July 20, 1984 / Vol. 33 / No. 28

- **393** ACIP: Rabies Prevention United States, 1984
- 408 Chromosomally Mediated Resistant Neisseria gonorrhoeae – United States
- 410 Fatalities from Occupational Heat Exposure
- 412 Tuberculosis United States, 1983

MORBIDITY AND MORTALITY WEEKLY REPORT

Recommendation of the Immunization Practices Advisory Committee (ACIP)

Rabies Prevention – United States, 1984

These revised recommendations of the Immunization Practices Advisory Committee (ACIP) on rabies prevention update the previous recommendations (MMWR 1980;29: 65-72,277-80) to reflect the current status of rabies and antirabies biologics in the United States. For assistance on problems or questions about rabies prophylaxis, call local or state health departments.*

INTRODUCTION

Although rabies rarely affects humans in the United States, every year, approximately 25,000 persons receive rabies prophylaxis. Appropriate managment of those who may have been exposed to rabies infection depends on the interpretation of the risk of infection and the efficacy and risk of prophylactic treatment. All available methods of systemic prophylactic treatment are complicated by instances of adverse reactions. These are rarely severe. Decisions on management must be made immediately; the longer treatment is postponed, the less likely it is to be effective.

Data on the efficacy of active and passive immunization after rabies exposure have come from both human and animal studies. Evidence from laboratory and field experience in many areas of the world indicates that postexposure prophylaxis combining local wound treatment, vaccine, and rabies immune globulin, is uniformly effective when appropriately used. However, rabies has occasionally developed in humans who had received postexposure antirabies prophylaxis with vaccine alone.

In the United States, rabies in humans has decreased from an average of 22 cases per year in 1946-1950 to zero to five cases per year since 1960. The number of rabies cases among domestic animals has decreased similarly. In 1946, more than 8,000 rabies cases were reported among dogs; 153 cases were reported in 1982. Thus, the likelihood of human exposure to rabies in domestic animals has decreased greatly, although bites by dogs and cats continue to be the principal reasons given for antirabies treatments.

The disease in wildlife—especially skunks, foxes, raccoons, and bats—has become more prevalent in recent years, accounting for approximately 85% of all reported cases of animal rabies every year since 1976. Wild animals now constitute the most important potential source of infection for both humans and domestic animals in the United States. Rabies among animals is present throughout the United States; only Hawaii remains consistently rabies-free.

Four of the six rabies fatalities in U.S. citizens occurring between 1980 and 1983 were related to exposure to rabid dogs outside the United States. In much of the world, including

^{*}If these are unavailable, call the Division of Viral Diseases, Center for Infectious Diseases, CDC ([404] 329-3095 during working hours, or [404] 329-2888 nights, weekends, and holidays).

ACIP: Rabies - Continued

most of Asia and all of Africa and Latin America, the dog remains the major source of human exposure.

RABIES IMMUNIZING PRODUCTS

There are two types of immunizing products: (1) vaccines that induce an active immune response, which requires about 7-10 days to develop but may persist for as long as a year or more, and (2) globulins that provide rapid passive immune protection, which persists for a short period of time, with a half-life of about 21 days. Both types of products should be used concurrently for rabies postexposure prophylaxis.

Vaccines for Use in the United States

Human diploid cell rabies vaccine (HDCV)[†]: HDCV is an inactivated virus vaccine prepared from fixed rabies virus grown in WI-38 or MRC-5 human diploid cell culture. The vaccine grown on WI-38 cells and developed in the United States is inactivated with tri-n-butyl phosphate and β -propiolactone (Wyeth Laboratories' WYVAC®), while that grown in MRC-5 cells and developed in Europe is inactivated with β -propiolactone (Merieux Institute's RABIES VACCINE®). Both vaccines are supplied as 1.0 ml, single-dose vials of lyophilized vaccine with accompanying diluent.

Globulins

Rabies Immune Globulin, Human (RIG): RIG (Cutter Laboratories' HYPERAB® and Merieux Institutes' IMOGAM®) is antirabies gamma globulin concentrated by cold ethanol fractionation from plasma of hyperimmunized human donors. Rabies neutralizing antibody content is standardized to contain 150 international units (IU) per ml. It is supplied in 2-ml (300 IU) and 10-ml (1,500 IU) vials for pediatric and adult use, respectively.

Antirabies Serum, Equine (ARS): ANTIRABLES SERUM® (Sclavo) is a refined, concentrated serum obtained from hyperimmunized horses. Neutralizing antibody content is standardized to contain 1,000 IU per vial. Volume is adjusted by the manufacturer on the basis of antibody potency in each lot. Currently, a 1,000-IU vial contains approximately 5 ml.

RATIONALE FOR CHOICE OF RABIES IMMUNIZING PRODUCTS

Both types of HDCV rabies vaccines are considered equally efficacious and safe when used as indicated on the labels. Only the Merieux Institute vaccine has been evaluated by the intradermal (ID) dose/route for preexposure immunization. No data are available on ID use with the Wyeth Laboratories vaccine. RIG is preferred over ARS, because the latter has a much higher risk of adverse reactions.

Vaccines

The effectiveness of rabies vaccines is measured by their ability to protect persons exposed to rabies and to induce antibodies to rabies virus. HDCV has been used concurrently with RIG or ARS to treat 45 persons bitten by rabid dogs or wolves in Iran, 31 persons bitten by a variety of rabid animals in Germany, and 511 persons bitten by a variety of rabid animals in the United States. In these studies, no person contracted rabies after receiving HDCV in combination with RIG.

All persons treated with RIG and five 1.0-ml intramuscular (IM) doses of HDCV and tested have developed a rabies antibody titer. The definition of a minimally acceptable antibody titer varies between laboratories and is influenced by the type of test conducted. CDC currently specifies a 1:5 titer by the rapid fluorescent-focus inhibition test (RFFIT) as acceptable. The World Health Organization (WHO) specifies a titer of 0.5 I.U.

Serious adverse reactions associated with rabies vaccines include systemic, anaphylactic, and neuroparalytic reactions. Serious adverse reactions occur at lower rates in the HDCV vaccine than with previously available types of rabies vaccine.

[†]Official name: Rabies Vaccine. The duck embryo vaccine which was used from 1957-1982 is no longer available in the United States.

Vol. 33/No. 28 ACIP: Rabies – Continued

Globulins

RIG and ARS are both effective; however, ARS causes serum sickness in over 40% of adult recipients. RIG rarely causes adverse reactions and should be the product of choice when available.

RATIONALE OF TREATMENT

Physicians must evaluate each possible rabies exposure. Local or state public health officials should be consulted if questions arise about the need for prophylaxis.

In the United States, the following factors should be considered before specific antirables treatment is initiated:

Species of Biting Animal

Carnivorous wild animals (especially skunks, raccoons, foxes, coyotes, and bobcats) and bats are the animals most commonly infected with rabies and have caused most of the indigenous cases of human rabies in the United States since 1960. Unless an animal is tested and shown not to be rabid, postexposure prophylaxis should be initiated upon bite or nonbite exposure to the animals. (See definition in "Type of Exposure" below.) If treatment has been initiated and subsequent testing in a competent laboratory shows the exposing animal is not rabid, treatment can be discontinued.

The likelihood that a domestic dog or cat is infected with rabies varies from region to region; hence, the need for postexposure prophylaxis also varies.

Rodents (such as squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, and mice) and lagomorphs (including rabbits and hares) are rarely found to be infected with rabies and have not been known to cause human rabies in the United States. In these cases, the state or local health department should be consulted before a decision is made to initiate postexposure antirabies prophylaxis.

Circumstances of Biting Incident

An unprovoked attack is more likely than a provoked attack to indicate the animal is rabid. Bites inflicted on a person attempting to feed or handle an apparently healthy animal should generally be regarded as provoked.

Type of Exposure

Rabies is transmitted by introducing the virus into open cuts or wounds in skin or via mucous membranes. The likelihood of rabies infection varies with the nature and extent of exposure. Two categories of exposure should be considered.

Bite: Any penetration of the skin by teeth.

Nonbite: Scratches, abrasions, open wounds, or mucous membranes contaminated with saliva or other potentially infectious material, such as brain tissue, from a rabid animal. Casual contact, such as petting a rabid animal (without a bite or nonbite exposure as described above), does not constitute an exposure and is not an indication for prophylaxis. There have been two instances of airborne rabies acquired in laboratories and two probable airborne rabies cases acquired in a bat-infested cave in Texas.

The only documented cases of rabies from human-to-human transmission occurred in four patients in the United States and overseas who received corneas transplanted from persons who died of rabies undiagnosed at the time of death. Stringent guidelines for acceptance of donor corneas should reduce this risk.

Bite and nonbite exposures from humans with rabies theoretically could transmit rabies, although no cases of rabies acquired this way have been documented. Each potential exposure to human rabies should be carefully evaluated to minimize unnecessary rabies prophylaxis.

MANAGEMENT OF BITING ANIMALS

A healthy domestic dog or cat that bites a person should be confined and observed for 10 days and evaluated by a veterinarian at the first sign of illness during confinement or before

ACIP: Rabies - Continued

release. Any illness in the animal should be reported immediately to the local health department. If signs suggestive of rabies develop, the animal should be humanely killed and its head removed and shipped, under refrigeration, for examination by a qualified laboratory designated by the local or state health department. Any stray or unwanted dog or cat that bites a person should be killed immediately and the head submitted, as described above, for rabies examination.

Signs of rabies in wild animals cannot be interpreted reliably; therefore, any wild animal that bites or scratches a person should be killed at once (without unnecessary damage to the head) and the brain submitted, as described above, for examination for evidence of rabies. If the brain is negative by fluorescent-antibody examination for rabies, the saliva can be assumed to contain no virus, and the bitten person need not be treated. If the biting animal is a particularly rare or valuable specimen and the risk of rabies small, consideration may be given to initiating postexposure treatment to the bitten person and delaying killing the animal for rabies testing.

POSTEXPOSURE PROPHYLAXIS

The essential components of rabies postexposure prophylaxis are local treatment of wounds and immunization, including administration, in most instances, of both globulin and vaccine (Tables 1 and 2).

Local Treatment of Wounds

Immediate and thorough washing of all bite wounds and scratches with *soap and water* is perhaps the most effective measure for preventing rabies. In experimental animals, simple local wound cleansing has been shown to reduce markedly the likelihood of rabies.

Tetanus prophylaxis and measures to control bacterial infection should be given as indicated.

Immunization

Postexposure antirabies immunization should always include administration of both antibody (preferably RIG) and vaccine, with one exception: persons who have been previously immunized with the recommended preexposure or postexposure regimens with HDCV or who have been immunized with other types of vaccines and have a history of documented adequate rabies antibody titer (See "RATIONALE FOR CHOICE OF RABIES IMMUNIZING PROD-UCTS") should receive only vaccine. The combination of globulin and vaccine is recommended for both bite exposures and nonbite exposures (as described under "RATIONALE OF TREATMENT"), regardless of the interval between exposure and treatment. The sooner treatment is begun after exposure, the better. However, there have been instances in which the decision to begin treatment was made as late as 6 months or longer after the exposure due to delay in recognition that an exposure had occurred.

