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Susan S. Addiss, MPH, MUrS, Commissioner

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RABIES UPDATE

The raccoon rabies epizootic was first detected in Connecticut in March 1991, when a rabid raccoon was found in Ridgefield, which borders New York state. As of August 31, 1992, the number confirmed animal rabies cases associated with the epizootic was 508, compared with 193 in 1991. Of the 1085 raccoons tested in 1992, 456 (42%) were positive for rabies. Rabies has occurred in domestic animals for the first time since the 1940s: eight cats, two sheep, and one dog. Cases have been confirmed from 64 of Connecticut's 169 cities (Figure 1). In June 1992, a case of raccoon rabies was confirmed in Scotland, less than 20 miles from the Rhode Island border.

Editorial Note: Raccoon rabies, epizootic among raccoons in the southeastern and mid-Atlantic states, has become an increasingly important problem in the northeastern United States. The extension of the epizootic was largely responsible for the 43% increase in the total number of reported cases of animal rabies in the United States from 1990 (4881) to 1991 (6975). In 1991, 3079 cases of rabies in raccoons were reported, the largest number reported in the history of animal surveillance in the United States.

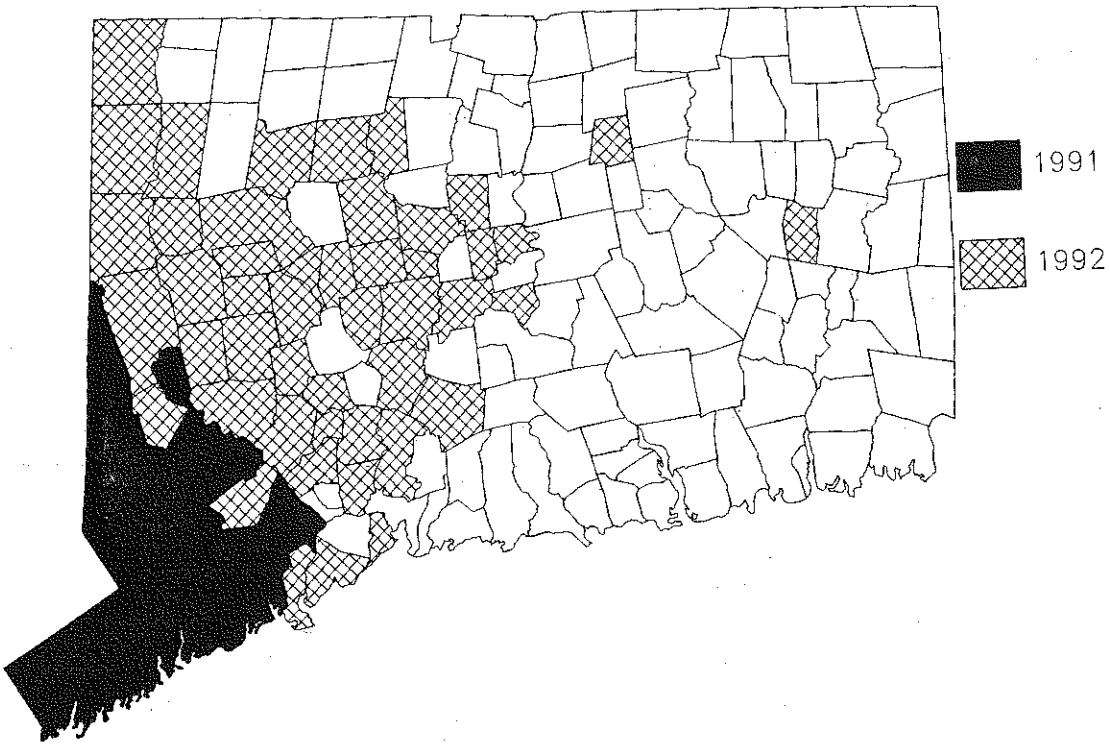
Raccoon rabies was probably introduced into the mid-Atlantic region in the mid 1970s when raccoons were transported from raccoon-rabies-enzootic regions of the southeastern United States to the mid-Atlantic area for replenishment of hunting stocks. The first cases occurred in West Virginia (1977), with subsequent spread to Virginia (1978), Maryland (1981), Pennsylvania (1982), Delaware (1987), New Jersey (1989), New York (1990), and Connecticut (1991); expansion to other New England states is expected during the next several years. With the recent identification of raccoon rabies in North Carolina (1991), raccoon rabies is now enzootic from Florida to Connecticut. Isolated reports of cases from Ohio and New Hampshire may indicate further expansion of the geographic limits of the epizootic to the West and North.

