration are collaborating to make such agents available in the United States (*10*).

Because clonal dissemination of *S. aureus* with reduced vancomycin susceptibility can occur, efforts must be intensified to prevent the transmission of such strains within and between facilities and to minimize the potential for these strains to become

Staphylococcus aureus — *Continued*

endemic. The recovery of *S. aureus* with presumptive reduced susceptibility to vancomycin should be reported immediately to state health departments and to CDC's Hospital Infections Program, National Center for Infectious Diseases, telephone (404) 639-6400. In addition, special infection-control precautions should be adhered to strictly (*10*), and an epidemiologic investigation should be initiated promptly.

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Interim Guidelines for Prevention and Control of Staphylococcal Infection Associated with Reduced Susceptibility to Vancomycin

Staphylococci are one of the most common causes of community- and hospital-acquired infection. In many U.S. hospitals, strains of staphylococci (i.e., *Staphylococcus aureus* or coagulase-negative staphylococci) are resistant to all available antimicrobials except vancomycin. Rare cases of infection in the United States (*1*) have been caused by coagulase-negative staphylococci with reduced susceptibility to vancomycin (minimum inhibitory concentration [MIC] ≥8 μg/mL)* (*2*).

In May 1996, an infection caused by a strain of *S. aureus* with reduced susceptibility to vancomycin (MIC=8 μg/mL) was diagnosed in a patient in a hospital in Japan (*3,4*); no such infections have been reported in the United States. Although the strain from Japan was not fully resistant to vancomycin (i.e., MIC ≥32 μg/mL), its appearance increases the likelihood that fully resistant strains may emerge. Because the

*National Committee for Clinical Laboratory Standards breakpoints: susceptible, ≤4 μg/mL or zone size ≥12 mm; intermediate, 8–16 μg/mL or zone size 10–11 mm; and resistant, ≥32 μg/mL or zone size ≤9 mm.

Guidelines — Continued

occurrence of fully vancomycin-resistant staphylococcal infection in a hospital could result in serious public health consequences, CDC and the Hospital Infection Control Practices Advisory Committee have developed interim guidelines to direct medical and public health responses when isolates of staphylococci with reduced vancomycin susceptibility are identified. This report describes these interim guidelines, which include steps to 1) decrease the likelihood that staphylococci with reduced vancomycin susceptibility will emerge; 2) recognize the occurrence of staphylococci with reduced vancomycin susceptibility; 3) obtain information about investigational antimicrobials for treating either patients infected with fully vancomycin-resistant staphylococci or patients infected with staphylococci with intermediate vancomycin resistance for whom conventional therapy fails; and 4) implement interim infection-control measures. To effectively implement these interim guidelines, each health-care facility should develop a plan based on these guidelines in which responsibilities for critical departments and personnel are clearly delineated.

Preventing the Emergence of Vancomycin Resistance

Antimicrobial use is a major risk factor for the emergence of antimicrobial-resistant pathogens. Reduction of overuse and misuse of antimicrobials will decrease the risk for emergence of staphylococci with reduced susceptibility to vancomycin. Medical and ancillary staff members who are responsible for pharmacy formulary decisions should review and restrict use of vancomycin (*5*) and ensure that use of other antimicrobials is appropriate.

Detecting Staphylococci with Reduced Vancomycin Susceptibility

Use of recommended laboratory methods (including media and incubation methods, antimicrobial susceptibility testing methods, and susceptibility breakpoints) for identifying such strains is essential.

1. The most accurate form of antimicrobial susceptibility testing for staphylococci is a minimal inhibitory concentration method (broth dilution, agar dilution, or agar-gradient diffusion) using a full 24-hour incubation. Strains of staphylococci with a MIC=8 μg/mL (classified as intermediate using National Committee for Clinical Laboratory Standards breakpoints) were not detected by using the current disk diffusion procedure.
2. All strains with a MIC ≥4 μg/mL should be considered candidate strains for reduced vancomycin susceptibility. Other than the isolate reported in Japan (*4*), all *S. aureus* strains with putative reduced vancomycin susceptibility sent to CDC for confirmation have been misidentified or mixed with other microorganisms. Therefore, the laboratory should ensure that the strain is in pure culture and reconfirm the genus and species of the organism; then repeat the susceptibility test for vancomycin using a minimal inhibitory concentration method.
3. After repeat testing, if species identification and vancomycin test results are consistent, immediately contact the state health department (SHD) and CDC's Hospital Infections Program, National Center for Infectious Diseases, telephone (404) 639-6400, to report the occurrence of a "presumptive" staphylococcal strain with reduced susceptibility to vancomycin and to obtain epidemiologic and laboratory assistance.

Guidelines — Continued

4. Retest staphylococci isolated from patients who fail to respond to vancomycin therapy because resistance may have emerged during therapy.

Obtaining Investigational Antimicrobials

The susceptibility pattern of a particular staphylococcus strain, the site of infection, and the response to conventional therapy is important in determining the need for investigational antimicrobials to treat infections caused by staphylococci with reduced vancomycin susceptibility. Several antimicrobial agents in clinical development may be useful in treating vancomycin-resistant enterococci and methicillin-resistant *S. aureus*. Some of these agents also may be useful in treating infections with *S. aureus* with reduced susceptibility to vancomycin. The usefulness of any antimicrobial agent will depend on the resistance mechanism and susceptibility pattern of the *S. aureus* strain. CDC and the Food and Drug Administration (FDA) are working to improve access by clinical providers to investigational agents that may be useful for treating patients with confirmed infections with *S. aureus* strains with reduced susceptibility to vancomycin. Physicians treating infections caused by staphylococci with reduced vancomycin susceptibility can obtain information about investigational drug therapies from FDA's Division of Anti-Infective Drug Products, telephone (301) 827-2120. The physician will be requested to send the isolate to CDC for microbiologic and epidemiologic evaluation.

