

**A LOG-LINEAR MODEL FOR PREDICTING
RISK FACTORS FOR RABIES POSITIVITY
IN RACCOONS IN VIRGINIA, 1984-1987**

by

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degree of**

**DOCTOR OF PHILOSOPHY
in
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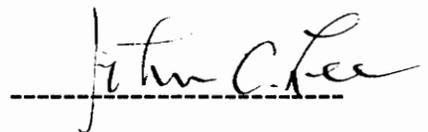
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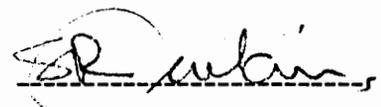
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**JULY 1990
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ABSTRACT

In response to an epidemic of rabies in the mid-Atlantic region, the Virginia Department of Health and the Consolidated Laboratory Services in 1982, redesigned their submission forms for animals being tested for rabies in an effort to elicit detailed information about the epidemiology of rabies in Virginia. The information collected from those submission forms was used in a mathematical model analysis of the epidemiology of raccoon rabies in Virginia for the years 1984 through 1987. Eleven explanatory variables and one response variable (positivity for rabies) were examined.

The objective of this study was to develop a model, through logistic regression, that would explain the epidemiology of rabies in raccoons in Virginia, and determine the risk factors for prediction for positivity for rabies in raccoons in Virginia. This information would aid further surveillance efforts, preventive education programs, and in formulating future oral vaccination programs in raccoons.

Multiway contingency tables were constructed (involving 2,3,4,5,and 6 way interactions), and log-linear models were fitted using an iterative fitting process to generate maximum likelihood estimates. The goodness of fit of each model was judged using the likelihood-ratio-chi-square p value (0.01). The backward stepwise model selection process was performed on logit models to find the best fitting model (0.01). The final model consisted of a combination of 17 four variable term models. Eight of the eleven explanatory variables remained important risk factors in the prediction of positivity for rabies in raccoons. To validate the model, it was applied to data collected in the years 1988 through July 1989. The model fit at the 0.01 level. Parameter estimates were calculated for each term in the model. All eight variables had main order effects (direct) on the response variable (positivity for rabies). Three second order effects were evident: age and season, behavior and season, and year and season. Future studies will involve applications of this model to other species to further explore the epidemiology of rabies, and to refine the model for practical applications.

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INTRODUCTION

Rabies few words in our lexicon have inspired such fear and dread throughout the ages, whose mark portended a death sentence on all who were so afflicted. Mad dogs and Englishmen, the whole world in fact, despaired that this scourge would never be banished, until Louis Pasteur developed and successfully tested the first rabies vaccine. With the enactment and enforcement of leash laws and effective vaccination programs in the 1950's, domestic rabies cases declined significantly in the U.S. Yet, rabies remains an important infectious disease today because of its existence in the wildlife population. The main reservoirs of rabies in the U.S. are the skunk, raccoon, bat, and fox, with other wild and domestic animals becoming infected through interaction with these rabid hosts. Because of the economic and public health significance of rabid domestic animals and the potential for human exposure, additional surveillance and control programs should be considered.

Rabies has become a geographically localized disease involving five major endemic foci in the U.S. Each foci usually involves only one primary host. One major foci is the Mid-Atlantic region where an outbreak of rabies in raccoons began in 1982. While the number of rabid raccoons has decreased, the disease continues to spread through Virginia and the surrounding states.

Additional information is needed about the epidemiology of the disease within the host population, how the virus affects the host, and the maintenance of the disease in the population and the geographical area. It has been difficult to obtain information regarding population dynamics and contact rates in the wild, though laboratory studies and the advent of monoclonal antibodies have allowed data to be collected about the virus itself. Computer simulation models and mathematical modeling have been developed in an attempt to understand and control rabies in the wild. The goals of mathematical modeling are to predict a dependent variable from a set of independent variables and to

estimate effect (through coefficients) in order to assess the relationship between the dependent variable and one or more independent variables [Greenberg and Kleinbaum, 1985]. Previous models have described the epidemiology of rabies within a population and theorized the effects of different control programs on that population. Models have been used extensively to evaluate the dynamics of rabies within the fox population [Bacon and MacDonald, 1980; Anderson et al., 1981; Voight and Tinline, 1985].

Programs that were originally developed to inhibit the spread of rabies within the wildlife population, were focused on eliminating the infected reservoir population. The most common methods of population reduction were by gassing (i.e., carbon monoxide pumped into fox burrows), poisoning, or trapping animals within the population, therefore, separating them from possible susceptible animals [Lewis, 1975]. However, in practice, these methods were not selective and researchers killed other animal species as well as healthy animals within the reservoir population [Lewis, 1975]. In addition, when the population density decreased, new and susceptible animals of that species often migrated or were born into the less populous area. This only served to propagate the epidemic of rabies within the area [MacDonald, 1980].

Because these control programs were not extremely successful, researchers examined other control options. Oral vaccination of the reservoir population emerged as one possible solution [MacInnes, 1988]. In 1983, field trials were conducted on fox populations in Europe using an oral vaccine hidden in "baits" (chicken heads or sausages), and immunization was accomplished by ingestion of the baits and absorption of the vaccine through the oral mucosa [Wandeler, 1988]. The attempt proved successful. Today, Switzerland is now free of rabies except for two small areas [Wandeler, 1988].

Due to the success of immunization in foxes, researchers have developed an oral vaccine for use in raccoons [Rupprecht et al., 1986].

They have conducted baiting trials (to determine the most attractive bait for raccoons) and are eagerly awaiting permission to conduct a controlled field trial [Rupprecht et al., 1988]. For this program or any oral vaccination program to be successful, careful attention must be given to the proper bait placement and timing of placement, and target population of the vaccine.

In order to select an appropriate control method, whether it be oral vaccination of the reservoir population or education of the public, epidemiologists have depended on surveillance methods to provide an understanding of the disease and the factors affecting its spread. Surveillance methods can provide important descriptive information about the epidemiology of the disease, the host, the environment, and their interactions. Accordingly, as the epidemic of raccoon rabies in Virginia became evident, the Department of Health established a surveillance system in 1982 by designing a detailed submission form that was filled out for each animal being submitted for rabies testing in Virginia. This detailed submission form is still being used by the Department of Health. The information collected on this form provides an excellent opportunity to explore the descriptive epidemiology of raccoon rabies over the subsequent seven years. It offers information for evaluation of temporal and seasonal patterns, geographical spread, and possible factors about the endemic and epidemic periods throughout this time.

These data were made available by the Department of Health to the author of this dissertation for analysis and interpretation. Mathematical modeling was used to evaluate the epidemiology of rabies in Virginia and to make some predictions about possible risk factors influencing the likelihood of rabies positivity in an area.

The purpose of this study was to develop a predictive model that would determine the possible risk factors affecting positivity of rabies in raccoons in Virginia from January 1, 1984 through December 31, 1987. The familiar

statistics of analysis of variance and linear regression are designed for continuous data. Because this study consists of frequency data, logistic regression techniques, specifically log-linear and logit models, are used. This predictive model would serve two purposes:

- 1) to contribute to the epidemiological explanation of how these factors interact and affect the course of the disease in raccoon populations; and
- 2) to contribute to the ability to predict whether a raccoon is positive for rabies.

The model developed in this study is a predictive model, but it may also describe epidemiological phenomena. By determining the risk factors, knowledge can be gained about temporal trends and the virus within the population. These trends may encompass both epidemic and endemic periods within the raccoon population. The resultant model can be applied to other species to determine differences or similarities in the risk factors, and may contribute to theories about the "compartmentalization" of rabies within one host species.

This study, and the resulting model, offers useful information for public health officials. The information will aid in decisions to be made about additional and more efficacious surveillance efforts, preventive education programs, and efforts in designing and implementing oral vaccination programs. Although this model will give both academic and practical information, it will take many more years of data collection and evaluation, to refine the model, increase its predictive and practical value, and to improve its accuracy of prediction. This study and others are necessary in light of the endemic reservoirs of rabies, especially if public health officials are to protect the public and possibly reduce the prevalence of rabies and its risk of infection of wild and domestic species.

LITERATURE REVIEW

HISTORY

Through over 2000 years of recorded existence, rabies has been a significant public health concern and an important infectious disease. Rabies was described as early as 322 B.C. by Aristotle. The first case of rabies described in the U.S. occurred in 1753, in a dog in Virginia [McLean, 1970]. Later an epidemic of fox rabies occurred in Massachusetts in 1812 [McLean, 1970]. Throughout the 19th century, epidemics of skunk rabies were recorded in California and Kansas, and fox rabies epidemics were documented in Alabama [Sikes, 1970].

In the first half of the 20th century, dogs became the most important animal host of rabies in the U.S. [Sikes, 1970]. The importance of rabies in dogs to human exposure overshadowed the continual presence of rabies in wildlife. However, when control measures in the 1950's reduced rabies in the dog population, the number of cases in wildlife surpassed those of domestic animals [Fishbein, 1988]. This was probably due to increased attention by researchers, as well as an actual rise in the number of wildlife cases [Winkler, 1986].

Rabid foxes predominated as the main source of rabies cases in wild animals until 1957, after which the majority of cases occurred in skunks [Carey, 1985]. Skunk cases increased to 1,909 in the U.S. in 1964, and have remained at high levels with peaks in 1971, 1972, and 1980 [Carey, 1985]. Rabid raccoons accounted for only a small portion of the wildlife rabies cases (2.7%) until 1962-1964 when an epidemic occurred in Florida [McLean, 1971]. By 1980, there were 393 cases of raccoon rabies reported in the U.S. [Carey, 1985]. During the mid-Atlantic raccoon epidemic in 1983, raccoons became the second most important animal reservoir behind skunks, with bat rabies being

third. Prior to this, bat rabies had consistently been the second highest animal reservoir in wildlife since 1974 [Carey, 1985]. In the U.S., there was a 17% decrease in wild animal cases in 1987, compared with cases in 1986 [Fishbein et al., 1988]. This decrease occurred in similar proportions in all three of the major wild animal hosts:

skunks (2033 cases in 1987, a 15% decrease from 1986

figures)

raccoons (1311 cases in 1987, a 17% decrease from 1986

figures)

bats (629 cases in 1987, a 20% decrease from 1986

figures) [Fishbein et al., 1988].

Today, dogs comprise less than 3% of the reported rabies cases as a result of vaccination and control programs, while wild animals account for 88% of reported rabies cases [Eng et al., 1989].

PATHOGENESIS

The pathogenesis of the rabies virus within the individual animal largely determines the behavior of the rabies virus within the host population [Murphy, 1988]. This bullet-shaped virus is a member of the Lyssa genus of the Rhabdoviruses, and is composed of a coiled RNA-containing core structure (nucleocapsid) surrounded by a lipoprotein membrane or envelope [Tordo and Poch, 1988]. Spike-like projections, which are 9nm long and separated by 5nm intervals, extend to the outside surface [Tordo and Poch, 1988]. The protein, glycoprotein(G), forms the spikes on the surface which are important for attachment of virions to susceptible cells [Tordo and Poch, 1988]. In addition, these spikes carry the key antigenic determinants which elicit the production of protective (neutralizing) antibodies when incorporated in a rabies vaccine

[Murphy, 1986]. One of the most unique features of the rabies virus is that it is exclusively neurotropic, that is, it specifically attacks and travels through the nervous system [Charlton, 1988]. Once the virus is introduced through a bite wound or laceration, the virus begins its migration via peripheral nerves to the central nervous system (CNS). After several replications, it spreads throughout the CNS, and begins centrifugal neural transport to other tissues of the body [Charlton, 1988]. The greatest concentration of virus is found in those organs with the richest nerve supply [Charlton, 1988].

Most of the clinical signs of rabies are considered to be an expression of neural dysfunction. Specifically, the virus often affects the limbic system which regulates an animal's behavior and actions, and is thus manifested clinically as passive or aggressive behavior [Kaplan, 1985]. The type of behavior may depend upon the species affected and upon the course of the infection over time. Depending upon the time of onset of the disease and the clinically manifested signs, these aberrant behaviors and actions can facilitate the spread of rabies [Bacon, 1985]. For instance, most people suspect that a skunk or fox which acts aggressively or attacks humans or other domestic animals is rabid. But even if an animal appears passive the virus still may be transmitted, especially if it is a time of communal denning or breeding [Bacon, 1985]. Because there is simultaneous centrifugal movements of infection to other tissues while the virus spreads through the CNS, the concentration of virus occurs in some tissues and fluids before the onset of clinical signs [Charlton, 1988]. Saliva from clinically normal animals may be infective for several days (from 1 to 8 days) before the onset of clinical signs [Charlton, 1988]. This knowledge is used as a basis for public health measures regarding quarantine and observation of biting animals [Charlton, 1988]. The incubation period for rabies in animals depends on the species and may have an average range of 2 to 12 weeks [Sikes, 1970]. Variability in the incubation period can also be

attributed to the quantity of virus in the saliva, its virulence, the species of the animal, and the area of the body which was bitten (inoculation site) [Tinline, 1988].

TRANSMISSION

The most common mode of transmission of rabies is through a bite wound. However, there is also evidence of airborne transmission to both humans and terrestrial animals within rabies-infected bat caves in the southwestern U.S. and in laboratories [Baer, 1975]. The virus was transmitted to sentinel animals placed in a poorly ventilated cave with large colonies of Mexican free-tailed bats (those insectivorous bats most infected in the U.S.). A wide range of mammals were infected through aerosol means: red and gray foxes, opossums, coyotes, and striped skunks [Constantine, 1962]. Winkler and Hopkins [1972] also reported an unusual outbreak of rabies in a laboratory animal colony through airborne transmission. This occurred at the Public Health Services' South West Rabies Investigational Station Las Cruces, New Mexico; 64 animals died of rabies, including 39 which had no known exposure history. There were two reports of workers being infected through aerosol transmission with the rabies virus while working in a laboratory. One occurred in 1972, and the other case occurred in 1977 [Winkler, 1973 ; CDC, 1977].

Another hypothesized mode of transmission of rabies is through oral ingestion of infected tissues [Fischman and Ward, 1968]. In 1970, Correa-Giron et al. reported mice becoming rabid after being fed rabies-infected tissues. It has also been reported that the virus may be transmitted transplacentally in cattle, mice, and skunks, but it seems an uncommon mode [Charlton, 1988]. Finally, there were five cases of rabies transmission in humans reported through transplants of rabies-infected corneas. Although non-bite transmission is reported, transmission through bites remains the most significant

epidemiologic mode.

TESTING

In the U.S., a direct immunofluorescence antibody (IFA) test is commonly used for postmortem diagnosis of rabies [Webster and Casey, 1988]. Portions of the thalamus, pons, hippocampus, and cerebellum are removed at necropsy and are used to make impression smears, which are stained with a fluorescein-rabies antibody conjugate/mouse brain suspension [Dean and Abelseth, 1973]. Both positive and negative controls are used for quality control purposes. These slides are then examined using a fluorescent microscope [Webster and Casey, 1988]. If the test results are negative, and there is human exposure, a mouse inoculation test or tissue culture inoculation is frequently performed [Lepine, 1973 and Koprowski, 1973]. The former consists of intracerebral inoculation of mice with a 10% suspension of brain tissue and either observing for mortality during a 30 day period, or sacrificing the mouse and testing the brain with the IFA test [Webster and Casey, 1988]. As its accuracy and reliability have improved, the murine neuroblastoma (NA) cell culture test is now gaining acceptance and being substituted for the mouse inoculation test [Webster and Casey, 1988]. This cell culture technique is much more sensitive to the street virus than are the mice used in the mouse inoculation test [Webster and Casey, 1988]. The IFA test, if done by properly trained, experienced personnel, is highly specific and rapid, and has become the standard test for rabies [Webster and Casey, 1988]. It is as reliable as the thirty day mouse inoculation test when used to examine fresh specimens, and is of greater sensitivity for testing decomposing tissues [Beran, 1981].

Monoclonal antibodies

The "compartmentalization" or localization of rabies in primary animal reservoirs as well as in certain geographic regions has perplexed researchers

for years. Until recently, there has been an inadequate explanation of this singular phenomenon. However, the development and use of monoclonal antibodies has allowed analysis of antigenic differences between strains of fixed and street viruses, as well as differences among isolates from different endemic geographic areas [Smith et al., 1984]. The Wistar Institute of Anatomy and Biology in Philadelphia, Pa., produced and characterized a panel of nineteen monoclonal antibodies specific for the rabies virus nucleocapsid antigen [Wiktor and Koprowski, 1978]. The monoclonal antibodies were obtained by fusion of mouse myeloma cells with spleen cells from mice immunized with complete rabies virus or viral components [Wiktor and Koprowski, 1978]. Antirabies antibodies produced by hybridomas expressed different specificities in their interaction with various strains of rabies virus [Wiktor and Koprowski, 1978]. Antigenic differences can be seen in both nucleocapsid protein and glycoprotein of the virus, though the nucleocapsid protein is the easiest to use and more easily found in the brain [Dietzschold et al., 1988]. By recording the immunofluorescence reaction of a panel of monoclonal antibodies to the nucleocapsid protein, characteristic reaction patterns of a virus from a given outbreak area can be defined, and antigenic differences in isolates from geographically separate outbreaks can be identified [Smith et al., 1984]. In addition, the use of monoclonal antibody patterns allows the epidemiologist to determine the possible source of infection for rabies cases that occur sporadically in atypical, nonreservoir hosts such as opossums, woodchucks, or humans [Rupprecht et al., 1987].

DISTRIBUTION

The geographic distributional patterns of wildlife rabies have expanded during the last decade. Cases seen in species other than the primary reservoir, are probably due to "spill-over" from the primary host species [Smith and Baer, 1988]. Many of the primary reservoir species appear to have a specific virus

(ecotype) which has been demonstrated through pathogenicity tests and antigenic patterns detected by monoclonal antibodies [Smith and Baer, 1988]. The compartmentalization of rabies within a primary species has been recognized repeatedly, but not completely explained. It may reflect the ecologic isolation between species, unrecognized virus strain differences, the specificity of strains for one host, or a combination of these and other factors [Smith, 1988]. Today, there are five major rabies endemic areas within the U.S. that have been established, each with a predominant reservoir animal species and certain viral ecotypes. They are:

- 1) the band of northcentral states stretching from Montana to Wyoming in the northwest, to Tennessee and Kentucky in the southeast (skunks)
 - 2) the southcentral states including Oklahoma, Texas, northwest Louisiana, Arkansas, and southern Missouri (skunks, but different rabies ecotype from above)
 - 3) northern California (third focus of skunks with similar rabies ecotype to northcentral states)
 - 4) the southeast states: Florida, Georgia, Alabama, South Carolina (raccoons)
 - 5) the mid-Atlantic states: Maryland, Virginia, West Virginia, Pennsylvania, Delaware, and D.C. (raccoons)
- [Smith et al., 1986].

MAJOR HOSTS

Fox

Foxes are the most susceptible species to rabies infection although cases are infrequent or sporadic, and are highly susceptible to infection by

airborne transmission [Winkler, 1975]. Since they are so susceptible, it is unlikely that they develop much immunity or resistance to rabies [Blancou, 1988]. In Europe, epidemiologists have found that foxes are good reservoirs because of their adaptability in locating denning sites, food selection, and in their ability to pass natural and constructed barriers [Blancou, 1988]. In Canada and in Europe, seasonal peaks in March in rabies cases occur annually [Johnston and Beauregard, 1969; Wandeler et al., 1974]. A secondary peak of rabies cases in the fall has been reported in Canada related to the time of weaning and the dispersal of the young [Johnston and Beauregard, 1969; Tinline et al., 1982]. Cyclic (temporal) epidemics of fox rabies occur every 2 or 4 years in Europe [Toma and Andral, 1977] and every 3 years in the arctic fox populations in Canada [Johnston and Beauregard, 1969; Tinline, 1981]. The incubation period of the disease in foxes can be extremely variable, but it is usually 19-28 days [MacDonald and Voight, 1985]. The most common clinical behavioral signs are fearlessness, aggression, and often confusion [Winkler, 1975]. A high number of attacks on livestock and other domestic animals occur during the expression of these behavioral signs [Winkler, 1975].

The fox epidemic that occurred in the Appalachian region (involving the Valley and Ridge Provinces, the Appalachian plateaus, and the Blue Ridge regions) in the 1960's has been extensively studied [Carey, 1985]. Beck [1967] reported that the epidemics lasted for 25-60 months, and that peaks usually covered 2 summers and one breeding season. Giles et al. [1972] noted that the rabies cases started to increase in October, which coincided with the dispersal of young foxes and competition for new territories. Finally, Carey [1974] observed a six month pattern, and noted that the epidemic seemed to spread to neighboring counties in a circular pattern. Often the pattern returned to the original county which was then reporting a decrease in numbers. Finally, Carey

[1974] pointed out that the Blue Ridge Mountains acted as a barrier for the epidemic spread. The epidemic spread east or west along the mountains but did not seem to cross it.

Skunks

In 1985, of a total of 8,113 reported positive rabies cases in the U.S., skunks accounted for 40% and raccoons were second at 19% [Charlton, 1988]. The striped skunk has been the most commonly reported rabid wild animal since 1961 [Fishbein et al., 1988]. Skunk saliva can contain 1000 times more virus per unit volume than that of foxes; thus, it has a greater potential to be infective [Prather et al., 1975]. Yet skunks are more resistant than foxes to the virus and to development of infection [Parker, 1975]. The incubation period of rabies in skunks is usually 14-90 days [Bacon, 1985]. The combination of long periods of viral shedding with a high virus titer in the saliva, and frequency of infection makes the striped skunk an efficient reservoir and disseminator of rabies in the U.S. [Parker, 1975]. Skunks were the source of virus responsible for 3 of the 6 human rabies deaths between 1966 and 1970 [Parker, 1975].

The clinical signs of rabies in skunks include aggression, incoordination, and hyperesthesia [Parker, 1975]. During these periods, the animals react violently to almost any external stimuli such as sound or movement, and often attack aggressively [Parker, 1975]. The tenacity with which rabid skunks bite and hold on to animals, probably contributes to the high transmissibility of skunk rabies to cattle [Parker, 1975]. They are normally nocturnal, but when rabid, increase their daytime movement and activities [Parker, 1975]. The skunk breeding season occurs in late February and March, at which time there is a peak in rabies cases. Skunks' denning behavior and their local scavenging

activity increase the potential for intraspecies spread of rabies [MacDonald and Voight, 1985]. While there is no measure or specific explanation for fox-skunk transmission rates, there are three endemic areas where fox-skunk rabies is predominant:

- 1) central and southern plains of the U.S. [Parker, 1975]
- 2) eastern woodlands of the U.S. [Carey, 1985]
- 3) southern Ontario in Canada [MacDonald and Voight, 1985].

The temporal occurrence of skunk rabies usually lags 3 to 6 months behind fox rabies [MacDonald and Voight, 1985].

Regarding the raccoon rabies that is endemic in the mid-Atlantic region, Jenkins and Winkler [1987] reported that at least 50 rabid skunks from 1977 to 1985 were from a 3 county area in southwest Virginia in which skunk rabies has been endemic for approximately 15 years. This appears to be an extension of endemic skunk rabies from the midwestern U.S. [Jenkins and Winkler, 1987]. When monoclonal antibody studies were done, the isolate from one of the southwest Virginia skunks matched isolates from skunks in the midwestern U.S., but not the raccoon virus ecotype of the mid-Atlantic region [Jenkins and Winkler, 1987].

Bats

The first case of bat rabies in the U.S. was reported in 1953 [Venters et al., 1954]. Bats may be responsible for causing isolated, sporadic cases in domestic and wild species [Smith et al., 1984]. This hypothesis has been developed through use of monoclonal antibody techniques to identify rabies

virus ecotypes [Smith, 1988]. Often a rabies case in a "rabies free" area has the same or similar viral ecotype as bat rabies. Bats are ubiquitous, with the most intensively investigated bats found in large colonies in caves in the southwestern U.S. [Carey and McLean, 1983]. Their incubation periods range from 2-25 weeks [Carey and McLean, 1983]. The increase in the reported number of rabid bats in North America probably reflects greater interest in rabies testing because of public fear, rather than a true increase within the bat population [Burnett, 1989]. The numbers of bat rabies cases seem to remain endemic at relatively constant levels [Baer, 1975]. Seasonal patterns of reported rabies cases in bats occur between May and October [Baer, 1975]. The most important question for epidemiologists regarding bats is their possible role in transmission to terrestrial wildlife and in the maintenance of rabies virus in the environment [Baer, 1975].

Raccoons

The first known case of raccoon rabies was reported in the U.S. in California in 1936 [McLean, 1975]. Although there were several reports in other states involving sporadic raccoon outbreaks, Florida became the first focus for a primary epidemic of rabies in raccoons [McLean, 1975]. The first reported raccoon rabies case in Florida was on the east central coast in 1947 [McLean, 1975]. From 1947 to 1953, 28 cases were reported from 13 northern counties in Florida [McLean, 1975]. Endemic raccoon rabies spread both north and south along major waterways between 1951 and 1958, eventually involving 31 counties [McLean, 1975]. By the 1960's rabid raccoons had spread into Georgia [McLean, 1975]. By 1984, all of Florida, most of South Carolina, Georgia, and eastern Alabama, were involved in the outbreak [Jenkins and Winkler, 1984]. There were 4,088 raccoons submitted for testing from 1957 to 1973, of which 19% were found to be rabid [Prather et al., 1975].

