

## **EPIDEMIOLOGY OF RACCOON RABIES IN VIRGINIA, 1984 to 1989**

Authors: Torrence, Mary E., Jenkins, Suzanne R., and Glickman, Lawrence T.

Source: Journal of Wildlife Diseases, 28(3) : 369-376

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-28.3.369>

---

BioOne Complete ([complete.BioOne.org](https://complete.BioOne.org)) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at [www.bioone.org/terms-of-use](https://www.bioone.org/terms-of-use).

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

---

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

## EPIDEMIOLOGY OF RACCOON RABIES IN VIRGINIA, 1984 to 1989

Mary E. Torrence,<sup>1</sup> Suzanne R. Jenkins,<sup>2</sup> and Lawrence T. Glickman<sup>1</sup>

<sup>1</sup> Center for Applied Ethology and Human-Animal Interaction and Section of Epidemiology,  
Department of Pathobiology, School of Veterinary Medicine, Purdue University,  
West Lafayette, Indiana 47907, USA

<sup>2</sup> Office of Epidemiology, Virginia Department of Health, P.O. Box 2448, Richmond, Virginia 23218, USA

**ABSTRACT:** Geographical and temporal trends in reports of rabid raccoons (*Procyon lotor*) in Virginia were summarized for 1984 to 1989; 3,256 raccoons were submitted for rabies testing, of which 1,053 (32.3%) had rabies. Both the absolute number of rabid raccoons and the percent of rabid raccoons (number rabid divided by number submitted) were examined for seasonal and yearly trends. Geographically, the epidemic moved eastward and southward in the state. The seasonal trend showed bimodal peaks in late winter and early fall and a seasonal low in summer. The percent of rabies positive raccoons peaked 1 mo earlier than the absolute number of rabies positive raccoons. The peak in the number of rabies positive raccoons occurred in 1987, while the percent of rabies positive raccoons peaked in 1986. These trends were used to recommend timing and placement of oral vaccine as one strategy to control raccoon rabies in wildlife.

**Key words:** Raccoon, rabies, Virginia, epidemiology, oral vaccination, temporal trends, *Procyon lotor*, survey.

### INTRODUCTION

An epidemic of rabies in raccoons (*Procyon lotor*) began in the mid-Atlantic states (Virginia, West Virginia, Maryland, Pennsylvania, District of Columbia) in 1977 (Centers for Disease Control, 1978, 1981). Reported cases peaked in 1983 (1,608 cases); thereafter the number of reports in the mid-Atlantic states declined through 1988 (except 1987) even though the geographic distribution of reports increased (Fishbein et al., 1988). By 1987, the epidemic had moved into Delaware, eastern Pennsylvania, and southern Virginia (Fishbein et al., 1988). New Jersey and New York first reported rabid raccoons in 1989 and 1990, respectively (Reid-Sanden et al., 1990).

Several surveys (Kappus et al., 1970; Bigler et al., 1973; McLean, 1975; Carey and McLean, 1983; Jenkins et al., 1988) found rabies-neutralizing antibody in raccoons in epidemic and endemic areas. There is still controversy regarding interpretation of rabies-neutralizing antibodies in raccoons and the most accurate method of measuring these antibodies (Winkler and Jenkins, 1991). A minimum virus-neutralizing antibody titer that indicates protec-

tion has not been defined; thus, it is impossible to predict if a seropositive raccoon is incubating rabies or is protected from the disease (Rupprecht et al., 1986, 1988, 1989; Winkler and Jenkins, 1991).

Properties of rabies virus (e.g., transmissibility and infectivity) and attributes of the host population (e.g., density, size, and number of susceptibles) are thought to determine the likelihood of perpetuation of a virus (Yorke et al., 1979). Seidensticker et al. (1988) theorized that variable incubation periods are responsible for the maintenance of the virus in the raccoon population. For example, there might be a short incubation period during communal denning and a long incubation period during solitary life.