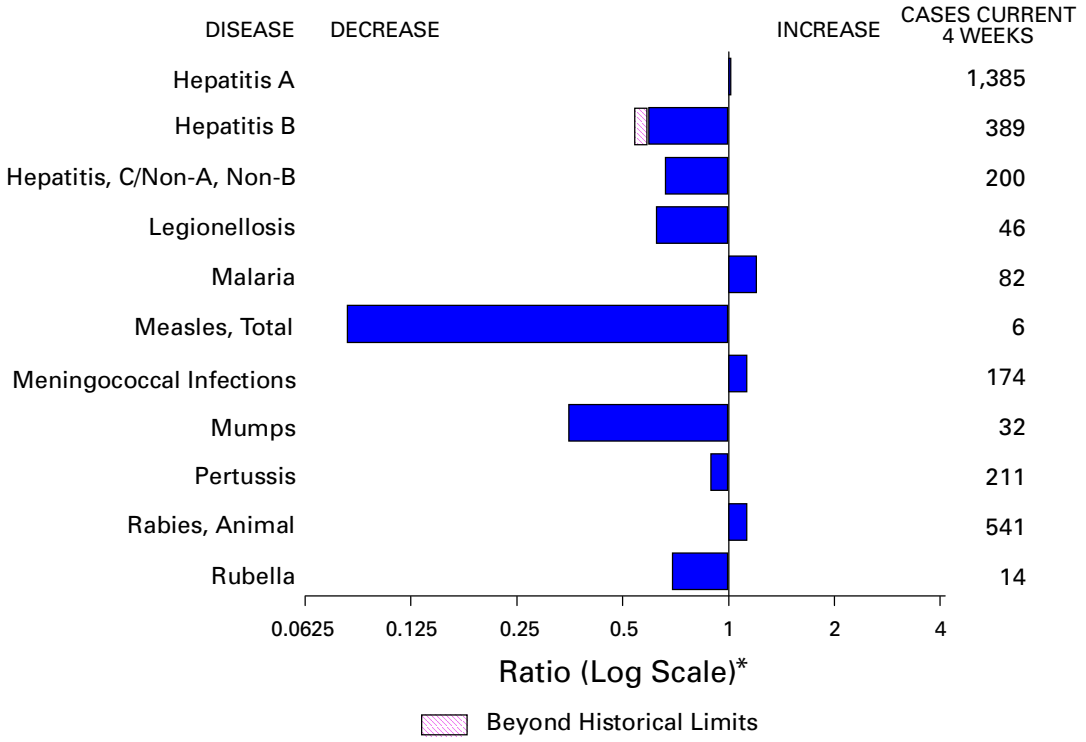
Preventing the Spread of Staphylococci with Reduced Vancomycin Susceptibility

To prevent the spread of staphylococci with reduced susceptibility to vancomycin within and between facilities and to minimize the potential for the organism to become endemic, the following steps should be taken whenever such an organism is isolated:

1. The laboratory should immediately notify infection-control personnel, the clinical unit, and the attending physician.
2. Infection-control personnel, in collaboration with appropriate authorities (including the SHD and CDC), should initiate an epidemiologic and laboratory investigation.
3. Medical and nursing staff should
   a. isolate the patient in a private room and use contact precautions (gown, mask, glove, and antibacterial soap for handwashing) as recommended for multidrug-resistant organisms (*6*);
   b. minimize the number of persons with access to colonized/infected patients; and
   c. dedicate specific health-care workers to provide one-on-one care for the colonized/infected patient or the cohort of colonized/infected patients.
4. Infection-control personnel should
   a. inform all personnel providing direct patient care of the epidemiologic implications of such strains and of the infection-control precautions necessary for their containment;
   b. monitor and strictly enforce compliance with contact precautions and other recommended infection-control practices;

(Continued on page 635)

FIGURE I. Selected notifiable disease reports, comparison of provisional 4-week totals ending July 5, 1997, with historical data — United States



*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.

TABLE I. Summary — provisional cases of selected notifiable diseases, United States, cumulative, week ending July 5, 1997 (27th Week)

	Cum. 1997		Cum. 1997
Anthrax	-	Plague	2
Brucellosis	28	Poliomyelitis, paralytic	-
Cholera	3	Psittacosis	21
Congenital rubella syndrome	2	Rabies, human	2
Cryptosporidiosis*	605	Rocky Mountain spotted fever (RMSF)	121
Diphtheria	5	Streptococcal disease, invasive Group A	867
Encephalitis: California*	4	Streptococcal toxic-shock syndrome*	21
eastern equine*	-	Syphilis, congenital¶	125
St. Louis*	1	Tetanus	21
western equine*	1	Toxic-shock syndrome	60
Hansen Disease	53	Trichinosis	3
Hantavirus pulmonary syndrome*†	6	Typhoid fever	136
Hemolytic uremic syndrome, post-diarrheal*	21	Yellow fever	-
HIV infection, pediatric*§	131		

-:no reported cases
*Not notifiable in all states.
†Updated weekly from reports to the Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases (NCID).
§Updated monthly to the Division of HIV/AIDS Prevention–Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP), last update June 24, 1997.
¶Updated from reports to the Division of STD Prevention, NCHSTP.

