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C.M.G. Buttery, M.D., M.P.H., Commissioner
Grayson B. Miller, Jr., M.D., Epidemiologist
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Rabies*

by Keith A. Clark, DVM, PhD

A family returning from vacation stopped at a fast food shop in south central Texas, where the 2 daughters were approached by an especially friendly kitten. It was thin, but alert, and obviously homeless. The girls persuaded their parents to let them "adopt" the kitten, which blissfully accompanied them the remaining few hours of their drive home. The next morning, the kitten was taken to a veterinarian for vaccinations and worming. The veterinarian judged the kitten to be approximately 4 months old and, although thin, in apparent good health. A taeniafuge was administered, and the kitten was vaccinated against feline panleukopenia, rhinotracheitis, and calicivirus. Inactivated rabies vaccine was administered IM in a hind limb.

For the rest of that day, the cat was subdued and somewhat withdrawn, but was docile and tolerant of being handled. The next morning, the kitten could not be found; it was later discovered to be hiding in a closet. It did not resist handling, but it seemed apprehensive and tried to escape and hide whenever it could. It wasn't eating or drinking much, if at all.

On the third day, the kitten was returned to the veterinary clinic. On arrival, it was agitated, but after a few minutes it was calm enough to be examined. Extreme nervousness and a slight hind limb paresis, or weakness without evidence of pain, were the only abnormal findings. After completing the examination, the veterinarian moved a few steps away. When she approached the cat again, it attacked violently, inflicting several bites and scratches to the veterinarian and a veterinary assistant.

Although the locality was essentially a rabies-free area, the kitten was euthanatized, and the head was submitted to the Texas Department of Health Laboratory, where the veterinarian's diagnosis of rabies was confirmed by immunofluorescence. Subsequent testing revealed the vital strain was probably of skunk origin.

The family was apprised of the diagnosis, and was advised to consult their physician to determine whether postexposure rabies prophylaxis was indicated. Both the veterinarian and the veterinary assistant, each of whom had received preexposure rabies vaccination, were given 2 doses each of human diploid cell rabies vaccine,[a] with an interval of 3 days between doses.

The family asked the following questions of the veterinarian:

From the Zoonosis Control Division, Texas Department of Health, Austin, TX 78756.

[a]*Rabies Vaccine, Institut Merieux, Miami, Fla.*

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Q: Isn't rabies rare in cats?

A: Rabies is rare in all domestic animals in the United States, but cats are more frequently affected than dogs. During the past decade, the total number of laboratory-confirmed cases of rabies in cats has exceeded the number in dogs by greater than 10%.[1] In this country, rabies is primarily a disease of wild animals; less than 10% of the reported cases are in domestic animals.[1]

Q: Is this a typical case?

A: Yes, although the range of signs may be quite extensive, they typically involve behavioral changes and unexplained paralysis. In this instance, a friendly and docile kitten became withdrawn, anorectic, apprehensive, agitated, and vicious; there also was an indication of partial hind limb paresis, which would probably have progressed rapidly had the cat not been euthanatized.

Q: Is rabies in cats different from rabies in other animals?

A: Cats, unlike dogs, skunks, raccoons, foxes, and bats, do not have an independent rabies cycle (ie, cat-to-cat transmission is not known to occur); therefore, rabies in cats is acquired from other species. When rabies develops in a species that does not have an independent rabies cycle, it is called spillover.

Q: Does this mean that cats cannot transmit rabies?

A: Absolutely not! Since 1946, at least 10 cases of rabies in human beings in the United States have been associated with bites of rabid or suspected rabid cats.[2,3]

Q: What is the importance of independent rabies cycles?

A: Rabies tends to be compartmentalized or confined principally to a particular species in a particular locality.[1] This species maintains the infection in nature, and fluctuations of population are integrally related to characteristic enzootic-epizootic cycles. Compartmentalization may result naturally from biological characteristics of the viral strain (ecotype), behavioral patterns of the host species, or both. Natural or induced resistance to infection on the part of other potential hosts in the area also may favor compartmentalization by reducing spillover. For example, the increase in the number of cats affected by wildlife-transmitted rabies in relation to the number of dogs affected by wildlife-transmitted rabies may be attributable to greater inherent susceptibility, to behavioral traits that increase exposures, or to a lower vaccination rate in cats.