Contact rates between infected and susceptible wildlife are influenced by season, weather, and geographical features (Carey et al., 1978). Fluctuations in contact rates due to breeding behavior and dispersal of the young may cause a characteristic seasonal variation in the incidence of fox (*Vulpes vulpes*) rabies (Johnston and Bearegard, 1969; Wandeler et al., 1974; Toma and Andral, 1977; Carey, 1982). This pattern may also occur in raccoons. Tem-

poral trends in wildlife rabies are probably influenced by population dynamics and the changing number of susceptibles. For example, in fox populations, rabies appears to spread in a wavelike pattern with regular epidemics every 2 to 4 yr (average of 3 yr) (Toma and Andral, 1977; Voight et al., 1985; Tinline, 1988). In contrast, a temporal pattern in raccoon rabies has not been reported.

While population reduction methods were successful for skunk rabies in Alberta, Canada from 1971 to 1979, similar attempts for other species in other countries have failed to control wildlife rabies (MacInnes, 1988; Baer, 1988). Since 1968, the emphasis of rabies control has shifted away from population reduction to a more humane and cost-effective control method, vaccination of primary reservoirs (MacInnes, 1988; Wandeler, 1988). Oral vaccination for fox rabies in Europe has been successful. The fox rabies vaccine is not effective in other species. But, an oral vaccine for raccoon rabies has been developed in the United States (Rupprecht et al., 1986). Ideally, oral vaccination of wildlife should be planned with knowledge of seasonal and yearly trends and implemented when contact rates and susceptible/immune ratios are at their lowest point (Wandeler et al., 1988).

The objective of this study is to describe the seasonal and geographical epidemiology of raccoon rabies in Virginia from 1984 to 1989. This information may be used to make recommendations for the timing and placement of oral vaccination programs.

#### MATERIALS AND METHODS

In response to the raccoon rabies outbreak in Virginia in 1982, the Office of Epidemiology in the Virginia Department of Health (P.O. Box 2448, Richmond, Virginia 23218, USA) and the Division of Consolidated Laboratory Services (DCLS) in the Virginia Department of General Services (Bureau of Microbiological Science, 109 Governor St., Richmond, Virginia 23219, USA) redesigned and standardized the rabies submission form that accompanies each animal specimen to be tested for rabies.

Rabies animal submission forms were completed by personnel in local health departments or animal control organizations. These data were computerized and entered by the Office of Epidemiology (Virginia Department of Health, P.O. Box 2448, Richmond, Virginia 23218, USA) using SAS (Statistical Analysis Systems 1983 version, Statistical Analysis Institute Inc., SAS Circle, P.O. Box 8000, Cary, North Carolina 27511, USA) for the years 1984 to 1987. Later data was entered into the Epi Info Computer Program (Centers for Disease Control, Division of Surveillance and Epidemiologic Studies, Epidemiology Program Office, Atlanta, Georgia 30087, USA). Laboratory tests were performed by the DCLS laboratory in Richmond, Virginia, two regional DCLS laboratories, or two district health department laboratories in Virginia. Immunofluorescent antibody testing of brain tissue was used to confirm rabies infection, although an occasional positive diagnosis was based solely on the presence of Negri bodies in histological sections of brain tissue (Dean and Abelseh, 1973; Lepine, 1973). When human exposure occurred and brain tissue from the animal was negative by immunofluorescent antibody testing, the tissue was subsequently tested by intracerebral mouse inoculation (Lepine, 1973; Koprowski, 1973). The results were recorded as either positive or negative for rabies. Those results reported by the laboratory as non-specific or unsatisfactory were considered as negative for rabies.

The total number of raccoons submitted for rabies testing, the number positive for rabies, and the percent positive (number positive/number tested) were recorded for each month and summed over the 6 yr period from 1984 to 1989. Yearly patterns of percent rabies positive raccoons and the number positive for rabies were compared for each county and for the state. Maps were prepared to describe the pattern of geographic spread of rabies from 1984 to 1989.

#### RESULTS AND DISCUSSION

There were 3,256 raccoons submitted for rabies testing from 1984 to 1989, of which 1,053 (32.3%) were positive. The number of rabid raccoons was consistently higher in the northern counties than in many of the other counties in Virginia from 1984 to 1989 (Fig. 1). The number of raccoon rabies cases in the central and coastal counties in Virginia increased beginning in 1987.