HDCV: HDCV is the only type of vaccine currently available in the United States and should be administered in conjunction with RIG at the beginning of postexposure therapy, as described below. In 1977, WHO established a recommendation for six IM doses of HDCV based on studies in Germany and Iran of a regimen of RIG or ARS and six doses of HDCV. When used in this way, the vaccine was safe and effective in protecting 76 persons bitten by proven rabid animals. The vaccine also induced an excellent antibody response in all recipients. Studies conducted by CDC in the United States have shown that a regimen of one dose of RIG and five doses of HDCV was safe and induced an excellent antibody response in all recipients. Of 511 persons bitten by proven rabid animals and so treated, none developed rabies.

Five 1-ml doses of HDCV should be given intramuscularly (for example, in the deltoid region). Other routes of administration, such as the ID route, have not been adequately evaluated for postexposure prophylaxis and should not be used. The first dose should be given as

Vol. 33/No. 28 ACIP: Rabies — Continued

soon as possible after exposure; an additional dose should be given on days 3, 7, 14, and 28 after the first dose. (WHO currently recommends a sixth dose 90 days after the first dose.) Because the antibody response following the recommended vaccination regimen with HDCV has been so satisfactory, routine postvaccination serologic testing is not recommended. In unusual instances, as when the patient is known to be immunosuppressed, serologic testing is indicated. Contact state health department or CDC for recommendations.

RIG (or ARS if RIG is not available): RIG is administered only once, at the beginning of antirabies prophylaxis, to provide immediate antibodies until the patient responds to HDCV by active production of antibodies. If RIG was not given when vaccination was begun, it can be given up to the eighth day after the first dose of vaccine was given. From about the eighth day on, RIG is not indicated, since an antibody response to the vaccine is presumed to have occurred. The recommended dose of RIG is 20 IU/kg or approximately 9 IU/lb of body weight. (When ARS must be used, the recommended dose is 40 IU/kg, approximately 18 IU/lb or 1,000 IU/55 Ib body weight.) If anatomically feasible, up to half the dose of RIG should be thoroughly infiltrated in the area around the wound, the rest should be administered intramuscularly. Because RIG may partially suppress active production of antibody, no more than the recommended dose of RIG should be given.

TABLE 1. Rabies postexposure prophylaxis guide - July 1984

The following recommendations are only a guide. In applying them, take into account the animal species involved, the circumstances of the bite or other exposure, the vaccination status of the animal, and presence of rabies in the region. Local or state public health officials should be consulted if questions arise about the need for rabies prophylaxis.

	Animal species	Condition of animal at time of attack	Treatment of exposed person*
DOMESTIC	Dog and cat	Healthy and available for 10 days of observation Rabid or suspected rabid Unknown (escaped)	None, unless animal develops rabies [†] RIG [§] and HDCV Consult public health officials. If treatment is indicated, give
WILD	Skunk, bat, fox, coyote raccoon, bobcat, and other carnivores	Regard as rabid unless proven negative by laboratory tests ¶	RIG [§] and HDCV RIG [§] and HDCV
OTHER	Livestock, rodents, and lagomorphs (rabbits and hares)		questions about the need squirrels, hamsters, guinea pigs, other rodents, rabbits, and hares

*All bites and wounds should immediately be thoroughly cleansed with soap and water. If antirabies treatment is indicated, both rabies immune globulin (RIG) and human diploid cell rabies vaccine (HDCV) should be given as soon as possible, *regardless* of the interval from exposure. Local reactions to vaccines are common and do not contraindicate continuing treatment. Discontinue vaccine if fluorescentantibody tests of the animal are negative.

[†]During the usual holding period of 10 days, begin treatment with RIG and HDCV at first sign of rabies in a dog or cat that has bitten someone. The symptomatic animal should be killed immediately and tested.

 § If RIG is not available, use antirabies serum, equine (ARS). Do not use more than the recommended dosage.

 \P The animal should be killed and tested as soon as possible. Holding for observation is not recommended.

TABLE 2. Rabies immunization — June 1984

I. PREEXPOSURE IMMUNIZATION. Preexposure immunization consists of three doses of HDCV, 1.0 ml, IM (i.e., deltoid area), one each on days 0, 7, and 28. (See text for details on use of 0.1 ml HDCV ID as an alternative dose/route.) Administration of routine booster doses of vaccine depends on exposure risk category as noted below. Preexposure immunization of immunosuppressed persons is not recommended.

	Criteria for Preexposure Immunization										
Risk category	Nature of risk	Typical populations	Preexposure regimen								
Continuou s	Virus present continuously, often in high concentrations. Aerosol, mucous membrane, bite, or nonbite exposure possible. Specific exposures may go unrecognized.	Rabies research lab workers.* Rabies biologics production workers.	Primary preexposure immunization course. Serology every 6 months. Booster immunization when antibody titer falls falls below acceptable level.*								
Frequent	Exposure usually episodic, with source recognized, but exposure may also be unrecognized. Aerosol, mucous membrane, bite, or nonbite exposure.	Rabies diagnostic lab workers,* spelunkers, veterinarians, and animal control and wildlife workers in rabies epizootic areas.	Primary preexposure immunization course. Booster immunization or serology every 2 years. [†]								
Infrequent (greater than population- at-large)	Exposure nearly always episodic with source recognized. Mucuous membrane, bite, or nonbite exposure.	Veterinarians and animal control and wildlife workers in areas of low rabies endemicity. Certain travelers to foreign rabies epizootic areas. Veterinary students.	Primary preexposure immunization course. No routine booster immunization or serology.								
Rare (population- at-large)	Exposure always episodic, mucous membrane, or bite with source recognized.	U.S. population-at-large, including individuals in rabies-epizootic areas.	No preexposure immunization.								

II. POSTEXPOSURE IMMUNIZATION. All postexposure treatment should begin with immediate thorough cleansing of all wounds with soap and water.

Persons not previously immunized:	RIG, 20 I.U./kg body weight, one half infiltrated at bite site (if possible), remainder IM; 5 doses of HDCV, 1.0 ml IM (i.e., del- toid area), one each on days 0, 3, 7, 14 and 28.
Persons previously immunized ${}^{\hat{S}}$:	Two doses of HDCV, 1.0 ml, IM (i.e., deltoid area), one each on days 0 and 3. RIG should not be administered.

*Judgment of relative risk and extra monitoring of immunization status of laboratory workers is the responsibility of the laboratory supervisor (see U.S. Department of Health and Human Service's *Biosafety in Microbiological and Biomedical Laboratories*, 1984).

[†]Preexposure booster immunization consists of one dose of HDCV, 1.0 ml/dose, IM (deltoid area). Acceptable antibody level is 1:5 titer (complete inhibition in RFFIT at 1:5 dilution). Boost if titer falls below 1:5.

§Preexposure immunization with HDCV; prior postexposure prophylaxis with HDCV; or persons previously immunized with any other type of rabies vaccine and a documented history of positive antibody response to the prior vaccination.

Vol. 33/No. 28 ACIP: Rabies — Continued

TREATMENT OUTSIDE THE UNITED STATES

If postexposure is begun outside the United States with locally produced biologics, it may be desirable to provide additional treatment when the patient reaches the United States. State health departments should be contacted for specific advice in such cases.

PREEXPOSURE IMMUNIZATION

Preexposure immunization may be offered to persons in high-risk groups, such as veterinarians, animal handlers, certain laboratory workers, and persons spending time (e.g., 1 month or more) in foreign countries where rabies is a constant threat. Persons whose vocational or avocational pursuits bring them into contact with potentially rabid dogs, cats, foxes, skunks, bats, or other species at risk of having rabies should also be considered for preexposure prophylaxis.

Preexposure prophylaxis is given for several reasons. First, it may provide protection to persons with inapparent exposures to rabies. Second, it may protect persons whose postexposure therapy might be expected to be delayed. Finally, although it does not eliminate the need for additional therapy after a rabies exposure, it simplifies therapy by eliminating the need for globulin and decreasing the number of doses of vaccine needed. This is of particular importance for persons at high risk of being exposed in countries where the available rabies immunizing products may carry a higher risk of adverse reactions.

Preexposure immunization does not eliminate the need for prompt postexposure prophylaxis following an exposure; it only reduces the postexposure regimen.

Human Diploid Cell Rabies Vaccine

Three 1.0 ml injections of HDCV should be given intramuscularly (for example, in the deltoid area), one on each of days 0, 7, and 28. In a study in the United States, more than 1,000 persons received HDCV according to this regimen; antibody was demonstrated in the sera of all subjects when tested by the RFFIT. Other studies have produced comparable results. Because the antibody response following the recommended vaccination regimen with HDCV has been so satisfactory, routine postvaccination serology is not recommended.

Booster Doses of Vaccine

Persons who work with live rabies virus in research laboratories or vaccine production facilities and are at risk of inapparent exposure should have the rabies antibody titer of their serum determined every 6 months; booster doses of vaccine should be given, as needed, to maintain an adequate titer (See "RATIONALE FOR CHOICE OF RABLES IMMUNIZING PROD-UCTS"). Other laboratory workers, such as those doing rabies diagnostic tests, spelunkers, and those veterinarians, animal control and wildlife officers in areas where animal rabies is epizootic should have boosters every 2 years or have their serum tested for rabies antibody every 2 years and, if the titer is inadequate, have a booster dose. Veterinarians and animal control and wildlife officers, if working in areas of low rabies endemicity, do not require routine booster doses of HDCV after completion of primary preexposure immunization (Table 2).

Postexposure Therapy of Previously Immunized Persons

When an immunized person who was vaccinated by the recommended regimen with HDCV or who had previously demonstrated rabies antibody is exposed to rabies, that person should receive two IM doses (1.0 ml each) of HDCV, one immediately and one 3 days later. RIG should not be given in these cases. If the immune status of a previously vaccinated person who did not receive the recommended HDCV regimen is not known, full primary postexposure antirabies treatment (RIG plus five doses of HDCV) may be necessary. In such cases, if antibody can be demonstrated in a serum sample collected before vaccine is given, treatment can be discontinued after at least two doses of HDCV.

ACIP: Rabies – Continued Intradermal Use of HDCV

HDCV produced by the Merieux Institute has been used for preexposure immunization in a regimen of three 0.1 ml doses given ID in the lateral aspect of the upper arm over the deltoid area, one dose each on days 0, 7, and 28. Experience gained with over 2,000 persons vaccinated in the United States by the ID route has shown that antibody was produced in all recipients, although the mean response was somewhat lower and may be of shorter duration than with comparable IM immunization. Antibody response in some groups vaccinated outside the United States has been found to be inadequate for reasons not yet determined.

Current data provide a sufficient basis to recommend the 0.1 ml ID dose/route as an alternative to the 1.0 ml IM dose/route for preexposure immunization in the United States. Postvaccination serology is not necessary following ID (or IM) immunization, except for persons suspected of being immunosuppressed. The manufacturer has not yet met the packaging and labeling requirements necessary to obtain approval by the U.S. Food and Drug Administration for the ID route. Since the 1.0-ml vial presently available is intended for IM use and contains no preservatives, the reconstituted vaccine must be used immediately. Data on ID immunization are not available for Wyeth Laboratories' vaccine, and it should not be used for ID vaccination.