TABLE II. Provisional cases of selected notifiable diseases, United States, weeks ending July 5, 1997, and July 6, 1996 (27th Week)

Reporting Area	AIDS		Chlamydia		*Escherichia coli* O157:H7		Gonorrhea		Hepatitis C/NA,NB	
					NETSS†	PHLIS§				
	Cum. 1997*	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1997	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996
UNITED STATES	30,463	34,082	208,706	206,400	661	327	130,054	152,114	1,558	1,845
NEW ENGLAND	1,277	1,384	8,369	8,342	60	27	2,803	3,205	31	50
Maine	28	22	485	U	6	-	29	22	-	-
N.H.	17	42	383	371	4	3	58	72	6	4
Vt.	23	10	201	231	3	1	25	30	-	15
Mass.	467	648	3,637	3,349	38	23	1,135	1,080	21	28
R.I.	85	94	1,021	1,042	1	-	234	267	4	3
Conn.	657	568	2,642	3,349	8	-	1,322	1,734	-	-
MID. ATLANTIC	9,745	9,439	29,047	34,558	42	11	17,032	21,254	170	154
Upstate N.Y.	1,645	1,163	N	N	27	4	2,741	3,711	132	122
N.Y. City	4,978	5,302	15,258	18,913	7	-	6,697	8,292	-	3
N.J.	1,973	1,786	4,448	6,729	8	5	3,147	4,184	-	-
Pa.	1,149	1,188	9,341	8,916	N	2	4,447	5,067	38	29
E.N. CENTRAL	2,041	2,762	29,720	44,701	111	41	18,163	29,114	289	272
Ohio	396	618	6,345	10,514	31	15	4,140	7,372	8	10
Ind.	361	389	4,378	4,878	23	10	2,917	3,242	7	7
Ill.	765	1,203	5,643	12,659	27	-	2,757	8,438	32	53
Mich.	386	401	9,173	11,158	30	6	6,529	7,618	242	202
Wis.	133	151	4,181	5,492	N	10	1,820	2,444	-	-
W.N. CENTRAL	565	811	11,609	16,079	99	64	5,434	7,429	90	50
Minn.	101	157	U	2,702	42	27	U	1,099	2	-
Iowa	70	57	2,351	1,980	16	8	643	504	18	24
Mo.	237	398	5,770	6,814	18	22	3,757	4,396	57	12
N. Dak.	7	9	417	508	3	3	28	14	2	-
S. Dak.	4	8	631	688	6	-	67	97	-	-
Nebr.	61	55	489	1,056	9	-	126	220	2	5
Kans.	85	127	1,951	2,331	5	4	813	1,099	9	9
S. ATLANTIC	7,504	8,521	43,860	25,736	72	35	42,222	48,627	151	93
Del.	144	165	-	563	2	2	601	742	-	-
Md.	950	1,022	3,711	U	6	3	6,713	5,018	10	1
D.C.	538	599	N	N	-	-	1,535	2,275	-	-
Va.	651	542	5,686	5,554	N	15	3,975	4,797	11	8
W. Va.	57	65	1,543	1,079	N	-	469	367	9	7
N.C.	428	466	8,783	U	19	12	8,148	9,531	29	27
S.C.	410	439	6,150	U	1	-	5,586	5,696	26	15
Ga.	965	1,279	5,781	6,327	19	-	6,513	10,926	U	-
Fla.	3,361	3,944	12,206	12,213	25	3	8,682	9,275	66	35
E.S. CENTRAL	1,022	1,132	16,908	15,487	48	7	16,849	16,367	184	341
Ky.	177	173	3,492	3,525	15	-	2,186	2,097	8	20
Tenn.	418	444	6,292	6,684	24	7	5,245	5,738	120	265
Ala.	237	323	4,133	4,337	6	-	5,860	6,752	6	2
Miss.	190	192	2,991	941	3	-	3,558	1,780	50	54
W.S. CENTRAL	3,187	3,297	28,107	10,906	28	5	17,442	10,223	194	168
Ark.	120	144	648	901	4	1	1,333	2,128	-	4
La.	545	777	4,142	3,630	4	3	3,865	3,839	117	101
Okla.	166	139	3,744	3,942	2	1	2,377	2,428	4	1
Tex.	2,356	2,237	19,573	2,433	18	-	9,867	1,828	73	62
MOUNTAIN	881	971	12,271	12,972	81	45	3,761	3,988	201	327
Mont.	22	14	477	636	5	-	20	13	12	10
Idaho	28	23	754	794	12	8	57	55	24	84
Wyo.	13	3	284	335	4	-	26	14	80	100
Colo.	210	298	1,896	989	30	16	1,058	916	24	30
N. Mex.	79	56	1,844	2,150	5	4	640	438	33	40
Ariz.	227	281	4,806	5,782	N	13	1,442	1,945	21	37
Utah	68	102	847	799	22	-	124	156	3	12
Nev.	234	194	1,363	1,487	3	4	394	451	4	14
PACIFIC	4,241	5,765	28,815	37,619	120	89	6,348	11,907	248	390
Wash.	380	380	4,711	5,136	23	22	986	1,116	16	34
Oreg.	162	266	1,991	2,856	37	40	309	438	4	6
Calif.	3,643	5,016	20,564	28,165	57	24	4,606	9,867	150	237
Alaska	22	14	731	538	3	-	211	228	-	2
Hawaii	34	89	818	924	N	3	236	258	78	111
Guam	2	4	31	220	N	-	3	36	-	6
P.R.	1,021	1,047	U	U	23	U	337	326	62	94
V.I.	52	14	N	N	N	U	-	-	-	-
Amer. Samoa	-	-	-	-	N	U	-	-	-	-
C.N.M.I.	1	-	N	N	N	U	16	11	2	-

N: Not notifiable U: Unavailable -: no reported cases C.N.M.I.: Commonwealth of Northern Mariana Islands

*Updated monthly to the Division of HIV/AIDS Prevention–Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention, last update June 24, 1997.