A bimodal pattern with peaks of rabies activity in late winter and early fall was

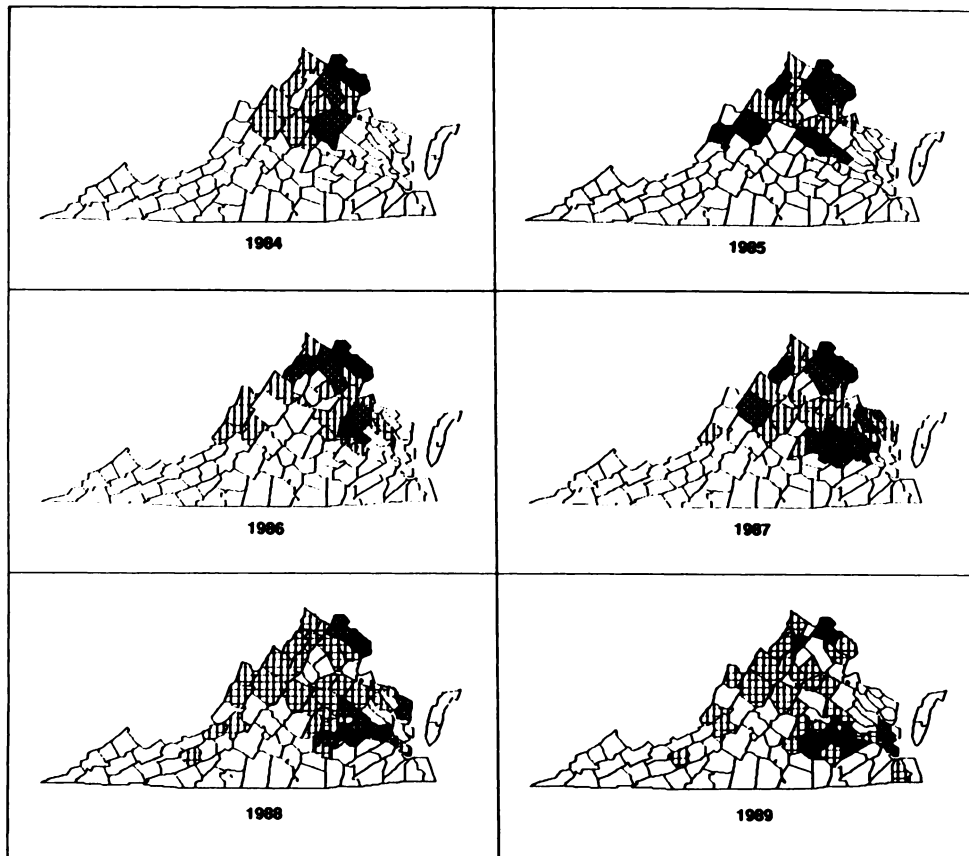


FIGURE 1. Geographic distribution of rabies positive raccoon cases from 1984 to 1989 in Virginia. The number of rabies positive raccoon cases are represented by shaded areas (□ 1-4; ■ 5-20; ■ > 20).

noted in the absolute number of rabies positive raccoons and in the percent of rabies positive raccoons (Fig. 2). Using the percent of rabies positive raccoons as an indicator of rabies activity, a rise in rabies occurred 1 mo earlier than indicated by the absolute number positive. When examining temporal trends, the percent of rabies positive raccoons showed a rise 1 yr earlier (1986) than did the absolute number of rabies positive raccoons (1987) (Fig. 3). Five counties in Virginia showed 2 or 3 yr cyclic peaks in rabies by percent rabies positive measure in contrast to no yearly cyclic trends seen in the absolute number of rabies positive raccoons (data not shown).

The persistence of rabies in wildlife continues to be an important issue for the public, public health officials, and epidemi-

ologists. Rabid animals pose a source of infection for humans and domestic animals. Vaccination of domestic animals has reduced the potential for rabies exposure and infection in man. However, potentially the most efficient control methods for wildlife rabies such as oral vaccination for raccoons and skunks are still considered as experimental in the United States. The difficulty in developing effective control strategies is due in part to inadequate knowledge of the ecology of rabies in wildlife populations, especially the mechanisms of rabies persistence, the actual number of rabid animals, and the variable incubation period of rabies in wild animals.