The second major raccoon rabies epidemic, the mid-Atlantic outbreak, probably originated with one or more rabid raccoons transported out of Florida. During 1977 and 1981, 3,500 raccoons were translocated to the mid-Atlantic region for restocking purposes [Jenkins et al., 1988]. Evidence of this connection between the rabies virus from the two areas can be found in monoclonal antibody studies. These studies have shown a close similarity in the antigenic characteristics of rabies virus isolates from the mid-Atlantic area and those from the southeast U.S. [Smith et al., 1984]. Of the 1,311 rabid raccoons reported in the U.S. in 1987, 99% were from the south east and mid-Atlantic states [Fishbein et al., 1988]. From a peak of 1,604 cases in 1983, the number of cases of raccoon rabies in the mid-Atlantic states has declined in each subsequent year except 1986 [Fishbein et al., 1988]. Yet there has been a substantial increase in the geographic distribution of the outbreak. By 1987, the epidemic had moved into Delaware, eastern Pennsylvania, and southern Virginia [Fishbein et al., 1988]. By 1989, it had moved into New Jersey [Sorhage, 1989].

There have been several theories for the continual spread and maintenance of rabies within the raccoon population. Carey and McLean [1983] and others [McLean, 1975; Bigler et al., 1973; Jenkins et al., 1988] theorized that the raccoon virus may not always be lethal in that species since antibodies have been found in apparently healthy raccoon populations. McLean [1975] demonstrated neutralizing antibody prevalence in epidemic areas averaging 16.4%. Populations in endemic areas demonstrated an average of 7.2% antibody prevalence which was significantly higher than the average neutralizing antibody prevalence (2.6%) found in raccoon populations in other parts of the U.S. [Carey and McLean, 1983]. Titers have been reported in raccoons from a variety of outbreak and non-outbreak areas, from raccoons experimentally infected, and in experimental vaccine trials [Kappus et al., 1970;

Bigler et al., 1973]. Seidensticker et al.[1983] suggested that a mixture of incubation periods may be needed for rabies maintenance in a raccoon population. The usual incubation period of rabies in a raccoon population is three weeks [Bacon, 1985]. A short incubation period would allow transmission of the rabies virus during the 5 months raccoons are active in foraging and co-denning behaviors, while a long incubation period would ensure the maintenance of the virus through the 7 month period when raccoons are dispersed in a solitary existence. If chronic, latent infections occur they may be attributed to low concentrations of virus in the saliva, low virulence, or transmission by routes other than a bite [McLean, 1975]. In a study by Winkler [1985] in which he examined raccoon salivary glands in 1982-1983 from the mid-Atlantic outbreak, he found a greater concentration of virus in raccoon salivary glands than previously reported. McLean [1975] suggested a stress-associated epidemic may occur due to breeding, "overcrowding", or concentration at certain locations because of habitat destruction due to encroachment of construction projects. However, there is still controversy about the most accurate method of evaluating rabies antibodies in wildlife, and whether results are due to actual low titers or some non-specific or cross reaction [Jenkins et al., 1988].

Other factors, besides the possibility of latent infections, must be considered which could affect the maintenance of the rabies virus within the raccoon population. Behavioral patterns may affect contact rates between raccoons and ultimately affect the spread of rabies within this species. For example, when raccoons feed, they may travel in groups of 2 or 3, giving them more dominance than would be exhibited by a single feeding raccoon [Sharp and Sharp, 1956]. These groups and mothers with their young usually feed early (before midnight) with singles feeding later. There is a decrease in feeding and consequently a decrease in contact among raccoons, if there are

winds greater than 10-15 mph, a large snowfall, or the temperatures drop much below 40F [Sharp and Sharp, 1956].

Behavioral patterns may also affect interspecies contact. Opossums are likely to compete with raccoons because they, too, are mainly nocturnal omnivores that use the same habitat [Hoffman and Gottschang, 1977]. In addition, cats, with the similar nocturnal patterns, have been found scavenging garbage cans and preying upon small mammals, so there is potential for them to be infected by raccoons [Hoffman and Gottschang, 1977]. While these two species are most likely to have contact with and become infected by raccoons, reports of dogs being exposed to raccoons are higher in number. This may be due to better reporting of dogs as compared to the other species. From 13 to 34% of rabid raccoons had contact with dogs in the southeastern states, as compared to 29 to 37% in the mid-Atlantic states [McLean, 1975; Hubbard, 1985; Jenkins and Winkler, 1987]. The positivity rates of raccoons interacting with a domestic animal in the mid-Atlantic epidemic were 82% from one study and 49% from another study [Jenkins and Winkler, 1989]. In a study of Virginia raccoons examined from 1984 to 1985, of the raccoons that exposed other animals, 49% of them in the Virginia study were positive [Jenkins and Winkler, 1988].

In the same study, Jenkins and Winkler [1988] found other factors that may affect the epidemiology of rabies in the raccoon population. They were:

- 1) Rabies positivity rates for males were 41% versus 30% for females.
- 2) The majority of the raccoons submitted for testing were captured during daylight hours. Positive rates in animals captured during daylight hours were 38% compared to 34% in animals captured at dusk.

- 3) Sixty percent of raccoons submitted were encountered in yards or residential areas. Positive rates ranged from 15% in commercial areas to 68% in cultivated land (best habitat for raccoons).
- 4) Those acting abnormally were positive in 38% of the cases submitted.
- 5) The group least likely to be positive were those killed in the road or trapped (0-9%).

HUMAN EXPOSURE

Although the incidence of rabies in dogs has declined in the U.S., it still remains the species responsible for most of the exposures that result in human antirabies treatment administered today [Helmick, 1983]. This is probably due to the close association of dogs with humans, and the high incidence of animal bites in the U.S. annually. In the 1950's, an average of 11 persons died of rabies each year in the U.S., and all of them were exposed by animals within the U.S. An average of one person per year acquired rabies from 1980 through 1988, and 73% of those infected were exposed to rabies outside the U.S. [Eng, 1989]. Human rabies postexposure prophylaxis is an important problem when considering cost, number of persons treated, or the public health resources required [Helmick, 1983]. Exposure is defined as: any penetration of skin by the teeth of a potentially infectious material of a potentially rabid animal [ACIP, 1981]. Approximately 400,000 doses of human diploid cell rabies vaccine have been administered as postexposure prophylaxis to an estimated 100,000 persons from 1980 to 1983 [CDC, 1984].

Although skunks are the wildlife species responsible for most of the human exposures, the likelihood of human exposure due to close association with raccoons needs to be considered. During a 10 year period of rabies in

Florida, (1963-1972), 75 human exposures from a total of 611 rabid raccoons were recorded [McLean, 1975]. Sixty-five percent of the human exposures to rabid wildlife reported in Florida from 1962 through 1972 occurred with raccoons [Bigler et al., 1973]. In a localized epidemic of raccoon rabies on an island in Florida in 1969, 45 rabid raccoons were observed and the observations recorded. Aggressive behavior was observed in 15 of these raccoons. Of these, seven attacked humans, six fought with dogs, and two were seen fighting with other raccoons [Kappus et al., 1970]. In contrast, Kappus [1976] found human exposure to raccoon bites following close or direct contact with unusually tame raccoons. The fact that rabid raccoon behavior is not predictable (i.e., always aggressive) makes the possibility of human exposure even higher. In Georgia, from 1967 to 1971, 90 human exposures to 418 rabid raccoons were reported. These exposures were reported as unprovoked attacks on humans by raccoons [Currier, 1972]. Finally, Helmick [1983] examined human post exposure prophylaxis in the U.S. during 1980 and 1981, and found 8% of the 5548 persons who received treatment had been exposed to a wild raccoon and 3% to a "pet" raccoon. There is no clear explanation for the lack of human cases from exposure to rabid raccoons, but it may be due to improved postexposure treatment following the emergence of raccoon rabies, low titers of virus in saliva, lower pathogenicity of the raccoon rabies virus, or lack of aggressive or unusual behavioral signs that would prevent their contact with humans [Schnurrenburger et al., 1969].

Raccoons make excellent use of artificial denning sites in close proximity to humans such as chimneys, abandoned houses, barns, garages, sheds, sewers, attics, and basements. Their presence at large artificial feeding stations established by people may increase scavenging, competition, and potential for human or domestic animal contact [Kappus, 1976]. In addition, raccoons adapt well to suburban and urban living, increasing their potential for closer

association with humans. Raccoons enjoy a "semidomesticated" status, as they are often taken in as pets, and thought of as cute, playful creatures. They are often fed as "community" pets, which increases their contact with other animals. Baer [1988] reported that urban-associated epidemics in the U.S. after the 1950's occurred exclusively in raccoons.

In a study by Jenkins and Winkler [1987] the case histories were examined of 133 persons who received rabies postexposure prophylaxis after exposure to a wild animal during the mid-Atlantic outbreak, during 1982-1983. They reported that 78 of these persons (59%) were exposed to a rabid raccoon; 43 (32%) were exposed to a raccoon that was unavailable for testing; 3 (2%) were exposed to a raccoon that subsequently tested negative; and that 5 were exposed to foxes, and 3 to skunks. The majority of exposures could have been prevented; only 16% of the exposures occurred as a result of an attack by an animal. The majority of exposures (75%) didn't require postexposure therapy. Bites, scratches, and contact of the animal's saliva with open wounds or mucous membranes accounted for only 25% of the exposures [Jenkins et al., 1988].

CONTROL PROGRAMS

Despite the control of rabies in domestic animals in the U.S., rabies continues to be a problem in wildlife. "Wildlife rabies presents both a public health hazard and an economic burden" [Lewis, 1975]. Economic burdens result from the loss of livestock or pets that become rabid or are exposed to rabid animals, the cost of follow-up on exposures by local health departments, the expense of postexposure prophylaxis in humans, lost work time for those requiring treatment, and the cost of population control programs. For example, in a study by Beck et al. [1987] the costs of animal testing, human treatment and basic administrative follow-up for 3 Maryland counties in 1982, were estimated

at 1 million dollars. Annual costs for Maryland control programs were estimated at 7 million dollars [Beck et al., 1987].

Previous control programs in wildlife have been expensive and ineffective. Poisoning and gassing are expensive control programs and pose the potential for killing other non target animals [Lewis, 1975]. Trapping is extremely expensive in terms of personnel costs, time, and other expense, although it is more selective [Lewis, 1975]. Finally, the wildlife population is capable of replacing the reduced numbers quickly, thus, making it necessary to continue the programs on an annual basis. In addition, MacDonald [1980] states, "wildlife reduction programs, may, therefore, have an adverse effect when neutralizing antibody protected animals are killed indiscriminately especially if these constitute a high proportion of the population." The best option now seems to be oral vaccination.

Since 1968, the direction of control of rabies in wildlife has shifted toward oral vaccination [MacInnes, 1988]. This emphasis has increased attention toward developing an effective oral vaccine that is safe for humans and other non-target species; developing a bait in which to put the vaccine; and determining the time and placement of the bait [MacInnes, 1988]. Placement and timing of the bait is crucial to the success of the oral vaccination program [Lewis, 1975]. The bait must be placed in areas where the target species will take it fairly quickly, and at a time critical for maximum control of the disease. The maximum point of control would be in conjunction with the impending rise of rabies in the population [Lewis, 1975]. In order to determine this, seasonal and temporal trends for rabies within the specific area and species must be determined [Wandeler et al., 1988]. To facilitate the placement of the bait, the ecology and behavior of the species, the density of the population, landscape, and patterns of the disease itself should be studied and defined [Lewis, 1975].

An effective oral vaccine for foxes has been developed and used in Europe, as well as in field trials in Ontario, Canada. For example, Switzerland, in 1982, attempted to eradicate fox rabies in the region of the Swiss Midlands [Wandeler et al., 1988]. Since the high mountain ranges naturally impeded the spread of the disease, the strategy of natural and artificial barriers was integrated in the oral vaccination program. Officials divided Switzerland into epidemiologic compartments delineated by natural and artificial obstacles to the spread of rabies, and vaccinated the fox population in each compartment [Wandeler et al., 1988]. Switzerland is now free of rabies except for an untreated area in the Jura Mountains bordering France, and an area in Canton Aargau [Wandeler et al., 1988]. They continue to maintain the "artificial barrier of immunization" along the border of areas where rabies is endemic [Wandeler et al., 1988].

ENVIRONMENTAL AND TEMPORAL CONSIDERATIONS

Because of the importance of oral vaccination and its potential for future use, other variables affecting rabies need to be examined. For example, many questions about the virus and its interaction with the host and the host's environment remain unexplained. In order to better understand these interrelationships, descriptive epidemiology involving knowledge about which individuals are affected (species, sex, age); when the disease occurs (seasonal and temporal trends); and where the disease occurs (biotic regions, climate, habitat, environment) needs to be addressed [Mausner and Bahn, 1985]. From this data, analytic epidemiology can be used to determine variables affecting the epidemiology of the disease, possible risk factors for animals being positive for rabies, and possible avenues of control [Mausner and Bahn, 1985].

As has been previously discussed, rabies has become a "compartmentalized" disease in the U.S. with specific geographical foci involving one primary host species. This observation has been strengthened by the use of monoclonal antibody studies [Smith et al., 1984]. However, the

reasons for compartmentalization within these specific geographic locations are still unclear. By mapping reported disease positivity rates, physiographic features, biotic regions, and land use, an estimate of where the disease may next occur, the pattern of its spread, and possible ecological characteristics with which the disease is associated can be determined [Carey, 1974]. Such knowledge is important to public health officials who must make decisions as to the possible risk of exposure to humans and domestic animals, and helping them plan budgets and allocate human and financial resources. In addition, natural barriers to the spread of the disease, as well as information regarding specific foci of the disease, are vital to any effective control program [Wandeler et al., 1988].

The seasonal trends in rabies infections would have an influence on control programs. Seasonal trends in a disease may suggest that seasonal influences can modify contact rates between animals or the numbers of susceptible animals available [Carey, 1974]. It is well known that during breeding seasons, the frequency of contact and movement of animals is increased, as well as the frequency of aggressive attacks [Anderson et al., 1981]. In addition, the dispersal of young after weaning, increases the number of susceptibles in the population and the frequency of contacts with other animals searching for new territories [Anderson et al., 1981]. Both of these events create characteristic seasonal variation in incidence [Johnston and Beauregard, 1969; Wandeler et al., 1974; Toma and Andral, 1977].

Seasonal trends may be due in part to the behavior of the host, but there are also environmental factors to consider. Weather and climate may affect the physiological, social, and behavioral state of the animal, thus modifying the means of transmission [Carey, 1974]. For example, air temperature is the major environmental influence on a raccoon's activity [Sharp and Sharp, 1956]. If it is extremely cold or wet, their activity will decrease, thus reducing the frequency of

contact with other animals or humans and reducing the potential for transmission. During the winter hibernation, bats are known to incubate the rabies virus until spring, when it is more easily transmitted [Baer, 1975].

Recognition and demonstration of seasonal trends are essential to aid in public health deliberations as well as control programs. For example, both humans and domestic animals are more likely to be exposed to wild animals during the spring, summer, and early fall months when people and animals are active outdoors [Helmick, 1983]. During these months, wild animals are also more active. The optimum time for oral vaccination programs is at the time of an impending rise in rabies, and at a time which will facilitate the immunization of the greatest number of susceptibles [Wandeler et al., 1988]. These times can be estimated through evaluating and recognizing seasonal patterns.

Although seasonal trends are easier to measure over a short period of time, temporal trends are just as important in the evaluation of the rate of spread of rabies and in establishing control programs [Wandeler et al., 1988]. In foxes, rabies appears to spread more in a wavelike epidemic pattern as compared to an extension of an endemic focus [Toma and Andral, 1977]. This epidemic outbreak followed by a quick "die-off" of the population creates a cyclic pattern of 2 to 4 years [Toma and Andral, 1977]. Carey [1974] described the spread of rabies in foxes in Virginia from one county to another as a "circular" pattern. Eventually, the original focus of the rabies outbreak which had reported low numbers over a period of time, experienced another epidemic [Carey, 1974].

On the other hand, raccoons appear to maintain the infection by extending the endemic focus. Serologic evidence of infection appears to precede the appearance of clinical rabies along the advancing front of the endemic focus [Beran, 1981]. This peripheral extension of endemic focus is scattered with occasional local epidemic outbreaks [Beran, 1981]. There is some speculation that these secondary epidemic waves occur as young

raccoons are born into endemic areas [Jenkins, 1989]. Supporting evidence for this was seen in several northern Virginia counties that reported an increase in the numbers of rabid raccoons in 1986, following a period of little or no rabies activity [Jenkins and Winkler, 1989].

MODELING

The earliest attempts at modeling of disease were made by William Farr in 1840 [Bailey, 1967]. Later in 1929, Soper invented a deterministic mathematical formula to describe the flow of hosts between compartments containing susceptibles, infected, and immune individuals [Hiorns and Cooke, 1981]. Because deterministic models often did not fit real data, stochastic or probability models were developed to deal with the elements of chance and variation. McKendrick in 1926 was the first to publish a true stochastic model for an epidemic [Bailey, 1957]. Instead of dealing with actual numbers of cases, it dealt with probabilities of new cases.

Multiple logistic regression models have become a standard method for data analysis in epidemiologic studies [Lemeshaw and Hosmer, 1982]. This model is most frequently used in studies involving dichotomous outcome variables (yes or no) that are related to a number of independent variables. It can easily be applied to stratified 2 x 2 tables of a case-control study [Lemeshaw and Hosmer, 1982]. Logistic regression techniques, specifically log-linear and logit models, are the statistics of choice for analysis of frequency data.

With respect to rabies, models were constructed mainly to explain the disease in fox populations. Many have been deterministic computer simulation models to explain rabies spread in foxes and the effects of different control methods in the disease [Preston, 1973; Bogel et al., 1981; Bacon and MacDonald, 1980; Anderson et al., 1981; Kallen et al., 1985; Garnerin et al.,

1986; Voight and Tinline, 1985]. Recently, Coyne et al. [1989] developed a deterministic, compartmental mathematical model to explain rabies in raccoons in the mid-Atlantic states.

One of the main disadvantages of modeling an epi-endemic disease is that the distribution of certain important epidemiologic parameters such as incubation period of the virus, distance, dispersion, and contact rate of the hosts, are not known [Garnerin et al., 1986]. It is known when an animal is removed from the population, but it is not known when it was infected. This demonstrates why surveillance reports are not good indicators of endemic incidence (less than 1% prevalence), but are fairly good indicators of epidemics (15% prevalence) [Bacon, 1985]. Yet, surveillance reports can demonstrate important geographical, environmental, and temporal trends of the virus and host, and can serve as a data base for modeling.

METHODS

In response to a raccoon rabies outbreak in Virginia in 1982, the Office of Epidemiology in the Virginia Department of Health, and the Division of Consolidated Laboratory Services in the Virginia Department of General Services, redesigned and standardized the rabies submission form that was to be submitted with each animal specimen that was tested for rabies (Fig. 1). This was done to increase the quality and quantity of clinical and epidemiologic information collected on each specimen. The following information was collected on each animal submitted for rabies testing:

- animal species
- date collected
- whether a human was exposed to the animal
- county
- whether the animal submitted was exposed to a known or suspect rabid animal
- the results of test performed on submitted animal
- date animal was submitted to the lab and the date the animal was tested

The following information was requested on the form but may or may not have been included, depending on the availability of the information:

- health/behavior status of animal
- how the animal was captured or found
- sex and age (adult or juvenile)
- exposure to other animals by the submitted animal
- time of day first sighted
- land use characteristics of site where collected
- type of exposures of tested animal to other animals and people.

Animal submission forms were filled out by personnel in local health departments or animal control organizations, and they recorded first hand information, if available. Prior instructions on how to fill out the submission forms and criteria for the categories were given to the personnel that would be recording the data. All of this data was collected and entered by the Office of Epidemiology in SAS (Statistical Analyses System, 1988) for the years 1984-1987. Data for 1988 to the present, were entered into the EpilInfo program. These data, collected for and entered by the Office of Epidemiology, were converted to SAS by the author for use in this study. Rabies diagnoses were performed at the Department of General Services, Division of Consolidated Laboratory Services(DCLS) central laboratory located in Richmond, Virginia, or at one of the two regional (DCLS) laboratories, or one of the two local health department laboratories experienced in rabies testing in Virginia. Immunofluorescent antibody testing of the brain tissue, as described in the Literature Review Section, was the most common method used, although an occasional positive diagnosis was based solely upon Negri body identification. When human exposure occurred, and brain tissue from the animal was negative by immunofluorescent antibody testing, the tissue was subsequently tested using the mouse inoculation test. The results were recorded as either positive or negative for rabies. Those results recorded as non-specific or unsatisfactory were considered as negative for rabies.

The original data from December 1983 through December 1987, contained information on 10,072 animals that were submitted for rabies testing. Because this represented 33 different species of animals, many of which would not have been likely to be exposed to rabies (i.e., indoor pet hamsters, guinea pigs, rats, mice, or gerbils), these species were automatically eliminated from analysis since they would not be good predictors of rabies in wildlife. The other wildlife species (with the exception of raccoons) were submitted in such small

numbers that the analysis of those few data points, and the ability to make predictions from such few data points, would diminish the reliability and validity of the model. Additionally, combining the data derived from these species with the larger data pool of raccoons would introduce additional unwanted variables in the model, since behaviors, habitat, and likelihood of human contact differ between the species. Therefore, the raccoon was selected as the species for analysis. It is the major reservoir of rabies in Virginia, and was submitted in greater numbers for testing.

The objectives for this log-linear model were to evaluate the epidemiology of rabies in raccoons in the years 1984 through 1987, and to perform several determinations of risk factors affecting the positivity of rabies in raccoons. This model would allow, through risk factor analysis, the ability to predict the probability of a raccoon being positive for rabies as well as the proportion of raccoons submitted that would be positive for rabies.

VARIABLE DESCRIPTION

The dichotomous variables used in this study were: whether the animal exposed a human, age (juvenile or adult) and sex of the raccoon, and whether the raccoon bit another animal. The polychotomous variables used in this study were: behavior, location where the animal was found, how it was captured, time of day it was found, county in which it was located, season and year it was collected. These variables were the eleven explanatory variables. The explanation of each variable and category can be found in Table 1. RABPOS (positive for rabies) was the one response variable.

VARIABLE SELECTION AND REDUCTION

Definitions-CASES were defined as animals testing positive for rabies, whether positive on the IFA test, Negri body test, or the mouse inoculation test. These were referred to as RABPOS, the response variable, in the model. Those animals recorded as negative were referred to as CONTROLS. Since the

population denominators (such as size, number of infected, and number of susceptibles) from which the sampling takes place were unknown, incidence or prevalence could not be calculated. However, positivity "odds" can allow an approximation. These were calculated as positivity "odds" for a dichotomous variable which equals: the number of positive animals divided by the number of negative animals.

A total of 2005 raccoons were examined from January 1, 1984 through December 31, 1987. Additionally, 1160 raccoons were examined from January 1, 1988 through July 31, 1989. Although the data had previously been entered on SAS, each submission form was reexamined and checked with the SAS output to identify potential entry mistakes or coding errors. Geographical maps were created to demonstrate total rabies cases and raccoon rabies cases from 1984 through 1987. Frequencies for each variable were calculated to recheck for coding problems, and to look for initial patterns (such as high frequencies in one variable, correlations between more than one variable) or inconsistencies in the data. Graphs of monthly trends for raccoon rabies cases and yearly trends were calculated.

Four variables containing multiple categories were collapsed to provide more frequencies per cell, and to eliminate potential problems of sparse cells. In the collapsing process, the decision was made to collapse the number of behavioral categories to those behaviors which were more likely to affect the potential for human or animal exposure to rabies, rather than those behaviors that represented the normal or natural behavior or habitat of the animals. Similarly, the location categories were collapsed to eliminate those categories which were least likely to affect the potential for human or animal exposure to rabies. In making this decision, the assumption was made that rabid animals were more likely to behave differently than unaffected animals. This direction of

study was primarily to explore the epidemiology of rabies and public health concerns moreso than describing a wide range of ecological variables which were considered not as relevant. Although collapsing was performed for more efficient analysis, the original categories were available for further examination. They consist of:

- 1) Location - originally had 11 categories. These were collapsed to 3 categories:

Residential - residential yard or residential building

Nonresidential - commercial or industrial

Iowa - a term coined to describe recreational, unimproved, agricultural, forested land or land within 100 feet of water.

- 2) Behavior - originally had 10 categories. These were collapsed into 6 categories:

Sick

Aggressive

Normal

Friendly

Neuro - wobbily or paralyzed

Flat - indifferent or

unresponsive

- 3) Counties and 16 major cities. These were collapsed into five categories coinciding with the five administrative regions established by the Virginia Department of Health. (See Figs. 2-4). These regions were established based primarily with regard to human population distribution. As of 1988, the populations for the regions were: Northwest - 785,590; Northern - 1,283,340; Southwest - 1,341,560; Central - 1,048,090; and the Eastern - 1,516,687.