Although raccoon rabies has not been responsible for any known human rabies case, the threat of transmission is great given the presence of large populations of raccoons in areas of high human population density and the ability of raccoons to coexist with humans in urban and suburban areas, as well as in rural habitats.

The rabies threat to humans is greatest when epizootics occur in domestic animals, especially the dog. Reduction of the number of human deaths from rabies in the United States has been largely attributed to vaccination of pets and stray animal control. These traditional control measures have been effective in breaking the chain of rabies transmission from domestic animals to humans, but do not reduce the vast reservoir of rabies infection present in wildlife of the United States.

The use of oral rabies vaccine has shown promise as a tool to curb the spread of wildlife rabies.[1,2] In the United States, a newly developed vaccinia-rabies glycoprotein (V-RG) recombinant vaccine for the oral vaccination of raccoons is being tested. Field trial studies of vaccine safety conducted in Virginia (1990) and Pennsylvania

Figure 1. Animal Rabies by Town and Year of First Identification
Connecticut , 1991 - September 10, 1992



(1991) showed no detrimental effects on the environment or in nontarget species.[3] As a result, the USDA recently gave permission for an efficacy field test with the vaccine to be conducted in a defined area of New Jersey.

Additional field trials of the oral rabies vaccine for raccoons are needed to establish the appropriate distribution method (airplane, helicopter, hand placement), minimum effective geographic area, bait density, frequency, and time(s) of year for vaccination in various habitats. Strategies may vary depending on the reason for an oral-vaccination program (i.e., eliminating rabies, preventing its introduction into an area, or reducing the number of rabid animals in an epizootic area). Until these concerns are addressed, the larger question of whether oral vaccination of wildlife is cost effective cannot be adequately answered.

In addition to threatening the health of humans, domestic animals, and other wildlife, the raccoon rabies epizootic has resulted in severe economic consequences for affected states. A recent study conducted in two counties in New Jersey indicated that private and public expenditures associated with the raccoon rabies epizootic increased from $405,565 per 100,000 population during a preepizootic period to $979,027 per 100,000 population during the epizootic period.[4] Extrapolated to the entire mid-Atlantic and New England regions, potential costs associated with prevention and control activities during the epizootic period could amount to hundreds of millions of dollars.

A major focus of the public health response to rabies has been education. Education of the public has emphasized ways to reduce the risk of exposure to wild animals in affected areas, the need to keep rabies immunizations for pet dogs and cats current, and the importance of seeking medical treatment if bitten by or exposed to a potentially rabid animal. Education of veterinarians, animal-control officers, and others in occu-

pations at high-risk for exposure to rabies has emphasized the importance of preexposure prophylaxis against rabies. Education efforts have also targeted physicians and other medical professionals because many physicians in these areas have never before given either preexposure or postexposure prophylaxis for rabies.

Information on rabies, including educational materials, is available form local health departments and from the Epidemiology Program, State of Connecticut, Department of Health Services (DHS), telephone 566-5058.

[Adapted from MMWR 1992;41:661-4.]

References

1. Schneider LG, Cox JH, Muller WW, Hohnsbeen KP. Current oral rabies vaccination in Europe: an interim balance. Rev Infect Dis 1988;10:S654-9.

2. Wandeler AI, Capt S, Kappeler A, Hauser R. Oral immunization of wildlife against rabies: concept and first field experiments. Rev Infect Dis 1988; 10:S649-53.

3. Rupprecht CE, Hanlon CA, Hamir A, Kiprowski H. Oral wildlife rabies vaccination: development of a recombinant virus vaccine. Transactions 57th North American Wildlife and Natural Resources Conference (in press).

4. Uhaa IJ, Data V, Sorhage F, et al. Epizootic raccoon rabies: cost of control and economic benefits of an oral rabies vaccine. J Am Vet Med Assoc (in press).