ACCIDENTAL INOCULATION WITH MODIFIED LIVE RABIES VIRUS

Individuals may be accidentally exposed to attenuated rabies virus while administering modified live rabies virus (MLV) vaccines to animals. While there have been no reported human rabies cases resulting from exposure to needlesticks or sprays with licensed MLV vaccines, vaccine-induced rabies has been observed in animals given MLV vaccines. Absolute assurance of a lack of risk for humans, therefore, cannot be given. The best evidence for a low risk, however, is the absence of recognized cases of vaccine-associated disease in humans despite frequent accidental exposures.

Currently available MLV animal vaccines are made with one of two attenuated strains of rabies virus: high egg passage (HEP) Flury strain or Street Alabama Dufferin (SAD) strain. The HEP Flury and SAD virus strains have been used in animal vaccines for over 10 years without evidence of associated disease in humans; therefore, postexposure treatment is not recommended following exposure to these types of vaccine by needlesticks or sprays.

Because the data are insufficient to assess the true risk associated with any of the MLV vaccines, preexposure immunization, and periodic boosters are recommended for all persons dealing with potentially rabid animals or frequently handling animal rabies vaccines.

ADVERSE REACTIONS

Human Diploid Cell Rabies Vaccine

Reactions after vaccination with HDCV are less common than with previously available vaccines. In a study using five doses of HDCV, local reactions, such as pain, erythema, and swelling or itching at the injection site, were reported in about 25% of recipients of HDCV, and mild systemic reactions, such as headache, nausea, abdominal pain, muscle aches, and dizziness were reported in about 20% of recipients. Two cases of neurologic illness resembling Guillain-Barré syndrome that resolved without sequelae in 12 weeks, and a focal subacute central nervous system disorder temporally associated with HDCV vaccine, have been reported.

Recently, a significant increase has been noted in "immune complex-like" reactions in persons receiving booster doses of HDCV. The illness, characterized by onset 2-21 days postbooster, presents with a generalized urticaria and may also include arthralgia, arthritis, angioedema, nausea, vomiting, fever, and malaise. In no cases were the illnesses life-threatening. Preliminary data suggest this "immune complex-like" illness may occur in up to 6% of persons receiving booster vaccines and much less frequently in persons receiving primary immuniza-

Vol. 33/No. 28

MMWR

ACIP: Rabies - Continued

tion. Additional experience with this vaccine is needed to define more clearly the risk of these adverse reactions.

Vaccines in Other Countries

Many developing countries use inactivated nerve tissue vaccines (NTV) or inactivated suckling mouse brain vaccine (SMBV). NTV is reported to provoke neuroparalytic reactions at a rate of about 1/2,000 vaccinees; the rate for SMBV is about 1/8,000.

Rabies Immune Globulin, Human

Local pain and low-grade fever may follow receipt of RIG. Although not reported specifically for RIG, angioneurotic edema, nephrotic syndrome, and anaphylaxis have been reported after injection of immune serum globulin (ISG). These reactions occur so rarely that the causal relationship between ISG and these reactions is not clear.

Antirabies Serum, Equine

ARS produces serum sickness in at least 40% of adult recipients; reaction rates for children are lower. Anaphylactic reactions may occur. When RIG is not available, and ARS must be used, the patient should be tested for sensitivity to equine serum. (See package circular for details.)

Because adverse reactions are associated more frequently with ARS than with RIG, and ARS might sensitize recipients to equine protein, ARS should be used only when RIG cannot be obtained.

Management of Adverse Reactions

Once initiated, rabies prophylaxis should not be interrupted or discontinued because of local or mild systemic adverse reactions to rabies vaccine. Usually such reactions can be successfully managed with anti-inflammatory and antipyretic agents (aspirin, for example).

When a person with a history of hypersensitivity must be given rabies vaccines, antihistamines may be given; epinephrine should be readily available to counteract anaphylactic reactions, and the person should be carefully observed immediately after immunization.

Serious systemic anaphylactic or neuroparalytic reactions occurring during the administration of rabies vaccines pose a serious dilemma for the attending physician. A patient's risk of developing rabies must be carefully considered before deciding to discontinue vaccination. Moreover, the use of corticosteroids to treat life-threatening neuroparalytic reactions carries the risk of inhibiting the development of active immunity to rabies. It is especially important in these cases that the serum of the patient be tested for rabies antibodies. Advice and assistance on the management of serious adverse reactions in persons receiving rabies vaccines may be sought from the state health department or CDC.

All serious systemic neuroparalytic or anaphylactic reactions to a rabies vaccine should be immediately reported to the state health department or the Division of Viral Diseases, Center for Infectious Diseases, CDC ([404] 329-3095 during working hours, or [404] 329-2888 at other times).

PRECAUTIONS AND CONTRAINDICATIONS

Immunosuppression

Corticosteroids, other immunosupressive agents, and immunosuppressive illnesses can interfere with the development of active immunity and predispose the patient to developing rabies. Immunosuppressive agents should not be administered during postexposure therapy, unless essential for the treatment of other conditions. When rabies postexposure prophylaxis is administered to persons receiving steroids or other immunosuppressive therapy, it is especially important that serum be tested for rabies antibody to ensure that an adequate response has developed.

ACIP: Rabies - Continued

Pregnancy

402

Because of the potential consequences of inadequately treated rabies exposure and limited data that indicate that fetal abnormalities have not been associated with rabies vaccination, pregnancy is not considered a contraindication to postexpsoure prophylaxis. If there is substantial risk of exposure to rabies, preexposure prophylaxis may also be indicated during pregnancy.

Allergies

Persons with histories of hypersensitivity should be given rabies vaccines with caution. When a patient with a history suggesting hypersensitivity to HDCV must be given that vaccine, antihistamines can be given; epinephrine should be readily available to counteract anaphylactic reactions, and the person should be carefully observed.

SELECTED BIBLIOGRAPHY

Anderson LJ, Nicholson KG, Tauxe RV, Winkler WG. Human rabies in the United States, 1960 to 1979: epidemiology, diagnosis, and prevention. Ann Intern Med 1984;728-35.

Anderson LJ, Sikes RK, Langkop CW, et al. Postexposure trial of a human diploid cell strain rabies vaccine. J Infect Dis 1980;142:133-8.

Baer, GM, ed. The natural history of rabies. New York: Academic Press, 1975.

(Continued on page 407)

	:	28th Week End	ing	Cumulat	ve, 18th Week	Ending
Disease	July 14, 1984	July 16, 1983	Median 1979-1983	July 14, 1984	July 16, 1983	Median 1979-198
Acquired Immunodeficiency Syndrome (AIDS)	79	N	N	2,130	N	N
Aseptic meningitis	114	248	193	2,345	2,863	2,515
Encephalitis: Primary (arthropod-borne						
& unspec.)	16	37	37	445	526	485
Post-infectious	5	1	1	60	54	54
Gonorrhea: Civilian	14,381	17,832	19,276	425,249	470,694	509,293
Military	544	388	375	10,979	12,762	14,562
Hepatitis: Type A	265	303	498	10,976	11,247	13,582
Туре В	401	430	399	13,141	12,449	10,670
Non A, Non B	55	69	N	1,950	1,821	N
Unspecified	69	99	163	3,118	3,799	5,351
Legionellosis	12	8	N	300	362	N
Leprosy	3	8	6	121	139	107
Malaria	25	34	34	438	394	539
Measles: Total" `	12	2	33	1,892	1,111	2,323
Indigenous	11	2	N	1,713	936	N
Imported	1	-	N	179	175	N
Meningococcal infections: Total	49	28	42	1,717	1,767	1,767
Civilian	49	28	42	1,713	1,751	1,751
Military	-	-	-	4	16	12
Mumps	15	23	44	1,959	2,136	3,964
Pertussis	28	74	27	1,042	1,058	628
Rubella (German measles)	11	12	31	449	697	1,784
Syphilis (Primary & Secondary): Civilian	400	520	495	14,703	17,148	15,929
Military	3	5	5	178	228	196
Toxic Shock syndrome	5	6	Ň	227	254	N
Tuberculosis	412	513	513	11.314	12,196	14,119
Tularemia	11	10	9	103	130	108
Typhoid fever	3	15	13	160	191	219
Typhus fever, tick-borne (RMSF)	26	62	54	347	470	479
Rabies, animal	56	107	107	2.669	3,509	3,509

TABLE I. Summary-cases specified notifiable diseases, United States

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1984		Cum. 1984
Anthrax Botulism: Foodborne Infant (Mont. 1, Calif. 1)	1 6 49	Plague Poliomyelitis: Total Paralytic	11 2 2
Other (Calif. 1) Brucellosis (Mont. 1) Cholera Congenital rubella syndrome	4 51 - 3	Psittacosis (Calif. 3) Rabies, human Tetanus - (Mo. 1) Trichinosis (Mass. 1, Ohio 1, Alaska 2)	46 25 44
Diphtheria Leptospirosis (Ohio 1)	10	Typhus fever, flea-borne (endemic, murine) (Calif. 1)	

*One of the 12 reported cases for this week was imported from a foreign country or can be directly traceable to a known internationally imported case within two generations.