- 4) Month animal was collected- originally had 12 categories.

These were collapsed into seasons as follows:

Season 1 - December, January, February

Season 2 - March, April, May

Season 3 - June, July, August

Season 4 - September, October, November.

Any response under any variable that was marked missing, other, or unknown, was dropped from the analysis. Other variables from the same submission form were retained in the analysis.

In several instances more than one category had been marked for the location and behavior variables. A frequency analysis of these duplicate counts was performed to evaluate the extent of this problem in the data. Of the categories recorded for the 10,072 original submissions, 8.4% of the categories under the variable location were marked more than once, and 26.3% of the categories under behavior were also marked more than once. To simplify analysis and to prevent duplication of counts, each category under these two variables was prioritized according to what category would be most likely to be a factor in human or animal exposure (location variable) or would be most likely a clinical sign consistent with a rabid animal. Only the highest priority category was counted. They are listed in descending order of priority:

Location:

Residential

Nonresidential

Iowa (refers to rural/agricultural/recreational areas)

and:

Behavioral:

Aggressive

Neuro

Flat

Friendly

Sick

Normal

The first four behaviors were higher in priority than sick because they were more descriptive of the actual behavior.

MODEL FITTING

With data this large, it was reasonable to develop a strategy to focus attention on a reduced set of models that give a reasonable fit to the data. As an indication of the complexity of this process, with just ten variables there are 3.5 million possible models of independence [Fienberg, 1980]. This is, in fact, fewer than the total number of different log-linear models. This strategy of reducing the number of model sets, was accomplished by looking at multiway tables and fitted log-linear models to determine if all twelve variables examined were significant for model selection and fitting. A criterion level of 0.01 was chosen to make the criteria more stringent for terms to remain in the model. While 0.01 is less conservative than 0.05, it was more important to eliminate any variables that might remain in further analysis only by chance.

Two way contingency tables were constructed for the twelve variables using SAS. As a result of this analysis, the explanatory variable, sex, was dropped from further analysis because it was independent of the other variables and of RABPOS. Also, the small number of responses, 701, would create sparse cells later. The BMDP4F computer program (1988) was used to form and analyze further hierarchical (3, 4, 5, and 6 way) contingency tables. To convert SAS data files for use in BMDP4F, all "character" variables were recoded to number values. BMDP4F fits log-linear models belonging to a hierarchical set in which inclusion of higher order terms automatically includes related lower order terms. Log-linear models provide a unified approach to analyzing categorical variables [Fienberg, 1980]. For data arranged in a multidimensional contingency table, a log-linear model represents the logarithm of the expected cell counts in terms of the parameters that denote the individual and joint effects of the variables forming the multiple cross-classification [Fienberg, 1980]. This computer program uses an iterative

fitting process to generate the maximum likelihood estimates of the expected cell frequencies of the specified models. The maximum process chooses estimators of the parameters that maximizes the likelihood function which represents the probability of observing the data obtained as a function of the unknown parameters [Fienberg, 1980]. The maximum likelihood estimators (MLE) have the desirable property of being those estimators that will most closely resemble the actual data for a given model [Fienberg, 1980]. They are the estimate of regression coefficients that can accommodate any kind of data.

The goodness-of-fit of each model was judged using the likelihood-ratio-chi-square p values. It is defined as:

$$G^2 = 2 \sum_{ijk} X_{ijk} \ln \left[\frac{X_{ijk}}{m_{ijk}} \right]$$

where G^2 = the likelihood-ratio test statistic, X_{ijk} = the observed values for the i th row, the j th column, and the k th layer in the table, \ln = logarithm, m_{ijk} = expected values for the i th row, the j th column, and the k th layer in the table, and Σ = summation over all cases in the table [Bishop, 1975]. The likelihood-ratio test determines how much the maximum likelihood value for one specific model changes based on the addition or deletion of one or more parameter to (or from) that given model. The selected log-linear model was the most parsimonious (simplest) model that provided a satisfactory explanation of the data at the desired criterion level (0.01). The general formula for a saturated three variable log-linear model is:

$$\log m_{ijk} = \lambda + \lambda_{1(i)} + \lambda_{2(j)} + \lambda_{3(k)} + \lambda_{12(ij)} + \lambda_{13(jk)} + \lambda_{23(jk)} + \lambda_{123(ijk)}$$

where m_{ijk} is the log of the expected values for i th row, j th column, and the k th layer, variable 1 takes level i ; variable 2 takes level j ; variable 3 takes level k ; and λ is the estimate of the parameter [Fienberg, 1980].

When the 3-way interactions were calculated, the variable, time, was found to be independent of RABPOS and the other variables; therefore, it was dropped from further analyses. Both the nonresidential category under location and the road kill category under captured had few responses (51 and 19, respectively); this created sparse cells. Therefore, these two categories were eliminated from further analysis. This improved the fit of the models.

Looking at 4 way interactions among the remaining ten variables, 80 models were fitted. In actual model fitting, the higher the p value, the better the fit because there is less variation unexplained. All but four models had p values above 0.01 for both the 3 way and 4 way interactions. Continuing the analysis, models using 5 way interactions were calculated. Of the possible 126 models, only those models using third order terms that fit poorly (less than 0.01) in previous models and that would produce less than 150 cells (to prevent sparse cell problems) were chosen. This left 30 possible models. During this analysis, there was a high proportion of sparse cells under the variable "behavior", so the six categories were collapsed into four:

- Aggressive
- Friendly
- Sick (to include flat and neuro)
- Normal

The categories flat and neuro were combined because most animals that would be paralyzed would be perceived by people as unresponsive, indifferent, or sick. Because of the large number of variables and sample size, it was unmanageable to examine higher interactions with all ten variables. Following examination of models with up to 5 way interactions, the highest order terms retained were third way. It was thus reasonable to infer that no higher terms were necessary.

The backward stepwise model selection procedure was performed on the resulting 30 models of four variables each. The backward stepwise procedure begins with the fitted model containing all the variables and deletes terms until the simplest model is found that fits the data. Each term is assessed by a conditional test of the parameter. By using the stepwise process, it permits reexamination, at every step, of the variables that were deleted in previous steps, thus permitting analysis of possible interactions between variables.

At this point, the models were examined as logit models (a subset of log-linear models). Logit models can be directly transformed from log-linear models because the three dimensional marginal totals from the tables are fixed and correspond to the explanatory variables. Logit models are the categorical response analog to regression models for continuous response variables [Fienberg, 1980]. The general formula for the logit model with three explanatory variables is:

$$\text{logit}_{ijk} = \omega + \omega_{1(i)} + \omega_{2(j)} + \omega_{3(k)} + \omega_{12(ij)} + \omega_{23(jk)} + \omega_{13(ik)} + \omega_{123(ijk)}$$

where ω notation is used when the variable of prime interest is the presence or absence of a response and the other variables are the presence or absence of various stimuli [Fienberg, 1980]. Logit (log-odds) models look at how the explanatory variables affect the response variable and not the relationships between the variables. Each term in the (linear) logit model expresses the influence of specific explanatory variables on the response (RABPOS); that is,

$$\omega_{13(ik)}$$

is the effect of variables 1 (at level 0) and 3 (at level k). This makes interpretation of a complex set of variables easier and more clear. Fourteen models required second order effects. In the rest, only first order (main) effects were required. Because location had no effect in most of the resulting models,

this variable was dropped, leaving 17 models to explain the data. Parameter estimates were calculated for these models to give estimates of the variables' magnitude and correlation with RABPOS.

VALIDATION

Because log-linear models are a relatively new statistic compared to other procedures, there is no cross-validation statistic such as R squared. Therefore, in an attempt to test the validity of these final models, they were applied to data covering the period from January 1, 1988 through July 31, 1989. Because the fourth season was missing for 1989, fixed zeros were placed into the analysis, so that the zeros in the cell were due to an actual zero probability for that cell. The resulting p values tested the fit. Parameter estimates were calculated to determine the similarity of patterns between the 1988-1989 data and the original data. The backward stepwise selection process was performed to examine how the resulting models for the 1988-1989 data would match the original models. As a final step, the data was combined for 1984-1989 and the same procedures used on the 1988-1989 data were performed.

RESULTS

The geographical distribution of laboratory confirmed rabies cases in all species submitted from January 1, 1984 through December 31, 1987 in Virginia can be seen in Figures 5 through 8. As is evident in these maps, rabies continued to be a major problem in the northern part of the state, and by 1987, rabies became more of a problem as far as numbers of cases (in comparison to previous years) in the central part of the state, with an extension into the coastal counties of Virginia. Throughout the years 1984 through 1987, there remained a small focus of rabies cases in the southwest counties of Virginia. These cases included primarily rabid skunks and other species as opposed to rabid raccoons. In Figures 9 through 12, the geographical distribution of reported raccoon rabies cases in Virginia can be seen for the same years. Since the rabies epidemic in Virginia consists primarily of rabid raccoons, the maps indicating the distribution of total rabies cases mirror the distribution of raccoon rabies cases. The only difference was the rabid skunk cases in the southwest counties of Virginia, which has been part of an endemic focus since the 1960's. In comparing the maps of 1987 for total rabies cases and rabid raccoons, there are a few counties with a higher number of total rabies cases than there were number of rabid raccoons reported. This could be attributed to a number of "spill-over" rabies cases in other species, and the possibility of rabies in bats, a species which could be found in any of the geographic regions of the state.

Monthly trends in the number of raccoons positive and negative for rabies from January 1, 1984 through December 31, 1988 can be found in Figures 13 through 17. For the years 1984 through 1986, there was an elevated number of raccoons negative for rabies (and thus, in the number of submissions) during the summer months, with a secondary peak in the fall months (i.e., October and

November). The largest number of rabid raccoons were reported in February or March in all five years. A secondary rise in number of rabid raccoons occurred in the early fall (i.e., August through October). In every year but 1986, the lowest number of rabid raccoons were found in June or July. In 1986, the lowest number of rabid raccoons was found in May. Fewer numbers of rabid raccoons were also reported in November and December in each year. Yearly trends for the same information can be found in Figure 18. The yearly trends show a high number of raccoons submitted and testing negative for rabies in 1984, with a decrease in numbers of raccoons submitted and testing negative in 1985 and 1986. Another increase in submissions and raccoon testing negative for rabies occurred in 1987 and 1988. In 1985, the lowest number of rabid raccoons were reported. The numbers of rabid raccoons increased in years 1986 and 1987. The number of rabid raccoons reported in 1988 decreased slightly in comparison to the number of rabid raccoons reported in 1987.

There were 2005 raccoons submitted during the study period listed above. Of these, 672 (34.1%) were positive for rabies. Frequencies of the other variables are found in Table 2. Of all the raccoons submitted :

- 1) 74.6% had no human exposure; 22.7% exposed a human
- 2) 19% were viewed as behaving aggressively; 31.4% were viewed as being friendly; 10.4% exhibited neurological signs (wobbily or paralyzed); 21.9% were viewed as acting normally; 11.5% were viewed as acting sick; and 6.0% were viewed as being indifferent or unresponsive.
- 3) 66.3% were found in residential areas; 2.6% were found in commercial or industrial locations; and 31% were found in such

locations as agricultural lands, forest, recreational areas, unimproved lands, and near water (Iowa)

- 4) 71.4% were killed; 16.5% were found dead; less than 1% were found dead by the road; and 5% were trapped
- 5) 32.5% did not bite another animal; 28.9% did bite another animal; and 38.7% of the data was missing for this category
- 6) 22.5% were juveniles (less than one year of age); 52% were adult; and 25% of the data was missing for this category
- 7) 14.9% were female; 20% were male; and 65% of the data was missing for this category
- 8) 8.3% were found at dawn; 54.2% were found during daylight hours; 9.7% were found at dusk; 13.5% were found at night; and 14.4% of the data was missing for this category
- 9) 23% were located in the central health region; 10.9% were found in the eastern health region; 36.2% were found in the northwest health region; 21.4% were located in the southwest health region; and 8.63% were found in the northern health region
- 10) 24.8% were reported in 1984; 15.5% were reported in 1985; 17.7% were reported in 1986; and 40.2% were reported in 1987
- 11) 16% were reported in the months of December through February; 30.3% were reported in the months of March through May; 26.8% were reported through the months of June through August; and 23.4% were reported through the months of September through November.

Positivity ratios for the explanatory variables are found in Table 3.

Model fitting

There were 17 final subset logit models that fit the data. The general

formula for this log-linear model including all of the variables is: (abbreviations defined below)

$$\text{BHXS} + \text{AHYS} + \text{BHYS} + \text{BCYS} + \text{ABYS} + \text{BCAS} + \text{ACYS} + \text{AHZS} + \text{ACXS} + \text{AHXS} + \text{BHAS} + \text{BCXY} + \text{BHXY} + \text{ACXY} + \text{BCAY} + \text{ACHZ} + \text{ABXY} + \text{RXS} + \text{RYS} + \text{RAS} + \text{RB} + \text{RH} + \text{RY} + \text{RS} + \text{RC} + \text{RA} + \text{RZ} + \text{RX}.$$

The general formula for this model in logit form is:

$$(\text{ABCHSXYZ}) \text{ AS,YS,XS,B,H,Y,S,C,A,Z,X}.$$

The logit approach expresses the model as the effects of the variables upon RABPOS, and reduces the complexity and difficulty of interpretation.

A simpler approach to examining the models is to look at each four term model individually. The abbreviations for the variables are as follows:

A= age of the raccoon

B= whether the raccoon bit another animal

C= method of capture

H= whether the raccoon exposed a human

S= season

X= behavior of the raccoon

Y= year

Z= health region in Virginia

To assist the reader, the following is a description of the first model

presented below. In the first model, (BHXS), B,H,XS, the four letters in the parentheses represent the four variables placed in the model; "bited", "human exposure", "behavior", and "season". The letters after the parentheses represent the terms (or variables) in the model that fit the data and that have an important effect on the response variable, RABPOS(positivity for rabies). When the variables BHXS are used in fitting the model to the data, the terms , B,H, and XS resulted. The variables "bited" and "human exposure" have a main effect on the positivity for rabies in raccoons (RABPOS). The term, XS, represents a second order effect of the variables, "behavior" and "season" together, on the positivity of rabies in raccoons. The other models listed can be interpreted in a similar manner. Any p value above 0.01 represents a fit to the data. The p values for the likelihood-ratio-chi-square test for each model are listed below.

	<u>p value</u>
1) (BHXS) B,H,XS	(0.09)
2) (AHYS) A,H,Y,S	(0.01)
3) (BHYS) B,H,S	(0.13)
4) (BCYS) B,C,Y,S	(0.01)
5) (ABYS) AS,B,YS	(0.02)
6) (BCAS) B,A,S,C	(0.03)
7) (ACYS) AS,C	(0.12)
8) (AHZS) A,H,Z,S	(0.07)
9) (ACXS) AS,X,C	(0.02)
10) (AHXS) AS,X,H	(0.03)
11) (BHAS) B,H,S,A	(0.09)
12) (BCXY) B,X,Y	(0.03)
13) (BHXY) B,H,X,Y	(0.07)
14) (ACXY) A,C,X,Y	(0.07)
15) (BCAY) B,A,Y	(0.02)
16) (ACHZ) A,H,Z	(0.55)
17) (ABXY) A,B,X	(0.28)

Most of the models contain only main order effects. Only three second order effects are present; behavior/season, age/season, and year/season.

Parameter estimates for each term in each of the 17 final subset logit models were calculated (see Tables 4 and 5). This procedure involved calculation of both a numerical value and a positive or negative sign to approximate the magnitude and association of each explanatory variable to the response variable, RABPOS. The values for all of these explanatory variables were strong (above 2.0). The signs of these values allowed interpretation of the relationship of the explanatory variable to RABPOS. A negative sign indicated an inverse relationship between the explanatory variable and the response variable, and a positive sign indicated a direct relationship. These sign patterns were consistent for each explanatory variable in all 17 subset models. This is demonstrated by examples of two different variables, "age" and "human exposure", in Table 6.

The main order effects calculated by the models for the data were:

- 1) Raccoons were more likely to be rabid if there was no human exposure.
- 2) Adult (more than one year of age) raccoons were more likely to be rabid.
- 3) Raccoons which bit another animal were more likely to be rabid.
- 4) Raccoons that were killed or found dead were more likely to be rabid.
- 5) Aggressive raccoons were more likely to be rabid.
- 6) Raccoons were more likely to be rabid during the years 1986 and 1987 than in the years 1984 and 1985.

- 7) Raccoons were more likely to be rabid in seasons one and two (December through May).
- 8) Raccoons were most likely to be rabid in every region except region 4 (southwest).

Second order effects that were demonstrated by the models were:

- 1) Juvenile raccoons were more likely to be positive in seasons one and two (December through May). Adult raccoons were more likely to be positive in seasons three and four (June through November).
- 2) Aggressive raccoons were more likely to be found in seasons three and four.
- 3) There was no consistent pattern between the variables season and year.

When these 17 subset logit models were fitted to the 1988-1989 data, all but three of the models had p values that increased, indicating a better fit (see Table 7). These three models (AHZS, ACYS, and ABYS) still fit at the 0.01 p level. When these same models were fitted to the combined data, 1984-1989, all but six models had equal or higher p values (see Table 7). Three models had slightly lower p values. Two models had marginal fits at the 0.01 p level, and the model BHYS did not fit at the 0.01 p level. Parameter estimates were calculated on these models for the 1988-1989 (see Table 8) and the 1984-1989 (see Table 10) data to determine if the magnitude and association patterns for each variable were consistent, or how they may have changed. Most of the main effects were the same; this was true for variables "age", "bited", "human exposure", "captured", and "behavior". Rabid raccoons were more likely to be found in the year 1988 but

not the year 1989. Rabid raccoons were more likely to be found in season two (March to May). Region three (northwest) was most likely to contain rabid raccoons. The only second order effect that was evident was age/season (see Table 9). Juvenile raccoons were more likely to be rabid during the seasons one and two (December to May) and adults were more likely to be rabid during seasons three and four (June through November).

When the data were combined (1984-1989) the majority of variables exhibited the same pattern. These included "age", "bited", "human exposure", "captured", "behavior", and "region". Seasons one and two were most likely to contain rabid raccoons. Rabid raccoons were most likely to be found in the time period between 1987-1989 than between 1984-1986. The second order effect that remained consistent was age and season (see Table 11).

When the backward stepwise selection process was calculated for the 1988 and 1989 data, all the resulting models became simpler; one or more variables were deleted in comparison to the resultant model. Only two variables, "bited" and "region" remained in every model. The explanatory variables "year" and "season" were deleted from each model. "Behavior" dropped out of all the final models, except one (BCXY). The second order terms were either deleted or were broken down into simpler main order terms. These models and the resultant p values can be seen in Table 12.

The backward stepwise selection process was calculated for the combined data for the years 1984 through 1989. In three models (BHXS, BHYS, BHXY), a new second order term developed (BH). In the majority of the remaining models, one or more variables were deleted from the final model. The variable, "season",

was deleted as a main order effect, but became a second order effect with "year" in several of the models. The resultant models and the p values can be found in Table 12. Conditional tests were run on each variable in each model to determine its significance. The general formula for the conditional test is:

$$G^2 = \frac{G_{(2)}^2 - G_{(1)}^2}{df_2 - df_1}$$

The results supported the findings of the modeling process involving eight final important variables. All eight variables were highly significant (p value less than 0.005, see Table 13), and consequently, could not be eliminated from any of the models.

DISCUSSION

The resulting model produced in this study for determining the risk factors affecting the positivity of rabies in raccoons was a combination of 17 four variable term subset models. Because the simultaneous examination of eleven explanatory variables would create an infinite combination of models, the alternative was to create a more manageable model which examined a few key variables, thus resulting in the 17 four variable term subset models instead of one large model containing all of the final variables. Although analysis using 17 models appears to be more complex an undertaking than just one model, the nature of the data is such that this is the only approach to adequately evaluate the data, and there are several results that validate their ability to describe the data. First of all, the parameter estimates have similar patterns (in sign and numerical value) in all 17 models (as seen in Table 6). Secondly, when these resulting models were applied to the existing 1988-1989 data, all of these models fit at the 0.01 p level. The 0.01 p level was chosen to make the model fitting process more conservative in order to eliminate any spurious associations. Finally, when the backward stepwise selection process was performed on the 1988-1989 data, no new terms resulted and similar models were calculated.

This study has fulfilled the objectives outlined in the introduction. Knowledge about the epidemiology of rabies in raccoons in Virginia was gained by determining the risk factors affecting the positivity of rabies in raccoons. For example, the fact that raccoons are more likely to be rabid when there is no human exposure, explains something about raccoon behavior as well as human behavior, yet raises several questions given the studies (as reported in the Literature Review Section) that describe the potential for human exposure to

rabid raccoons. Unlike rabid skunks or foxes, rabid raccoons are less likely to approach humans, thus, humans are less likely to come into contact with rabid raccoons. In addition, rabid raccoons may become more isolated from the rest of the raccoon population and their normal habitat. An explanation may be that the rabies endemic foci exist primarily in rural raccoons rather than in urban dwelling raccoons. If this is true, is it because there is more interaction between and food searching by rural raccoons or that they may be less healthy? McClintock [1981] reports that urban raccoons do little foraging for food and travel shorter distances than rural dwelling raccoons. Whatever the reasons, this knowledge is valuable for public health programs, since humans are less likely to be in contact with a rabid raccoon. According to the parameter estimates of the model, their pets are more likely to be bitten by rabid animals, making immunization of their pets vital to prevent the potential for rabies exposure from wildlife to pets to humans.

Another important point is that trapped raccoons are less likely to be rabid than those found dead or killed. This information provides a good argument against trapping raccoons as a method of eliminating rabies in an area affected by a rabies outbreak. It also provides additional information about the behavior of a rabid raccoon. Rabid raccoons may become indifferent to new situations, human populations, and trapping devices, and less attracted to bait due to loss of appetite or other changes in behavior due to the disease. This behavior would reinforce the fact that there is less human exposure. The fact that much of the trapping is done in urban settings may also support the theory that rabies is more common in rural raccoons. There is no information about those that are hunted for sport. The category "killed " under the variable "behavior" includes those animals killed by dogs, citizens, or animal control officers, and loses some of its potential usefulness in this analysis.

Risk factors were determined that can be used to predict positivity of rabies in raccoons. The major risk factors for predictability are age of the raccoon, whether the raccoon bit another animal, the behavior of the raccoon, and the season in which the animal was found. Although these factors are the best predictors, other variables such as "region", "year", and "human exposure" are important influences on whether the animal was rabid. Because of the influence of these variables, probability values that are calculated on only four variable models for predictability must be cautiously evaluated.

The distribution and spread of rabies in raccoons in Virginia occurred in an eastern direction within the state (see Figures 9 through 12). One concern regarding this pattern of spread is that eastern Virginia (the "Tidewater Area"), is an excellent habitat for raccoons. Once established, this rabies focus could pose a potential for raccoon rabies to spread down the coast to North Carolina. The southwest region of Virginia has remained relatively free from raccoon rabies. There may be several explanations for this. One may be that the area is unable to support a significant raccoon population. According to data from the Virginia Department of Games and Inland Fisheries, this area had the lowest raccoon concentration prior to the rabies outbreak in Virginia, and is the area most overhunted. It may also be due to the influence of the physiographic provinces in Virginia [studied by Hoffman, 1969]. While rabies in foxes has been known to travel along the ridges and valleys of the Blue Ridge mountains, rabies in raccoons does not seem to cross over to the Appalachian plateau (i.e., southwest Virginia). However, the disease has crossed over into the other physiographic provinces in Virginia. It has also continued to spread geographically throughout the entire mid-Atlantic region. In 1989, New Jersey reported its first rabies case

in raccoons [Sorhage, 1989]. Additional studies should be conducted to determine which factors could minimize or prevent the spread of raccoon rabies to southwest Virginia counties and to other extreme southern counties, and the potential spread to other states.

The monthly trends (seen in Figures 13 through 16) for the years 1984 through 1987 demonstrate a consistent peak in rabies cases in the months of February and March. This is consistent with other studies that have demonstrated seasonal rabies patterns, usually related to the breeding season. There is also a consistent trend in these same years with the lowest number of raccoons positive for rabies in mid-summer and in October through December. The low numbers of raccoons positive for rabies in the months of November and December are probably related to the ethology of this species (i.e, less activity in the winter months) resulting in a decreased likelihood of contact with positive animals. The low numbers of raccoons positive for rabies during the summer months are more difficult to explain. Berner and Gysel [1967] report that there is lower overall activity in the raccoons during the summer months since food is plentiful and the weather is mild. This could result in less traveling and less contact rates. There may be a lower overall number of susceptible raccoons, especially if a high number of rabies cases occur earlier in the year removing these animals through death from the population. The most likely explanation may be a long incubation period of rabies after contact with a rabid animal. For example, a raccoon may be infected in March or April, and not show clinical signs until October or longer. The highest number of submissions of rabies suspects are in the mid-summer months. This may be attributed to the fact that many people are more active outdoors during these months, and thus are more likely to come in contact with wildlife.