Q. How does behavior influence compartmentalization?

A: The inherent behavior patterns characteristic of a particular species may affect the likelihood of contracting rabies. For example, cats and skunks frequently share the same habitat (such as a barn), and do so in a state of peaceful coexistence, whereas any encounter between a dog and a skunk will invariably be antagonistic. After one such encounter, (which usually does not include physical contact with the skunk's body) most dogs will keep a safe distance from skunks, but most cats have not learned avoidance, and therefore are more vulnerable to attack by a rabid skunk. Furthermore, in the case of infected animals, behavior typical of the species may tend to promote or retard dissemination of the disease. For example, rabid foxes usually travel much farther than do rabid skunks.

Q: How are different strains of rabies virus detected?

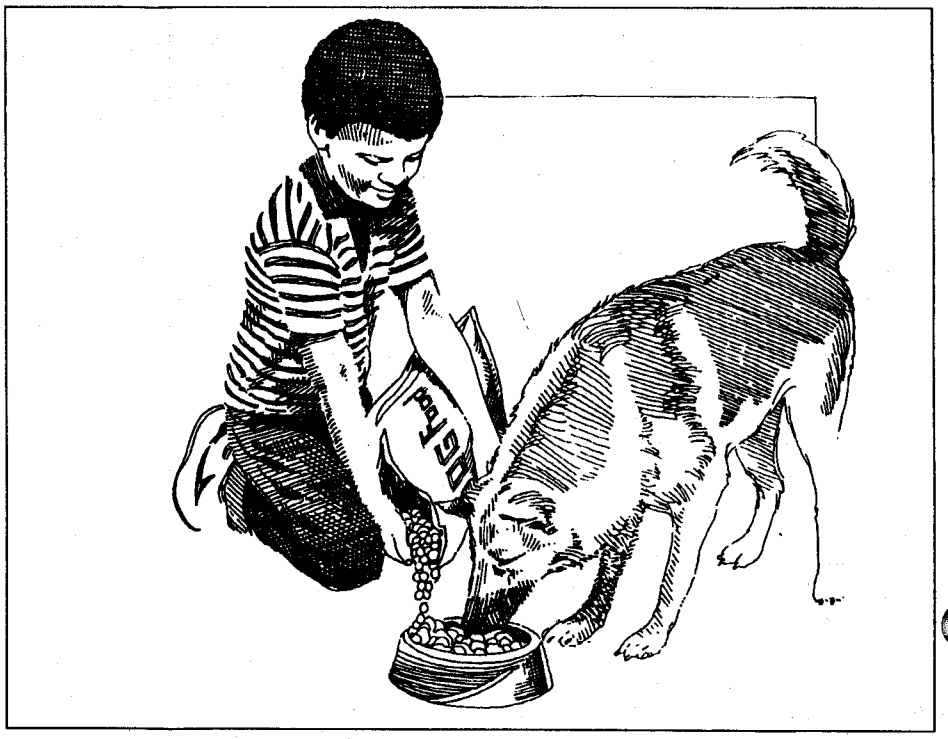
A: Rabies virus strains are detected by immunofluorescent microscopic examination, in much the same manner as routine diagnosis, using monoclonal antibody analysis, a sophisticated new techique.[4] The development of monoclonal antibodies, which are specific for one antigenic focus on the viral particle, has made it possible to identify antigenically different strains of rabies virus. The identifiable strains correlate well with the species and geographic distributions observed epizootiologically, and are stable over long periods.[5] Because this technique allows identification of the original source of infection, it is a valuable epidemiologic tool.

Q: How many strains are in this country?

A: At least 5 antigenically distinct strains have been isolated from terrestrial animals in the United States (2 from skunks, 1 from raccoons, and 1 each from gray and red foxes). Bat strains seem to be more variable, but it appears that at least 5 distinct strains can be matched with a like number of bat species.[5]

Q: Are these strains separated geographically?

A: Yes, for the most part. The red fox strain is found in Canada and



New York; the gray fox strain is in Arizona; one skunk strain is in the north central states and California; the other skunk strain is in the south central states; the raccoon strain is enzootic in the southeastern states and epizootic in the mid-Atlantic states. An epidemiologically distinct focus of gray fox rabies exists concurrently with skunk rabies in southwest Texas but, thus far, the viral isolates are indistinguishable by monoclonal antibody analysis from the northern skunk and urban Mexican dog strains. The northern skunk and Texas skunk strains coexist in Kansas, at the limits of their respective extensions.[b]

Q: What roles do insectivorous bats play in transmitting rabies to terrestrial animals?