Recommendations are often made using public health surveillance data, but sub-

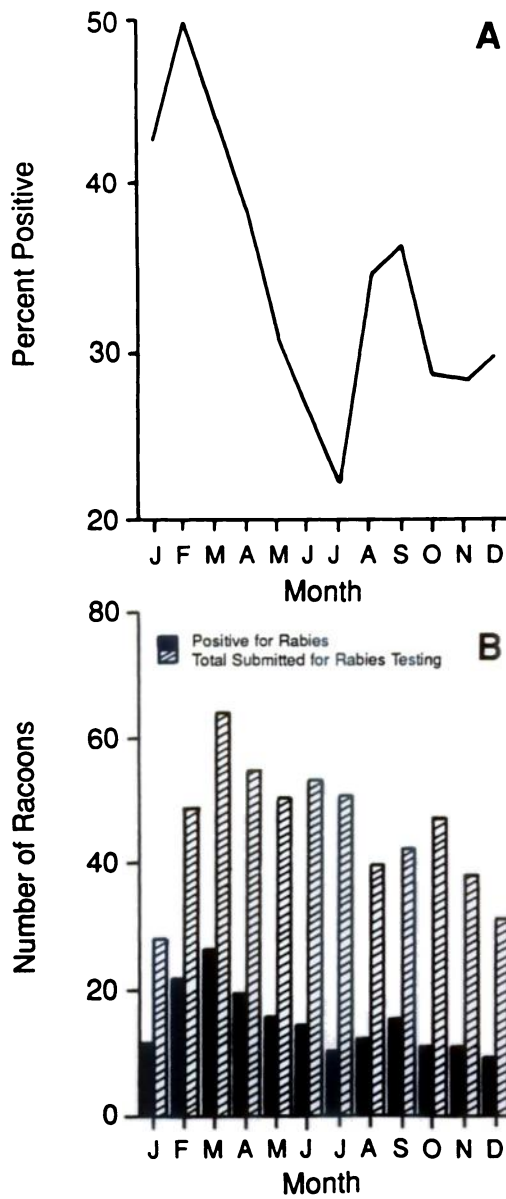


FIGURE 2. The average percent of rabies positive raccoons (A.) and the average absolute number of raccoon submissions and rabies positive raccoons (B.) by month from 1984 to 1989 in Virginia.

mission of animals for testing is nonrandom and influenced by regional differences in laboratory resources and testing policy, public awareness of the disease, and the stage of the rabies epidemic within a region. Where raccoons live in close proximity to humans, residents may be more