The presence of a higher number of rabid raccoons in 1986 and especially in 1987, as compared to 1984 and 1985 may signal the beginning of a cyclic pattern of rabies in raccoons. Care must be taken when evaluating increases in

rabid animals because of the difference in numbers of animals submitted. If a large number of animals are submitted, the chances of a rabid animal being submitted are probably higher, thus, resulting in higher numbers of rabid animals reported. However, in the year 1986, there seems to be a "true" increase in rabid animals, since both 1985 and 1986 had the same number of animals submitted, with 1986 reporting a 50% increase in rabid animals. Previously, a cyclic pattern of three years in fox rabies has been described [Toma and Andral, 1977; Tinline, 1988; Voight et al., 1985]. The peak year for raccoon rabies in Virginia was 1983. Further study of the years after the peak might demonstrate the existence or absence of a cyclic phenomenon. The difficulty will be to evaluate this on a statewide basis while the epidemic is still spreading.

The "odds" positivity ratios (see Table 3) are useful in that they support conclusions made about effects determined by the final model. For example, the positivity "odds" ratios for human exposure are: no exposure 603/893 (.68), and exposure 60/395 (.15). For the variable "behavior", the "odds" ratio for a raccoon biting another animal is 296/281 (1.05), and not biting another animal 152/500 (.30). Under the variable, "region" the northwest region has a positivity ratio of 232/197 (1.18) as compared to the southwest region with 5/168 (.03). These ratios are consistent with the data which demonstrate that rabid raccoons are less likely to have human exposure, more likely to bite other animals, and to be located in the northwest region of Virginia. Any number over 1.0 demonstrates a strong relationship. "Odds" ratios should be used to examine categories to see if they were collapsed correctly, and to determine if any important information was lost. Some caution is warranted in examining "odds" ratios. The actual number of cases may be a more reliable number to study, since the "odds" ratios will vary according to the number of submissions [thus, affecting the number of negatives (denominator)]. The "odds" ratio is extremely dependent on fluctuations in the

denominator.

The only second order effect that was reliable, despite the presence of two other second order effects in the models, was the interaction of age and season on positivity for rabies in raccoons. This effect and its parameter estimates remained consistent for all the models as well as for the fitting of the models using the 1988 through 1989 data. Juveniles were more likely to be rabid during the months December through May at a time when they were becoming old enough to disperse into and have greater contact with the main raccoon population. Adults were more likely to be rabid during the months of June through November. This may be due to the increased interaction in the fall months when raccoons start establishing permanent denning sites, and there is a dispersal of young adult raccoons. This may also be due to young raccoons becoming infected, incubating the virus for a long period of time, and not displaying clinical signs until they become an adult.

An important, yet inconsistent, pattern of the second order effect of year and season is understandable in light of epidemiologic patterns. The epidemiology of rabies may vary from year to year, altering its effect in the model. The same is true of season. Seasonal and temporal trends are common in rabies. As the number of years studied increases, (i.e., 1984-1989 models) "year" emerges more often in the models. In contrast, in the model process of 1988 through 1989, "year" became less important.

Finally, the second order effect of behavior and season is consistent with biological factors, because climate and breeding seasons often affect the behavior of the raccoons. There is no clear explanation as to the emergence of BH ("bited" and "human exposure") as a new second order effect in the 1984-1989 models. It is suspected that as additional data is collected, and the

modeling process continues, more second order effects may become apparent. In addition, this interaction may be possible because if a raccoon bites a pet animal, humans are much more likely to be concerned about exposure or be exposed.

Although the original rabies submission form was designed to gain as much detailed information about each animal submitted for testing to help define and evaluate the rabies epidemic in Virginia, the consistency and reliability of the entries on the forms varied with the background and training of the sanitarians, animal control officers, and others who submitted the forms. This variation was partly due to the design of the questions and the format of the submission form. It was only when statistical analysis was performed on this data that some of these problems became apparent. In evaluating the data, those animals reported as negative were purposefully labelled as controls. It was recognized that they were not true controls in the sense that they were not sampled randomly and matched with the positive rabies cases. However, with regards to a wildlife population in which the denominators are unknown, (i.e. size, age, sex, and number of susceptible and infected animals) it was fair to assume that the negatives in this study were reliable controls.

One of the biggest problems in evaluating the data was the number of categories within each variable. This posed two specific problems in analysis of the data. First, as more variables were combined in the models, sparse cells emerged. Those cells with less than 5 observations caused a poor fit of the model to the data. Any model that had more than 150 cells potentially led to sparse cells and poorly fitting models. This can be a frequent problem when there are several variables containing a large number of categories. The second problem, specifically involving the variables location and behavior, involved duplicate counts (more than one category within each variable had been marked). When this had occurred, the same animal would be counted two, three, or even more times by the computer.

In an attempt to correct these two problems, two procedures were performed. Multiple categories for individual variables were collapsed whenever possible. The collapse of the category "months" into a new category "seasons" did not cause a loss of information nor did it introduce bias. Several of the variables associated with rabies exhibited seasonal trends or patterns. Counties and cities recorded as locations of raccoon submissions were collapsed into five categories representing the five administrative regions of the Virginia Department of Health. This was thought to be the best option since collapsing the counties into the 37 health districts within these regions would also create too many categories. Collapsing the data was not without problems as it could lead to loss of potentially important information about focal rabies areas. "Region" of submission only appeared in two models, and was not found to interact with the variable "year". It was known that the counties differed as to the status and period of the rabies epidemic in their area and also as to the number of animals they had submitted. Some areas had submitted a larger number of animals even though the clinical signs of the animal were not consistent with the animal having rabies, whereas other areas had submitted only those animals which had exposure or contact with humans or pet animals. Urban areas were more likely to submit animals for testing, and were more likely to maintain an interest in adequately filling out submission forms, whereas rural areas submitted fewer animals and were often less thorough in recording entries on the submission forms. One method to examine this bias would be to perform a study on a specific area, analyzing rabies over time. One excellent area to study would be Loudoun and Fairfax counties, since they have submitted a large number of raccoons over the years, and rabies has remained persistent in their region for several years.

The other two variables necessitating collapsing of categories were "location" and "behavior". The eleven location categories were collapsed into just three: residential, nonresidential, and iowa (which was a term intended to represent agricultural/recreational/forest areas). Collapsing the "location" category may have caused some information to be lost, and in turn, caused

"location" to drop out of the model as an insignificant variable in its relationship to RABPOS. However, "location" may have dropped out because raccoons are fairly adaptable as to denning sites, and may be found in a wide variety of areas. Raccoons especially thrive around human dwellings. Chimneys, barns, and other buildings make good denning sites, and the human propensity to feed raccoons adds to the benefits of living in close proximity to humans. If free food is not available, raccoons exhibit great prowess in raiding trash cans. In contrast, skunks are not normally found around human dwellings and typically would be found there only if they were rabid. To reinforce the assertion that these two species differ in their "willingness" to live near humans, the raccoon model involving "location" would need to be fitted to the skunk data to see if "location" as a variable remained significant in the model. Skunks do live in human dwellings, but they are usually found in rural areas.

"Location" categories were collapsed in such a way that similar categories were combined. Categories left in the analysis were those locations most likely to represent potential for human or pet animal exposure, or those locations that rabid animals would inhabit. This same procedure was also used in collapsing the categories within the "behavior" variable into four categories: normal, sick, aggressive, and friendly. This process of collapsing likely caused the loss of valuable information about specific behavioral patterns. However, this may have eliminated some bias problems with the response categories listed under the "behavior" variable. Some personnel who submitted these forms had marked several responses under behavior, with some of the responses being incongruent with each other (i.e. marking normal and paralyzed, or normal and sick).

This incongruity in responses leads to another important problem which occurred with the variables "location" and "behavior", namely duplicate counts. In order to eliminate this problem, the categories were prioritized as to those

which were most important in the description of a rabid animal, its behavior, potential location, and those behaviors and locations that would most likely lead to human or animal exposure. Prioritization decisions were made on the assumption that most rabid animals act differently from unaffected animals. Decisions as to which "behavior" category was most valuable would vary according to the species being studied (i.e., aggressiveness is more common in rabid skunks or foxes in comparison with rabid raccoons). Aggressiveness is often a common clinical sign of a rabid animal since the virus attacks the limbic system, which controls behavior.

After these initial decisions involving collapsing categories, the analytical process of determining a model to fit the data was begun. Twelve variables (including RABPOS) prove too large a number to attempt a modeling process, since after four variables, the possible combinations of models describing the data ranges into the millions or higher. Therefore, some initial analyses were performed to determine if any of the variables were insignificant and could thus be dropped from further study. Using the multiway contingency tables and the maximum likelihood ratio tests on possible log-linear models from these contingency tables resulted in 3 variables being dropped: sex of the animal, location where the animal was found, and time of day the animal was found. The fact that "time" dropped out was surprising given the fact that rabid animals, especially skunks and foxes, are more likely to be seen during the daylight, as compared to their usual nocturnal patterns. After conducting 6 way interaction analyses, only 3 second order effects were found, providing reasonable assurance that it was not necessary to continue the arduous and difficult task of forming higher hierarchial contingency tables.

Another problem with the data was that some of the questions on the submission form were open-ended (asked the respondent to fill in information).

Answers to this type of question can not be appropriately analyzed, and may lack potentially beneficial information. For example, respondents may fill in the "other" blank rather than marking a specific category response because they think it might be more helpful in evaluating the data. However, since the information given varied with each respondent, the resulting answers were not counted or coded. Open-ended questions and category responses marked other or unknown were eliminated since they did not yield useful information.

Passive surveillance studies undoubtedly have inherent bias problems because of the nature of their methods. The actual rabies epidemiologic history of the county and the human population may influence the submission rates of animals as well as perhaps the number of rabid animals found. It is estimated that only 1-10% of the rabies cases are actually counted. This makes multiple regression analysis difficult for these studies. Mathematical modeling still must deal with the same bias, but can at least determine the problem and provide a rough estimate of the epidemiologic status in the state. In addition, log-linear models are the only method of analysis of nominal data (yes or no answers) that is represented in contingency tables. Although this method does not enable researchers to determine exact values for predictability, modeling can provide valuable information as to the epidemiology of disease, and areas for future study.

Data submissions in earlier years were less selective than in later years, thus introducing the possibility of another source of bias. Significant publicity which prompted public concern caused an increased submission of animals to be tested for rabies, even though it was unlikely they were infected with rabies. Although more animals were submitted than perhaps should have been, this

process provided more of a representative sampling than is the case with the animals that are being submitted now. In general, the health department's policy is to test only those animals that expose humans or that expose other animals. Therefore, it could be suggested that animal control officers are being more selective in the submission process, and are therefore more likely to submit a rabid animal. However, since 1160 (29% positive) raccoons were submitted during 1988 and the first seven months of 1989, as compared to 2005 (34% positive) raccoon submitted during the years 1984-1987, it is evident that the submission process is not totally selective, and that the sampling of the animals remains fairly representative. It is more likely that animals in rural areas are less likely to be submitted since there are fewer people in a given area with a lower likelihood of exposure, and those who might encounter these species are less concerned about the possibility of rabies.

The analysis of the rabies data derived from the submission form over the years 1984 through 1987 has indicated a need for further surveillance and information as well as improvements in the questionnaire to enhance the reliability and validity of the study. The author recognizes that the submission form and the subsequent collection of data was not for the primary purpose of academic or statistical analysis. The laboratory personnel testing for rabies use the information from the submission form to determine if rabies testing needs to be performed, and what tests should be used (i.e., if the mouse inoculation is needed). Local health departments use the information to determine if follow-up information is needed, to determine the significance of possible human exposure, the potential risk of rabies, and to make decisions about post-exposure prophylaxis. One of the main improvements should be to reduce the number of categories, keeping in mind that the resulting categories should not be applicable solely to raccoons, but should also be descriptive of other species as well, since

rabies causes different behaviors in different species. Under the variable, "behavior", the categories should be reduced to Normal, Unusually aggressive, Wobbily or Paralyzed, and Unresponsive to environment or people. Respondents should be instructed to select only ONE of those categories. This would eliminate duplicate counts, and facilitate consistency of responses. The variable "location" should have the categories reduced to Residential area (building or yard), Business/Industrial area (building or yard), Agricultural (farms, cropland, or livestock areas), Recreational (state parks, local parks, playgrounds), and School yards.

For the variable "human exposure", categories should be created for type of exposure to include: Bite by an animal, Scratch by an animal, Exposure to saliva of animal through mucous membranes or scratch. An additional variable should be included to indicate how many people were exposed (i.e., the categories should be ; One, Two to Five, Five to Ten, and over Ten people). Previous studies have shown that wild animal exposures involve large numbers of people. The variable, time of day, should be reworded, since the terms "dawn" and "dusk" have different meanings for different individuals. To clarify this, the following categories could be used: 6 am -8 am, 8:01 am - 5pm, 5:01 pm -7 pm, and 7:01 pm -5:59 am. Thought must be given to the fact that the time for dawn and dusk will change with the season. Finally, the query as to whether the animal submitted bit any other animals seemed to cause confusion among the respondents. For example, one filled in the blank and talked about a cat holding a bat in its mouth when it was the bat that was submitted. In that case, it was safe to assume that there was at least interaction with another animal, even though there was insufficient information provided to determine whether the other animal had been bitten. It would be better if the question were worded to elicit a yes or no answer about bite wounds to exposed animals, or consider rewording the question just to measure interaction activity with another animal. In addition, all

open ended questions (fill in the blank) and any category that was labeled unknown or other should be eliminated to prevent lack of uniformity or consistency of answers.

Some variables had few recorded responses. It is interesting to note that two of the variables thought to be most important in predicting the positivity of rabies in raccoons, "age" and "bited", are two of the three highest variables with nonresponses. "Age" has a 25% nonresponse rate, and "bited" has a 39% nonresponse rate. Only "sex" of the animal had a higher nonresponse rate (65%) which was probably a factor in it being insignificant in the model process. The importance of these two factors and the high nonresponse rate makes it worthwhile to consider how to improve the response rates and to determine why the nonresponse rate is high. Personnel filling out the submission forms received insufficient training in aging and sexing of wild animals, although charts and explanations were provided. In addition, these two variables were not of primary interest to the animal control officers or health department personnel. The questions regarding these two variables need to be reevaluated. It was not apparent whether this was due to difficulty in measuring, whether the respondents did not know the information, whether the question or form was too long or complicated, or whether the respondents took it upon themselves to determine what was or was not relevant information. Further investigation is needed to evaluate the reasons for inadequate responses and how to correct questions or respondent's behaviors to assure that adequate, appropriate information is gathered. One obvious solution should be to shorten the questionnaire, making the questions less wordy and easier to understand, and by placing the most important questions first. In addition, personnel should be better trained with emphasis as to the importance of the information, how it is being used, and how their contribution will help increase accuracy and reliability of the data, and improve the interpretation of the data. Follow-up telephone calls to

respondents would allow prompt resolution of discrepancies although this would increase the time involved and cost of the monitoring program. Another approach would be to decide which variables are the most important to obtain information about. Nonresponse has the potential to induce bias into the study and may have serious impact on the conclusions to be drawn. In designing the question, one should design a measure, not a conversational inquiry. It must maximize the relationship between the answers recorded and what the researchers are trying to measure.

There was an expectation when this study began that it would yield specific practical information. Although it has enabled a better understanding of the epidemiology of rabies in raccoons in Virginia, the nature of the data at this point does not lend itself to specific predictive values and a practical model for field work. However, it has provided a direction in which to develop methods to enhance this information. In an attempt to go from the full model, which contains a combination of 17 four variable term models, to one "practical" field model, several approaches were taken. First, a decision was made to drop the variables (out of the remaining eight) which were of least practical value to the clinical users. Following re-analysis of the original 17 models, their fit on the 1988-1989 data and the 1984-1989 data, and how many terms were either dropped or remained in the models, the explanatory variable, "region", was dropped from further analysis. The significance and influence of the "region" (or counties) can vary greatly with the epidemiology of rabies, and is thus not a good variable for prediction. Second order terms were also dropped from further analysis. Using the seven remaining variables, models containing five variable terms were constructed. These 21 models were entered in the backward stepwise model selection process, with inclusion of terms that were in the original models. This procedure was performed to increase the complexity of the model to determine if any variables were duplicating effects, with the realization that this process would

be hindered by the presence of sparse cells.

Because this additional analysis did not result in any definite information about variables that could be dropped to facilitate simplification of the models, a decision was made to determine the simplest model that might be of value to public health officials. The models with the highest p values for fitting (ACHZ, ABXY, ACYS) were examined to see if they would be valuable as field models. Consideration was given as to whether the variables were easily measured or more likely to be acknowledged (i.e., recorded by the respondents). As was stated before, the variable "region" varies greatly with the epidemiology of rabies and is not valuable as a predictor. The variable, "human exposure", is also not of much practical value since any animal that exposes a human is automatically tested for rabies, provided that the animal can be captured. Any model containing year was also dropped from further examination, since like the variable "region", it varies during the different stages of the epidemic, different climate situations, and the population dynamics of the raccoons. Therefore, none of the models with the highest p values demonstrate a high potential for use in a field model. The model with the most practical use would be the model that contained the following variables: age of the animal, whether it bit another animal, the behavior of the animal, and the season (ABXS). "Captured" is also a possible valuable variable. These variables were examined using the backward stepwise selection process (ABXS, ABCS, ABCX, and ABCXS). The resulting models for ABCS and ABCX fitting at the 0.01 p level, eliminated the term, C (captured). This supported the decision that the model containing ABXS was the most practical. In addition, the resulting model for ABCXS from the backward stepwise selection model process was the same as the resulting model for ABXS [(ABXS) BX, A, XS], further supporting the decision that ABXS would be the most practical model and the fact that the variable "capture" can be explained by another variable, most likely, "behavior".

Although the variables "year", "region", and "human exposure", are not valuable predictors, each were added separately to the model ABXS to determine their possible effects. When the variable "human exposure" was added to ABXS [ABHXS] the resulting model was the same as the model for ABXS except for the main order term, "human exposure". This demonstrates the importance of the variable and the fact that it is not explained by another variable in this model. When the variable "region" was added [ABZXS], the resulting model gave the same model as ABXS except for the addition of the main order effect, "region", and a new second order effect BS [bited and season]. This demonstrates that the variable "region" is an important variable by itself. When the variable "year" was added [ABYXS], the resulting model had the same terms as the model for ABXS with the addition of two second order terms, AS [age and season] and AY [age and year]. This demonstrates that although "year" is not as important by itself, it does create interactions and changes with other variables. The same is true of "region". In conclusion, "region" and "year" seem to have the most important influences on the other variables. Thus, the decision to analyze a regional study over time is an important one.

There is no simple explanation for the emergence of the second order term, BX [bited and behavior] in all of the models. There are two possible explanations for this interaction. Ecologically, the behavior of the raccoon would affect whether it might bite another animal [i.e., aggressive behavior]. On the other hand, those observers watching the interaction between the raccoon and another animal are more likely to view the raccoon as "aggressive" rather than being territorial or protective of its offspring, and are more likely to have the animal tested.

To determine the value of this particular model for prediction, an estimate of probability was determined on ABXS [see Table 17]. The definition of the probability of an animal being positive is the frequency definition. In other words,

the probability that an event happens on a single trial is the proportion of times it exists in the population. Therefore, the calculation of the estimated probability is the probability of an individual raccoon being positive for rabies given the specific variables, or the proportion of raccoons that are positive that have the same specific variable characteristics. The general formula for calculating these estimated probabilities is:

$$p = \frac{\text{expected frequency of being positive}}{\text{total expected frequency of a characteristic}}$$

In conclusion, this study has aided in the analysis of the epidemiology of rabies in raccoons in Virginia. It has offered useful suggestions regarding the redesign of the submission form to enhance reliable data collection, and future direction of the surveillance of this disease. Through continued surveillance and a more focused approach, the model will become more precise and offer more practical information. In addition, by acquiring additional knowledge regarding the risk factors associated with the positivity of rabies in raccoons, the model can be better refined, and new directions of research can be developed. For example, how can we better understand and measure the behavior of rabid raccoons? One approach would be to conduct a controlled study with infected raccoons and normal raccoons. This would provide more reliable behavioral discriminators which would be incorporated into the behavioral question on the submission form. Conversely, it might indicate that behavior is not a practical variable to try to measure. One advantage of surveillance studies is that they often allow the researcher the ability to "fine-tune" and improve the study to yield more precise and useful information. Finally, this model developed for prediction of positivity for rabies in raccoons, can be used to evaluate rabies data from other

species. Should the model work with the other species, other additional variables could be evaluated to understand how the disease interacts with the various species, the region in question, and time. This in turn, will help in the understanding of how these factors influence the potential for human and domestic animal exposure, provide approaches to protect the public health of the human population, and will influence control measures against the disease.

REFERENCES CITED

- Anderson, R.M., H.C. Jackson, R.M. May, and A.M. Smith. 1981. Population dynamics of fox rabies in Europe. *Nature*. 289:765-771.
- Bacon, P.J., and D.W. MacDonald. 1980. To control rabies: vaccinate foxes. *New Sci.* 87:640-645.
- Bacon, P.J. 1985. Chptr 5, Rabies in nonhematophagous bats. In *Population dynamics of rabies in wildlife*. Academic Press Inc. London. 385 pp.
- Baer, G.M. 1975. The natural history of rabies. vol 1 and 2. Academic Press Inc. New York. 841 pp.
- Baer, G.M. 1988. Oral rabies vaccination: an overview. *Reviews of Infectious Diseases*. 10 (4): 644-648.
- Bailey, N.T.J. 1957. The mathematical theory of epidemics. Charles Griffin and Co., London. 194 pp.
- Bailey, N.T.J. 1967. The simulation of stochastic epidemics in two dimensions. *Proc. Fifth Berkley Symp. Math Statis. and Prob.* , University of California, Berkley and Los Angeles. 4:237-256.
- Bailey, N.T.J. 1975. Mathematical theory of infectious disease and its applications. Hafner Press, N.Y., N.Y. 1002.
- Bartlett, M.S. 1956. Deterministic and stochastic models for recurrent epidemics. *Proc. Third Berkley Symp. Math. Stat. and Prob.* California Press, L.A., Calif. 4:81-109.
- Beck, J.W., 1967. Report on the evaluation of the Virginia rabies control program. Administrative report to the Virginia Dept. Health, Richmond, VA 7 pp. Mimeogr.
- Beck, A.M., S.R. Felser, and L.T. Glickman. 1987. An epizootic of rabies in Maryland, 1982-1984. *Am. J. Pub. Health* 77 (1): 42-44.
- Beran, G.W., 1981. Rabies and infections by rabies-related viruses. In J.H. Steele ed. *CRC handbook series in zoonoses*, section B., volume 2. Crc Press, Inc. Boca Raton, Florida. 57-135pp.

- Berner, A., and L.W. Gysel. 1967. Raccoon use of large tree cavities and ground burrows. *J. of Wildl. Mgt.* 31(4): 706-714.
- Bigler, W.J., R.G. McLean, and H.A. Trevino. 1973. Epizootiologic aspects of raccoon rabies in Florida. *Am. J. of Epidem.* 98 (5):326-335.
- Bigler, W.J., G.L. Hoff, J.S. Smith, R.G. McLean, H.A. Trevino, and J. Ingwersen. 1983. Persistence of rabies antibody in free-ranging raccoons. *J. Inf. Dis.* 148:610.
- Bishop, Y.M., S.E. Fienberg, P.W. Holland. 1975. *Discrete Multivariate Analysis: Theory and Practice.* MIT Press, Cambridge, Mass. 557 pg.
- Blancou, J., 1988. Ecology and epidemiology of fox rabies. *Review of Infectious Diseases.* 10 (4): 606-609.
- BMDP Statistical Software Inc., 1440 Sepulveda Blvd., Los Angeles, Calif. 90025, 1988.
- Bogel, K., H. Moegle, F. Steck, W. Krocza, and L. Andral. 1981. Assessment of fox control in areas of wildlife rabies. *Bull. of WHO.* 59 (2):269-279.
- Burnett, C.D. 1989. Bat rabies in Illinois: 1965-1986. *J. Wildl. Dis.* 25 (1): 10-19.
- Carey, A.B. 1974. An analysis of an apparent rabies epizootic in Rockbridge County, Virginia. Master of science, wildlife management. 168 pp.
- Carey, A.B., and R.G. McLean. 1983. The ecology of rabies: evidence of co-adaptation. *J. Appl. Ecology* 20:777-800.
- Carey, A.B. 1985. Chptr. 2. Multispecies rabies in the eastern United States. In P.J. Bacon ed. *Population dynamics of rabies in wildlife.* Academic Press, Orlando, Fla. 23-39.
- Centers for Disease Control. 1973. Zoonoses surveillance. Annual rabies report. Centers for Disease Control, Atlanta, Ga.
- Centers for Disease Control. 1977. *MMWR* 26:183-184.
- Centers for Disease Control. 1984. *MMWR* 33(14): 185-187

- Charlton, K.M. 1988. The pathogenesis of rabies. Chapter 5 in J.B. Campbell and K.M. Charlton eds. *Developments of virology: rabies*, volume 7. Kluwer Academic Publishing. Norwell. 431 pp.
- Constantine, D.G. 1862. Rabies transmission by a non-bite route. *Public Health Rep.* 77 (4):287-289.
- Correa-Giron, E.P., A. Rae, and S.E. Sulken. 1970. The infectivity and pathogenesis of rabies virus administered orally. *Am. J. of Epidem.* 91 (2):203-215.
- Coyne, M.J., G. Smith, and F.E. McAllister. 1989. Mathematic model for the population biology of rabies in raccoons in the mid-Atlantic states. *Am. J. Vet. Res.* 50 (12):2148-2154.
- Currier, R.W. 1972. Epidemic Intelligence Service Conference. Centers for Disease Control, Atlanta, Georgia.
- Dean, D.J., and M.C. Abelseth, 1973. The fluorescent antibody test. In M.M. Kaplan and H. Koprowski eds. *Laboratory techniques in rabies*. 3rd edition. Geneva, World Health Organization. 179-89.
- Dietzschold B., E.E. Rupprecht, M. Tollis, M. Lafon, J. Mattel, T.J. Wiktor, and H. Koprowski. 1988. Antigenic diversity of the glycoprotein and nucleocapsid proteins of rabies and rabies-related viruses: implications for epidemiology and control of rabies. *Reviews of Infectious Diseases* 10 (4):785-798.
- Eng, T.R., T.A. Hamaker, J.A. Dobbins, T.C. Tong, J.H. Bryson, and P.F. Pinsky. 1989. Rabies surveillance, U.S., 1988. *MMWR.* 38(1):1-19.
- Fienberg, S.E. 1980. *The analysis of cross-classified categorical data.* MIT Press, Cambridge, Mass.
- Fischman, H.R., and F.E. Ward. 1968. *Am. J. Epidem.* 88:132-138.
- Fishbein, D.B., J.G. Dobbins, J.H. Bryson, P.F. Pinsky, and J.S. Smith. 1988. Rabies surveillance, U.S., 1987 *MMWR.* 37 (4):1-17.