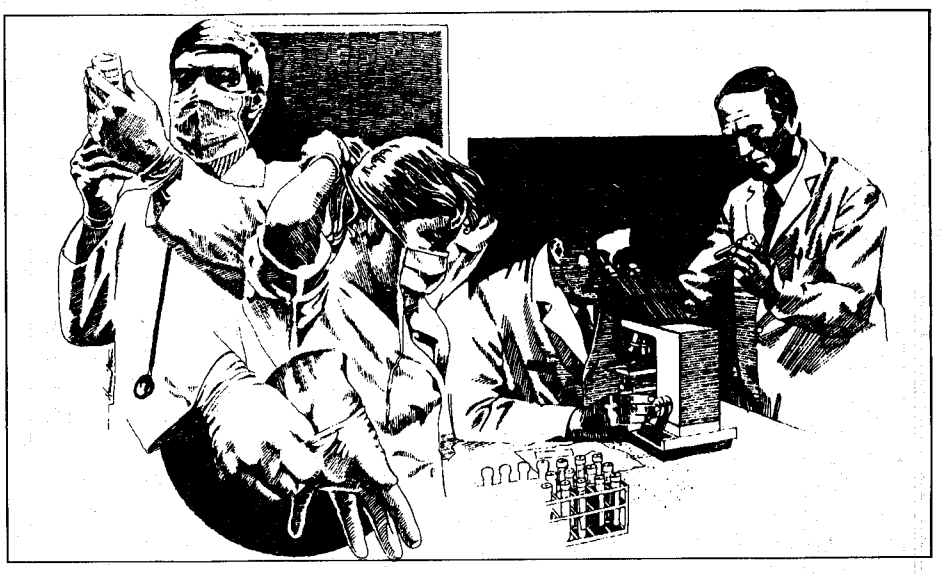
A: Bats infected with rabies may shed the virus in their saliva, and several species, including cats, cattle, and man have developed rabies after being bitten by rabid bats. However, monoclonal antibody analysis indicates that few rabid terrestrial animals are infected with bat strains.

Q: What about aerosol transmission?

A: Aerosol transmission of rabies has occurred, but only under specialized conditions in which the air contains a high concentration of suspended particles or droplets carrying viral particles. Such conditions are unusual, but have occurred in laboratory settings, caves with extremely high populations of colonial bats, and perhaps once in animal quarters where rabies-infected wild animals were housed near uninfected ones.[6] There is no danger of aerosol rabies transmission under most circumstances, and rabies virus has not been isolated from skunk musk (spray).[7]

Q: How long does rabies virus persist in the environment?

A: It depends on the ambient conditions, but rabies virus is not especially persistent under most normal conditions. It is destroyed rapidly at temperatures greater than 50 C,[8] and



generally is believed to survive no more than a few hours at room temperature, however, it will persist for years in frozen tissues.

Q: What tissues and secretions are infective?

A: The virus may be located in the central and peripheral nervous systems, the saliva, and sometimes in the urine of infected bats.[9]

Q: Is there a reliable method of rabies diagnosis applicable to live animals?

A: No. Although several techniques, including skin biopsy, corneal smears, and serologic tests have been promoted, an unacceptable level of false-negative results severely limits their usefulness.[c]

Q: What role do rodents play in rabies transmission?

A: Most rodents are highly susceptible to artificial infection with rabies, but are rarely found to be naturally infected, possibly because they rarely survive the bite of a rabid carnivore. They are not important in rabies transmission, and there has never been a report of human rabies transmitted by a rodent.

Q: How can wildlife rabies be controlled?

A: Disease control in wild populations is much more difficult than in domestic animals. Because wildlife rabies is a disease of overpopulation, population reduction programs have been advocated; however, these programs generally have proven to be impractical or not cost-effective, principally because a population explosion usually follows the reduction program unless it is sustained.