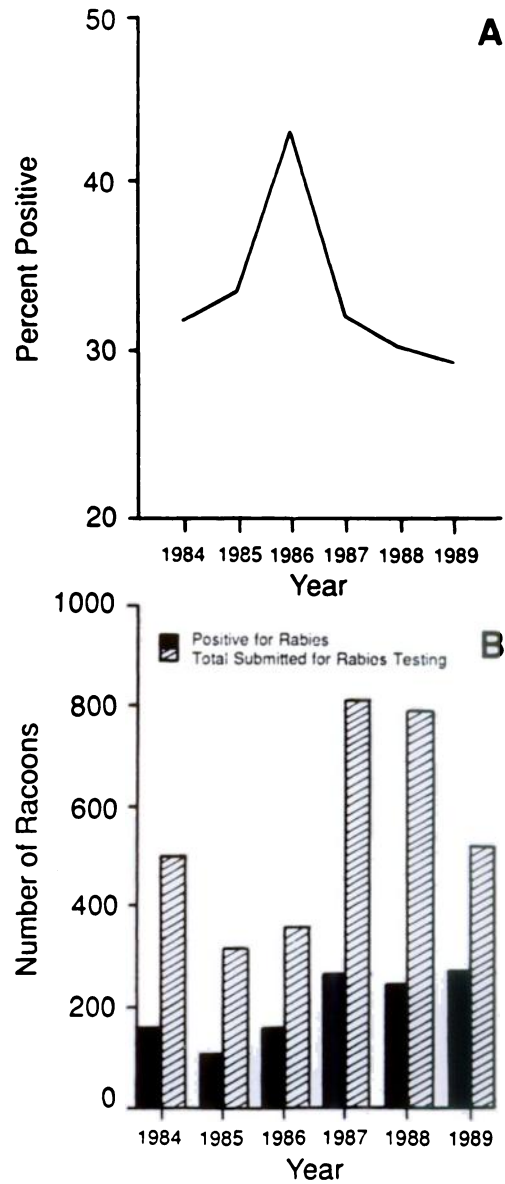


FIGURE 3. The average percent of rabies positive raccoons (A.) and the average absolute number of raccoon submissions and rabies positive raccoons (B.) by year from 1984 to 1989 in Virginia.

likely to submit animals for testing whereas residents in more sparsely populated areas may submit fewer animals (Jenkins and Winkler, 1987). As the number of submissions increase, so does the likelihood of diagnosing a rabid animal. In contrast, the number of submissions has less effect on the percent positive rabid animals.

Until an efficient method of enumerating the wildlife population and the population at risk of disease is established, passive surveillance will remain the primary method of rabies reporting. This surveillance data measures when an animal is removed from the population, but not when it is infected, or how many animals are infected and not reported. This is one reason why surveillance reports are less than perfect indicators of endemic incidence (<1% prevalence) but are fairly good indicators for epidemics (15% prevalence) (Bacon, 1985). However, surveillance reports can demonstrate important geographical, environmental, and temporal trends of rabies.

Patterns of rabies activity shown by the percent rabies positive raccoons differed from patterns in absolute number of rabies positive raccoons during 1984 to 1989 in Virginia. This difference is probably due to variations in animals submitted for testing and its implications for the study of the epidemiology of rabies is unclear. During the mid-Atlantic rabies epidemic, several counties in Virginia (e.g., Fairfax, Fauquier) (data not shown) demonstrated a peak in rabies activity in 1 yr using percent of rabies positive raccoons as a measure, whereas no peaks in rabies activity were observed in that same year using the absolute positive number of rabid raccoons. This may suggest that the percent positive is a more meaningful measure than the absolute number positive. This information (percent positive) should be examined before determining the timing and placement of oral vaccination.

Raccoon rabies in Virginia from 1984 to 1989 spread in an eastward and southward direction. One concern at the time regarding this pattern of spread was that eastern Virginia (the "Tidewater Area") was an excellent habitat for raccoons. In contrast, the southwest region of Virginia has remained relatively free of raccoon rabies despite an endemic area of skunk rabies in several counties. According to data from the Virginia Department of Game

and Inland Fisheries, the southwest area had the lowest raccoon concentration prior to the raccoon rabies outbreak and is the area most overhunted (Commonwealth of Virginia, Commission of Games and Inland Fisheries, Box 11104, Richmond, Virginia 23220, USA; Permit records, 1977–1981, 28 pp). The lack of raccoon rabies in the southwest region may also be due to the effects of the landscape ecology (Carey et al., 1978). From this current knowledge about regional rabies activity, it can be speculated that a bigger outbreak of rabies will occur in the Tidewater area than in the southwest. Rabies is only currently entering these areas.

The decrease in raccoon rabies shown by the percent positive measure (Fig. 2) during the summer months is consistent with current knowledge of raccoon rabies activity in the United States (Reid-Sanden et al., 1990). This suggests that at a time when humans and pets are more likely to come into contact with raccoons, the raccoons are less likely to be rabid. This decrease in rabies may be due to the facts that (1) raccoons are less communal in the spring and have decreased contact rates (MacClintock, 1981); (2) there is a smaller susceptible population because of greater rabies activity earlier in the year; (3) there is some period of time before rabies among a new susceptible population becomes evident using public health surveillance methods.