BABESIOSIS UPDATE

Babesiosis, a relatively uncommon protozoal infection of red blood cells, is transmitted by the bite of Ixodes dammini ticks and by blood transfusion. Infection is seasonal, with most cases occurring when Ixodes ticks feed in summer and early fall.

In 1992, 21 cases of babesiosis have been reported to DHS as of September 15. These cases have involved residents from the following towns or cities: Groton (1), Montville (1), New Haven (1), Salem (1), Woodbridge (1), East Lyme (2), Lyme (3), Stonington (3), Waterford (3), and Old Lyme (5). The number of cases reported so far this year excedes that reported in any year since babesiosis was added to the list of reportable diseases in October 1989.

The first documented endemic case of babesiosis in Connecticut was reported from Stonington in 1988. In 1989, eight cases of babesiosis were acquired in Connecticut; seven by residents of Stonington or Old Lyme, and one by blood transfusion to a central Connecticut resident[1]. In 1990, four cases were reported; three from Stonington and one from Montville. In 1991, three cases of babesiosis were reported; one each from East Haddam, Montville, Stonington).

The elderly, immunocompromised, and people who lack a functioning spleen are particularly susceptible to babesiosis. The illness is generally mild or subclinical in healthy children and adults; others may present with symptoms that include fever, chills, headache, and weakness accompanied by findings of anemia, thrombocytopenia, microscopic hematuria, and mild elevations of bilirubin, lactic dehydrogenase, and hepatic transaminases. Intra- erythrocytic parasites are often observed on careful examination of peripheral blood smears, though their absence does not exclude the diagnosis.

In 1989, a serosurvey was done in the New London area, using antibodies to Borrelia burgdorferi (the causative agent of Lyme disease) as a marker for Ixodes tick exposure.[2] Of the 74 persons tested by IFA for anti-Babesia antibodies, 11 (15%) had positive antibody titers ($\geq$1:64). Persons who lived in the same town as a clinical babesiosis case were 5 times more likely to be seropositive (37% vs 7%, relative risk 5.1, 95% confidence interval 1.7, 15.4). Another Connecticut study found a 9.5% seropositivity rate for Babesia microti antibody among persons who were seropositive for Lyme disease.[3]

Since 1976, the Connecticut Agricultural Experiment Station has captured and tested rodents for B. microti. To date, the parasite has

been recovered from mice in six towns: Stonington, Old Lyme, Lyme, and Montville in New London County; East Haddam in Middlesex County; and from a single mouse from West Hartford, Hartford County (personal communication, J. Anderson).

Physicians and laboratory personnel should consider babesiosis in patients with fevers, chills, and anemia of unknown origin, and should report cases to the Epidemiology Program (566-5058). An immunofluoresence test for antibodies to Babesia can be obtained through the State Bureau of Laboratory Services.

REFERENCES:

1. Mintz ED, Anderson JF, Cable RG, Hadler JL. Transfusion-transmitted babesiosis: A case report from a new endemic area. Transfusion 1991; in press.

2. Mintz ED, Anderson JF, Hadler JL, Cartter ML. Cluster of Babesiosis in Connecticut, 1989. Presented at Epidemic Intelligence Service 39th Annual Conference; April 23-27, 1990; Centers for Disease Control; Atlanta, GA.

3. Krause PJ, Telford SR, Ryan R, et al. Geographical and temporal distribution of babesial infection in Connecticut. J Clin Micro 1991; 29: 1-4.

Reports of Selected Communicable Diseases
Connecticut, Final Summary, 1990 - 1991

DISEASE	1991	1990	% Change From 1990
AIDS	533	427	+24.8%
Gonorrhea	6,607	8,621	-23.4%
Syphilis P&S	455	874	-47.9%
Measles	29	196	-85.2%
Rubella	1	3	-66.7%
Tuberculosis	148	164	-9.8%
Hepatitis A	122	137	-10.9%
Hepatitis B	190	257	-26.1%
Salmonellosis	1,063	916	+16.0%
Shigellosis	133	227	-41.4%

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EPIDEMIOLOGY SECTION
PREVENTABLE DISEASES DIVISION
State of Connecticut Department of Health Services
150 Washington Street
Hartford, CT 06106

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