†National Electronic Telecommunications System for Surveillance.

§Public Health Laboratory Information System.

TABLE II. (Cont'd.) Provisional cases of selected notifiable diseases, United States, weeks ending July 5, 1997, and July 6, 1996 (27th Week)

Reporting Area	Legionellosis		Lyme Disease		Malaria		Syphilis (Primary & Secondary)		Tuberculosis		Rabies, Animal
	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997
UNITED STATES	412	385	1,615	2,966	677	632	4,066	5,937	8,371	9,351	3,727
NEW ENGLAND	25	18	353	543	36	24	79	82	214	226	557
Maine	1	1	3	7	1	3	-	-	11	16	113
N.H.	3	-	7	13	1	1	-	1	6	8	21
Vt.	4	2	3	4	2	2	-	-	3	1	90
Mass.	7	9	59	32	14	7	38	39	127	98	117
R.I.	5	6	43	52	4	3	2	1	16	23	11
Conn.	5	N	238	435	14	8	39	41	51	80	205
MID. ATLANTIC	66	83	898	2,089	170	198	191	272	1,610	1,652	770
Upstate N.Y.	15	21	140	898	29	38	18	43	215	180	564
N.Y. City	2	4	16	115	91	109	40	88	841	852	-
N.J.	11	8	277	504	37	36	77	89	314	365	84
Pa.	38	50	465	572	13	15	56	52	240	255	122
E.N. CENTRAL	140	137	26	26	42	84	346	1,001	851	994	78
Ohio	75	47	20	12	9	7	107	376	156	155	59
Ind.	23	32	5	9	6	6	77	130	81	97	8
Ill.	3	17	1	5	5	42	34	272	414	540	2
Mich.	33	27	-	-	19	17	72	109	143	156	8
Wis.	6	14	U	U	3	12	56	114	57	46	1
W.N. CENTRAL	37	21	20	56	26	14	75	209	263	243	241
Minn.	1	1	15	3	9	3	U	25	68	62	23
Iowa	9	3	1	9	8	2	3	13	30	34	83
Mo.	10	5	2	24	4	7	50	149	106	89	12
N. Dak.	2	-	-	-	2	-	-	-	5	3	34
S. Dak.	2	2	-	-	-	-	-	-	7	13	32
Nebr.	9	8	1	-	1	-	1	7	12	13	1
Kans.	4	2	1	20	2	2	21	15	35	29	56
S. ATLANTIC	62	52	187	140	150	95	1,705	2,003	1,680	1,754	1,579
Del.	6	4	16	62	2	2	15	19	11	27	33
Md.	14	7	135	32	45	28	480	340	154	152	288
D.C.	3	3	7	1	9	5	45	84	52	73	2
Va.	11	12	4	7	32	16	144	234	165	149	312
W. Va.	N	N	1	4	-	1	1	2	29	27	45
N.C.	6	5	8	27	7	10	364	550	196	247	495
S.C.	2	4	1	2	9	4	211	224	180	196	83
Ga.	-	1	1	-	14	8	285	346	305	338	159
Fla.	20	15	14	5	32	21	160	204	588	545	162
E.S. CENTRAL	22	23	34	38	15	15	912	1,368	557	730	141
Ky.	2	2	4	13	3	3	80	70	97	120	19
Tenn.	14	9	15	12	4	6	386	444	154	254	81
Ala.	2	2	4	1	5	3	242	287	212	233	41
Miss.	4	10	11	12	3	3	204	567	94	123	-
W.S. CENTRAL	7	2	28	29	6	13	580	605	1,055	988	166
Ark.	-	-	4	15	2	-	60	143	107	102	25
La.	2	-	1	1	4	2	200	289	-	5	1
Okla.	2	2	11	3	-	-	59	99	97	83	63
Tex.	3	-	12	10	-	11	261	74	851	798	77
MOUNTAIN	26	25	8	4	36	29	72	73	283	325	60
Mont.	1	1	-	-	2	3	-	-	7	14	14
Idaho	2	-	2	-	-	-	-	1	7	4	-
Wyo.	1	3	2	3	2	2	-	2	2	3	18
Colo.	8	6	2	-	18	14	3	22	50	44	-
N. Mex.	1	1	-	-	5	1	-	4	16	51	4
Ariz.	7	7	1	-	4	3	59	38	144	114	22
Utah	5	2	-	1	2	4	3	2	11	34	-
Nev.	1	5	1	-	3	2	7	4	46	61	2
PACIFIC	27	24	61	41	196	160	106	324	1,858	2,439	135
Wash.	6	2	2	3	8	9	7	6	99	133	-
Oreg.	-	-	9	10	10	11	4	4	82	92	2
Calif.	20	22	50	27	173	134	93	313	1,546	2,069	115
Alaska	-	-	-	-	3	2	1	-	46	46	18
Hawaii	1	-	-	1	2	4	1	1	85	99	-
Guam	-	1	-	-	-	-	-	3	5	55	-
P.R.	-	-	-	-	3	-	124	127	88	105	31
V.I.	-	-	-	-	-	-	-	-	-	-	-
Amer. Samoa	-	-	-	-	-	-	-	-	-	-	-
C.N.M.I.	-	-	-	-	-	-	5	1	-	-	-