			July	/ 14, 19	84 and Jul	y 16, 1983	(28th \	Neek)				
		Aseptic	Encer	halitis	Gong	orrhea	н	epatitis (V	'iral), by ty	Lagional		
Reporting Area	AIDS	Menin- gitis	Primary	Post-in- fectious		ilian)	A	В	NA,NB	Unspeci- fied	Legionel- losis	Leprosy
	Cum. 1984	1984	Cum. 1984	Cum. 1984	Cum. 1984	Cum. 1983	1984	1984	1984	1984	1984	Cum. 1984
UNITED STATES	2,130	114	445	60	425,249	470,694	265	401	55	69	12	121
NEW ENGLAND Maine	71	8	29	1	12,030	11,677	5	16	2	14	1	5
N.H.	1	3	4	-	492 336	600 360		1 5	1	-	-	-
Vt. Mass.	37	1	2 15	-	200 4,598	223 5,050	4	6	ī	12	i	4
R.I.	4	-	-	-	832	638	-	-	-	-	-	1
Conn.	29	2	8	1	5,572	4,806	1	4	-	2	-	-
MID ATLANTIC Upstate N.Y.	963 83	9 4	55 20	6 5	58,665 8,880	59,886 9,327	21 5	50 7	4	2	-	24 2
N.Y. City	695	3	3	-	24,627	24,493	9	29	-	2	-	22
N.J. Pa.	138 47	2	15 17	1	9,947 15,211	11,149 14,917	7	14	4		2	-
E.N. CENTRAL		-										
Ohio	104 14	12 4	96 34	15 7	56,677 14,788	67,448 18,061	14 5	30 8	4	4	2 1	6 2
Ind. III.	16 54	4	20 14	-	6,734	7,016	4	6	1	3	1	-
Mich.	14	4	23	6	12,185 16,302	18,941 17,708	3	9	1 2	1	-	2 2
Wis.	6	-	5	2	6,668	5,722	-	-	-	-	-	-
W.N. CENTRAL	21	5	16	-	20,380	21,995	6	17	2	-	-	1
Minn. Iowa	5 1	-	6 7	-	3,018 2,288	3,099 2,415		2 3	-	-	:	1
Mo.	10	4	i	-	9,829	10,755	4	7	2	-	-	-
N. Dak. S. Dak.		-	-	-	194 520	228 611	1	2		-	-	
Nebr. Kans.	2 3	1	1 1	-	1,321 3,210	1,361 3,526	1	3	2	2	-	:
S. ATLANTIC	301	33	80	14	108,574	120,812	20	114	8	6	5	5
Del. Md.	4 19	- 3	1 19	-	1,959 12,094	2,159 15,507	-	1 16	1	-	2	-
D.C.	42	-	-	-	7,891	8,182	-	6	-	-	-	1
Va. W. Va.	17	5 2	19 5	5	10,297 1,297	10,400 1,284	1	9	-	-	2	3
N.C.	6	6	16	7	17,114	17,689	:	19	1	2	2	-
S.C. Ga.	6 28	3	2 2	1	10,689 20,864	11,479 25,255	1 4	16 18	1	1	1	-
Fla	175	14	16	1	26,369	28,857	14	27	5	3	•	1
E.S. CENTRAL Ky.	14 7	15	22 3	6	36,885 4,474	39,680 4,598	9 5	45 2	6	7	-	-
Tenn.	3	1	6	1	15,326	16,255	2	10	2	1	-	-
Ala. Miss.	3 1	12 2	12 1	5	11,797 5,288	12,309 6,518	2	31 2	4	4	-	:
W.S. CENTRAL	115	2	32	4	57,931	66,598	32	19	3	19	1	7
Ark. La.	18	1	4	2	4,919 13,230	5,059 11,928	1 20	14	2	3 12	-	-
Okla. Tex.	4 93	1	10 18	1	6,343 33,439	7,827 41,784	6 5	5	1	1 3	1	ī
MOUNTAIN	32	7	17	7	13,752	14,594	28	29	3	7	2	, 7
Mont.	-	-	-	-	578	635	-	-	-	-	1	-
ldaho Wyo	1	-	-	-	663 402	662 384	-	4	1	2	-	-
Colo.	19	5	7	-	3,979	4,110	6 1	6	1	-	-	-
N. Mex. Ariz.	6	2	4	3	1,548 3,753	1,771 4,073	13	2 10	1	3	-	- 5
Utah Nev.	3 3	-	6	4	667 2,162	721 2,238	4	7	-	4	1	1 1
		-	-	-					-		-	
PACIFIC Wash	509 25	23 5	98 3	7	60,355 4,131	68,004 5,197	130	81 4	23 6	10	1	66 3
Oreg Calif.	3 476	15	93	-7	3,589 50,126	3,533 56,176	23 102	4 72	2	10	1	1 47
Alaska	-	1	-	<i>'</i> -	1,501	1,685	-	- 12	15	- 10	-	47
Hawaii	5	2	2	-	1,008	1,413	1	1	-	-	-	15
Guam P.R.	33	U 2	-	ī	95 1,835	94 1,524	U 13	U 13	U	U 12	U	- 1
V.I.	-	-	-	-	228	154	-	-	-	-	-	-
Pac. Trust Terr.	-	U	-	-	-	-	U	U	U	U	U	-

TABLE III. Cases of specified notifiable diseases, United States, weeks ending July 14, 1984 and July 16, 1983 (28th Week)

N: Not notifiable

			J	uiy 14	, 1984	and .	July 16, '	983	(28th \	Week)				
Reporting Area	Malaria	Indig	Meas	ies (Rub		Total	Menin- gococcal Infections	Mui	mps		Pertussis			Rubella	
	Cum. 1984	1984	Cum. 1984	1984	Cum. 1984	Cum. 1983	Cum. 1984	1984	Cum. 1984	1984	Cum. 1984	Cum. 1983	1984	Cum. 1984	Cum. 1983
UNITED STATES	438	11	1,713	1	179	1,111	1,717	15	1,959	28	1,042	1,058	11	449	697
NEW ENGLAND Maine	28	1	98	-	9	15	102	-	60 16	2	20	38 4	-	28	11
N.H. Vt.	-	:	33	-	3	3	6	-	13	-	4	6	-	1	3
Mass.	2 15	1	3 52	-	3	4	23 36	-	3	2	14	.7	-	-	3
R.I. Conn.	4	-	10	-	3		9 27	-	14 5 9	-	1 1	17 4	-	27	5
MID ATLANTIC	70	4	94	1	22	77	285	1	228	3	92	244	4	145	123
Upstate N.Y. N.Y. City	19 16	1	18	-	7	6	102	1	52	š	55	78	-	97	20
N.J.	21	3	72 4	1†	9 2	41 27	49	-	12	-	3	36	4	36	86
Pa.	14	-	-	-	4	3	57 77	-	126 38	:	5 29	15 115	-	11 1	3 14
E.N. CENTRAL Ohio	33 7	2	565 2	-	67 5	613 78	270 94	3	814	19	286	258	-	67	109
ind.		-	2	-	1	393	36	3	417 40	2 16	51 195	75 20	-	2 2	22
III. Mich.	10	:	159	-	1	136	53	-	155		15	110	-	38	45
Wis.	6 10	2	392 10	:	54 6	5 1	52 35	:	152 50	1	13 12	12 41	2	18 7	15 26
W.N. CENTRAL Minn.	12	-	2	-	3	1	110	1	80	1	80	63	1	28	30
lowa	2	:	-	-	3	1	21	-	3	-	9	22	-	2	6
Mo.	6	-	2	-		-	18 32	-	17	1	4 12	5 11	1	1	-
N. Dak. S. Dak.	1	-	-	-	-	-	1		í	-	12	1	-	3	-
Nebr.	1	:	:	-	-	-	7	-	-	-	5	3	-	-	-
Kans.	i	-	-	-	-	-	9 22	ī	3 49	-	2 48	21	-	22	24
S. ATLANTIC Del.	78 4	:	10	-	17	177	359	2	137	1	75	152	-	20	84
Md.	19	-	4	:	- 5	- 5	3 29	-	2 27	-	2 4	2 25	-	1	1
D.C. Va	1	-	-	-	5	-	25	-		-	-	25	-	-	-
W. Va.	19 1	-	1	-	1	22	41	1	13	-	9	42	-	-	1
N.C.	5	-	-	:	:	-	5 51	1	27 15	-	7 17	5 17	-		9
S.C. Ga.	1	-	-	-	-	4	34	-	2	-	'í	ii	-	-	.1
Fla.	6 22	-	5	-	- 6	8 138	71 120	ī	17 34	1	5 30	31 19	:	2 17	11 61
E.S. CENTRAL Ky.	3	-	1	-	2	6	100	-	37	-	6	11	-	7	10
Tenn.	-	:	1	-	2	1	38	-	8	-	1	3 3	-	3	9
Ala.	3	-	-	-		- 5	24 26	-	12 5	-	2	3	-	1	1
Miss.	-	-	-	-	•	-	12	-	12	-	3	2	-	3	-
W.S. CENTRAL Ark.	34	-	362	-	22	70 10	184 27	-	105	-	231	143 13	-	13 3	88
La.	5	-	-	-	-	25	35		5	-	11	2	-	-	9
Okla. Tex.	5 24	-	362	:	7 15	1 34	23 99	N	N 100	:	206 11	104 24	-	10	79
MOUNTAIN	16	-	91	· _	10	3	58	2	193	1	74	104	1	13	27
Vont. daho	1	-	-	-	-	-	1	-	4	-	17	1	-	ī	3 8
Wyo.	2	-	-		-	-	6 2	-	8	-	3	3	2	ż	ž
Colo.	1	-	-	-	-	2		-	13	1	26	70	-	2	-
N. Mex. Ariz.	1	:	68	-	8	ī	.7	N	N	-	5	8 9	-	1	6
Jtah Nev.	3	-	23	-	2		14	2	161 5	-	13	9	1	6 1	7
ACIFIC	- 164	4	- 490	-	- 27	- 149	3 249	-	1	-	2	- 45	5	128	215
Nash.	5	-	107	-		4	249	6	305 32	1	178 33	45	-	1	8
Dreg. Calif.	8	•	-	-		7	37	N	N	-	11	6	2	123	13 194
Alaska	148	-	244	-	24	137	168 7	5 1	254 5	:	65 -	31	5	1	-
ławaii	3	4	139	-	3	1	1	-	14	-	69	-	-	3	-
Suam P.R.	1 3	U -	83	U -	2	2 81	1 3	U 1	5 92	U -	:	8	U -	2 6	3
/.l. Pac. Trust Terr.	-	Ū	:	Ū	-	5	-	Ū	3	Ū	-	-	Ū	-	1

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending July 14, 1984 and July 16, 1983 (28th Week)

*For measles only, imported cases includes both out-of-state and international importations.

		July	14, 1984 a	and July	16, 1983	(28th We	ek)		
Reporting Area	Syphilis (Primary &	(Civilian) Secondary)	Toxic- shock Syndrome	Tuber	culosis	Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1984	Cum. 1983	1984	Cum. 1984	Cum. 1983	Cum. 1984	Cum. 1984	Cum. 1984	Cum. 1984
UNITED STATES	14,703	17,148	5	11,314	12,196	103	160	347±	2,669
NEW ENGLAND	294	383	-	316	352	2	7	1	21
Maine N.H.	3 7	10 16	-	17 22	20 25	-	-	- /	10 4
Vt.	1	1	-	7	4	:	-	-	-
Mass. R.I.	172 11	236 13	-	165 25	179 28	2	5	1 4	- 5
Conn.	100	107	-	80	96	-	2	-	2
MID ATLANTIC	2,007	2,171		2,076	2,199	-	23	4	171
Upstate N.Y.	134	175	-	356	342	-	9	3	, 16
N.Y. City N.J.	1,255 369	1,280 419	-	826 459	892 462	-	6 4	1	4
Pa	249	297	-	435	503		4	-	151
E.N. CENTRAL	636	942	1	1,484	1,571	1	22	16	117
Ohio	131	247	i	285	251	-	4	12	11
Ind. III	74	73	-	168	146	-	2	2	13
Mich.	177 210	465 113	-	620 317	689 404	1	8 2	2	48 13
Wis.	44	44	-	94	81	-	6	-	32
W.N. CENTRAL	223	210	_	322	396	28	6	26	446
Minn.	67	88		58	80	-	2	-	45
lowa Mo.	10	9	-	34	37		3	1 ·	88 37
N. Dak.	109	. 74	-	156 8	207 5	18		- 4	87
S. Dak.	2	 9		11	28	10	-	3	116
Nebr. Kans	11	11	-	16	11	-	1	2 16	31 42
	19	18	-	39	28	-		10	
S. ATLANTIC Del.	4,404	4,490	1	2,362	2,436	4	19	160	768 4
Md.	16 266	19 286	-	31 263	20 189	-	-	13	438
D.C.	174	191	-	· 88	94	-	6	-	
Va. W. Va.	227 10	321		234 76	242 81	-	4	26 5	133 23
N.C	437	15 417	1	349	334	1	1	57	10
S.C.	406	278	-	282	231	-	1	42 16	26 86
Ga. Fla.	751 2,117	835 2,128	-	322 717	453 792	3	6	1	48
							5	34	136
E.S. CENTRAL Ky	978 57	1,169 67	-	1,041 238	1,121 272	2	2	5	_ 35
Tenn	276	326	-	339	332	2	. 2	18	56
Ala. Miss.	313	483	-	314	294 223	-	1	6 5	45
	332	293	-	150	223	-			
W.S. CENTRAL Ark.	3,535	4,503	-	1,290	1,451	46 30	9	99 18	569 61
La.	89 649	108 951	-	137 165	160 254	30	1	1	23
Okla.	121	121	-	127	126	13	2	61	68
Tex.	2,676	3,323	-	861	911	-	6	19	417
MOUNTAIN	336	375	-	289	340	15	10	5 -	118
Mont. Idaho	2	5	-	14	34	4	1	5	62
Wyo.	14	6 7	-	18	18 8	4	-	-	-
Colo.	78	81	-	25	33	5	2	-	21
N. Mex. Ariz.	44 131	115	-	56 133	72 136	1 2	3 3	-	9 21
Utah	11	90 13	-	27	23	2	-	-	-
Nev.	52	58	-	16	16	1	1	-	5
PACIFIC	2,290	2,905	3	2,134	2,330	5	59	2	323
Wash.	72	105	-	107	112	-	1	1	1
Oreg. Calif.	70 2,104	60 2,696	1 2	88 1,789	100 1,945	2 3	53	-	315
Alaska	3	7	-	33	33	-	1	1	6
Hawaii	41	37	-	117	140	-	3	-	-
Guam	-	-	U	5	4	-	-	-	
P.R. V.I.	459	598	-	217	263	-	3 3	-	34
Pac. Trust Terr.	8	10	U U	2	1	-	-	-	-
	-	-	•						