Oral vaccination of susceptible reservoir species appears to hold the most promise for the future. Foxes can be successfully vaccinated by the oral route, using modified live virus rabies vaccine.[10,11] Fox rabies has been controlled in Switzerland by aerial distribution of chicken necks injected with modified live virus rabies vaccine. A recombinant vaccine with potential usefulness in several species, made by linking a highly immunogenic portion of the rabies virus to vaccinia virus, appears especially promising in preliminary studies.[12]

Q: If 90% of rabies in the United States is in wild animals, why is vaccination of dogs and cats so important?

A: Pets frequently are the targets of attacks by rabid wild animals, and pets with rabies are a much greater hazard to human health than are rabid wild animals. Data accumulated by the Texas Department of Health indicates that the average rabid domestic animal exposes 5 times as many people to rabies as the average rabid wild animal. Furthermore, a vaccinated pet population affords a protective barrier between rabid wildlife and man.

Continued to page 4

[b] *Smith JS, Rabies Laboratory, Centers for Disease Control, Lawrenceville, Ga: Personal communication, 1986.*

[c] *Baer GM, Rabies Laboratory, Centers for Disease Control, Lawrenceville, Ga: Personal communication, 1985.*

Continued from page 3

Q: Do the vaccines available currently protect against all known indigenous strains of rabies?

A: Yes.

Q: Did our cat have feline rabies?

A: The preferred terminology would be that this was a case of skunk rabies in a cat. The species name used as an adjective should be restricted to indicate the host that maintains the cycle, if known. The term canine rabies should be reserved to describe rabies that is transmitted from dog to dog; raccoon rabies should mean rabies transmitted by raccoons. Conversely, if rabies spills over into the dog population from a non-canine source, it should be called wildlife rabies in a dog, or fox (skunk, raccoon, etc) rabies in a dog if the viral strain is determined. This convention is more accurate and more informative for those involved in control measures.

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National Childhood Vaccine Injury Act: Reporting Requirements

Since March 21, 1988, health-care providers who administer certain vaccines and toxoids are required by law to record permanently certain information and to report certain events.* The vaccines and toxoids to which these requirements apply follow: diphtheria and tetanus toxoids and pertussis vaccine (DTP); pertussis vaccine (P); measles, mumps, and rubella single-antigen vaccines and combination vaccines (MMR, MR); diphtheria and tetanus toxoids (DT); tetanus and diphtheria toxoids (Td); tetanus toxoid (T); poliovirus vaccine live, oral (OPV); and poliovirus vaccine inactivated (IPV) (Table 1). The requirements also will apply to DTP combined with inactivated poliovirus vaccine (DTP/Polio combined) if it becomes available.

Requirements for Recording

Specifically, a health-care pro-

**The National Childhood Vaccine Injury Act of 1986, as Section 2125 of the Public Health Service Act as codified at 42 U.S.C. § 300aa-25 (Supp. 1987).*

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Table 1. Reportable events following vaccination

Vaccine/Toxoid	Event	Interval from Vaccination
DTP, P, DTP/Polio Combined	A. Anaphylaxis or anaphylactic shock	24 hours
	B. Encephalopathy (or encephalitis)*	7 days
	C. Shock-collapse or hypotonic-hyporesponsive collapse*	7 days
	D. Residual seizure disorder*	(See Aids to Interpretation*)
	E. Any acute complication or sequela (including death) of above events	No limit
	F. Events in vaccinees described in manufacturer's package insert as contraindications to additional doses of vaccines† (such as convulsions)	(See package insert)
Measles, Mumps, and Rubella; DT, Td, Tetanus Toxoid	A. Anaphylaxis or anaphylactic shock	24 hours
	B. Encephalopathy (or encephalitis)*	15 days for measles, mumps, and rubella vaccines; 7 days for DT, Td, and toxoids
	C. Residual seizure disorder*	(See Aids to Interpretation*)
	D. Any acute complication or sequela (including death) of above events	No limit
	E. Events in vaccinees described in manufacturer's package insert as contraindications to additional doses of vaccine†	(See package insert)
Oral Polio Vaccine	A. Paralytic poliomyelitis	
	—in a non-immunodeficient recipient	30 days
	—in an immunodeficient recipient	6 months
	—in a vaccine-associated community case	No limit
	B. Any acute complication or sequela (including death) of above events	No limit
	C. Events in vaccinees described in manufacturer's package insert as contraindications to additional doses of vaccine†	(See package insert)
Inactivated Polio Vaccine	A. Anaphylaxis or anaphylactic shock	24 hours
	B. Any acute complication or sequela (including death) of above event	No limit
	C. Events in vaccinees described in manufacturer's package insert as contraindications to additional doses of vaccines†	(See package insert)

***Aids to Interpretation:**

Shock collapse or hypotonic-hyporesponsive collapse may be evidenced by signs or symptoms such as decrease in or loss of muscle tone, paralysis (partial or complete), hemiplegia, hemiparesis, loss of color or turning pale white or blue, unresponsiveness to environmental stimuli, depression of or loss of consciousness, prolonged sleeping with difficulty arousing, or cardiovascular or respiratory arrest.