The bimodal peak in seasonal rabies activity (Fig. 2) probably reflects underlying biological phenomena. For example, the rise early in the year may correlate with the January to March breeding season (MacClintock, 1981) and increased contact rates and aggression (Bigler et al., 1973). The second seasonal rise in rabies may be related to dispersion of the young into the adult population (MacClintock, 1981). Both peaks in rabies activity were evident 1 mo earlier when using the percent of rabies positive raccoons as the measure of rabies activity compared with the absolute number of rabies positive animals. The differ-

ences in peaks of rabies activity may be explained by changes in human behavior rather than by raccoon biology. For example, as more rabid animals are reported in the media, people may submit more raccoons for rabies testing, thus increasing the total number of positive raccoons, but not the percent of raccoons that test positive. Both peaks were followed by a decrease in rabies activity possibly due to raccoons dying from the disease, thus temporarily reducing the total raccoon population.

There was a peak in rabies activity in 1986 (Fig. 3) using the percent of rabies positive raccoons as the measure, 1 yr before the peak in the absolute number of rabies positive raccoons. This too may be explained by increased human awareness of rabies in wildlife that leads to inflated submissions for rabies testing rather than a change in rabies virus biology or population dynamics. A cyclic pattern of rabies activity was not obvious from 1984 through 1989. The fact that the positive percent of rabid raccoons was still decreasing in 1989 may mean that rabies will continue to decline to an endemic level then disappear, or assume a cyclic pattern over a longer period of time than was analyzed in this study.

The seasonal and geographic trends (1984 to 1989) in the raccoon rabies epidemic in Virginia have provided useful information for planning control method strategies. Rabies might not persist in a raccoon population if its fatal outcome removes a sufficient number of susceptible animals from that population. For an epidemic to persist, there must be a threshold ratio of susceptible to immune animals and a minimum level of population density (Yorke et al., 1979). Seasonal increases in disease may indicate an increase in the susceptible population or an increase in contact rates. Thus, a seasonal low in rabies activity might be the best time for oral vaccination in order to further decrease the number of susceptibles below some critical point before contact rates may increase.

If it is assumed that rabies is rapidly fatal in raccoons as in most other animals, then the number of susceptible raccoons would approximate the total raccoon population in an area. Conversely, if there is a possibility of the existence of immune raccoons in a population, then the threshold number for perpetuation of rabies would depend on the ratio of susceptible to immune animals. The number of susceptibles is probably correlated to the number of animals moving into an affected area or born into the population. The only low point in seasonality apparent in the rabies epidemic in Virginia was during the summer months. When the number of susceptible animals moving into a new area increased in the fall (dispersal of young) and the number of raccoons born increased in late winter, the number of rabies cases increased. In previous studies, the positivity rates of males, females, adult, and juvenile raccoons varied with the different studies; no distinct patterns were seen (Jenkins and Winkler, 1987; Jenkins et al., 1988; Hubbard, 1985). Therefore, oral vaccination might best be implemented during the early summer months.

Geographic and yearly trends could direct attention to control method strategies. For example, because of the spread of rabies toward the coastal area of Virginia and the potential for spread southward into North Carolina and into other unaffected coastal counties in Virginia, oral vaccination could be implemented along the southeast border of Virginia to provide a "barrier" to further spread. Several counties in the northern part of Virginia (Fairfax, Frederick, Prince William, Loudoun, and Fauquier) (data not shown) where rabies has been consistently high throughout the epidemic, showed 2 or 3 yr cycles based on the percent of rabies positive raccoons. These cycles of rabies activity are probably due to an increase in susceptible raccoons due to new births, but may also be due to movement of raccoons. A containment strategy including oral vaccination programs could be implemented. This strategy might diminish the raccoon popula-

tion below the critical level needed for maintenance of the virus.

If oral vaccination of wildlife becomes a feasible control method, a measure of its efficacy must be developed. Wandeler (1991) suggested 3 techniques to measure efficacy: extensive surveillance, monitoring bait uptake, and determining seroprevalence. Monitoring bait uptake measures the amount of vaccine consumed, but does not indicate the number of raccoons vaccinated. There is still uncertainty as to what rabies-neutralizing antibodies mean in wildlife. The antibodies may indicate active disease or may indicate immunity to rabies.