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending July 14, 1984 and July 16, 1983 (28th Week)

U: Unavailable

TABLE IV. Deaths in 121 U.S. cities,* week ending July 14, 1984 (28th Week Ending)

		All Caus	es. By A	ge (Year					All Causes, By Age (Years)						
Reporting Area	All Ages	≥65	45-64	İ –		<1	P&I** Total	Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	P&I** Total
NEW ENGLAND	677	484	125	37	14	17	41	S. ATLANTIC	1,298	773	303	128	56	38	32
Boston, Mass.	176	121	37	10	5	3	16	Atlanta, Ga.	142 249	88 154	27 61	17 17	6 11	4 6	5
Bridgeport, Conn. Cambridge, Mass.	38 29	22 25	12 3	3 1	-	1	2	Baltimore, Md. Charlotte, N.C.	249	53	18	'7	2	4	1
Fall River, Mass.	37	30	5	i	1	-	-	Jacksonville, Fla.	79	50	17	4	4	4	3
Hartford, Conn.	73	45	16	7	3	2	1	Miami, Fla.	173	106	44	13	8 4	2	1
Lowell, Mass. Lynn, Mass.	33	24 13	5 2	3	1	-	4	Norfolk, Va. Richmond, Va.	47 85	26 44	13 31	3 6	2	2	
New Bedford, Mas	15 s. 32	26	4	2	-	2	1	Savannah, Ga	33	18	7	5	3	-	3
New Haven, Conn.	58	38	10	5	1	4	2	St. Petersburg, Fla.		94	16	3	1	1	7
Providence, R.I.	57	45	7	1	-	4	7	Tampa, Fla.	67 181	40 73	18 40	6 45	1 12	2 11	6 3
Somerville, Mass. Springfield, Mass.	4 36	4 26	6	1	1	2	1	Washington, D.C. Wilmington, Del.	43	27	11	2	2	1	3
Waterbury, Conn.	33	23	7	i	2	-	2	-							36
Worcester, Mass.	56	42	11	2	Ξ.	1	4	E.S. CENTRAL	708	444	181	39 8	18 1	25 5	, 2
MID. ATLANTIC								Birmingham, Ala. Chattanooga, Tenn	107 60	67 34	26 22	2	1	1	11
Albany, N.Y.	2,569 52	1,664 34	566 9	212 3	72 2	55 4	95	Knoxville, Tenn.	82	48	23	5	3	3	7
Allentown, Pa.	33	18	14	-	1	1	-	Louisville, Ky	87	53	23	4	2	5	3
Buffalo, N.Y.	116	71	34	6	3	2	10	Memphis, Tenn	141	94 49	27 16	10 3	6 2	3 3	5 5
Camden, N.J. Elizabeth, N.J.	40 26	22	11	5	-	2	-	Mobile, Ala. Montgomery, Ala.	73 51	49	13	3	-	-	2
Erie, Pa.†	31	19 19	47	3 2	2	1	-	Nashville, Tenn.	107	64	31	4	3	5	11
Jersey City, N.J.	46	32	8	3	ĩ	2	-					407	76	67	38
	1,365	860	294			24	47	W.S. CENTRAL	1,365 44	755 28	330 6	137 3	/0 5	2	4
Newark, N.J. Paterson, N.J.	58 37	33 21	13 7	5 4	4	3	4 2	Austin, Tex. Baton Rouge, La	44	28	7	4	4	2	-
Philadelphia, Pa.†	294	191	66	24	2 5	3 8	12	Corpus Christi, Tex	36	18	11	4	3	-	-
Pittsburgh, Pa.†	49	30	17	-	ĭ	ĭ	2	Dallas, Tex.	209	110	59	25 4	8 6	7 5	4
Reading, Pa.	30	20	8	-	2	-	-	El Paso, Tex	69 100	37 64	17 21	47	4	4	3
Rochester, N.Y. Schenectady, N.Y.	128 32	99 26	20	5	2	2	6 1	Fort Worth, Tex. Houston, Tex.	427	208	118	60	20	21	5
Scranton, Pa.†	28	20	6 7	1	-	:		Little Rock, Ark.	59	30	17	3	4	5	4
Syracuse, N.Y.	118	83	25	4	3	3	6	New Orleans, La.	92	50	18 35	14 9	7 8	3 13	13
Trenton, N.J.	27	16	7	3	1	-	3	San Antonio, Tex.	177 37	112 22	35	2	2	2	
Utica, N.Y. Yonkers, N.Y.	24 35	20 30	4 5	-	-	-	1	Shreveport, La. Tulsa, Okla.	70	48	12		5	3	1
		1,396	531	161	77	82	72	MOUNTAIN	651	391	147	57	35 2	21 3	32 6
Akron, Ohio	57	31	15	8	3	-	-	Albuquerque, N Me		49 26	12 7	12 2	3	1	5
Canton, Ohio Chicago, III	40 547	30 305	6 153	2 42	1 19	1 28	10	Colo. Springs, Colo Denver, Colo.	132	72	34	11	11	4	6
Cincinnati, Ohio	115	80	19	9	2	5	3	Las Vegas, Nev	72	39	19	7	6	1	3
Cleveland, Ohio	162	100	42	10	4	6	2	Ogden, Utah	32	20	5	4	1	2	1
Columbus, Ohio	129	73	35	11	4	6	6	Phoenix, Ariz. Pueblo, Colo.	147 25	86 17	43	7	2		2
Dayton, Ohio Detroit, Mich.	105 252	60 149	27 64	4 20	9 11	5 8	7 6	Salt Lake City, Utal		19	9			1	-
Evansville, Ind.	47	37	5	20	2	-	1	Tucson, Ariz	95	63	14	10	6	2	7
Fort Wayne, Ind.	60	33	13	5	3	6	4				400	147	61	53	90
Gary, Ind.	15	8	3	3	1	-	1	PACIFIC Barkelow Calif	2,045 29	1,340 23	439	147		55	- 50
Grand Rapids, Micl Indianapolis, Ind.	h 45 183	35 107	10 46	17	7	- 6	1 2	Berkeley, Calif. Fresno, Calif.	75	47	11	3	6	8	3
Madison, Wis.	40	28	7	3	í	ĭ	6	Glendale, Calif.	28	21	4	2	1	-	2
Milwaukee, Wis.	131	91	21	11	2	6	3	Honolulu, Hawaii	83	49	23		1	2	5 3
Peoria, III.	50	35	11	2	2	-	7	Long Beach, Calif.	80 565	55 331	23 145		17	12	11
Rockford, III.	36 58	28 41	3 15	3 2	2	-	1	Los Angeles, Calif. Oakland, Calif.	71	45	19		2	1	10
South Bend, Ind. Toledo, Ohio	108	75	22	5	3	3	5	Pasadena, Calif.	42	32	7	3	-	-	2
Youngstown, Ohio	67	50	14	1	1	1	2	Portland, Oreg.	121	80	32		1	4 6	6 16
					~ .	~~		Sacramento, Calif.	151 150	101 116	28 21	11	5 3	3	11
W.N. CENTRAL	808 91	545 66	165 13	45 7	21	32 5	28 7	San Diego, Calif. San Francisco, Cali		104	25		2	2	3
Des Moines, Iowa Duluth, Minn.	39	29	9	í		-	í	San Jose, Calif.	205	139	36	16	9	5	10
Kansas City, Kans.	33	19	8	3	2	1	- 1	Seattle, Wash	146	101	26		5	6	2
Kansas City, Mo.	103	59	29	9	1	5	2	Spokane, Wash. Tacoma, Wash.	59 88	37 59	14 19		4 5	3	4
Lincoln, Nebr.	35 78	28 55	5 15	1 3	1 2	3	- 4	racoma, wasn.			19	+	5	'	
Minneapolis, Minn. Omaha, Nebr.	78 96	64	17	7	3	5	5	TOTAL	12,368	7,792	2,787	963	430	390	464
St. Louis, Mo.	181	115	34	13	9	10	-								
St. Paul, Minn.	67	54	10	1	1	1	3								
Wichita, Kans.	85	56	25	-	2	2	6								

* Mortality data in this table are voluntarily reported from 121 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

The cause of changes in reporting methods in these 4 Pennsylvania cities, these numbers are partial counts for the current week. Com-plete counts will be available in 4 to 6 weeks.
Total includes unknown ages.

Cause of	Years of potential life lost before		ated mortality ruary 1984	Estimated number
morbidity or mortality (Ninth Revision ICD, 1975)	age 65 by persons dying in 1982* [†]	Number* [§]	Annual Rate/100,000*§	of physician contacts February 1984 ^{•¶}
ALL CAUSES (TOTAL)	9,429,000	165,960	920.6	104,800,000
Accidents and adverse effects (E800-E949)	2,367,000	6,270	34.8	5,400,000
Malignant neoplasms (140-208)	1,809,000	35,390	196.3	1,500,000
Diseases of heart (390-398, 402, 404-429)	1,566,000	62,680	347.7	5,200,000
Suicides, homicides (E950-E978)	1,314,000	3,520	19.5	-
Cerebrovascular diseases (430-438)	256,000	13,340	74.0	600,000
Chronic liver disease and cirrhosis (571)	252,000	2,340	13.0	100,000
Pneumonia and influenza (480-487)	118,000	5,700	31.6	3,300,000
Chronic obstructive pulmonary diseases and allied conditions (490-496)	114.000	5,770	32.0	2,400,000
Diabetes mellitus (250)	106,000	2,850	15.8	2,200,000
Prenatal care*				2,900,000
Infant mortality*††		3,700	12.9 /1,000	live births

TABLE V. Years of potential life lost, deaths, and death rates, by cause of death, and estimated number of physician contacts, by principal diagnosis, United States

*For details of calculation, see footnotes for Table V, MMWR 1984;33:2.