- Garnerin Ph., S. Hazout, and A.J. Valleron. 1986. Estimation of two epidemiological parameters of fox rabies: the length of incubation period and the dispersion distance of cubs. *Ecolog. Modelling*. 33:123-135.
- Giles, R.H. Rabies control trapping program, 1954-1968. Dept. of Forestry and Wildlife, Virginia Polytechnic Institute and State University, Blacksburg, Va. 22 pp. Multilith.
- Greenberg, R.S. and D.G. Kleinbaum. 1985. Mathematical modeling strategies for the analysis of epidemiologic research. *Ann. Rev. Public Health*. 6:223-245.
- Helmick, C.G. 1983. The epidemiology of human rabies postexposure prophylaxis, 1980-1981. *JAMA* 250 (15): 1990- 1996.
- Hiorns, R.W. and D. Cooke (ed.). 1981. The mathematical theory of the dynamics of biological populations II. Academic Press, N.Y., N.Y. 1003.
- Hoffman, C.D. and J.L. Gottschang. 1977. Numbers, distribution, and movements of a raccoon population in a suburban residential community. *J. Mamm.* 58 (4):623-636.
- Hoffman, R.L. 1969. The biotic regions of Virginia. In *The insects of Virginia*. No.1 Research Div. Bull. 48, Virginia Polytechnic Institute and State University, Blacksburg, Virginia. 23-32.
- Hubbard, D.R. 1985. A descriptive epidemiologic study of raccoon rabies in a rural environment. *J. Wildl. Dis.* 21:105.
- Jenkins, S.R. and W.G. Winkler. 1984. Mid-Atlantic states raccoon rabies outbreak. Public Health Service, CDC, Atlanta. *Epi* 83-6-2.
- Jenkins, S.R. and W.G. Winkler. 1987. Descriptive epidemiology from an epizootic of raccoon rabies in the middle Atlantic states, 1982-1983. *Am. J. of Epidem.* 126 (3):429-437.

- Jenkins, S.R., B.D. Perry, and W.G. Winkler. 1988. Ecology and epidemiology of raccoon rabies. *Reviews of Infectious Diseases*. 10 (4):620-625.
- Jenkins, S.R. and W.G. Winkler. 1989. Raccoon rabies. In *The Natural History of Rabies*. CRC Press, Inc., Boca Roton, Fla., in press.
- Johnston, D., and M. Beauregard. 1969. Rabies epidemic in Ontario. *Bull. Wild. Dis. Ass.* 5:357-370.
- Kallen, A., P. Arcuri, and J.D. Murray. 1985. A simple model for the spatial spread and control of rabies. *J. Theor. Biol.* 116:377-393.
- Kaplan, C. 1985. Chptr. 1. Rabies: a worldwide disease. In P.J. Bacon ed. *Population dynamics of rabies in wildlife*. Academic Press, Orlando, Fla. 1-20.
- Kappus, K.D., W.J. Bigler, R.G. McLean, and H.A. Trevino. 1970. The raccoon as an emerging rabies host. *J. Wildl. Dis.* 6:507-9.
- Kappus, K.D. 1976. Canine rabies in the U.S., 1971-1973: a study of reported cases with reference to vaccination history. *Am. J. Epidem.* 103 (2):242-249.
- Kissling, R.E. 1975. The fluorescent antibody test in rabies. Chapter 20 in G.M. Baer ed. *The natural history of rabies*, vol. 1. Academic Press. New York.
- Koprowski, H. 1973. The mouse inoculation test. In M.M. Kaplan and H. Koprowski eds. *Laboratory techniques in rabies*. 3rd edition. Geneva, World Health Organization. 85-93.
- Lemeshaw, S. and D.W. Hosmer. 1982. A review of goodness of fit statistics for use in the development of logistic regression model. *Am. J. Epidem.* 115 (1):92-106.
- Lepine, P. 1973. Histopathological diagnosis. In M.M. Kaplan and H. Koprowski eds. *Laboratory techniques in rabies*. 3rd edition. Geneva, World Health Organization. 56-71.

- Lewis, J.C. 1975. Control of rabies among terrestrial wildlife by population reduction. Chapter 15 in G.M. Baer ed. The natural history of rabies, vol. 2 Academic Press. New York.
- Linebeck, N.G. 1980. A model of rabies diffusion. *Southeastern Geographer*. 20 (1):1-5.
- MacClintock, D. 1981. A natural history of raccoons. Charles Scribner's Sons, New York, N.Y.
- MacDonald, D.W. 1980. Rabies and wildlife: a biologist's perspective. Oxford Univ. Press. Oxford.
- MacDonald, D.W. and P.J. Bacon. 1982. Fox society, contact rate, and rabies epizootiology. *Comp. Immun. Microbiol. Infect. Dis.* 1 (1-3):247-256.
- MacDonald, D.W., and D.R. Voight. 1985. Chptr. 4. The biological basis of rabies models. In P.J. Bacon ed. Population dynamics of rabies in wildlife. Academic Press, Orlando, Fla. 71-103.
- MacInnes, C.D. 1988. Chptr. 17. Control of wildlife rabies: the Americas. In J.B. Campbell and K.M. Charlton eds. Rabies: developments in veterinary virology. Kluwer Academic Publishers, Boston, Mass. 381-347.
- Mausner, J.S., and S. Kramer. 1985. Mausner and Bahn: Epidemiology- an introductory text. W.B. Saunders Co., Philadelphia, Pa. 360 pp.
- McLean, R.G. 1970. Wildlife rabies in the United States: recent history and current concepts. *J. Wildl. Dis.* 6:229-233.
- McLean, R.G. 1971. Rabies in raccoons in the southeastern U.S.. *J. Inf. Dis.* 123:680.
- McLean, R.G. 1975. Raccoon rabies. Chapter 4 in G.M. Baer ed. The natural history of rabies, vol. 2. Academic Press. New York.
- Murphy, F.A. 1988. The pathogenesis of rabies virus infection: an abstract. *Reviews of Infectious Diseases* 10 (4):732.

- Murphy, F.A. 1986. The rabies virus and pathogenesis of the disease. Chpt 2. in D.B. Fishbein, L.A. Sawyer, W.G. Winkler eds. Rabies concepts for medical professionals. Merieux Institute, Inc. Miami, Florida.
- Parker, R.L. 1975. Rabies in skunks. Chapter 3 in G.M. Baer ed. The natural history of rabies, vol. 2. Academic Press. New York.
- Prather, E.C., W.J. Bigler, G.L. Hoff, and J.A. Tomas. 1975. Rabies in Florida: history: status: trends. Div. of Health, State of Fla., Monograph Series #14, Jacksonville, Fla.
- Preston, E. 1973. Computer simulated dynamics of a rabies-controlled fox population. J. Wildli. Mgt. 37 (4):501-512.
- Recommendation of the Immunization Practices Advisory Committee (ACIP). 1981. MMWR 30;535-536.
- 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. Proceedings of the National Academy of Science, U.S.A. 83:7947-7950.
- Rupprecht, C.E., L.T. Glickman, P.A. Spencer, and T.J. Wiktor. 1987. Epidemiology of rabies virus variants: differentiation using monoclonal antibodies and discriminant analysis. Am. J. Epidem. 126:298-309.
- Rupprecht, C.E., 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 (4):803-809.
- SAS Institute Inc., SAS Circle, P.O. Box 8000, Cary, N.C. 27512-8000, 1988.
- Schnurrenberger, P.R., R.S. Martin, G.L. Meerdink, N.J. Rose. 1969. Pub. Health Rep. 84:1078-1084.
- Seidensticker, J., A.J.T. Johnsing, R. Ross, G. Saunders, and M.B. Webb. 1983. Ecological and behavioral considerations for raccoons in relation to the epidemiology of wildlife rabies. Presented at the North American Symposium on Rabies in Wildlife, Baltimore, November.

- Sharp, W.M. and L.H. Sharp. 1956. Nocturnal movements and behavior of wild raccoons at a winter feeding station. *J. Mamm.* 37 (2):170-177.
- Sikes, R.K. 1970. Rabies. Chapter 1 in J.W. Davis, L.H. Karstad, D.O. Trainer, eds. *Infectious diseases in wild mammals*. Iowa State University Press. Ames, Iowa.
- Smith, J.S., J.W. Sumner, L.F. Roumilat, G.M. Baer, and W.G. Winkler. 1984. Antigenic characteristics of isolates associated with a new epizootic of raccoon rabies in the U.S. *J. Inf. Dis.* 149 (5):769-774.
- Smith, J.S., F.L. Reid-Sanden, L.F. Rouimallat, C. Trimarchi, K. Clark, G.M. Baer, and W.G. Winkler. 1986. Demonstration of antigenic variation among rabies virus isolates by using monoclonal antibodies to nucleocapsid proteins. *J. Clin. Microbiol.* 24:573-580.
- Smith, J.S. 1988. Monoclonal antibody studies of rabies in insectivorous bats of the U.S. *Reviews of Infectious Diseases.* 19 (4):637-643.
- Smith, J.S., and G.M. Baer. 1988. Chptr. 12. Epizootiology of rabies: the Americas. In J.B. Campbell and K.M. Charlton eds. *Rabies: developments in veterinary virology*. Kluwar Academic Publishers, Boston, Mass. 267-301.
- Sorhage, F. 1989. New Jersey Health Department. (personal communication).
- Steele, J.H. 1988. Rabies in the Americas and remarks on global aspects. *In* *Reviews of Infectious Diseases.* 10(4):585-598.
- Tinline, R.R., D.R. Voigt, L.H. Broekhoven. 1982. Third International Symposium in Vet. Epidmiology and Economics. 581-589.
- Tinline, R.R. 1988. Chptr. 13. Persistence of rabies in wildlife. In J.B. Campbell and K.M. Charlton eds. *Rabies: developments in veterinary virology*. Kluwar Academic Publishers, Boston, Mass. 301-323.
- Toma, B. and L. Andral. 1977. Epidemiology of fox rabies. *Adv. Virus Res.* 21:1-36.

- Tordo, N. and O. Poch. 1988. Structure of rabies virus. Chapter 2 in J.B. Campbell and K.M. Charlton eds. *Developments of virology: rabies: volume 7*. Kluwar Academic Publishing. Norwell.
- Venters, H.D., D.R. Heffert, J.E. Scatterday, and A.V. Hardy. 1954. Rabies in bats in Florida. *Am. J. of Public Health* 44:182-185.
- Voight, D.R., R.R. Tinline, and L.H. Broekhoven. 1985. Chpt 13 in *Population dynamics of rabies in wildlife*. Academic Press, London, pp. 311-349.
- Wandeler, A., 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. *Zbl. Vet. Med. B.* 21:735-760.
- Wandeler, A.I., S. Capt, A. Kappeler, and R. Hauser. 1988. Oral immunization of wildlife against rabies: concept and first field experiments. *Reviews of Infectious Diseases*. 10 (4):649-653.
- Warrell, D.A., and M.J. Warrell. 1988. Human rabies and its prevention: an overview. *Reviews of Infectious Diseases* 10 (4): 720-731.
- Webster, W.A., G.A. Casey, A. Tabel, and A.H. Corner. 1974. Skunk rabies in Ontario. *Can. Vet. J.* 15:163-167.
- Webster, W.A. and G.A. Casey. 1988. Diagnosis of rabies infection. Chapter 9 in J.B. Campbell and K.M. Charlton eds. *Developments of virology: rabies: volume 7*. Kluwar Academic Publishing. Norwell.
- Wiktor, T.J. and H. Koprowski. 1978. Monoclonal antibodies against rabies virus produced by somatic cell hybridization: detection of antigenic variants. *Proc. Natl. Acad. Sci. USA.* 75 (8): 3938-3942.
- Winkler, J.P. 1973. Airborne rabies transmission in a laboratory worker. *JAMA.* 226:1219.
- Winkler, W.G., E.F. Baker, and C.C. Hopkins. 1972. An outbreak of non-bite transmitted rabies in a laboratory animal colony. *Am. J. of Epidem.* 95 (3):267-277.

- Winkler, W.G. 1975. Chptr. 1. Vol 2. Fox rabies. In G.M.Baer ed. The natural history of rabies. Academic Press Inc., London. 3-21.
- Winkler, W.G. 1986. Chptr.3 Current Status of Rabies in the United States. In D.B. Fishbein, L.A. Sawyer, W.G. Winkler eds. Rabies Concepts for Medical Professionals. Merieux Institute, Inc., Miami, Fla. 17-28.
- Winkler, W.G., J.S. Shaddock, C. Bowman. 1985. Rabies virus in salivary glands of raccoons (Procyon lotor). J. Wildli. Dis. 21 (3):297-298.

APPENDIX

**COMMONWEALTH OF VIRGINIA
DEPARTMENT OF GENERAL SERVICES
DIVISION OF CONSOLIDATED LABORATORY SERVICES
BUREAU OF MICROBIOLOGICAL SCIENCE**

RABIES

Lab. No. _____
Date Received: _____
___ Central Lab.
___ Shenandoah Regional
___ Southwest Regional

Kind of Animal: _____ Pet ___ Stray ___ Wild
 Owner (if pet): _____
 Address _____
 County: _____
 Grid Number of Location: _____
 Date Collected: _____
 Date Sent: _____

REPORT TO:

Name _____
 Street _____
 City _____ Zip _____

TELEPHONE NUMBER _____

I. HUMAN EXPOSURE:

A. Was there human exposure to the animal being submitted: YES NO

- Name of person(s) exposed: _____ Phone: _____
- Describe type of exposure (bite, scratch, contact with saliva, etc.), location of bite on the body, and circumstances surrounding exposure: _____

II. ANIMAL HISTORY, EXPOSURE, AND BEHAVIOR:

A. Check beside any of the following which apply to the behavior of the animal being submitted:

1. ___ APPARENTLY NORMAL	4. ___ OVERLY FRIENDLY	6. ___ UNRESPONSIVE TO STIMULI
2. ___ APPEARED SICK	5. ___ INDIFFERENT TO HUMAN EXPOSURE	7. ___ WOBBLY GAIT
3. ___ AGGRESSIVE		8. ___ PARALYZED
9. OTHER _____		

- B. Was animal: KILLED FOUND DEAD, NOT ROAD KILL ROAD KILL CAUGHT IN TRAP
- C. Current rabies vaccination: YES NO NOT APPLICABLE
- D. Animal bitten by a proven or suspected rabid animal: YES NO UNKNOWN
- E. Submitted animal's Age: ADULT JUVENILE Sex: MALE FEMALE
- F. Did the animal bite any other animals: YES NO. If yes, please describe the circumstances surrounding exposure: _____
- G. Time of day the animal was first sighted: DAWN DAYLIGHT DUSK NIGHT
- H. Characteristics of the site where animal was collected:
- | | | |
|--|--|--|
| (1) RESIDENTIAL: <input type="checkbox"/> YARD <input type="checkbox"/> BUILDING | (2) <input type="checkbox"/> COMMERCIAL | (3) <input type="checkbox"/> INDUSTRIAL |
| (4) <input type="checkbox"/> RECREATIONAL AREA | (5) <input type="checkbox"/> UNIMPROVED LAND | (6) <input type="checkbox"/> AGRICULTURAL (TYPE) _____ |
| (7) <input type="checkbox"/> FOREST (TYPE) _____ | (8) <input type="checkbox"/> WITHIN 100 FT. OF WATER | (9) <input type="checkbox"/> UNKNOWN |
| (10) <input type="checkbox"/> OTHER (DESCRIBE) _____ | | |

FOR REPORT RESULTS CALL CENTRAL LAB (804) 786-5142; SHENANDOAH (703) 743-6326; SOUTHWEST (703) 628-3136

III. LABORATORY REPORT OF RABIES EXAMINATION (to be filled in by lab personnel only)

A. Microscopic examination for rabies:

- "NEGATIVE". This result does not exclude the possibility of rabies in this animal.
- "POSITIVE". Evidence of rabies.
- "NOT PERFORMED".

Date Reported: _____ Additional test will follow No additional testing

B. Fluorescent rabies antibody results:

- "POSITIVE". Evidence of rabies virus.
- "NEGATIVE". No evidence of rabies virus.
- "NEGATIVE, BUT BRAIN DECOMPOSED". This result does not exclude the possibility of rabies in this animal.
- "NON-SPECIFIC". Brain tissue shows non-specific fluorescence, results are uncertain.
- "UNSATISFACTORY". No test performed due to: _____

Date Reported: _____ Additional test will follow No additional testing

C. Mouse Inoculation test:

- MICE SURVIVED 21 OR 30 DAYS WITH NO RABIES SYMPTOMS.
- MICE DIED WITH EVIDENCE OF RABIES.
- NOT PERFORMED.
- TEST NOT COMPLETED OR INCLUSIVE.

Date Reported: _____

Figure 1. Standard submission form for animals being submitted for testing for rabies in Virginia.

TABLE 1. Codes and definitions for all variables and categories measured by the submission form for rabies testing in Virginia.

1. HUMAN EXPOSURE (Hum Exp)

0 = no human exposure

1 = there was human exposure

2. HOW THE ANIMAL WAS CAPTURED (Capture)

1 = animal was killed

2 = animal was found dead but was not killed on the road

3 = animal was killed on the road

4 = animal was caught in a trap

3. AGE OF THE ANIMAL (Age) (determined by dentition)

1 = juvenile (J) (less than one year of age)

2 = adult (A) (over one year of age)

4. SEX OF THE ANIMAL (Sex)

1 = female (F)

2 = male (M)

5. TIME OF DAY THE ANIMAL WAS SIGHTED (Time)

1 = dawn

2 = daylight

3 = dusk

4 = night

6. LAND USE CHARACTERISTICS WHERE THE ANIMAL WAS FOUND

(Location)

residential yard	within 100 ft. of water
residential building	forest
commercial	agricultural
industrial	
recreational area	0= no
unimproved land	1= yes

7. BITES ANOTHER ANIMAL (Bited)

0 = no

1 = yes

8. BEHAVIOR OF THE ANIMAL (Behav)

Normal (nor)

Sick (sick) 0 = no

Aggressive (aggr) 1 = yes

Friendly (fri)

Indifferent (indif)

Unresponsive (unresp)

Wobbily (wob)

Paralyzed (para)

9. YEAR (Year)

1 = 1984 4 = 1987

2 = 1985 5 = 1988

3 = 1986 6 = 1989

10. SEASON (Season)

- 1 = December - February
- 2 = March - May
- 3 = June - August
- 4 = September - November

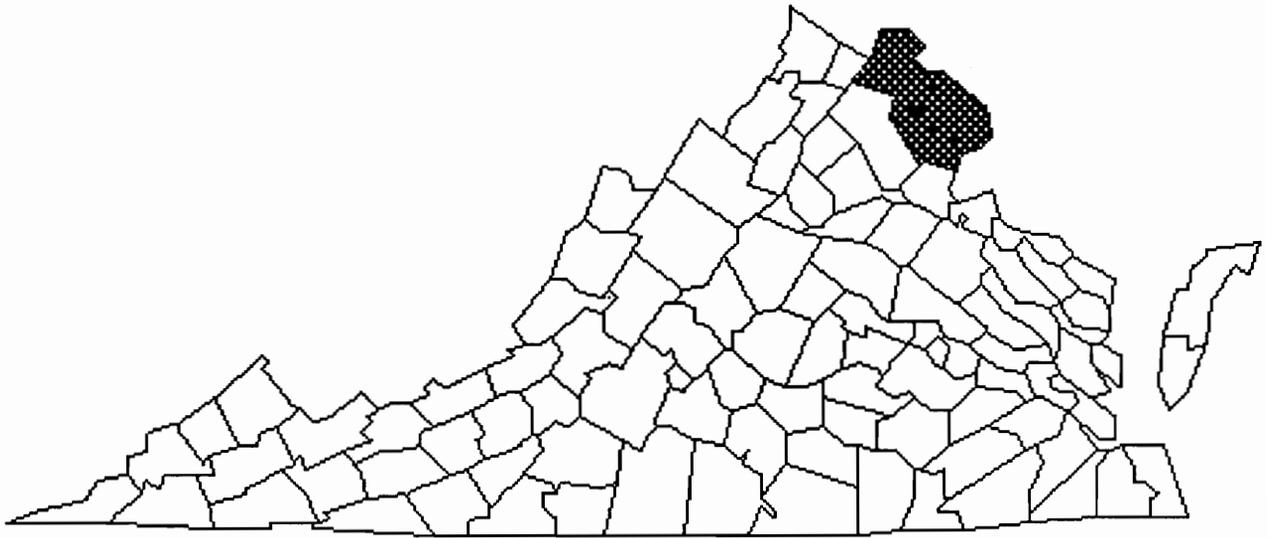
11. REGION (Region)

- 1 = central health region (center)
- 2 = eastern health region (east)
- 3 = northwestern health region (nwest)
- 4 = southwestern health region (swest)
- 5 = northern health region (north)

12. TEST RESULT (either by micro, FA, or mit test)

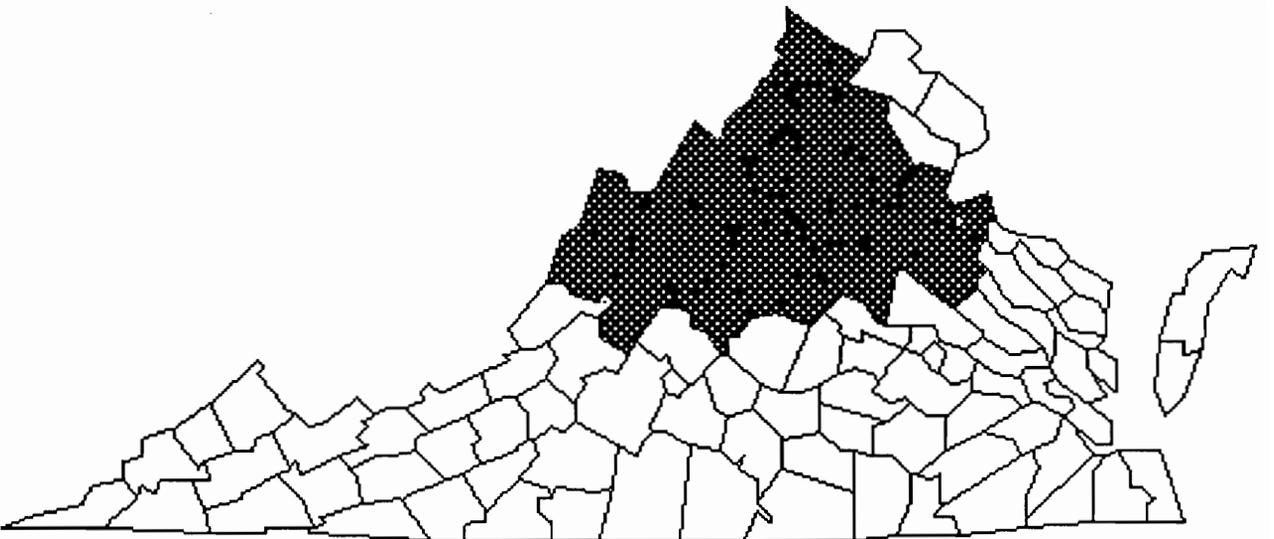
- 0 = negative for rabies (RABNEG or CONTROL)
- 1 = positive for rabies (RABPOS or CASE)