The most reasonable technique to measure efficacy is probably extensive surveillance. If surveillance is established before control is introduced and then sustained, some estimate of change in rabies activity will be measured. If a decrease in rabies activity occurs, it may be due to vaccine-induced immunity, a decrease in population due to disease, or a decrease in epidemic cases. A measure such as the percent of rabies positive animals is essential to allow the earliest indication of rabies activity in an area and the most complete measure of continuing rabies activity during the epidemic and endemic stages of a rabies outbreak.

#### ACKNOWLEDGMENTS

The authors acknowledge the Office of Epidemiology, Virginia Department of Health, for providing their time, expertise, and the rabies surveillance data and the local health department personnel and animal control officers who filled out the submission forms. This study was funded in part by a grant from Merieux Institute, Inc. to the senior author and was part of the dissertation of the senior author.

#### LITERATURE CITED

- BACON, P. J. 1985. Rabies in nonhematophagous bats. *In* Population dynamics of rabies in wildlife, P. J. Bacon (ed.). Academic Press, Inc., London, England, pp. 109–130.
- BAER, G. M. 1988. Oral rabies vaccination: An overview. *Reviews of Infectious Diseases* 10: S644–648.
- BIGLER, W. J., R. G. MCLEAN, AND H. A. TREVINO. 1973. Epizootiological aspects of raccoon rabies in Florida. *American Journal of Epidemiology* 98: 326–335.
- CAREY, A. B., R. H. GILES, JR., AND R. G. MCLEAN. 1978. The landscape epidemiology of rabies in Virginia. *American Journal of Tropical Medicine and Hygiene* 27: 573–580.
- . 1982. The ecology of red foxes, gray foxes, and rabies in the eastern United States. *Wildlife Society Bulletin* 10: 18–26.
- , AND R. G. MCLEAN. 1983. The ecology of rabies: Evidence of co-adaptation. *Journal of Applied Ecology* 20: 777–780.
- CENTERS FOR DISEASE CONTROL. 1978. Annual Summary 1977. Rabies Surveillance. U.S. Department of Health and Human Services, Atlanta, Georgia, 2 pp.
- . 1981. Annual Summary 1978. Rabies Surveillance. U.S. Department of Health and Human Services, Atlanta, Georgia, 2 pp.
- . 1989. Division of surveillance and epidemiologic studies. Epidemiology Program Office, Atlanta, Georgia, Version 3, 157 pp.
- DEAN, D. J., AND M. C. ABELSETH. 1973. The fluorescent antibody test. *In* Laboratory techniques in rabies, 3rd ed., M. M. Kaplan and H. Koprowski (eds.). World Health Organization, Geneva, Switzerland, pp. 179–189.
- FISHBEIN, D. B., J. G. DOBBINS, J. H. BRYSON, P. F. PINSKY, AND J. S. SMITH. 1988. Rabies surveillance, U.S., 1987. *Morbidity and Mortality Weekly Record* 37: 1–17.
- HUBBARD, D. R. 1985. A descriptive epidemiological study of raccoon rabies in a rural environment. *Journal of Wildlife Diseases* 21: 105–110.
- JENKINS, S. R., AND W. G. WINKLER. 1987. Descriptive epidemiology from an epizootic of raccoon rabies in the mid-Atlantic states, 1982–1983. *American Journal of Epidemiology* 126: 429–437.
- , B. D. PERRY, AND W. G. WINKLER. 1988. The ecology and epidemiology of raccoon rabies. *Reviews of Infectious Diseases* 10: S620–625.
- JOHNSTON, D., AND M. BEAUREGARD. 1969. Rabies epidemic in Ontario. *Bulletin of Wildlife Disease Association* 5: 357–370.
- KAPPUS, K. D., W. J. BIGLER, R. G. MCLEAN, AND H. A. TREVINO. 1970. The raccoon as an emerging rabies host. *Journal of Wildlife Diseases* 6: 507–509.
- KOPROWSKI, H. 1973. The mouse inoculation test. *In* Laboratory techniques in rabies, 3rd ed., M. M. Kaplan and H. Koprowski (eds.). World Health Organization, Geneva, Switzerland, pp. 85–93.
- LEPINE, P. 1973. Histopathological diagnosis. *In* Laboratory techniques in rabies, 3rd ed., M. M. Kaplan and H. Koprowski (eds.). World Health Organization, Geneva, Switzerland, pp. 56–71.
- MACINNES, C. D. 1988. Control of Wildlife rabies: The Americas. *In* Rabies: Developments in veterinary virology, Vol. 7, J. B. Campbell and K. M. Charlton (eds.). Kluwer Academic Publishers, Boston, Massachusetts, pp. 381–405.