[†]Years of potential life lost for persons between 1 year and 65 years old at the time of death are derived from the number of deaths in each age category as reported by the National Center for Health Statistics, *Monthly Vital Statistics Report* (MVSR), Vol. 31, No. 13, October 5, 1983.

⁹National Center for Health Statistics, *Monthly Vital Statistics Report* (MVSR), Vol. 33, No. 3, June 21, 1984, pp. 8-9.

[¶]IMS America National Disease and Therapeutic Index (NDTI), Monthly Report, February 1984, Section III.

⁺⁺MVSR Vol. 33, No. 2, May 23, 1984, p. 1.

ACIP: Rabies - Continued

Bahmanyar M, Fayaz A, Nour-Salehi S, Mohammadi M, Koprowski H. Successful protection of humans exposed to rabies infection: postexposure treatment with the new human diploid cell rabies vaccine and antirabies serum. JAMA 1976;236:2751-4.

Bernard KW, Smith PW, Kader FJ, Moran MJ. Neuroparalytic illness and human diploid cell rabies vaccine. JAMA 1982;248:3136-8.

Boe E, Nyland H. Guillain-Barré syndrome after vaccination with human diploid cell rabies vaccine. Scand J Infect Dis 1980;12:231-2.

CDC. Systemic allergic reactions following immunization with human diploid cell rabies vaccine. MMWR 1984;33:185-7.

Corey L, Hattwick MAW. Treatment of persons exposed to rabies. JAMA 1975;232:272-6.

Greenberg M, Childress J. Vaccination against rabies with duck-embryo and Semple vaccines. JAMA 1960;173:333-7.

Helmick CG: The epidemiology of human rabies postexposure prophylaxis, 1980-1981. JAMA 1983;250:1990-6.

ACIP: Rabies - Continued

Hattwick MAW. Human rabies. Public Health Reviews 1974;3:229-74.

- Hattwick MAW, Rubin RH, Music S, Sikes RK, Smith JS, Gregg MB. Postexposure rabies prophylaxis with human rabies immune globulin. JAMA 1974;227:407-10.
- Peck FM Jr, Powell HM, Culbertson CG. A new antirabies vaccine for human use. J Lab Clin Med 1955;45:679-83.
- Rubin RH, Hattwick MAW, Jones S, Gregg MB, Schwartz VD. Adverse reactions to duck embryo rabies vaccine. Range and incidence. Ann Intern Med 1973;78:643-9.
- Tierkel ES, Sikes RK. Preexposure prophylaxis against rabies. Comparison of regimens. JAMA 1967;201:911-4.
- Tint H, Rosanoff El. Clinical responses to T(n)BP-disrupted HDCS (WI-38) rabies vaccine. Dev Biol Stand 1976;37:287-9.
- Wiktor TJ, Plotkin SA, Koprowski H. Development and clinical trials of the new human rabies vaccine of tissue culture (human diploid cell) origin. Dev Biol Stand 1978;40:3-9.
- World Health Organization. Sixth report of the Expert Committee on Rabies. Geneva, Switzerland: World Health Organization, 1973 (WHO technical report no. 523).
- Sinnecker H, Atanasiu P, Bahmanyar M, Selimov M, Wandeler AI, Bogel K (Working Group 2, World Health Organization). Vaccine potency requirements for reduced immunization schedules and preexposure treatment. Dev Biol Stand 1978;40:268-70.

Epidemiologic Notes and Reports

Chromosomally Mediated Resistant *Neisseria gonorrhoeae* — United States

During 1983-1984, an increasing number of cases of β -lactamase negative, penicillinresistant *Neisseria gonorrhoeae* were reported to CDC. Unlike penicillinase-producing *N. gonorrhoeae* (PPNG), which have plasmid-mediated resistance to penicillin, these β -lactamase negative, resistant gonococci have chromosomally mediated resistance based on available data.

The first reported outbreak of chromosomally mediated (β -lactamase negative) resistant *N. gonorrhoeae* (CMRNG) in the United States occurred in Durham County, North Carolina (1). Since this outbreak, in which more than 200 cases were eventually detected, 16 other states have reported cases with resistant gonococci. Of these, Tennessee, New Mexico, and Oregon have reported more sustained outbreaks.

Cases in these outbreaks were detected either by routine screening of all gonococcal isolates (New Mexico) or screening of primary treatment failure isolates (Tennessee, Oregon) for susceptibility to penicillin at the local or state levels. Screening was performed by disk agar diffusion or by growth on penicillin-containing media. Gonococcal isolates that grew on media containing 1.6 μ g/ml of penicillin or produced a zone of inhibition less than 26 mm, with a 10 μ g penicillin disk, were submitted to CDC for confirmation of resistance. Minimum inhibitory concentrations by the agar dilution susceptibility test were determined for antimicrobials that included penicillin, ampicillin, tetracycline, cefotaxime, cefuroxime, cefoxitin, spectinomycin, and trimethoprim/sulfamethoxazole. Isolates resistant to penicillin and ampicillin were equally resistant to tetracycline by agar dilution susceptibility testing.

Of all CMRNG isolates submitted to CDC for agar dilution susceptibility testing during 1983-1984, 11.0% were susceptible to less than 2 μ g/ml of penicillin; none were susceptible to less than 2 μ g/ml of tetracycline; and only 47.0% were susceptible to less than 0.5 μ g/ml trimethoprim and 9.5 μ g/ml sulfamethoxazole (trimethoprim/sulfamethoxazole). All isolates were susceptible to spectinomycin, cefoxitin, cefuroxime, and cefotaxime. Immunologic characterization demonstrated that all CMRNG isolates were serogroup IIb (the majority of the same serovariant) based on serotyping by experimental monoclonal antibodies to major outer membrane protein (2). Of the 18 New Mexico cases, two distinctly different serovariants were detected within serogroup IIb.

Neisseria gonorrhoeae - Continued

Vol. 33/No. 28

Clinical and epidemiologic information were obtained for patients whose isolates were tested. Excluding North Carolina, of the 16 other reporting states, over half of the CMRNG cases were from Tennessee, New Mexico, and Oregon.

Tennessee: All the 14 Tennessee patients were heterosexuals, and two patients could be linked to interstate travel to Virginia or North Carolina. Strains from the Tennessee cases were immunologically similar and had similar antimicrobial susceptibility patterns consistent with continued endemic transmission within the state.

New Mexico: Of the 18 CMRNG patients from New Mexico, seven were heterosexual (three males, four females), and 11 were homosexual males. All heterosexual patients and seven homosexual patients were infected with gonococcal strains immunologically identical, with similar antimicrobial susceptibility patterns. Strains from these cases were more resistant to penicillin than strains from the other four homosexual patients. Heterosexual CMRNG patients could not be linked to homosexual CMRNG patients by sexual history or naming of sexual contacts. All homosexual patients were clustered within Albuquerque; heterosexual patients were more widely distributed throughout the state. Based on immunologic studies of the gonoccoci recovered from these individuals and examination of temporal and geographic variables for heterosexuals versus homosexuals, at least two separate outbreaks with no demonstrable common source occurred in New Mexico. No evidence for interstate or foreign transmission into New Mexico could be identified for any of the cases.

Oregon: Of the eight cases reported from Oregon, all occurred among homosexual males. Gonococcal strains from these individuals shared identical immunologic and antimicrobial susceptibility patterns. No epidemiologic evidence for interstate or foreign transmission could be documented for any of these cases, suggesting only endemic transmission within the homosexual community in Oregon. No additional cases have been reported from Oregon since March 1984.

Reported by M Kimberly, DrPh, State Laboratory Director, W DeVault, CE Chapman, MD, G Conrad, Venereal Disease Control, RH Hutcheson, Jr, MD, State Epidemiologist, Tennessee State Dept of Health; JM Mann, MD, L Nims, Scientific Laboratory, A Chowning, E Montes, Venereal Disease Control, HF Hull, MD, State Epidemiologist, Health Svcs Div, New Mexico Dept of Health and Environment, L Foster, MD, D Harger, H Horton, Venereal Disease Control, C Schade, MD, JA Googins, MD, State Epidemiologist, State Health Div, Oregon Dept of Human Resources; Sexually Transmitted Diseases Laboratory Program, Center for Infectious Diseases, Div of Sexually Transmitted Diseases, Center for Prevention Svcs, Div of Field Svcs, Epidemiology Program Office, CDC.

Editorial Note: Seventeen states, including North Carolina, have reported cases of CMRNG to CDC since 1983. The majority of these cases were detected as primary therapeutic failures to the penicillins or tetracyclines. Gonococcal strains from the majority of U.S. outbreaks and cases have generally been immunologically similar (serogroup IIb) with similar antimicrobial susceptibilities.

Based on epidemiologic data, foreign importation has been infrequently documented for these CMRNG strains in the United States (3). In contrast, foreign importation contributes to the largest proportion of PPNG in the United States, although domestic transmission became more important after 1976 (4).

Cases of CMRNG may be detected by screening for penicillin resistance at the local or state levels to guide appropriate therapy and permit rapid follow-up of cases. Screening by disk agar diffusion or with penicillin-containing media will identify chromosomally mediated resistance to penicillin. Disk susceptibility testing to tetracycline and trimethoprim/sulfameth-oxazole should be performed only by standardized procedures using appropriate controls (5, 6). Inconsistent results to these two antimicrobials may be seen with disk susceptibility testing (5, 6).

Based on agar dilution susceptibility testing, infections caused by CMRNG should clinically respond to therapy with recommended dosages of spectinomycin, cefoxitin, cefotaxime, or ce-

Neisseria gonorrhoeae - Continued

furoxime. CDC treatment guidelines for PPNG infections provide the recommended schedules for these antimicrobials and emphasize the importance of the immediate use of spectinomycin as primary therapy for gonorrhea cases when treatment failures are suspected (7).

Since 1975, gonorrhea has generally declined in the United States (8). PPNG increased dramatically between 1976 and 1982 but decreased in 1983 (8). Unfortunately, cases of CMRNG have been reported with increasing frequency since the North Carolina outbreak. Because the extent and prevalence of CMRNG infections are not yet fully understood, screening of all β -lactamase negative (nonpenicillinase-producing) primary treatment failure gonococcal isolates for penicillin susceptibility (1) is encouraged at the local and state levels to improve surveillance and guide appropriate therapy. Screening at the community level should be most cost-effective, since the majority of these CMRNG strains are equally resistant to tetracycline, thereby preventing unnecessary and usually ineffective retreatment with a tetracycline. Because of high secondary treatment failure rates with tetracycline, theracycline should not be used as the drug of choice for either PPNG or CMRNG infections that have failed primary therapy with penicillin or ampicillin. Spectinomycin, cefoxitin, or cefotaxime should be used to treat CMRNG infections at dosages recommended for PPNG (7).

More active surveillance for these CMRNG infections will be required to determine their accurate prevalence, and support control activities.