NORTHERN REGION



Cities : Alexandria

NORTHWEST REGION



Cities : Fredericksburg; Charlottesville

Figure 2. Northern and northwest administrative regions of the Virginia Department of Health

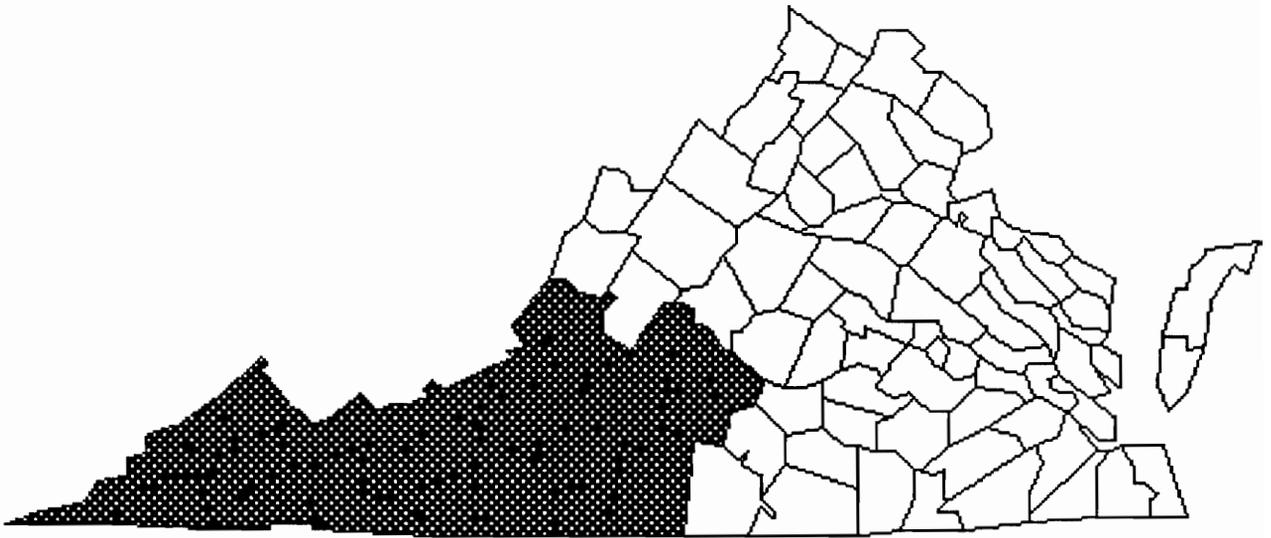
COUNTIES INCLUDED IN EACH REGIONNORTHWEST

Augusta
Bath
Highland
Rockbridge
Rockingham
Frederick
Clarke
Page
Shenandoah
Warren
Caroline
King George
Spotsylvania
Stafford
Orange
Culpepper
Fauquier
Madison
Rappahannock
Albermarle
Fluvanna
Greene
Louisa
Nelson
Page

NORTHERN

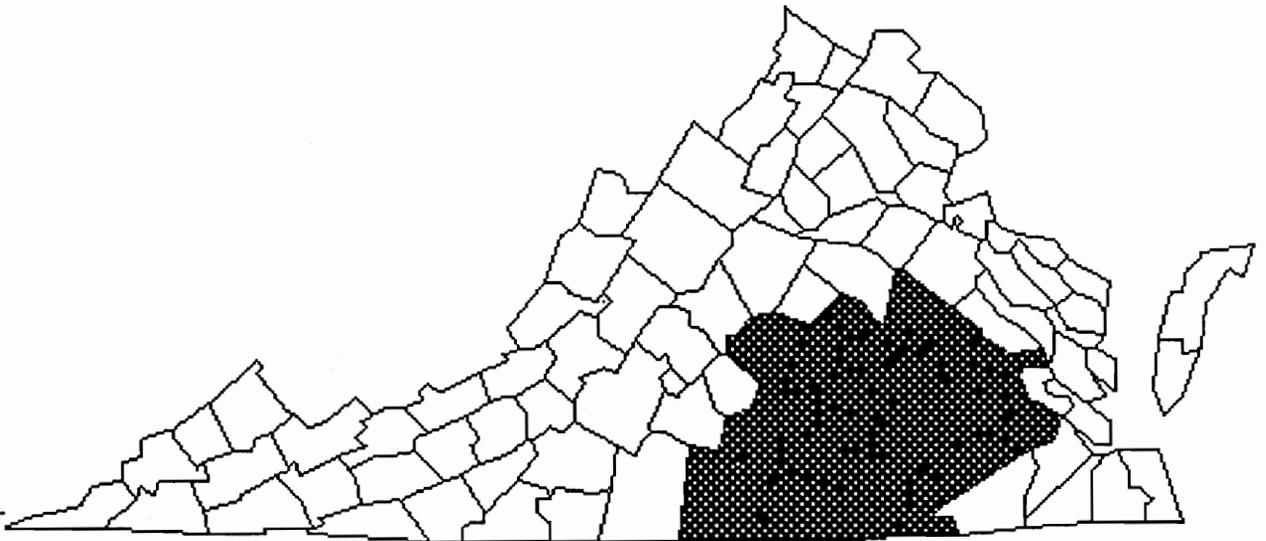
Fairfax
Loudoun
Prince William

SOUTHWEST REGION



Cities : Roanoke; Danville; Lynchburg

CENTRAL REGION



Cities : Richmond; Petersburg; Hopewell

Figure 3. Southwest and central administrative regions of the Virginia Department of Health

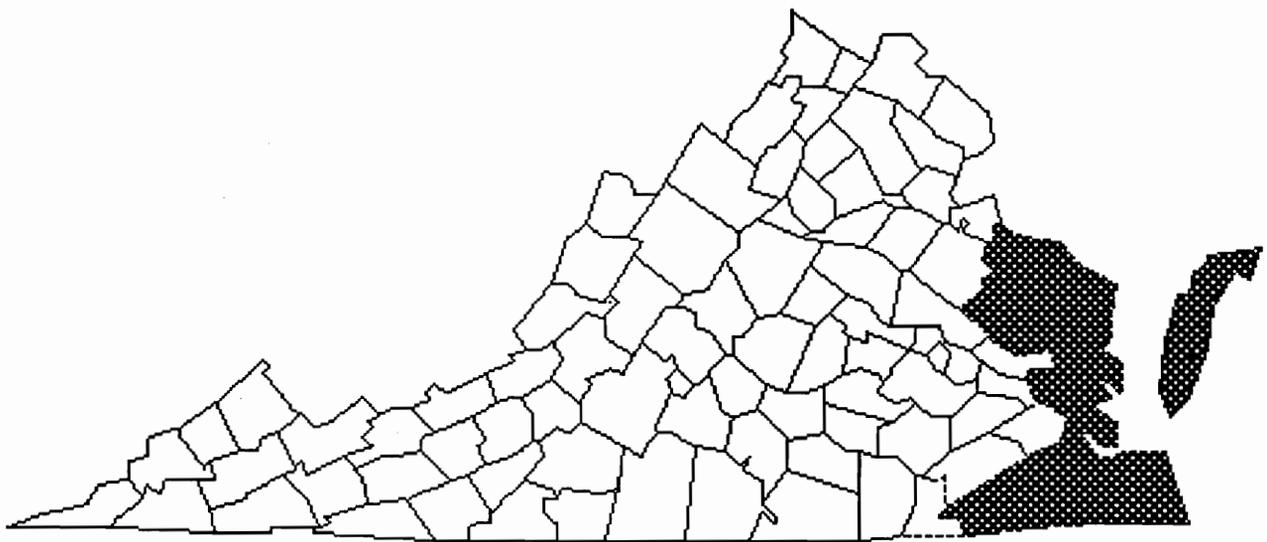
COUNTIES INCLUDED IN EACH REGIONCENTRAL

Chesterfield
Powhatan
Dinwiddie
Prince George
Surry
Sussex
Hanover
Charles City
Goochland
New Kent
Henrico
Amelia
Buckingham
Charlotte
Cumberland
Lunenburg
Nottoway
Prince Edward
Mecklenberg
Brunswick
Halifax
Greensville

SOUTHWEST

Roanoke
Alleghany
Botetourt
Craig
Amherst
Appomatox
Bedford
Campbell
Russell
Buchanan
Dickenson
Tazewell
Pittsylvania
Henry
Franklin
Patrick
Scott
Lee
Wise
Smyth
Bland
Carroll
Grayson
Washington
Wythe
Floyd
Giles
Montgomery
Pulaski

EASTERN REGION



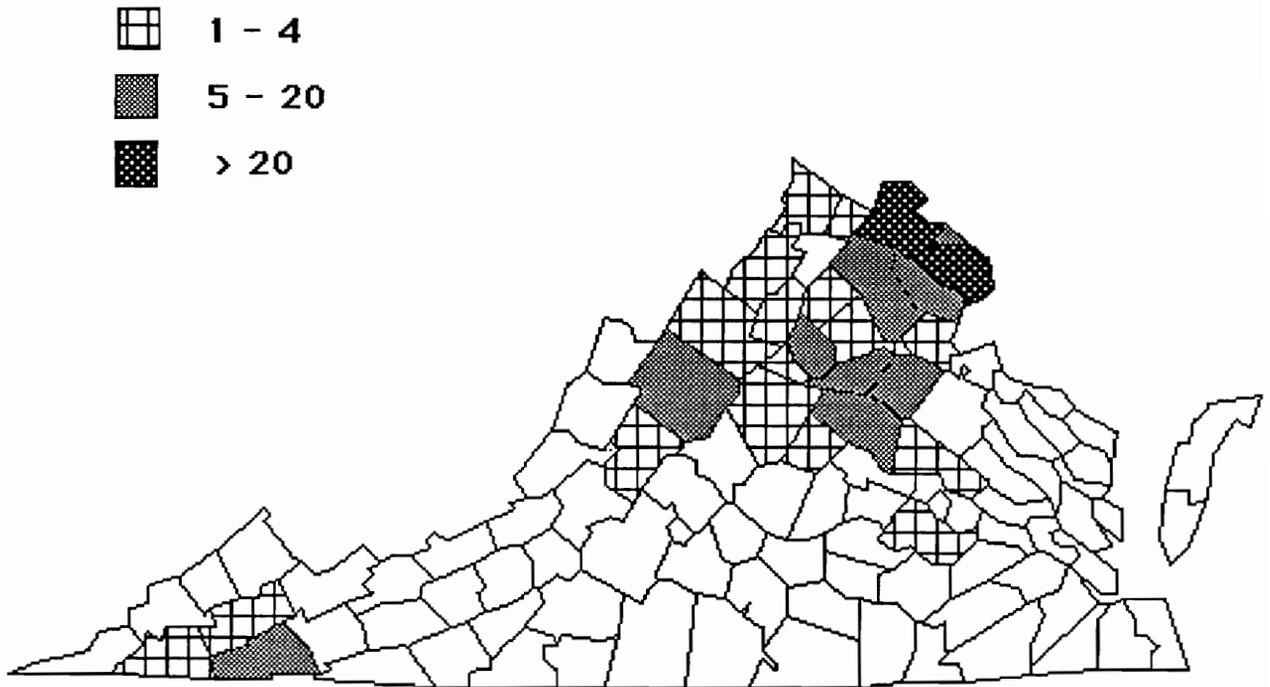
Cities : Virginia Beach; Chesapeake; Hampton; Norfolk; Newport News;
Portsmouth; Suffolk

Figure 4. Eastern administrative region of the Virginia Department of Health

COUNTIES INCLUDED IN EACH REGION

EASTERN

Accomack
Northampton
Middlesex
Essex
Gloucester
King and Queen
King William
Mathews
Richmond
Lancaster
Northumberland
Westmoreland
James City
York
Franklin
Southampton
Isle of Wight



1984

16 cases in Alexandria; 1 case in Fredericksburg; 1 case in Hopewell

Figure 5. Geographical distribution of positive rabies cases in all species in Virginia in 1984

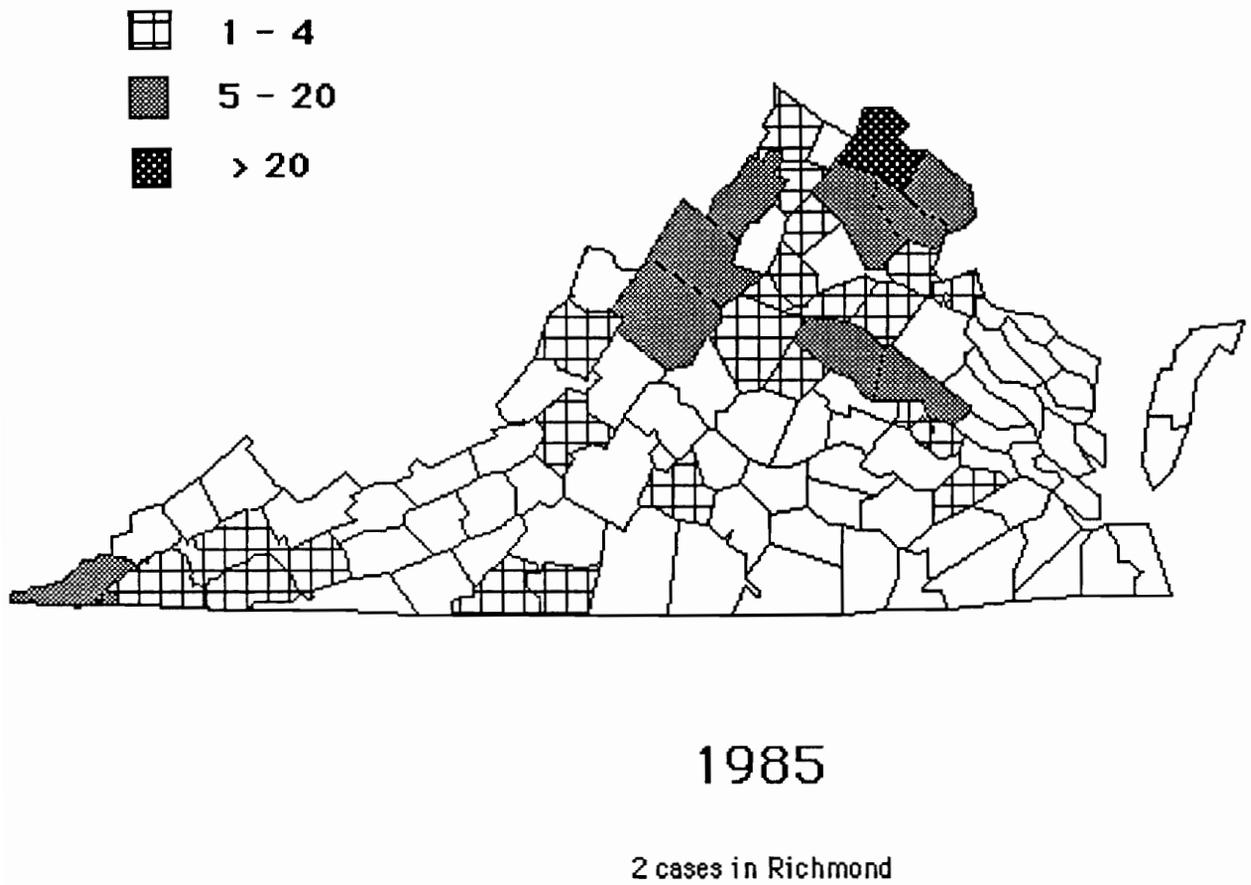


Figure 6. Geographical distribution of positive rabies cases in all species in Virginia in 1985

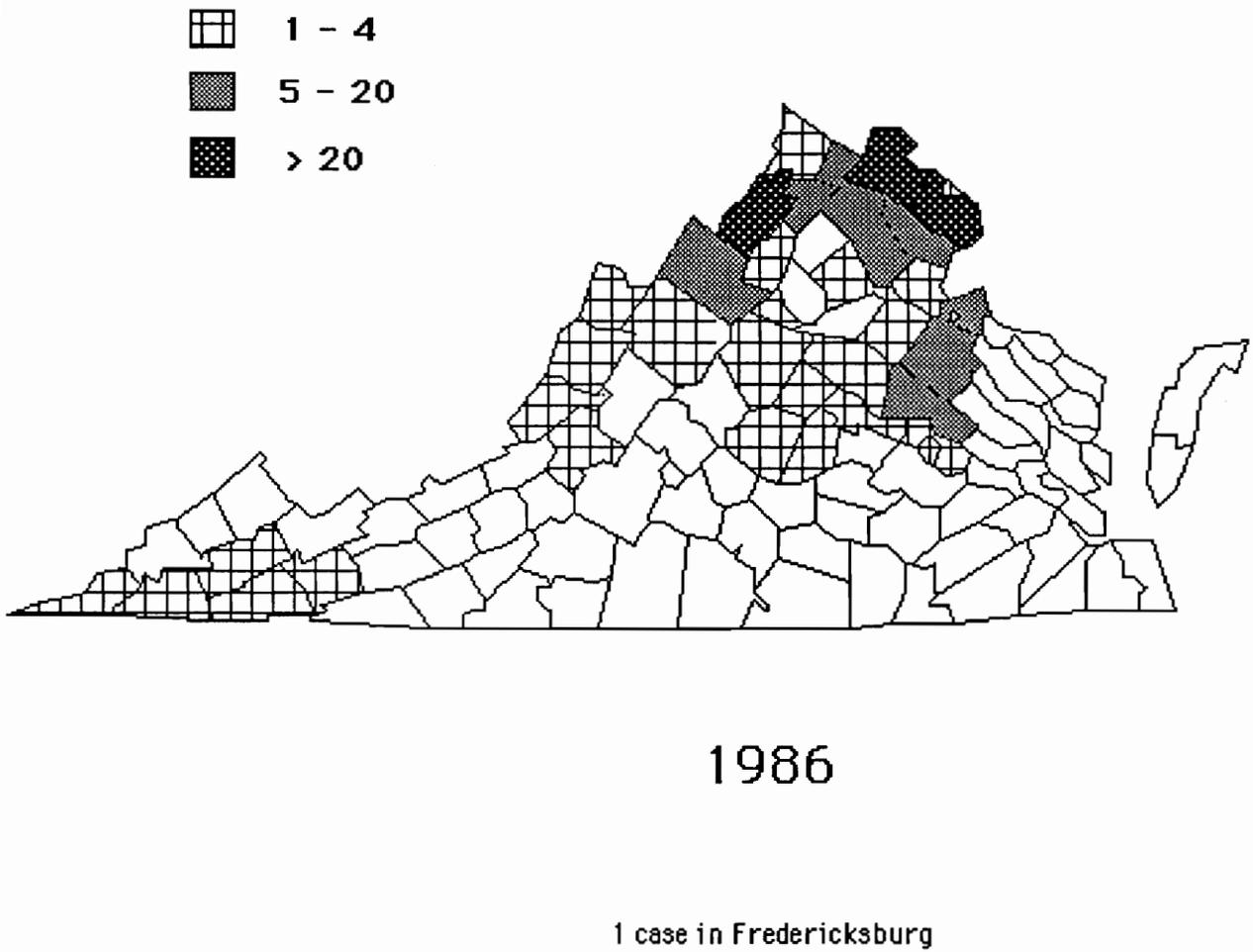


Figure 7. Geographical distribution of positive rabies cases in all species in Virginia in 1986

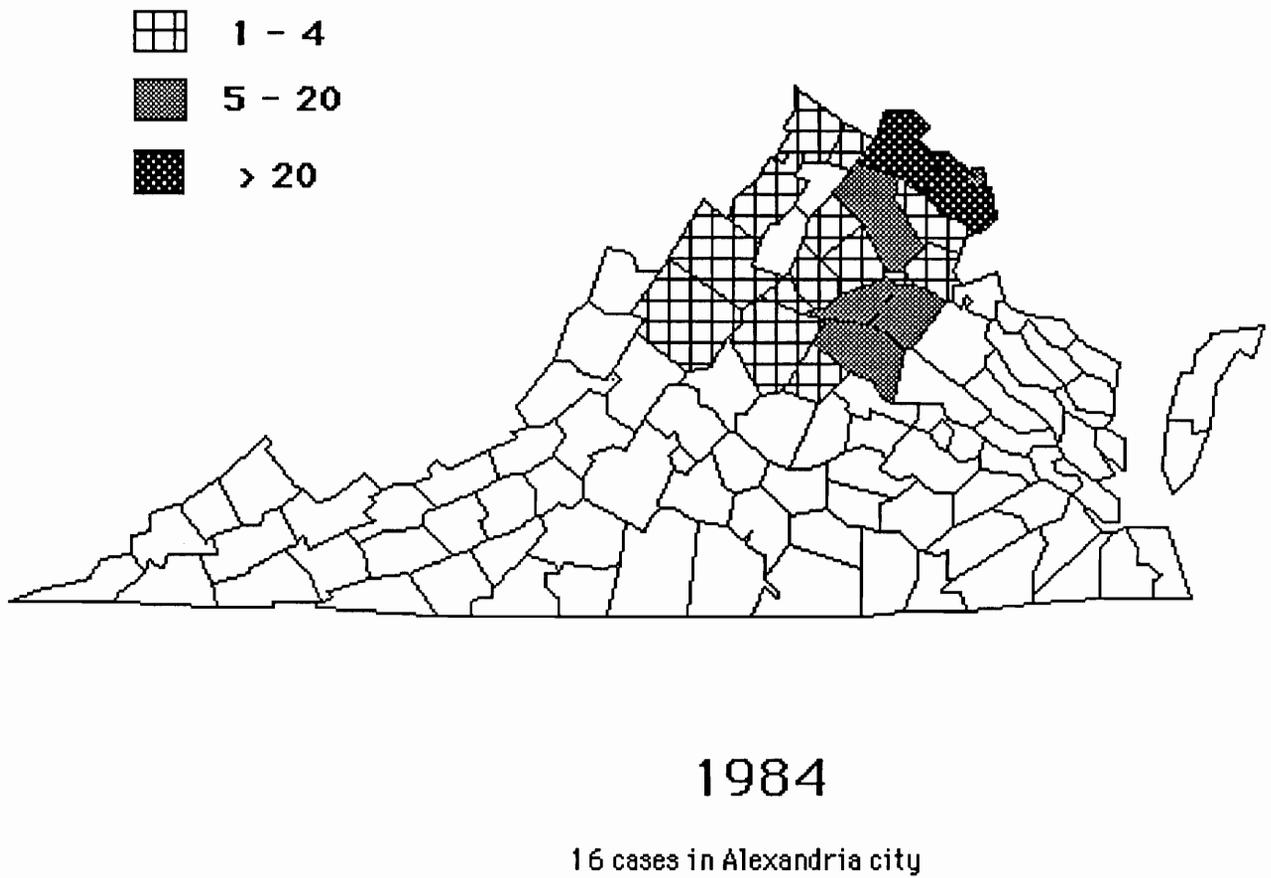


Figure 9. Geographical distribution of positive rabies cases in raccoons in Virginia in 1984

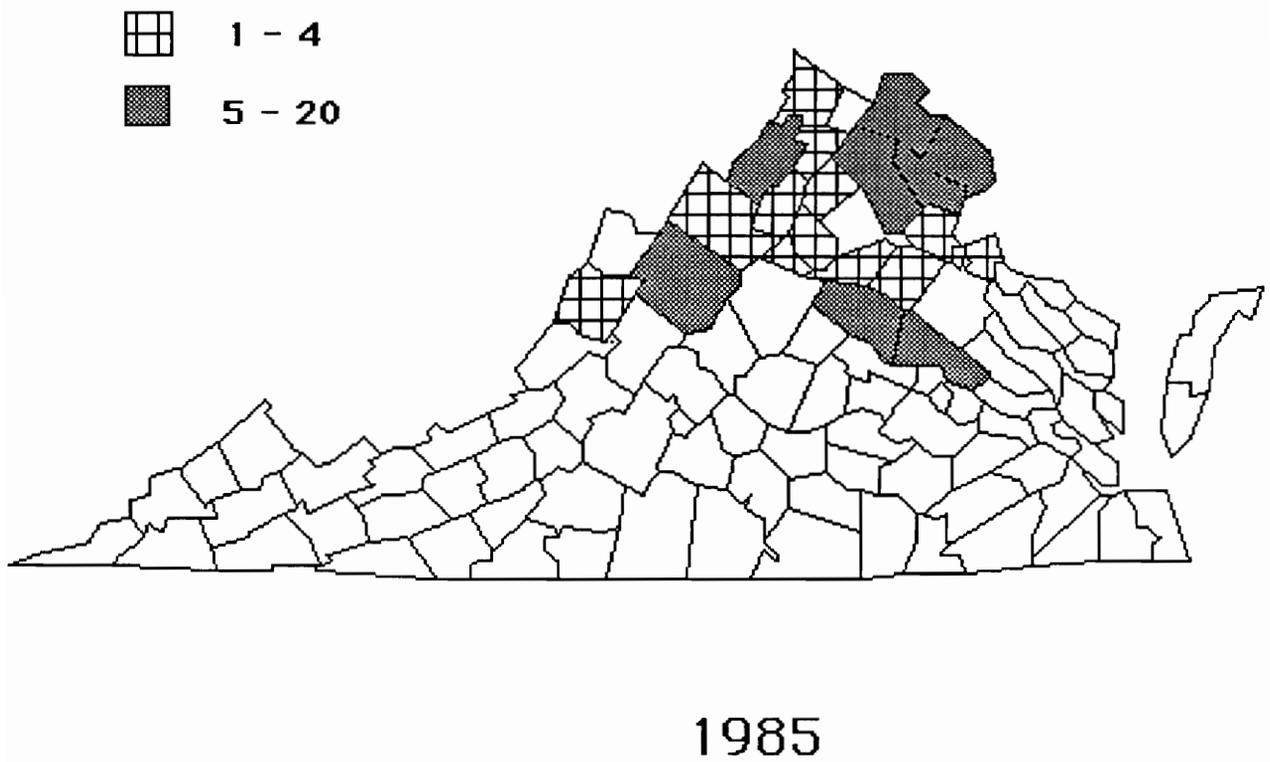


Figure 10. Geographical distribution of positive rabies cases in raccoons in Virginia in 1985