N: Not notifiable U: Unavailable -: no reported cases

TABLE III. Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending July 5, 1997, and July 6, 1996 (27th Week)

Reporting Area	*H. influenzae*, invasive		Hepatitis (Viral), by type				Measles (Rubeola)					
			A		B		Indigenous		Imported†		Total	
	Cum. 1997*	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	1997	Cum. 1997	1997	Cum. 1997	Cum. 1997	Cum. 1996
UNITED STATES	602	620	13,729	13,877	4,308	4,802	-	50	-	21	71	286
NEW ENGLAND	34	16	306	160	76	101	-	9	-	1	10	11
Maine	3	-	41	12	7	2	-	-	-	-	-	-
N.H.	4	9	18	7	5	8	-	1	-	-	1	-
Vt.	3	-	7	3	2	8	-	-	-	-	-	1
Mass.	21	6	129	79	32	30	-	8	-	-	8	9
R.I.	2	1	47	7	9	6	-	-	-	-	-	-
Conn.	1	-	64	52	21	47	U	-	U	1	1	1
MID. ATLANTIC	69	130	1,003	934	596	777	-	12	-	4	16	25
Upstate N.Y.	12	33	147	208	117	180	-	2	-	3	5	4
N.Y. City	20	34	354	297	210	281	-	4	-	1	5	9
N.J.	27	35	176	210	127	159	-	1	-	-	1	1
Pa.	10	28	326	219	142	157	-	5	-	-	5	11
E.N. CENTRAL	98	107	1,403	1,261	460	566	-	5	-	3	8	16
Ohio	59	56	204	470	44	64	-	-	-	-	-	2
Ind.	8	7	159	161	49	77	-	-	-	-	-	-
Ill.	22	32	299	318	111	171	-	5	-	1	6	3
Mich.	8	7	665	203	241	202	-	-	-	2	2	2
Wis.	1	5	76	109	15	52	-	-	-	-	-	9
W.N. CENTRAL	29	21	1,052	1,069	255	242	-	9	-	2	11	16
Minn.	19	10	90	50	23	19	-	-	-	2	2	14
Iowa	3	3	179	208	29	29	-	-	-	-	-	-
Mo.	3	5	565	543	175	156	-	1	-	-	1	1
N. Dak.	-	-	10	28	1	-	-	-	-	-	-	-
S. Dak.	2	1	14	39	-	-	U	8	U	-	8	-
Nebr.	1	1	47	82	10	16	-	-	-	-	-	-
Kans.	1	1	147	119	17	22	-	-	-	-	-	1
S. ATLANTIC	119	108	874	561	637	643	-	2	-	4	6	5
Del.	-	1	12	6	3	4	-	-	-	-	-	1
Md.	47	37	141	103	95	82	-	-	-	1	1	-
D.C.	2	5	14	18	21	26	-	-	-	1	1	-
Va.	7	4	100	82	63	80	-	-	-	-	-	2
W. Va.	3	4	6	12	9	14	-	-	-	-	-	-
N.C.	17	18	106	68	123	182	-	-	-	1	1	-
S.C.	4	3	66	30	60	43	-	-	-	-	-	-
Ga.	20	27	190	41	57	7	-	-	-	-	-	1
Fla.	19	9	239	201	206	205	-	2	-	1	3	1
E.S. CENTRAL	34	18	339	799	361	411	-	-	-	-	-	-
Ky.	4	5	44	16	22	40	-	-	-	-	-	-
Tenn.	22	7	209	558	234	240	-	-	-	-	-	-
Ala.	8	5	51	101	37	27	-	-	-	-	-	-
Miss.	-	1	35	124	68	104	U	-	U	-	-	-
W.S. CENTRAL	31	27	2,855	2,586	540	524	-	3	-	1	4	5
Ark.	1	-	141	254	31	45	-	-	-	-	-	-
La.	6	2	114	82	68	60	-	-	-	-	-	-
Okla.	19	22	859	1,088	17	24	-	-	-	-	-	-
Tex.	5	3	1,741	1,162	424	395	U	3	U	1	4	5
MOUNTAIN	61	33	2,125	2,255	475	587	-	5	-	-	5	80
Mont.	-	-	52	67	5	6	-	-	-	-	-	-
Idaho	1	1	77	136	15	62	-	-	-	-	-	1
Wyo.	1	-	20	20	20	22	-	-	-	-	-	-
Colo.	9	7	241	208	93	64	-	-	-	-	-	6
N. Mex.	7	8	171	257	159	196	-	-	-	-	-	6
Ariz.	24	12	1,078	861	105	137	-	5	-	-	5	8
Utah	3	5	352	504	55	60	-	-	-	-	-	54
Nev.	16	-	134	202	23	40	-	-	-	-	-	5
PACIFIC	127	160	3,772	4,252	908	951	-	5	-	6	11	128
Wash.	2	2	285	286	41	55	-	-	-	-	-	37
Oreg.	22	22	200	560	59	61	-	-	-	-	-	7
Calif.	97	130	3,194	3,329	787	824	-	2	-	6	8	19
Alaska	1	4	23	28	13	4	-	-	-	-	-	63
Hawaii	5	2	70	49	8	7	-	3	-	-	3	2
Guam	-	-	-	6	1	-	U	-	U	-	-	-
P.R.	-	1	172	111	691	530	-	-	-	-	-	2
V.I.	-	-	-	24	-	21	U	-	U	-	-	-
Amer. Samoa	-	-	-	-	-	-	U	-	U	-	-	-
C.N.M.I.	5	10	1	1	21	5	U	1	U	-	1	-