Residual seizure disorder may be considered to have occurred if no other seizure or convulsion unaccompanied by fever or accompanied by a fever of less than 102°F occurred before the first seizure or convulsion after the administration of the vaccine involved,

AND, if in the case of measles-, mumps-, or rubella-containing vaccines, the first seizure or convulsion occurred within 15 days after vaccination OR in the case of any other vaccine, the first seizure or convulsion occurred within 3 days after vaccination,

AND, if two or more seizures or convulsions unaccompanied by fever or accompanied by a fever of less than 102°F occurred within 1 year after vaccination.

The terms seizure and convulsion include grand mal, petit mal, absence, myoclonic, tonic-clonic, and focal motor seizures and signs. Encephalopathy means any significant acquired abnormality of, injury to, or impairment of function of the brain. Among the frequent manifestations of encephalopathy are focal and diffuse neurologic signs, increased intracranial pressure, or changes lasting at least 6 hours in level of consciousness, with or without convulsions. The neurologic signs and symptoms of encephalopathy may be temporary with complete recovery, or they may result in various degrees of permanent impairment. Signs and symptoms such as high-pitched and unusual screaming, persistent unconsolable crying, and bulging fontanel are compatible with an encephalopathy, but in and of themselves are not conclusive evidence of encephalopathy. Encephalopathy usually can be documented by slow wave activity on an electroencephalogram.

†The health-care provider must refer to the CONTRAINDICATION section of the manufacturer's package insert for each vaccine.

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vider who administer one or more of these vaccines or toxoids are required to ensure that there is recorded in the vaccine recipient's permanent medical record (or in a permanent office log or file) the date the vaccine was administered, the manufacturer and lot number of the vaccine, and the name, address, and title of the person administering the vaccine. The term health-care provider is defined as any licensed health-care professional, organization, or institution, whether private or public (including federal, state, and local departments and agencies), under whose authority a specified vaccine is administered.

Requirements for Reporting

Health-care providers are required to report to the U.S. Department of Health and Human Services (DHHS) selected events occurring after vaccination. Reportable events applicable to the previously mentioned vaccines and toxoids are shown in Table 1 and include events described in the vaccine manufacturer's package insert as contraindications to receiving additional doses of the vaccine.

Methods for Reporting

In the United States, vaccines are either publicly or privately purchased. Publicly purchased vaccines are bought with federal, state, and/or local government funds. At present, the method and route for reporting adverse events depend on whether the vaccine administered is publicly or privately purchased. Events occurring after receipt of publicly purchased vaccines are reported through local, county, and/or state health departments to the Centers for Disease Control (CDC) on its Report of Adverse Events Following Immunization (CDC form 71.19). Events occurring after receipt of a privately purchased vaccine usually are reported directly to the Food and Drug Administration (FDA) on its Adverse Reaction Report (FDA form 1639) by the health-care provider or the manufacturer.

For the time being, these two systems for reporting adverse events are to be used to implement the requirement of Title XXI of the Public Health Service Act for reporting adverse events to DHHS (Table 2).

Reportable events occurring after receipt of a publicly purchased vaccine shall be reported to local, county, and/or state health departments through channels currently in place at those institutions. The Report of Adverse Events Following Immunization, available at each state health department, shall be completed and sent by the state health department to CDC.

Reportable events occurring after receipt of a privately purchased vaccine shall be reported by the health-care provider directly to the FDA on the Adverse Reaction Report (FDA form 1639). Health-care providers will need to ensure that the name of the vaccine manufacturer, the lot number of the vaccine, and the interval between vaccination and onset of the reaction are included on this form. FDA form 1639 can be obtained directly from Food and Drug Administration, HFN-730, Rockville, Maryland 20857. The form also is printed in *FDA Drug Bulletin*, the physician's edition of the *Physician's Desk Reference, USP Drug Information for Health Care Providers*, and *AMA Drug Evaluations* and can be duplicated.