References

- 1. CDC. Penicillin-resistant gonorrhea-North Carolina. MMWR 1983;32:273-5.
- Tam MR, Buchanan TM, Sandstrom EG, Holmes KK, et al. Serological classification of *Neisseria* gonorrhoeae with monoclonal antibodies. Infect and Immun 1982;36:1042-53.
- 3. CDC. Gonorrhea surveillance reports, 1975-1984.
- Jaffe HW, Biddle JW, Johnson SR, Wiesner PJ. Infections due to penicillinase producing Neisseria gonorrhoeae in the United States: 1976-1980. J Infect Dis 1981;144:191-7.
- Biddle JW, Swenson JM, Thornsberry C. Disc agar diffusion antimicrobial susceptibility tests with beta-lactamase producing *Neisseria gonorrhoeae*. J Antibiot 1978;31:352-8.
- 6. Barry AL. "Diffusion test procedures." The antimicrobic susceptibility test: principles and practices. Philadelphia, Pennsylvania: Lea & Sebiger 1976:180-207.
- 7. CDC. Sexually transmitted diseases treatment guidelines, 1982. MMWR Supplement, 1982;31: 35S-62S.
- 8. CDC. Gonorrhea-United States, 1983. MMWR 1984;33:361-3.

Fatalities from Occupational Heat Exposure

Presented below are two of several fatalities from occupational heat stroke reported to the National Institute for Occupational Safety and Health (NIOSH) since 1977.

Indiana: In July 1980, a 24-year-old white male, who was employed at a surface coal mine, collapsed and later died after performing heavy labor in a hot environment. The worker, 5 feet 9 inches tall and weighing about 200 pounds, had been employed at the mine for $1\frac{1}{2}$ weeks. On the day of the reported incident, he was assigned to load 40-pound bags of explosives into vertically drilled holes in preparation for blasting the material overlying the coal seam. He began work at 6:00 a.m., and at 3:40 p.m., informed a co-worker that he did not feel well. He walked about 50 yards to a shady area and collapsed. The outdoor dry bulb temperature was 39.4 C (103 F).

The worker was moved to a nearby hospital where his rectal temperature registered 42.2 C (108 F). By the time he was transferred to the intensive care unit (ICU), his temperature exceeded 43.3 C (110 F). He was treated with an ice pack and intravenous fluids but died at 6:30 p.m. The autopsy report listed systemic hyperthermia with extreme generalized dilation of capillaries (cardiovascular shock) and cerebral edema as the immediate causes of death.

Vol. 33/No. 28

MMWR

Occupational Heat Exposure - Continued

Wisconsin: In September 1981, a 39-year-old black male, 5 feet 7 inches tall and weighing 165 pounds, was employed as a furnace attendant at an aluminum foundry. He had worked at the foundry for 2 weeks and was responsible for turning on and attending a furnace used to melt aluminum. On the afternoon of the reported incident, he had pressed the wrong button and accidentally spilled molten aluminum on the floor. He spent about 15 minutes removing the spill and wore a silver reflective suit for protection against the radiant heat emanating from the metal. The outdoor dry bulb temperature was 28.3 C (83 F), and the worksite temperature was about 29.4 C (84 F); the estimated temperature of the molten aluminum in the furnace was 982.2 C (1,800 F).

After removing the spilled material, the worker described the accident to his supervisor and, still wearing the suit, left the workplace without explanation. He was discovered 15 minutes later having seizures in the foundry parking lot. Paramedics transported him to a hospital at 5:40 p.m.; on arrival, his body temperature was 41.7 C (107 F). Medication controlled the seizures, but he remained comatose. He was treated with rubbing alcohol and an ice pack, and at 7:00 p.m., when his body temperature was 35.6 C (96 F), he was placed on a hyperthermic machine in the ICU. He began bleeding from the rectum at 9:30 p.m., and fresh, frozen plasma was administered. The bleeding apparently stopped but then recurred with hematuria. He died the next day at 9:30 a.m. in cardiac arrest. The autopsy report listed the causes of death as hyperthermia, disseminated intravascular coagulation, and coronary arteriosclerosis.

The worker had a history of treatment for alcoholism and reportedly had been drinking heavily in the days before his death; however, at the time of hospitalization, he had no alcohol in his blood. Four days before the heatstroke, he had severely lacerated his toes in a lawn-mower accident and was treated with antibiotics and tetanus toxoid.

Reported by Div of Respiratory Disease Studies, Div of Biomedical and Behavioral Science, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Illness and death from environmental heat are important public health problems (1). This is especially true in the occupational setting when workers performing physical labor outdoors are exposed to higher-than-normal ambient temperatures and when such temperatures have an additive effect on heat generated by the jobs themselves. The fatalities reported here illustrate, in both outdoor and indoor settings, the circumstances that may lead to heatstroke and, subsequently, to death.

Occupational heat-related conditions include heat cramps, heat exhaustion, dehydration, and skin disorders. In addition, the risk of unintentional injuries increases substantially with exposure to heat stress (2). An estimated six million workers in the United States may be exposed to occupational heat stress. Estimates of deaths and illnesses associated with occupational heat exposures are difficult to obtain, because worksite conditions and occupation are usually not listed on hospital records or death certificates; moreover, heatstroke may not be recognized as the primary cause of illness or death. However, for 1973-1976, annual reports from the California Department of Health Services alone show seven fatalities among 1,128 acute occupational heat-related illnesses (3). About 10%-15% of these patients required hospitalization, and an additional 40% were absent from work for varying periods after their illnesses; the remainder returned to work after medical treatment.

The health status of a worker is important in determining the response to heat exposure (4). Certain preexisting conditions can render a person more susceptible to heatstroke; these include obesity, drug abuse, alcoholism, acute or chronic illnesses, fatigue, poor physical condition, overeating, use of anticholinergic and certain psychotropic drugs, lack of sleep, and lack of acclimatization (5). The first worker described here was moderately obese and in poor physical condition; the second had a history of treatment for alcoholism and may have been affected by the wound and the medication he received 4 days before his death.

Occupational Heat Exposure - Continued

In 1969, an international panel of scientists convened by the World Health Organization recommended keeping a worker's deep body temperature at or below 38 C (100.4 F) to prevent heat illnesses (6). In response to this, NIOSH developed in 1972 a Criteria Document for Occupational Exposure to Hot Environments, which recommended the following preventive measures (7): (1) acclimatizing new workers and workers returning from vacation or absence because of illness; (2) implementing a work/rest regimen matched to the severity of the workers' heat exposure. (The Threshold Limit Value for Heat Stress adopted by the American Conference of Governmental Industrial Hygienists can be used as a guide to establish a suitable work/rest regimen [8]); (3) scheduling hot operations for the coolest part of the day; (4) making drinking water and salt readily available to replace the water and salt lost by sweating; (5) making protective clothing available to workers, as appropriate; (6) reducing environmental heat by engineering controls; (7) monitoring environmental heat at the job site; (8) performing pre-employment and periodic medical examinations to define those at increased risk; and (9) instructing workers and supervisors about preventive measures and early recognition of the symptoms of heat-related disorders.

References

- 1. CDC. Illness and death due to environmental heat—Georgia and St. Louis, Missouri, 1983. MMWR 1984;33:325-6.
- Ramsey JD, Burford CL, Beshir MY, Hensen RC. Effects of workplace thermal conditions on safe work behavior. Journal of Safety Research 1983;14:105-14.
- 3. State of California, Department of Health, Occupational Health Branch. Occupational disease in California. Annual Reports 1973-1976.
- 4. Dukes-Dobos FN. Hazards of heat exposure. A review. Scand J Work Environ Health 1981;7:73-83.
- 5. Bartley JD. Heat stroke: is total prevention possible? Milit Med 1977;142:528,533-5.
- 6. World Health Organization. Health factors involved in working under conditions of heat stress. WHO Technical Report Series 1969; no. 412.
- National Institute for Occupational Safety and Health criteria for a recommended standard. Occupational exposure to hot environments. Cincinnati: National Institute for Occupational Safety and Health 1972 (Document #HSM 72-10269).
- 8. American Conference for Governmental Industrial Hygienists. TLV's Threshold Limit Values for chemical substances and physical agents in the work environment with intended changes for 1983-1984. Heat Stress 62-9.

Current Trends

Tuberculosis – United States, 1983

In 1983, 23,846 cases of tuberculosis were reported to CDC, for a rate of 10.2 cases per 100,000 population. Compared with 1982, this represents a 6.6% decrease in the number of cases reported and a decline of 7.3% in the rate.

Rates for the 50 states ranged from 23.1/100,000 in Hawaii to 1.3/100,000 in North Dakota (Table 3). The rate increased in 13 states, remained unchanged in one, and decreased in 36.

The rate among persons living in 56 cities with populations of 250,000 or more was 21.2/100,000—more than twice the national rate (Table 4). Urban rates ranged from 58.4/100,000 in Miami, Florida, to 2.5/100,000 in Toledo, Ohio. Eight cities had rates at least three times the national rate: Miami, Florida; Newark, New Jersey; Atlanta, Georgia; San Francisco, California; Tampa, Florida; Honolulu, Hawaii; Washington, D.C.; and Oakland, California.

TABLE 3. Tuberculosis cases and rates – United States, 1983 and 1982

State	Tubercul	osis cases	Cas	e rate		cording rate	Population
	1983	1982	1983	1982	1983	1982	July 1, 1983
United States	23,846	25,520	10.2	11.0	•	•	233,981,000
Alabama	522	631	13.2	16.0	11	7	3,959,000
Alaska Arizona	98	96	20.5	21.9	2	2	479,000
Arkansas	264 414	300 412	8.9 17.8	10.5 18.0	22 3	21 3	2,963,000 2,328,000
California	3,469	3,606	13.8	14.6	7	9	25,174,000
Colorado	108	113	3.4	3.7	42	41	3,139,000
Connecticut	194	155	6.2	4.9	33	36	3,138,000
Delaware District of Columbia [†]	65	55	10.7	9.1 36.1	17	23	606,000 623,000
Florida	202 1,457	228 1,467	32.4 13.6	14.1	9	11	10,680,000
Georgia	808	830	14.1	14.7	5	8	5,732,000
Hawaii	236	252	23.1	25.4	1	1	1,023,000
Idaho	35	31	3.5	3.2	40	45	989,000
Illinois Indiana	1,380	1,653	12.0	14.4 7.3	15 27	10 31	11,486,000 5,479,000
lowa	411	399	7.5 2.2	2.5	47	47	2,905,000
Kansas	65 76	73 92	3.1	3.8	44	39	2,425,000
Kentucky	523	605	14.1	16.5	6	4	3,714,000
Louisiana	439	471	9.9	10.8	19	19	4,438,000
Maine	39	57	3.4	5.0	43	35	1,146,000 4,304,000
Maryland Massachusetts	409	540	9.5	12.7 8.7	20 30	16 26	5,767,000
Michigan	389 790	503 864	6.7 8.7	9.5	23	22	9,069,000
Minnesota	165	157	4.0	3.8	38	40	4,144,000
Mississippi	414	333	16.0	13.1	4	14	2,587,000
Missouri	399	390	8.0	7.9	26	28 37	4,970,000 817,000
Montana Nebraska	47	37	5.8	4.6 2.0	35 49	37 49	1,597,000
Nevada	25 52	32 67	1.6 5.8	7.6	34	29	891,000
New Hampshire	38	33	4.0	3.5	39	43	959,000
New Jersev	809	804	10.8	10.8	16	18	7,468,000
New Mexico	116	122	8.3	9.0	24 12	25 15	1,399,000 17,667,000
New York North Carolina	2,309	2,268	13.1 12.8	12.8 13.4	12	12	6,082,000
North Dakota	780 9	806 16	1.3	2.4	50	48	680,000
Ohio	519	621	4.8	5.8	37	33	10,746,000
Oklahoma	331	335	10.0	10.5	18	20	3,298,000
Oregon	182	194	6.8	7.3	28	30 24	2,662,000 11,895,000
Pennsylvania Rhode Island	972 60	1,080 34	8.2 6.3	9.1 3.5	25 32	42	955,000
South Carolina	443	513	13.6	16.0	10	6	3.264,000
South Dakota	443	36	6.6	5.2	31	34	700,000
Tennessee	645	747	13.8	16.1	8	5	4,685,000
Texas Utah	1,965	2,045	12.5 2.8	13.4 3.3	14 45	13 44	15,724,000 1,619,000
Vermont	46	51		3.3 2.5	45	44	525,000
Virginia	11 520	13 672	2.1 9.4	12.5	21	17	5,550,000
Washington	239	301	5.6	7.1	36	32	4,300,000
West Virginia	133	162	6.8	8.3	29	27	1,965,000
Wisconsin Wyoming	164 14	208 10	3.5 2.7	4.4 2.0	41 46	38 50	4,751,000 514,000
					40		· · · · · · · · · · · · · · · · · · ·
American Samoa§ Guam§	7 48	4 49	20.4 45.4	12.1 46.3	:		34,298 105,821
Northern Mariana Is.§	48	75	441.0	40.5	•	•	16,780
Puerto Rico ⁹	452	473	13.9	14.8	•	•	3,261,000
Trust Terr. Pacific Is.9 U.S. Virgin Is.9	188 2	209 0	160.7 2.0	178.6	•	:	116,973
•Network	2	U	2.0	0.0			101,500