N: Not notifiable U: Unavailable -: no reported cases

*Of 128 cases among children aged <5 years, serotype was reported for 65 and of those, 26 were type b.

†For imported measles, cases include only those resulting from importation from other countries.

TABLE III. (Cont'd.) Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending July 5, 1997, and July 6, 1996 (27th Week)

Reporting Area	Meningococcal Disease		Mumps			Pertussis			Rubella		
	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996
UNITED STATES	2,000	1,934	3	326	379	44	2,496	1,940	-	64	129
NEW ENGLAND	123	81	-	7	1	5	521	435	-	-	24
Maine	12	9	-	-	-	-	6	13	-	-	-
N.H.	13	3	-	-	-	1	60	19	-	-	-
Vt.	2	3	-	-	-	2	173	10	-	-	2
Mass.	62	30	-	2	1	2	260	388	-	-	20
R.I.	9	8	-	4	-	-	12	-	-	-	-
Conn.	25	28	U	1	-	U	10	5	U	-	2
MID. ATLANTIC	175	213	-	30	53	3	173	125	-	3	7
Upstate N.Y.	44	53	-	6	15	-	52	62	-	1	3
N.Y. City	31	31	-	-	13	-	40	19	-	2	2
N.J.	40	46	-	-	2	-	5	7	-	-	2
Pa.	60	83	-	24	23	3	76	37	-	-	-
E.N. CENTRAL	284	278	2	34	85	3	188	257	-	4	3
Ohio	110	97	2	16	27	3	77	82	-	-	-
Ind.	32	37	-	4	5	-	29	15	-	-	-
Ill.	85	83	-	7	17	-	28	61	-	1	1
Mich.	34	29	-	7	35	-	31	22	-	-	2
Wis.	23	32	-	-	1	-	23	77	-	3	-
W.N. CENTRAL	149	148	-	12	5	3	146	71	-	-	-
Minn.	19	14	-	5	1	3	99	43	-	-	-
Iowa	33	32	-	6	-	-	16	3	-	-	-
Mo.	75	61	-	-	2	-	19	15	-	-	-
N. Dak.	1	2	-	-	2	-	2	1	-	-	-
S. Dak.	4	7	U	-	-	U	2	2	U	-	-
Nebr.	5	13	-	1	-	-	3	2	-	-	-
Kans.	12	19	-	-	-	-	5	5	-	-	-
S. ATLANTIC	362	293	-	46	54	7	248	186	-	33	22
Del.	5	2	-	-	-	-	-	13	-	-	-
Md.	35	35	-	4	18	1	79	64	-	-	-
D.C.	1	4	-	-	-	-	2	-	-	-	1
Va.	33	35	-	6	5	-	25	20	-	1	2
W. Va.	14	12	-	-	-	-	4	2	-	-	-
N.C.	64	49	-	7	11	-	68	34	-	22	8
S.C.	41	38	-	10	5	-	11	7	-	9	1
Ga.	69	81	-	4	2	-	7	9	-	-	-
Fla.	100	37	-	15	13	6	52	37	-	1	10
E.S. CENTRAL	149	136	-	16	15	1	54	149	-	-	2
Ky.	35	19	-	3	-	-	11	128	-	-	-
Tenn.	54	41	-	3	1	-	22	12	-	-	-
Ala.	44	40	-	6	3	1	13	4	-	-	2
Miss.	16	36	U	4	11	U	8	5	U	-	N
W.S. CENTRAL	199	222	-	34	28	5	48	65	-	4	7
Ark.	25	26	-	-	1	1	10	2	-	-	-
La.	37	41	-	11	10	-	11	4	-	-	1
Okla.	23	20	-	-	-	4	10	5	-	-	-
Tex.	114	135	U	23	17	U	17	54	U	4	6
MOUNTAIN	116	116	-	43	16	7	721	184	-	5	6
Mont.	8	5	-	-	-	-	9	7	-	-	-
Idaho	8	16	-	2	-	1	510	60	-	1	2
Wyo.	1	3	-	1	-	1	5	1	-	-	-
Colo.	32	19	-	3	3	1	141	41	-	-	2
N. Mex.	18	20	N	N	N	1	32	32	-	-	-
Ariz.	32	29	-	29	1	3	18	12	-	4	1
Utah	11	11	-	6	2	-	4	6	-	-	-
Nev.	6	13	-	2	10	-	2	25	-	-	1
PACIFIC	443	447	1	104	122	10	397	468	-	15	58
Wash.	54	57	-	12	17	10	192	191	-	3	12
Oreg.	91	77	-	-	-	-	18	33	-	-	1
Calif.	295	307	1	80	86	-	180	231	-	7	42
Alaska	1	4	-	2	2	-	1	1	-	-	-
Hawaii	2	2	-	10	17	-	6	12	-	5	3
Guam	-	3	U	1	4	U	-	-	U	-	-
P.R.	8	9	-	4	1	-	-	2	-	-	-
V.I.	-	-	U	-	1	U	-	-	U	-	-
Amer. Samoa	-	-	U	-	-	U	-	-	U	-	-
C.N.M.I.	-	-	U	4	-	U	-	-	U	-	-