Health-care providers are requested not to provide the names and other personal identifiers of patients on FDA form 1639. Such information will be reported for publicly purchased vaccines to state and local health departments, which in turn will remove the names and personal identifiers when submitting CDC form 71.19 to CDC.

Reported by: National Vaccine Program, Office of the Assistant Secretary of Health. Office of Biologics, Office of Epidemiology and Statistics, Food and Drug Administration. Div of Immunization, Center for Prevention Services, CDC.

Reprinted from MMWR 1988;37: 197–200.

Table 2. Reporting of events occurring after vaccination

	Vaccine Purchased with Public Money	Vaccine Purchased with Private Money
Who Reports:	Health-care provider who administered the vaccine	Health-care provider who administered the vaccine
What Products To Report:	DTP, P, Measles, Mumps, Rubella, DT, Td, T, OPV, IPV, and DTP/Polio Combined	DTP, P, Measles, Mumps, Rubella, DT, Td, T, OPV, IPV, and DTP/Polio Combined
What Reactions To Report:	Events listed in Table 1 including contraindicating reactions specified in manufacturers' package inserts	Events listed in Table 1 including contraindicating reactions specified in manufacturers' package inserts
How To Report:	Initial report taken by local, county, or state health department. State health department completes CDC from 71.19	Health-care provider completes Adverse Reaction Report-FDA form 1639 (include interval from vaccination, manufacturer, and lot number on form)
Where To Report:	State health departments send CDC form 71.19 to: MSAEFI/IM (E05) Centers for Disease Control Atlanta, GA 30333	Completed FDA form 1639 is sent to: Food and Drug Administration (HFN-730) Rockville, MD 20857
Where To Obtain Forms:	State health departments	FDA and publications such as *FDA Drug Bulletin*

National Conference on Clustering of Health Events

Thursday and Friday, February 16–17, 1989 • Atlanta, Georgia—Hotel InterContinental Atlanta

Sponsors: Centers for Disease Control, Agency for Toxic Substances and Disease Registry, and Association of State and Territorial Health Officials.

Purpose: To provide a forum for the comprehensive consideration of the phenomenon of clustering and to furnish public health workers, the media, and others with a theoretical and practical basis for addressing clusters of adverse health events. The detection and interpretation of clusters of adverse health events in space and time is of considerable public health concern. Evaluating the importance of observed clusters is a scientific and social challenge that can provide clues to risk factors and etiology.

Format: The conference will consist of 2 days of invited plenary talks and the presentation of contributed papers, grouped by subject and discipline. There is no registration fee. Papers will be presented in the following areas:

1. Public health response to reports of clusters
2. Statistical considerations in the aggregation of events
3. Epidemiologic considerations in disease aggregation
4. Risk perception and public information
5. Cluster investigations

CME Credits: The Centers for Disease Control (CDC) is accredited by the Accreditation Council for Continuing Medical Education (CME) to sponsor continuing medical education for physicians. CDC will offer Category 1 CME credits for designated conference sessions to physicians who have responsibilities for planning, directing, or coordinating environmental public health activities.

To preregister, please write Martha S. Brocato, Center for Environmental Health and Injury Control, Centers for Disease Control (MS-F10), 1600 Clifton Road, N.E., Atlanta, GA 30333, or telephone her at (404) 488-4251 (FTS 236-4251).

Note: A block of rooms has been reserved at the Hotel InterContinental Atlanta at Lenox Square, 300 Lenox Road, Atlanta, Georgia 30316. Room rates for conference attendees will not exceed approved Federal Government maximum lodging rate, inclusive of all taxes ($69 currently). Hotel reservations should be made directly with the hotel before January 15, 1989—telephone: 404/365-8840: after September 1, 1988, 404/262-3344. After January 15, rooms will be based on availability.