- MACCLINTOCK, D. 1981. A natural history of raccoons. Charles Scribner's Sons, New York, New York, 144 pp.
- MCLEAN, R. G. 1975. Raccoon rabies. *In* The natural history of rabies, Vol. 2, G. M. Baer (ed.). Academic Press, New York, New York, pp. 53-78.
- REID-SANDEN, F. L., J. G. DOBBINS, J. S. SMITH, AND D. B. FISHBEIN. 1990. Rabies surveillance in the United States during 1989. *Journal of the American Veterinary Medical Association* 197: 1571-1583.
- RUPPRECHT, C. E., T. J. WIKTOR, D. H. JOHNSTON, A. N. HAMIR, B. DIETZSCHOLD, W. H. WUNNER, L. T. GLICKMAN, AND H. KOPROWSKI. 1986. Oral immunisation and protection of raccoons (*Procyon lotor*) with a vaccinia-rabies glycoprotein recombinant virus vaccine. *In* Proceedings of the National Academy of Science, United States 83: 7947-7950.
- , A. N. HAMIR, D. H. JOHNSTON, AND H. KOPROWSKI. 1988. Efficacy of a vaccinia-rabies glycoprotein recombinant virus vaccine in raccoons (*Procyon lotor*). *Reviews of Infectious Diseases* 10: S803-809.
- , B. DIETZSCHOLD, J. H. COX, AND L. G. SCHNEIDER. 1989. Oral vaccination of raccoons (*Procyon lotor*) with an attenuated (SAD-19) rabies virus vaccine. *Journal of Wildlife Diseases* 25: 548-554.
- SEIDENSTICKER, J., A. J. T. JOHNSINGH, R. ROSS, G. SANDERS, AND M. B. WEBB. 1988. Raccoons and rabies in appalachian mountain hollows. *National Geographic Research* 4: 359-370.
- TINLINE, R. R. 1988. Persistence of rabies in wildlife. *In* Rabies: Developments in veterinary virology, Vol. 7, J. B. Campbell and K. M. Charlton (eds.). Kluwer Academic Publishers, Boston, Massachusetts, pp. 301-323.
- TOMA, B., AND L. ANDRAL. 1977. Epidemiology of fox rabies. *Advances in Virus Research* 21: 1-36.
- VOIGHT, D. R., R. R. TINLINE, AND L. H. BROEKHOVEN. 1985. *In* Population dynamics of rabies in wildlife, P. J. Bacon (ed.). Academic Press, London, England, pp. 311-349.
- WANDELER, A. 1991. Oral immunization of wildlife. *In* Natural history of rabies, 2nd ed., George M. Baer (ed.). CRC Press Inc., Boca Raton, Florida, pp. 485-503.
- , G. WACHENDORFER, U. FORSTER, H. KREKEL, W. SCHULE, J. MULLER, AND F. STECK. 1974. Rabies in wild carnivores in Europe. III. Ecology and biology of the fox in relation to control operations. *Zeitschrift für Veterinarmedizin* 21: 735-760.
- , S. CAPT, A. KAPPELER, AND R. HAUSER. 1988. Oral immunization of wildlife against rabies: Concept and first field experiments. *Reviews of Infectious Diseases* 10: S649-653.
- WINKLER, W. G., AND S. R. JENKINS. 1991. Raccoon Rabies. *In* Natural history of rabies, 2nd ed., G. M. Baer (ed.). CRC Press Inc., Boca Raton, Florida, pp. 325-340.
- YORKE, J. A., N. NATHANSON, G. PIANIGIANI, AND J. MARTIN. 1979. Seasonality and the requirements for perpetuation and eradication of viruses in populations. *American Journal of Epidemiology* 109: 103-123.

*Received for publication 7 March 1991.*