N: Not notifiable U: Unavailable -: no reported cases

TABLE IV. Deaths in 122 U.S. cities,* week ending July 5, 1997 (27th Week)

Reporting Area	All Causes, By Age (Years)						P&I† Total
	All Ages	>65	45-64	25-44	1-24	<1	
NEW ENGLAND	465	324	84	35	15	7	34
Boston, Mass.	141	90	32	12	5	2	11
Bridgeport, Conn.	32	20	8	2	2	-	2
Cambridge, Mass.	18	15	3	-	-	-	2
Fall River, Mass.	29	23	3	3	-	-	-
Hartford, Conn.	41	27	4	6	1	3	1
Lowell, Mass.	13	9	3	1	-	-	2
Lynn, Mass.	8	5	3	-	-	-	1
New Bedford, Mass.	13	12	1	-	-	-	1
New Haven, Conn.	20	10	5	3	1	1	1
Providence, R.I.	42	31	6	2	3	-	8
Somerville, Mass.	6	5	-	1	-	-	-
Springfield, Mass.	33	23	6	2	2	-	-
Waterbury, Conn.	26	23	1	2	-	-	1
Worcester, Mass.	43	31	9	1	1	1	4
MID. ATLANTIC	2,095	1,435	388	166	57	48	81
Albany, N.Y.	46	36	6	1	2	1	2
Allentown, Pa.	18	12	3	3	-	-	-
Buffalo, N.Y.	62	48	11	1	1	1	3
Camden, N.J.	25	15	6	1	-	3	1
Elizabeth, N.J.	14	11	3	-	-	-	-
Erie, Pa.	35	29	3	1	-	2	1
Jersey City, N.J.	77	52	16	6	2	1	-
New York City, N.Y.	1,014	689	188	89	25	23	25
Newark, N.J.	61	25	22	9	4	-	4
Paterson, N.J.	13	11	1	-	1	-	-
Philadelphia, Pa.	401	253	90	35	17	6	22
Pittsburgh, Pa.§	46	37	7	1	-	1	5
Reading, Pa.	7	5	1	1	-	-	-
Rochester, N.Y.	101	79	16	3	1	2	6
Schenectady, N.Y.	21	16	3	2	-	-	4
Scranton, Pa.	35	29	2	3	-	1	2
Syracuse, N.Y.	70	53	3	7	3	4	4
Trenton, N.J.	30	22	3	2	-	3	2
Utica, N.Y.	19	13	4	1	1	-	-
Yonkers, N.Y.	U	U	U	U	U	U	U
E.N. CENTRAL	1,882	1,239	369	162	64	48	97
Akron, Ohio	30	17	7	4	1	1	-
Canton, Ohio	31	24	5	-	1	1	3
Chicago, Ill.	461	274	106	46	22	13	22
Cincinnati, Ohio	74	54	16	4	-	-	8
Cleveland, Ohio	96	52	27	8	4	5	-
Columbus, Ohio	157	105	31	13	4	4	9
Dayton, Ohio	94	68	13	11	2	-	7
Detroit, Mich.	172	109	37	19	4	3	4
Evansville, Ind.	29	24	4	1	-	-	1
Fort Wayne, Ind.	74	47	14	8	3	2	1
Gary, Ind.	17	13	1	1	2	-	1
Grand Rapids, Mich.	51	39	4	3	4	1	1
Indianapolis, Ind.	161	90	43	14	7	7	12
Lansing, Mich.	32	24	6	1	-	1	3
Milwaukee, Wis.	122	94	17	7	2	2	7
Peoria, Ill.	30	18	5	2	3	2	3
Rockford, Ill.	38	26	6	3	1	2	2
South Bend, Ind.	60	46	4	7	1	2	6
Toledo, Ohio	96	71	16	6	2	1	4
Youngstown, Ohio	57	44	7	4	1	1	3
W.N. CENTRAL	678	472	121	47	12	17	45
Des Moines, Iowa	89	63	19	4	3	-	11
Duluth, Minn.	36	27	8	-	1	-	4
Kansas City, Kans.	13	7	4	1	-	1	-
Kansas City, Mo.	93	58	16	8	1	1	3
Lincoln, Nebr.	25	20	2	3	-	-	2
Minneapolis, Minn.	154	113	19	14	2	6	10
Omaha, Nebr.	61	37	12	6	2	4	3
St. Louis, Mo.	96	69	16	7	-	4	5
St. Paul, Minn.	50	38	11	1	-	-	5
Wichita, Kans.	61	40	14	3	3	1	2