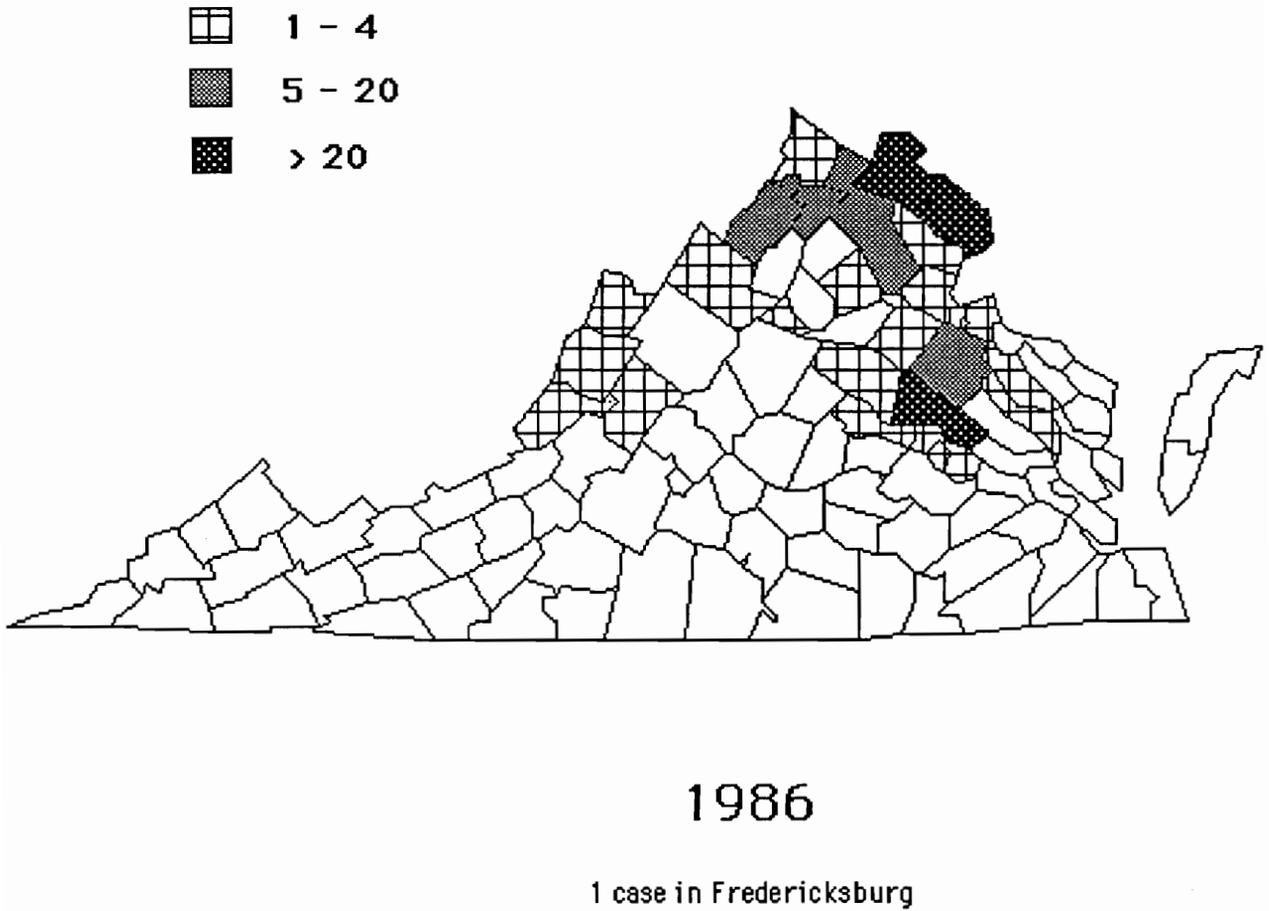
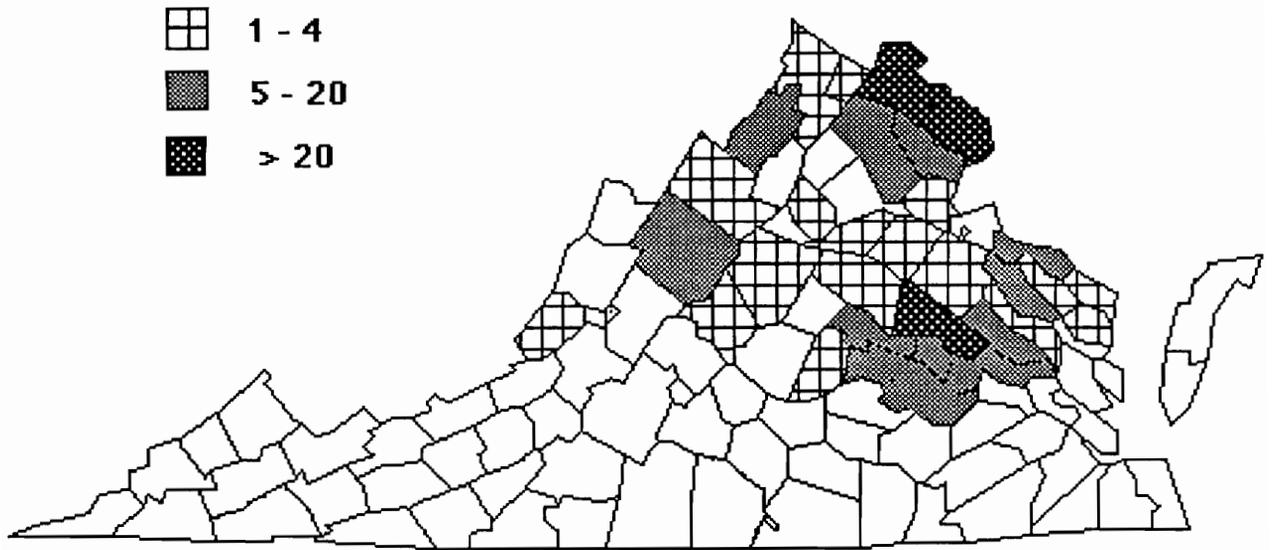


Figure 11. Geographical distribution of positive rabies cases in raccoons in Virginia in 1986



1987

1 case in Fredericksburg; 1 case in Portsmouth; 2 cases in Richmond

Figure 12. Geographical distribution of positive rabies cases in raccoons in Virginia in 1987

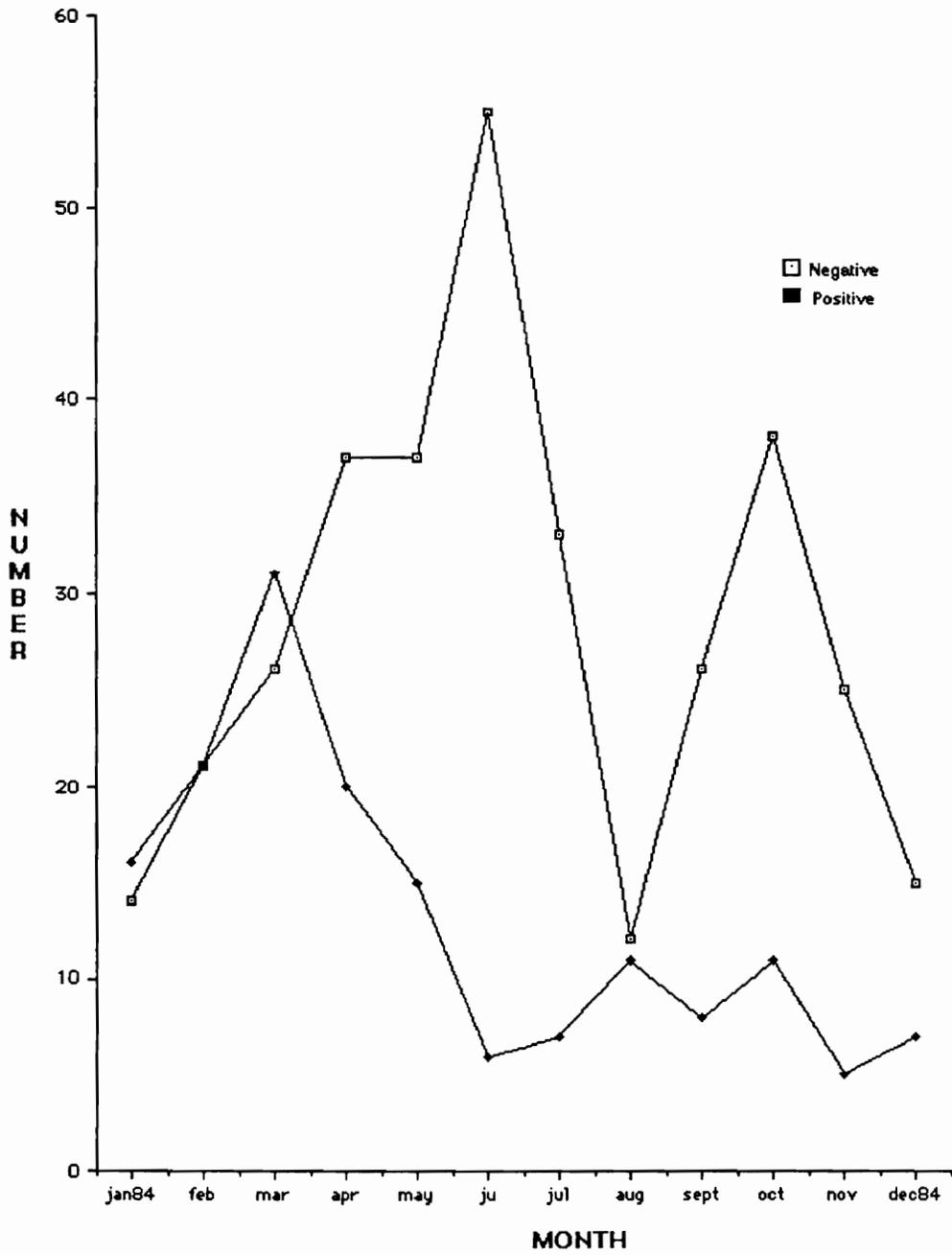


Figure 13. Number of raccoons testing positive and negative for rabies in Virginia during 1984

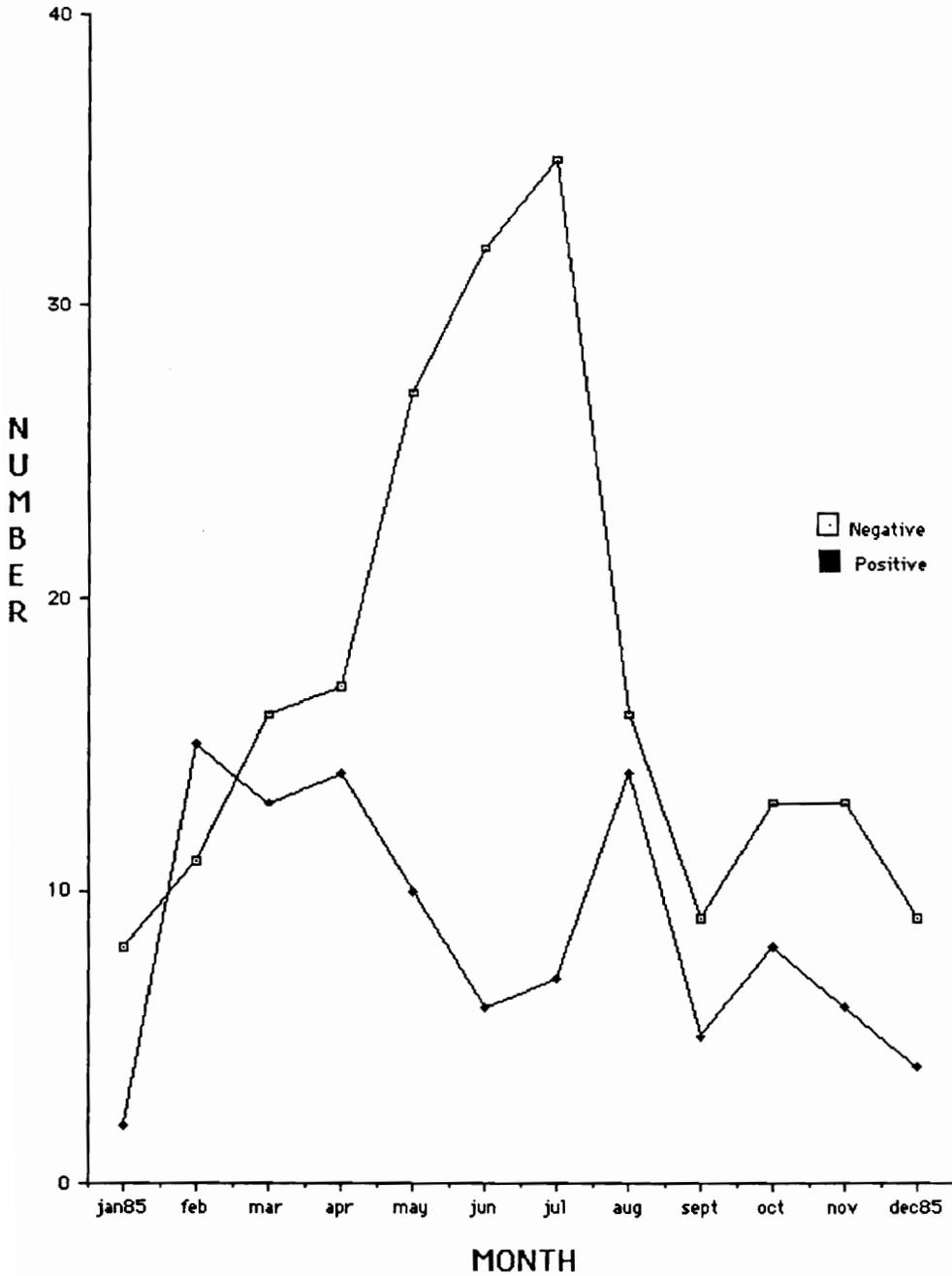


Figure 14. Number of raccoons testing positive and negative for rabies in Virginia during 1985

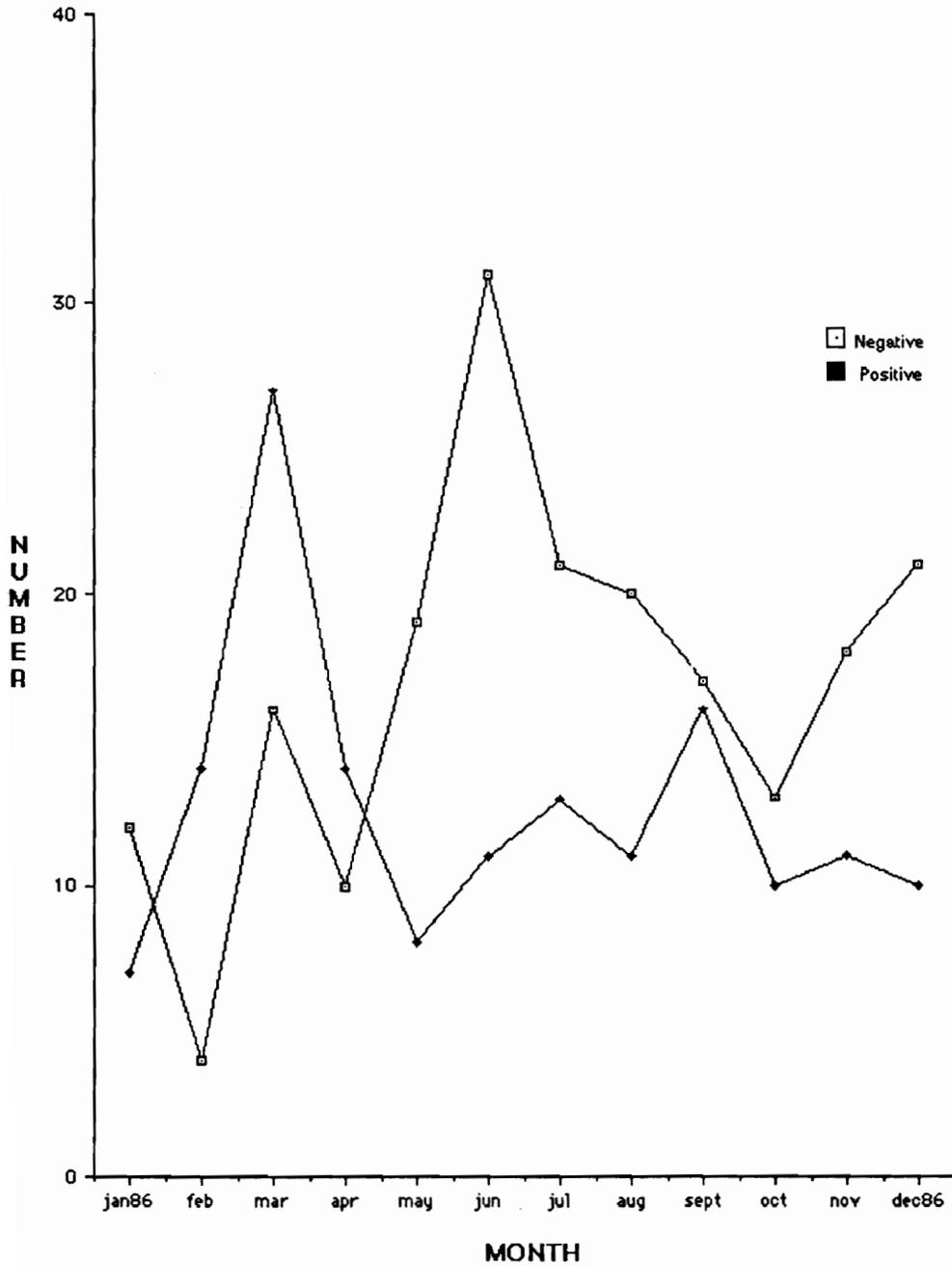


Figure 15. Number of raccoons testing positive and negative for rabies in Virginia during 1986

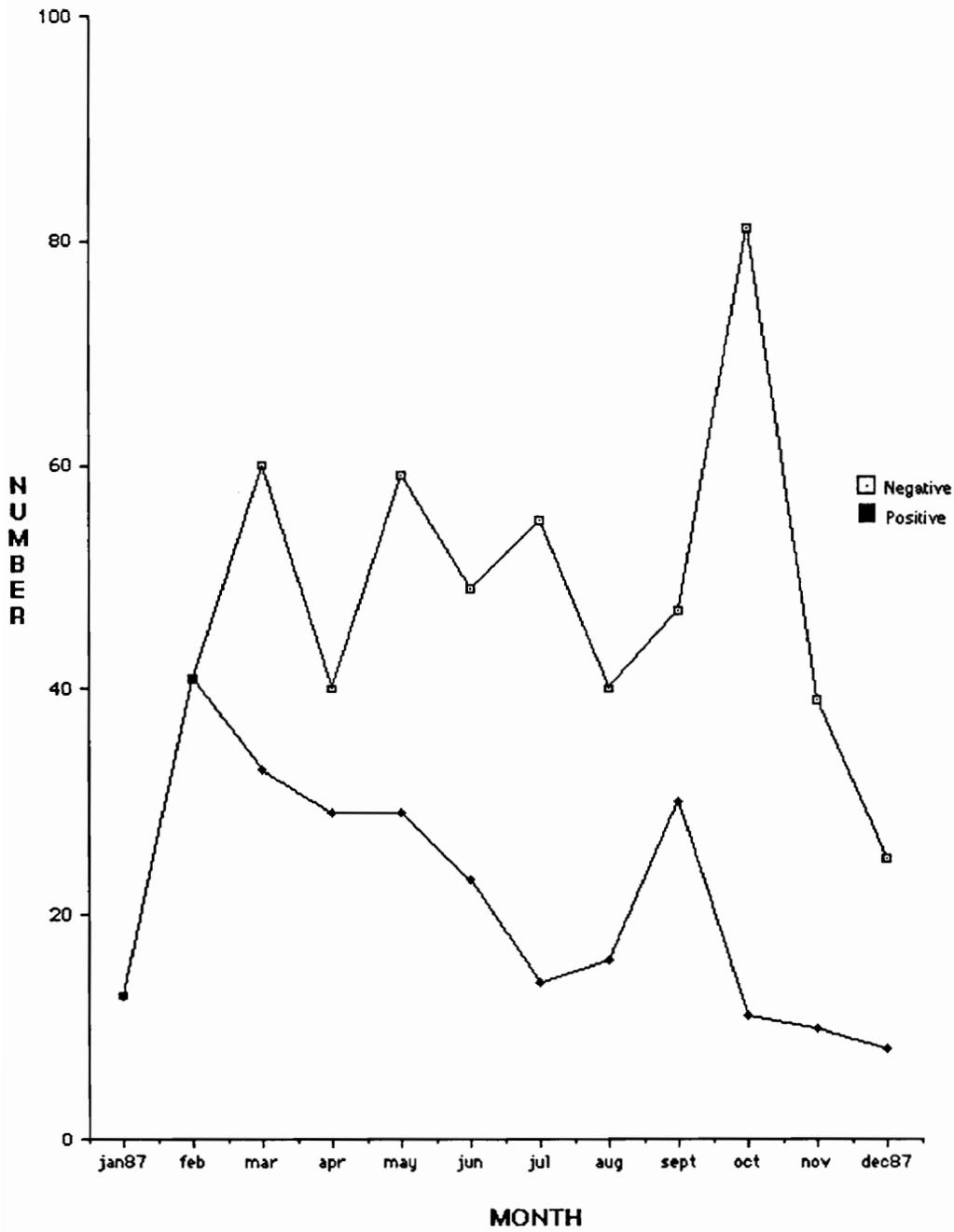


Figure 16. Number of raccoons testing positive and negative for rabies in Virginia during 1987

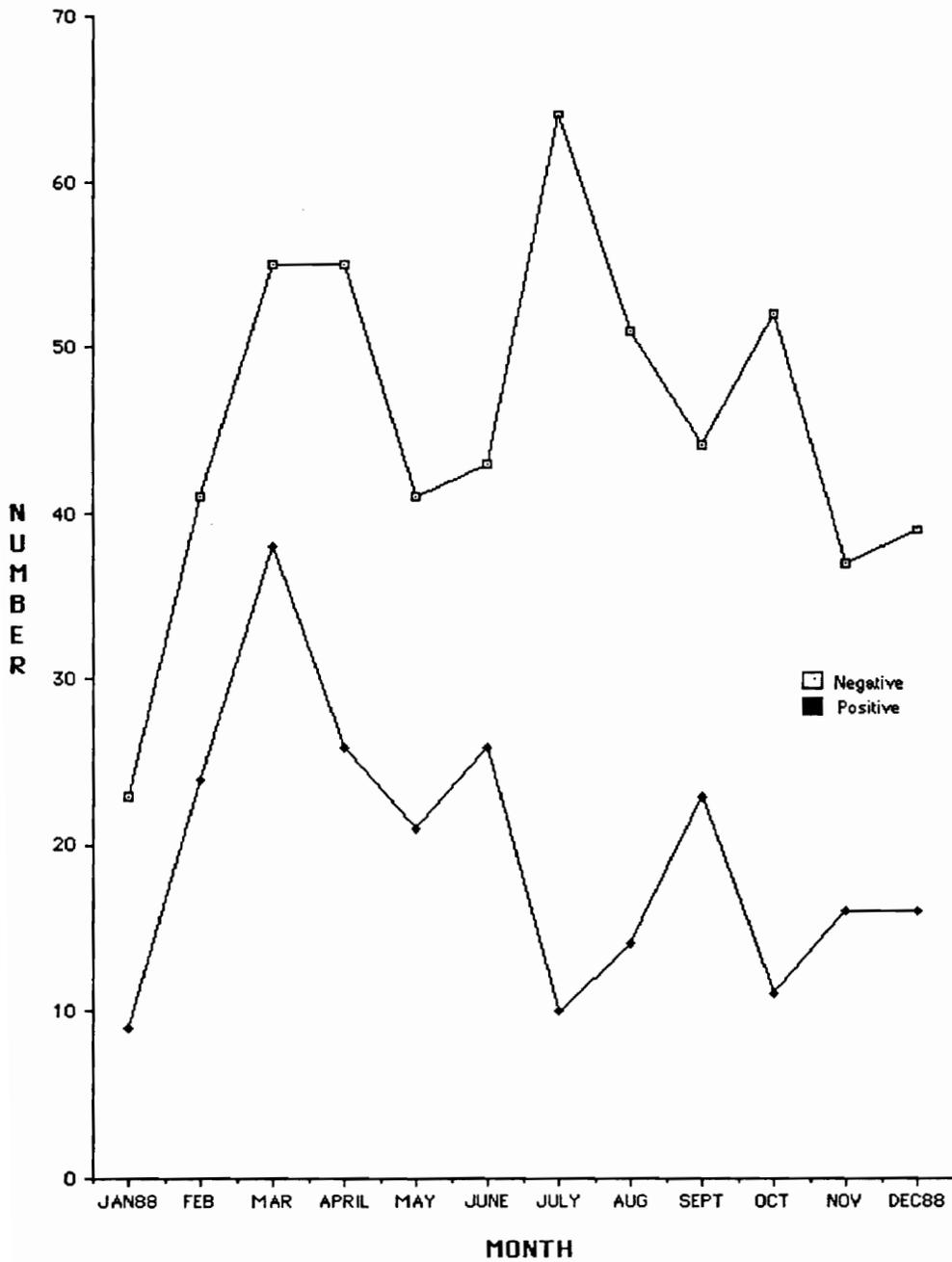


Figure 17. Number of raccoons testing positive and negative for rabies in Virginia during 1988