Not ranked.

[†]District of Columbia is not ranked with the States but is included in totals.

§Not included in totals.

Tuberculosis - Continued

414

TABLE 4. Tuberculosis cases and rates: cities with populations of 250,000 or more - United States, 1983 and 1982

State	Tuberculo	osis cases	Cas	e rate	Rank acc to ra	•	Population estimates
	1983	1982	1983	1982	1983 1		1983
Albuquerque, N.M.	25	30	7.0	8.6	53	46	357,600
Atlanta, Ga.	191	•	43.8	•	3	•	436,000
Austin, Tex.	33	40	8.8	11.0	51	39	375,500
Baltimore, Md.	148	221	19.7	29.0	18	10	750,000
Birmingham, Ala.	74	74	26.2	25.8	11	13	282,500
Boston, Mass.	137	150	24.3	26.6	15	11	563,000
Buffalo, N.Y. Charlotte, N.C.	50	42	14.8	12.4	32	35	338,100
Chicago, III.	45	59	13.7	18.7	38	25	328,400
Cincinnati, Ohio	871 60	1,069 78	29.0	35.6	10	6	3,005,100
Cleveland, Ohio	88		15.6	20.2	27	20	385,500
Columbus, Ohio	88 43	125	15.3	21.8	28	17	573,800
Dallas, Tex.	215	51 190	11.8 22.6	9.0 20.4	43 17	44 19	364,900 949,600
Denver, Colo.	49	56	9.8	11.2	48	38	500,600
Detroit, Mich.	286	312	25.1	25.9	12	12	1,138,700
El Paso, Tex.	66	76	14.0	16.7	36	29	471,600
Ft. Worth, Tex.	76	76	18.7	19.1	19	23	406,300
Honolulu, Hawaii	135	131	35.3	35.3	6	7	382,200
Houston, Tex.	517	648	29.4	38.1	9	4	1,760,000
Indianapolis, Ind.	102	92	14.4	13.0	33	34	706,800
Jacksonville, Fla.	82	91	14.8	16.5	31	30	554,400
Kansas City, Mo.	43	42	9.6	9.4	49	43	448,200
Long Beach, Cal.	60	95	16.1	25.6	26	14	373,100
Los Angeles, Cal. Louisville, Ky.	769	684	25.0	22.5	13	16	3,071,100
•	74	•	24.8	•	14	•	298,700
Memphis, Tenn. Miami, Fla.	89	•	13.6	•	40	•	655,600
Milwaukee, Wisc.	225	269	58.4	61.4	1	1	385,100
Minneapolis, Minn.	65 40	63	10.5	10.0	45	42	618,200
Nashville, Tenn.	75	31	11.0 16.3	8.5	44 25	47	364,700 459,900
Newark, N.J.	159	145	49.9			2	
New Orleans, La.	99	121	49.9	44.4 21.5	2 22	18	318,800 567,200
New York, N.Y.	1.651	1,594	23.3	21.5	16	15	7,086,100
Norfolk, Va.	37	52	13.9	19.4	37	23	266,900
Oakland, Cal.	110	35	31.7	10.1	8	41	347,300
Oklahoma City, Okla.	55	46	13.4	11.3	41	37	409,700
Omaha, Nebr.	12	12	3.8	3.8	55	52	312,900
Philadelphia, Pa.	297	335	17.8	19.8	21	21	1,665,400
Phoenix, Ariz. Pittsburgh, Pa.	87	89	10.3	10.8	46	40	841,200
	65	75	15.3	17.7	29	28	424,000
Portland, Ore. Sacramento, Cal.	67	66	18.4	17.9	20	27	365,000
St. Louis, Mo.	42	94	14.4	32.9	34	8	292,600
St. Paul, Minn.	57 27	59 22	13.6	13.4	39	33	419,800
San Antonio, Tex.	136	133	10.1 16.4	8.2 16.3	47 24	48 31	267,300
San Diego, Cal.	131	138					830,400
San Francisco, Cal.	303	299	14.2 42.9	15.4 43.2	35 4	32	925,000
San Jose, Cal.	101	118	42.9	43.2	30	3 26	705,700 671,800
Seattle, Wash.	85	97	17.4	19.7	23	22	489,700
Tampa, Fla.	100	83	36.5	30.7		- 9	274,300
Toledo, Ohio	9	26	2.5	7.3	56	50	354,600
Tucson, Ariz.	47	41	13.1	11.6	42	36	359,900
Tulsa, Okla.	34	32	9.3	8.8	50	45	365,900
Virginia Beach, Va.	19	17	6.7	6.2	54	51	282,600
Washington, D.C. Wichita, Kans.	202	228	32.4	36.1	7	5	623,000
		23	7.1	8.2	52	49	279,800
Total – 56 Cities	8,685	8,775	21.2	22.3	+	+	41,052,100
San Juan, P.R.	79	80	17.8	18.4	+	+	443,600

Not available, because in 1982, the reporting area included city-county data.

[†]Not ranked.

Vol. 33/No. 28

MMWR

Tuberculosis - Continued

In 1983, 1,360 tuberculosis cases were reported among children under 15 years of age, including 818 cases among children less than 5 years of age; in 1982, there were 1,349 and 789 such cases, respectively.

Final tuberculosis mortality data for 1981 show 1,937 deaths. Compared with the final totals of 2,007 and 1,978 deaths in 1979 and 1980 and the 1982 provisional estimate of 1,980 deaths by the National Center for Health Statistics, there was essentially no change in tuberculosis mortality over the 4-year period 1979-1982.

Reported by Div of Tuberculosis Control, Center for Prevention Svcs, CDC.

Editorial Note: From 1968 through 1978, the average annual decrease in tuberculosis cases in the United States was 5.6%. From 1978 through 1981, when there was a large influx of Southeast Asian refugees, the average annual decline was only 1.4%. A 6.8% decrease in the number of cases in 1982 and the 6.6% decrease in 1983 indicate the previous downward trend has resumed.

Three factors may have contributed to the decreased number of tuberculosis cases reported in 1983: (1) There was an increase in the number of states using the new individual case reporting system, which requires more accurate verification of cases before they are counted; (2) the number of refugees arriving in the United States with tuberculosis declined; and (3) the number of indigenous tuberculosis cases may have actually declined.

Despite the decline in reported cases in 1983, tuberculosis persists as a public health problem. Transmission of infection continues, as evidenced by the continued occurrence and lack of decline of disease in young children. Tuberculosis mortality has not declined; moreover, in 1980, tuberculosis was the leading cause of death among 38 notifiable diseases for which mortality data were reported (1). The number of tuberculosis deaths that year exceeded the combined total of deaths for the other 37 notifiable diseases. It is estimated that more than 10 million persons in this country are infected with tubercle bacilli. They have a lifelong risk of developing disease, which can be minimized by giving preventive treatment. Additional cases will occur in new residents of this country who come from areas of the world where tuberculosis infection rates are much higher than in the United States. Unless otherwise contraindicated, these persons should receive a course of preventive therapy (2).

State and local health departments are responsible for ensuring the control of tuberculosis in the community. It is estimated that 40,000 persons on health department registers are currently under treatment or medical supervision for tuberculosis and that each year, approximately 200,000 persons exposed to new cases must be examined. Many of these persons are placed on preventive treatment. Tuberculosis control has been complicated by the global emergence of organisms resistant to antituberculous drugs (3). Community outbreaks continue to occur in the United States (4,5).

References

- 1. CDC. Annual summary 1982: reported morbidity and mortality in the United States. MMWR 1983;31:148.
- American Thoracic Society/Centers for Disease Control (ATS/CDC). Treatment of tuberculosis and other mycobacterial diseases. Am Rev Respir Dis 1983;127:790-6.
- Kleeberg HH, Boshoff MS. A world atlas of initial drug resistance. Tuberculosis Research Institute of the South African Medical Research Council, Pretoria, South Africa, 1980.
- 4. CDC. Interstate outbreak of drug-resistant tuberculosis involving children—California, Montana, Nevada, Utah. MMWR 1983;32:516-8.
- 5. Silverman PR. An outbreak of tuberculosis in southern Delaware: the meaning of surveillance and containment. Del Med J 1984;56:156-8.

The Morbidity and Mortality Weekly Report is prepared by the Centers for Disease Control, Atlanta, Georgia, and available on a paid subscription basis from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, (202) 783-3238.

The data in this report are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the succeeding Friday.

The editor welcomes accounts of interesting cases, outbreaks, environmental hazards, or other public health problems of current interest to health officials. Such reports and any other matters pertaining to editorial or other textual considerations should be addressed to: ATTN: Editor, *Morbidity and Mortality Weekly Report*, Centers for Disease Control, Atlanta, Georgia 30333.

Director, Centers for Disease Control James O. Mason, M.D., Dr.P.H. Director, Epidemiology Program Office Carl W. Tyler, Jr., M.D. Editor Michael B. Gregg, M.D. Assistant Editor Karen L. Foster, M.A.

*U.S. Government Printing Office: 1984-746-149/10004 Region IV

DEPARTMENT OF HEALTH & HUMAN SERVICES Public Health Service Centers for Disease Control Atlanta GA 30333

Official Business

Penalty for Private Use \$300

S *HCRH NEWV75 8129 DR VERNE F NEWHOUSE VIROLOGY DIVISION CID 7-B14 X

Postage and Fees Paid U.S. Dept. of H.H.S. HHS 396