Reporting Area	All Causes, By Age (Years)						P&I† Total
	All Ages	>65	45-64	25-44	1-24	<1	
S. ATLANTIC	1,144	714	253	124	35	18	46
Atlanta, Ga.	160	101	37	17	4	1	4
Baltimore, Md.	168	108	32	20	6	2	14
Charlotte, N.C.	59	41	10	5	1	2	4
Jacksonville, Fla.	112	70	26	9	7	-	-
Miami, Fla.	99	59	18	20	1	1	-
Norfolk, Va.	47	33	8	3	1	2	1
Richmond, Va.	50	29	14	7	-	-	2
Savannah, Ga.	59	32	16	5	2	4	2
St. Petersburg, Fla.	69	48	11	7	1	2	2
Tampa, Fla.	153	105	30	13	4	1	13
Washington, D.C.	147	79	40	17	8	3	4
Wilmington, Del.	21	9	11	1	-	-	-
E.S. CENTRAL	670	464	119	39	30	18	53
Birmingham, Ala.	177	131	27	9	6	4	12
Chattanooga, Tenn.	54	38	10	2	2	2	5
Knoxville, Tenn.	85	66	12	3	2	2	14
Lexington, Ky.	53	29	15	2	2	5	5
Memphis, Tenn.	141	93	24	12	10	2	13
Mobile, Ala.	45	30	7	4	2	2	-
Montgomery, Ala.	19	15	2	-	2	-	1
Nashville, Tenn.	96	62	22	7	4	1	3
W.S. CENTRAL	1,228	806	232	107	55	28	67
Austin, Tex.	43	29	7	3	3	1	1
Baton Rouge, La.	48	32	9	5	2	-	-
Corpus Christi, Tex.	48	35	6	4	2	1	4
Dallas, Tex.	122	65	37	11	8	1	1
El Paso, Tex.	58	44	7	6	1	-	6
Ft. Worth, Tex.	93	64	18	4	4	3	3
Houston, Tex.	306	195	59	31	12	9	23
Little Rock, Ark.	63	43	9	6	2	3	4
New Orleans, La.	95	56	13	12	13	1	-
San Antonio, Tex.	139	98	25	9	5	2	9
Shreveport, La.	80	56	11	7	2	4	5
Tulsa, Okla.	133	89	31	9	1	3	11
MOUNTAIN	616	420	109	48	20	19	34
Albuquerque, N.M.	90	68	16	3	2	1	7
Boise, Idaho	33	28	5	-	-	-	3
Colo. Springs, Colo.	45	31	9	2	2	1	2
Denver, Colo.	92	50	19	8	7	8	6
Las Vegas, Nev.	112	70	25	12	2	3	8
Ogden, Utah	17	14	1	1	-	1	-
Phoenix, Ariz.	94	63	12	10	5	4	-
Pueblo, Colo.	20	17	2	1	-	-	-
Salt Lake City, Utah	U	U	U	U	U	U	U
Tucson, Ariz.	113	79	20	11	2	1	8
PACIFIC	1,429	1,011	239	102	42	35	104
Berkeley, Calif.	7	6	-	-	-	1	1
Fresno, Calif.	70	48	14	1	4	3	3
Glendale, Calif.	37	32	4	-	-	1	4
Honolulu, Hawaii	53	36	9	1	2	5	3
Long Beach, Calif.	63	46	9	3	4	1	9
Los Angeles, Calif.	460	332	71	39	11	7	21
Pasadena, Calif.	12	9	3	-	-	-	2
Portland, Oreg.	112	72	20	12	4	4	5
Sacramento, Calif.	U	U	U	U	U	U	U
San Diego, Calif.	100	69	17	6	2	6	13
San Francisco, Calif.	125	84	25	13	2	1	11
San Jose, Calif.	170	117	30	14	7	2	14
Santa Cruz, Calif.	19	13	4	2	-	-	4
Seattle, Wash.	99	69	16	8	5	1	3
Spokane, Wash.	52	41	7	1	1	2	5
Tacoma, Wash.	50	37	10	2	-	1	6
TOTAL	10,207¶	6,885	1,914	830	330	238	561

U: Unavailable -: no reported cases

*Mortality data in this table are voluntarily reported from 122 cities in the United States, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included.

†Pneumonia and influenza.

§Because of changes in reporting methods in this Pennsylvania city, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

¶Total includes unknown ages.

Guidelines — Continued

c. determine whether transmission has already occurred by obtaining baseline cultures (before initiation of precautions) for staphylococci with reduced susceptibility to vancomycin from the anterior nares and hands of all health-care workers, roommates, and others with direct patient contact;
d. assess efficacy of precautions by monitoring health-care personnel for acquisition of staphylococci with reduced susceptibility to vancomycin as recommended by consultants from SHD or CDC;
e. avoid transferring infected patients within or between facilities, and if transfer is necessary, fully inform the receiving institution or unit of the patient's colonization/infection status and appropriate precautions; and
f. consult with SHD and CDC before discharge of the colonized/infected patient.

Reported by: Hospital Infection Control Practices Advisory Committee. Div of Anti-Infective Drug Products and Div of Special Pathogens and Immunologic Drug Products, Center for Drug Evaluation and Research, Food and Drug Administration. Hospital Infections Program, National Center for Infectious Diseases, CDC.

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Contributors to the Production of the *MMWR* (Weekly)

Weekly Notifiable Disease Morbidity Data and 122 Cities Mortality Data

Denise Koo, M.D., M.P.H.

State Support Team

Robert Fagan
Jill Andrews
Karl A. Brendel
Siobhan Gilchrist, M.P.H.
Harry Holden
Gerald Jones
Felicia Perry
Svati Shah, M.P.H.

CDC Operations Team

Carol M. Knowles
Deborah A. Adams
Willie J. Anderson
Christine R. Burgess
Patsy A. Hall
Myra A. Montalbano
Angela Trosclair, M.S.

Desktop Publishing and Graphics Support

Morie M. Higgins
Peter M. Jenkins

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