Have an Idea for the *Bulletin?*

The editor welcomes any reports of cases, outbreaks, or public health problems of interest to the Bulletin's *readers. Such accounts and any other comments or suggestions regarding the* Bulletin *should be addressed to: Editor,* Epidemiology Bulletin, *Office of Epidemiology, Room 700, 109 Governor Street, Richmond, Virginia 23219.*

Epidemiologists Switch Jobs

Dr. Jay Jones has left the Office of Epidemiology to become the District Health Director for Hanover Health District, which includes Hanover, Charles City, Goochland, and New Kent Counties. As a Commissioned Officer in the U.S. Public Health Service, Dr. Jones was assigned to the Office of Epidemiology in 1986 by the Centers for Disease Control (CDC). During the two years of his assignment he served as the Assistant State Epidemiologist, assisting local health departments in the investigation of outbreaks and conducting surveillance studies of communicable diseases and occupational injuries. We will miss Dr. Jones and his expertise, but we are glad that the State, and Hanover County in particular, will continue to benefit from his service.

Replacing Dr. Jones as the U.S. Public Health Service assignee to Virginia is Dr. Lynne Pemberthy. Dr. Pemberthy joined the Office of Epidemiology at the end of July after completing the Epidemic Intelligence Service (EIS) Course as CDC in Atlanta. Prior to her joining the EIS Service, she was in a General Preventive Medicine Residency at Johns Hopkins University. She has a Masters in Public Health degree from the same university (majoring in epidemiology). Dr. Pemberthy received her medical degree from the University of Michigan at Ann Arbor. She has already initiated several epidemiologic studies in cooperation with three District Health Directors and she is looking forward to meeting and working with all of the remaining Directors at some time during her two year assignment. Dr. Pemberthy encourages District Directors and practicing physicians to call her (804/786-6261) for any questions about, or discussions of, epidemiologic issues.

Cases of selected notifiable diseases, Virginia, for the period August 1, through August 31, 1988.

Disease	State					Regions				
			Total to Date		Mean 5 Year	This Month				
	This Month	Last Month	1987	1988	To Date	N.W.	N.	S.W.	C.	E.
Measles	0	9	1	143	23	0	0	0	0	0
Mumps	15	8	68	119	38	12	1	0	1	1
Pertussis	3	0	44	19	29	1	0	1	0	1
Rubella	0	0	1	11	1	0	0	0	0	0
Meningitis—Aseptic	13	17	140	80	142	0	8	1	2	2
*Bacterial	12	13	116	109	159	2	2	2	0	6
Hepatitis A (Infectious)	14	62	166	270	103	0	1	0	6	7
B (Serum)	11	57	301	210	337	1	6	0	3	1
Non-A, Non-B	3	10	36	54	52	0	0	0	0	3
Salmonellosis	311	191	1139	979	949	47	87	43	83	51
Shigellosis	67	77	128	308	99	3	17	3	17	27
Campylobacter Infections	130	99	409	417	403	21	37	21	20	31
Tuberculosis	40	22	296	266	274	5	12	5	10	8
Syphilis (Primary & Secondary)	21	33	194	267	264	2	2	1	10	6
Gonorrhea	1325	1373	9729	8982	12274	—	—	—	—	—
Rocky Mountain Spotted Fever	1	8	14	13	30	1	0	0	0	0
Rabies in Animals	20	36	261	252	230	7	1	3	6	3
Meningococcal Infections	2	4	56	41	51	0	0	0	0	2
Influenza	0	21	1227	2420	1622	0	0	0	0	0
Toxic Shock Syndrome	0	0	0	0	5	0	0	0	0	0
Reye Syndrome	0	0	0	0	3	0	0	0	0	0
Legionellosis	0	0	7	6	14	0	0	0	0	0
Kawasaki's Disease	0	3	20	11	21	0	0	0	0	0
Acquired Immunodeficiency Syndrome	66	35	150	277	—	3	25	8	11	19

Counties Reporting Animal Rabies: Botetourt 2 bats; Buckingham 1 skunk; Caroline 1 raccoon; Chesterfield 1 bat, 2 raccoons; Craig 1 raccoon; Fairfax 1 raccoon; Fauquier 1 fox; Frederick 1 raccoon; Hanover 1 skunk; Henrico 1 fox; Northumberland 1 cat, 2 raccoons; Page 2 skunks; Shenandoah 1 skunk; Warren 1 raccoon.

Occupational Illnesses: Asbestosis 9; Carpal Tunnel Syndrome 7; Dermatitis 1; Loss of Hearing 8; Pneumoconioses 55.

*other than meningococcal

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