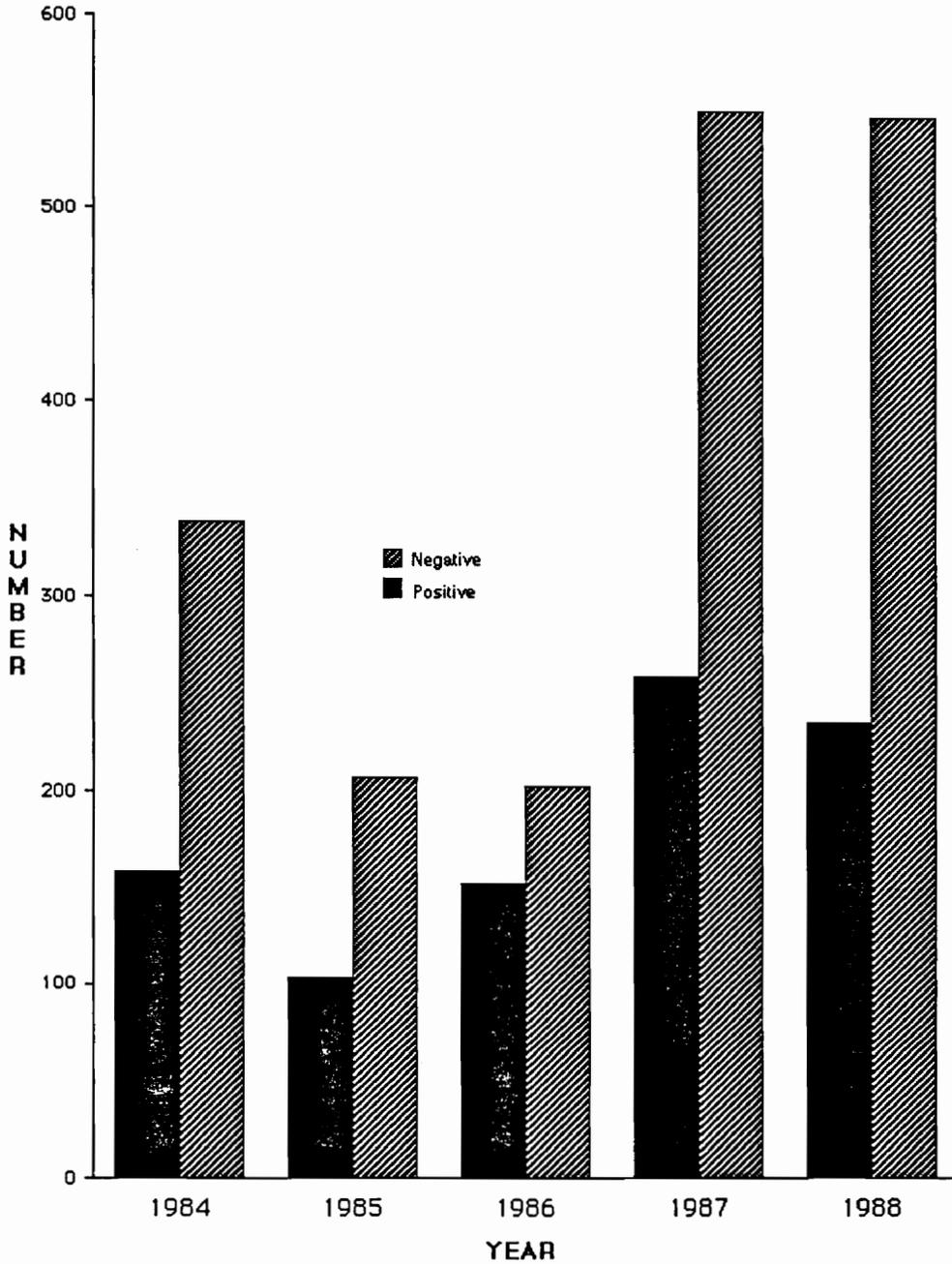


Figure 18. Number of raccoons testing positive and negative for rabies in Virginia from January 1, 1984 through December 31, 1988.

Table 2. Frequencies of explanatory variables measured from January 1, 1984 through December 31, 1987.

<u>VARIABLE</u>	<u>NUMBER</u>	<u>PERCENT</u>
<u>HUM EXP</u>		
NO (0)	1496	(74.62)
YES (1)	455	(22.69)
MISSING	54	(2.69)
<u>BEHAVIOR</u>		
AGGR	381	(19)
FLAT	119	(5.94)
FRIEND	629	(31.37)
NEURO	208	(10.37)
NORMAL	438	(21.85)
SICK	230	(11.47)
<u>LOCATION</u>		
RESIDENTIAL	1330	(66.33)
NONRESID	51	(2.55)
IOWA	624	(31.12)
<u>CAPTURED</u>		
KILLED	1432	(71.42)
FOUND DEAD	331	(16.51)
ROAD KILL	19	(0.94)
TRAPPED	104	(5.19)
MISSING	19	(5.94)
<u>BITED</u>		
NO (0)	652	(32.52)
YES (1)	577	(28.78)
MISSING	776	(38.70)
<u>AGE</u>		
JUVENILE (1)	452	(22.54)
ADULT (2)	1045	(52.12)
MISSING	508	(25.33)
<u>SEX</u>		
FEMALE (1)	298	(14.86)
MALE (2)	403	(20.10)
MISSING	1304	(65.04)
<u>TIME</u>		
DAWN (1)	166	(8.28)
DAYLIGHT (2)	1086	(54.16)
DUSK (3)	194	(9.68)
NIGHT (4)	270	(13.47)
MISSING	289	(14.41)
<u>REGION</u>		
CENTER (1)	460	(22.94)
EAST (2)	218	(10.87)
NWEST (3)	725	(36.16)
SWEST (4)	429	(21.4)
NORTH (5)	173	(8.63)
<u>YEAR</u>		
1984 (1)	497	(24.79)
1985 (2)	310	(15.46)
1986 (3)	354	(17.65)
1987 (4)	806	(40.20)
MISSING	38	(1.90)
<u>SEASON</u>		
DEC-FEB (1)	321	(16.01)
MARCH-MAY (2)	607	(30.28)
JUNE-AUG (3)	538	(26.83)
SEPT-NOV (3)	470	(23.44)
MISSING	69	(3.44)

See Table 1 for explanation of codes.

Table 3. Positivity ratios for the explanatory variables from January 1, 1984 through December 31, 1987.

<u>VARIABLE</u>	<u>RABPOS</u>	<u>RABNEG</u>	<u>POS.RATIO</u>
<u>HUM EXP</u>			
NO (0)	603	893	0.68
YES (1)	60	395	0.15
TOTAL	663	1288	0.51
<u>BEHAVIOR</u>			
AGGR	179	202	0.89
FLAT	46	73	0.63
FRIEND	218	411	0.53
NEURO	70	138	0.51
NORMAL	91	347	0.26
SICK	75	155	0.48
TOTAL	679	1326	0.51
<u>LOCATION</u>			
RESIDENTIAL	490	840	0.58
NONRESID	6	45	0.13
IOWA	183	441	0.41
TOTAL	679	1326	0.51
<u>CAPTURED</u>			
KILLED (1)	539	893	0.6
FOUND DEAD (2)	96	235	0.41
ROAD KILL (3)	2	17	0.12
TRAPPED (4)	17	87	0.2
TOTAL	679	1232	0.53
<u>BITED</u>			
NO (0)	152	500	0.3
YES (1)	296	281	1.05
TOTAL	448	781	0.57
<u>AGE</u>			
JUVENILE (1)	420	625	0.67
ADULT (2)	91	361	0.25
TOTAL	511	986	0.52
<u>TIME</u>			
DAWN (1)	58	108	0.54
DAYLIGHT (2)	394	692	0.57
DUSK (3)	80	114	0.7
NIGHT (4)	78	192	0.41
TOTAL	610	1106	0.55
<u>REGION</u>			
CENTER (1)	138	322	0.43
EAST (2)	48	170	0.28
NWEST (3)	232	197	1.18
SWEST (4)	5	168	0.03
NORTH (5)	256	469	0.55
TOTAL	679	1326	0.51
<u>YEAR</u>			
1984 (1)	158	339	0.47
1985 (2)	104	206	0.5
1986 (3)	152	202	0.75
1987 (4)	257	549	0.47
TOTAL	671	1296	0.52
<u>SEASON</u>			
DEC-FEB (1)	151	170	0.89
MARCH-MAY (2)	243	364	0.67
JUNE-AUG (3)	139	399	0.35
SEPT-NOV (4)	131	339	0.39
TOTAL	664	1272	0.52

See Table 1 for explanation of codes.

TABLE 5. PARAMETER ESTIMATES FOR SECOND ORDER EFFECTS IN THE FINAL SUBSET MODEL.														
RATIO OF THE LOG-LINEAR PARAMETERS (SECOND ORDER)														
MODEL	AGE	SEASON	RAB POS		YEAR	SEASON	RAB POS		BEHAV	SEASON	RAB POS			
			0	1			0	1			0	1		
(ABYS) AS,B,YS	1	1	-2.884	2.884	2	1	-2.157	2.157						
			2.884	-2.884			0.515	-0.515						
	2				3		1.658	-1.658						
							-0.638	0.638						
	1	2		-0.54	0.54	1	2	-1.009	1.009					
				0.54	-0.54			-0.237	0.237					
	1	3		1.731	-1.731	2		1.052	-1.052					
				-1.731	1.731			0.065	-0.065					
	2					3		1.255	-1.255					
								0.7	-0.7					
	1	4		1.783	-1.783	1	3	-0.47	0.47					
				-1.783	1.783			-1.732	1.732					
	2					2		1.721	-1.721					
								-0.912	0.912					
						3		-2.556	2.556					
								2.427	-2.427					
					4									
(ACXS) AS, X, C	1	1	-3.458	3.458	2	1								
			3.458	-3.458										
	2					2	2	-1.219	1.219					
								1.219	-1.219					
	1	3		2.85	-2.85	2		2.85	-2.85					
				-2.85	2.85									
	1	4		2.015	-2.015	2		2.015	-2.015					
				-2.015	2.015									
	(ACYS) C, Y, AS	1	1	-4.024	4.024	2	1							
				4.024	-4.024									
		1	2		-0.819	0.819	2	2	0.819	-0.819				
					0.819	-0.819								
1		3		3.225	-3.225	2		3.225	-3.225					
				-3.225	3.225									
1		4		1.896	-1.896	2		1.896	-1.896					
				-1.896	1.896									
(AHXS) AS,X,H		1	1	-3.314	3.314	2	1							
				3.314	-3.314									
		1	2		-1.212	1.212	2	2	1.212	-1.212				
					1.212	-1.212								
	1	3		2.774	-2.774	2		2.774	-2.774					
				-2.774	2.774									
	1	4		1.962	-1.962	2		1.962	-1.962					
				-1.962	1.962									
	(BHXS) B,H,XS									1	1	-1.377	1.377	

TABLE 6. PARAMETER ESTIMATES FOR AGE AND HUMAN EXPOSURE FOR THE FINAL SUBSET MODELS.								
RATIO OF THE LOG-LINEAR PARAMETERS								
MODEL	AGE	RAB POS		MODEL	HUM EXP	RAB POS		
		0	1			0	1	
(AHXS) AS,X,H	1	3.471	-3.471	(AHXS) AS,X,H	0	-7.28	7.28	
	2	-3.471	3.471		1	7.28	-7.28	
(ACYS) C,Y,AS	1	5.264	-5.246	(BHXS) B,H,XS	0	-4.729	4.729	
	2	-5.246	5.246		1	4.729	-4.279	
(ABYS) AS,B,YS	1	4.065	-4.065	(BHYS) B,H,Y,S	0	-5.409	5.409	
	2	-4.065	4.065		1	5.409	-5.409	
(ACXS) AS,X,C	1	4.419	-4.419	(BHXY) B,H,X,Y	0	-4.842	4.842	
	2	-4.419	4.419		1	4.842	-4.842	
(ACXY) A,C,X,Y	1	5.858	-5.858	(BHAS) B,H,A,S	0	-4.815	4.815	
	2	-5.858	5.858		1	4.815	-4.815	
(ACHZ) A,H,Z	1	5.249	-5.249	(ACHZ) A,C,H,Z	0	-7.56	7.56	
	2	-5.249	5.249		1	7.56	-7.56	
(AHZS) A,H,Z,S	1	4.573	-4.573	(AHZS) A,H,Z,S	0	-7.632	7.632	
	2	-4.573	4.573		1	7.632	-7.632	
(AHYS) A,H,Y,S	1	4.827	-4.827	(AHYS) A,H,Y,S	0	-8.256	8.256	
	2	-4.827	4.827		1	8.256	-8.256	
(ABXY) A,B,X,Y	1	4.332	-4.332					
	2	-4.332	4.332					
(BCAY) B,A,C,Y	1	5.133	-5.133					
	2	-5.133	5.133					
(BCAS) B,C,A,S	1	4.545	-4.545					
	2	-4.545	4.545					
SEE TABLE 1 FOR EXPLANATION OF CODES.								

TABLE 7. P VALUES OF THE FINAL 17 SUBSET MODELS BEING FITTED ON THE DATA FROM JANUARY 1, 1988 THROUGH JULY 31, 1989 AND ON THE DATA FROM JANUARY 1, 1984 THROUGH JULY 31, 1989.

<u>MODEL</u>	<u>1988-1989</u> <u>p value</u>	<u>1984-1989</u> <u>p value</u>
(BHXS) B,H,XS (0.09)	0.05	0.02
(AHYS) A,H,Y,S (0.01)	0.06	0.08
(BHYS) B,H,Y,S (0.13)	0.17	0
(BCYS) B,C,Y,S (0.01)	0.2	0.01
(ABYS) AS,B,YS (0.02)	0.05	0.06
(BCAS) B,A,C,S (0.03)	0.11	0.04
(ACYS) AS,C,Y (0.12)	0.08	0.1
(AHZS) A,H,Z,S (0.07)	0.01	0.01
(ACXS) AS,X,C (0.02)	0.2	0.2
(AHXS) AS,X,H (0.03)	0.5	0.5
(BHAS) B,H,A,S (0.09)	0.35	0.35
(BCXY) B,X,Y (0.03)	0.64	0.06
(BHXY) B,H,X,Y (0.07)	0.67	0.08
(ACXY) A,C,X,Y (0.07)	0.54	0.54
(BCAY) B,A,C,Y (0.02)	0.35	0.35
(ACHZ) A,H,Z,C (0.55)	0.32	0.32
(ABXY) A,B,X (0.28)	0.09	0.09

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 6. PARAMETER ESTIMATES FOR EACH FINAL MODEL FITTED ON DATA FROM JANUARY 1, 1968 THROUGH JULY 31, 1968.

MODEL	BATED		BASE FOR		AGE		SEASON		YEAR		REGION		NUM EXP		CAPTURE		REHAY				
	0	1	0	1	0	1	0	1	0	1	0	1	0	1	0	1	0	1			
(ABYS) AS,B,Y,S	0	3.456	-3.456	2.903	-2.903	1	-1.097	1.097	1	1.334	-1.334										
	1	-3.456	3.456	2.903	-2.903	2	-1.464	1.464	2	-1.334	1.334										
						3	0	0													
						4	1.428	-1.428													
(ACRY) A,C,X,Y				1	4.046	-4.046			1	0.976	-0.976					1	-1.523	1.523	1	2.364	-2.364
				2	-4.046	4.046			2	-0.976	0.976					2	-2.459	-2.459	2	1.682	-1.682
																4	2.085	-2.085	5	0	0
																6	-2.397	2.397	6		
(AHZS) A,H,Z,S				1	3.014	-3.014	1	0.3	-0.3			1	0.76	-0.76	0	-3.951	3.951				
				2	-3.014	3.014	2	-1.3	1.3			2	2.357	-2.357	1	3.951	-3.951				
							3	0.421	-0.421			3	-5.194	5.194							
							4	0.339	-0.339			4	1.866	-1.866							
												5	0.602	-0.602							
(ACRZ) A,C,H,Z				1	3.099	-3.099						1	1.061	-1.061	0	-3.939	3.939				
				2	-3.099	3.099						2	2.36	-2.36	1	3.939	-3.939				
											3	-5.165	5.165								
											4	1.573	-1.573								
											5	0.653	-0.653								
(ACRS) A,R,X,C				1	3.423	-3.423	1	-1.571	1.571												
				2	-3.423	3.423	2	-2.098	2.098												
							3	2.212	-2.212												
							4	1.021	-1.021												
(ACRY) A,R,C,Y				1	3.696	-3.696	1	-1.612	1.612	1	-0.943	0.943									
				2	-3.696	3.696	2	-1.851	1.851	2	0.943	-0.943									
							3	2.26	-2.26												
							4	0.638	-0.638												
(AHRS) A,R,X,H				1	3.05	-3.05	1	-1.075	1.075												
				2	-3.05	3.05	2	-1.964	1.964												
							3	1.756	-1.756												
							4	0.938	-0.938												
(BHY) B,H,Y,S				0	3.732	-3.732				1	0.101	-0.101									
				1	-3.732	3.732				2	-0.912	0.912									
(BCKY) B,X,Y				0	3.894	-3.894				1	-1.087	1.087									
				1	-3.894	3.894				2	1.087	-1.087									
(BHAS) B,A,H,S				0	3.253	-3.253	1	2.876	-2.876	1	0.762	-0.762									
				1	-3.253	3.253	2	-2.876	2.876	2	-2.152	2.152									

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 8. PARAMETER ESTIMATES FOR EACH MODEL FITTED ON DATA FROM JANUARY 1, 1969 THROUGH JULY 31, 1969 (cont.)

MODEL	BITEP		AGE		SEASON		YEAR		REGION		RMM EXP		CAPTURE		RAB FOR		BEHAV		
	0	1	0	1	0	1	0	1	0	1	0	1	0	1	0	1	0	1	
[BHCY] B,H,A,Y	0	3.014	-3.014																
	1	-3.014	3.014																
	0																		
[BCHS] B,C,Y,S	0	4.19	-4.19																
	1	-4.19	4.19																
	0																		
	1																		
[BCAS] B,A,C,S	0	3.028	-3.028																
	1	-3.266	3.266																
	0																		
	1																		
[BCAV] B,A,C,V	0	3.293	-3.293																
	1	-3.293	3.293																
	0																		
[ABCV] A,B,C,V	0	3.06	-3.06																
	1	-3.06	3.06																
[BHCY] B,H,A,Y	0	3.076	-3.076																
	1	-3.076	3.076																
	0																		
	1																		
[AHYS] A,H,Y,S	1	2.046	-2.046																
	2	-3.646	3.646																
	0																		
	1																		

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 9. PARAMETER ESTIMATES FOR SECOND ORDER EFFECTS IN MODELS FITTED ON DATA FROM JANUARY 1, 1966 THROUGH JULY 31, 1969.

RATIO OF THE LOG-LINEAR PARAMETERS (SECOND ORDER)												
MODEL	AGE	SEASON	RAB POS		YEAR	SEASON	RAB POS		BEHAV	SEASON	RAB POS	
			0	1			0	1			0	1
(ABYS) AS,B,YS	1	1	-2.737	2.737	1	1	-1.986	1.986				
	2		2.737	-2.737	2		1.986	-1.986				
	1	2	-1.669	1.669	1	2	-2.097	2.097				
	2		1.669	-1.669	2		2.097	-2.097				
	1	3	0	0	1	3	0	0				
	2		0	0	2		0	0				
	1	4	2.443	-2.443	1	4	2.247	-2.247				
	2		-2.443	2.443	2		-2.247	2.247				
	(ACKS) AS,X,C	1	1	-2.338	2.338							
2			2.338	-2.338								
1		2	-1.121	1.121								
2			1.121	-1.121								
1		3	2.19	-2.19								
2			-2.19	2.19								
1		4	0.96	-0.96								
2			-0.96	0.96								
(ACYS) AS,C,Y		1	1	-2.578	2.578							
	2		2.578	-2.578								
	1	2	-0.987	0.987								
	2		0.987	-0.987								
	1	3	2.367	-2.367								
	2		-2.367	2.367								
	1	4	0.832	-0.832								
	2		-0.832	0.832								
	(AHDK) AS,X,H	1	1	-1.994	1.994							
2			1.994	-1.994								
1		2	-1.275	1.275								
2			1.275	-1.275								
1		3	1.98	-1.98								
2			-1.98	1.98								
1		4	0.957	-0.957								
2			-0.957	0.957								
(BHDK) B,H,XS										1	1	0
									2		0.442	-0.153
									5		-0.071	0.027
									6		-0.083	0.083
									1	2	0.147	-0.147
									2		0	0
									5		0	0
									6		-0.147	0.147
									1	3	0	0
									2		0	0
									5		0	0
									6		0	0
								1	4	-0.147	0.147	
								2		-0.153	0.153	
								5		0.027	-0.027	
								6		0.12	-0.12	

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 16. PARAMETER ESTIMATES FOR EACH MODEL FITTED ON DATA FROM JANUARY 1, 1984 THROUGH JULY 31, 1988 (cont.).

MODEL	BITED		AGE		SEASON		YEAR		REGION		HUMIDITY		CAPTURE		BEHAV		RMR POS		
	0	1	0	1	0	1	0	1	0	1	0	1	0	1	0	1	0	1	
(BDCY) B.H.J.V	0	6.102	-4.102				1	-2.519	2.519			0	-4.92	4.92			1	5.146	-5.146
	1	-6.102	6.102				2	-1.618	1.618			1	4.92	-4.92			2	-1.567	1.567
							3	-4.103	4.103								5	-3.816	3.816
							4	1.824	-1.824								6	-0.101	0.101
							5	3.474	-3.474										
							6	2.851	-2.851										
(BCV8) B.C.V.S	0	9.612	-9.612				1	-3.09	3.09			1	-2.762	2.762			1	-2.845	2.845
	1	-9.612	9.612				2	-3.03	3.03			2	-2.105	2.105			2	-2.054	2.054
							3	3.509	-3.509			3	-3.734	3.734			4	2.91	-2.91
							4	3.462	-3.462			4	0.963	-0.963					
							5	3.279	-3.279										
							6	5.435	-5.435										
(BCA8) B.C.A.S	0	3.678	-3.678				1	2.423	-2.423								1	-1.332	1.332
	1	-3.678	3.678				2	-2.423	2.423								2	-1.005	1.005
							3	0.55	-0.55								4	1.946	-1.946
							4	0.235	-0.235										
(BCV1) B.C.V.A	0	3.272	-3.272				1	2.769	-2.769								1	0	0
	1	-3.272	3.272				2	-2.769	2.769								2	1.738	-1.738
(ABDY) A.B.J.V	0	3.744	-3.744				1	2.352	-2.352								1	0	0
	1	-3.744	3.744				2	-2.352	2.352								2	1.738	-1.738
							3	-3.589	3.589								5	0	0
							4	0.775	-0.775								6	0	0
							5	3.11	-3.11										
							6	5.226	-5.226										
(BDC8) B.H.X.B	0	7.355	-7.355				1	-2.789	2.789			0	-4.737	4.737			1	4.699	-4.699
	1	-7.355	7.355				2	-2.542	2.542			1	4.737	-4.737			2	1.393	-1.393
							3	3.285	-3.285								5	-3.035	3.035
							4	1.672	-1.672								6	-2.106	2.106
(AHY8) A.H.S.V	1	3.715	-3.715				1	0	0			0	-4.466	4.466					
	2	-3.715	3.715				2	0	0			1	4.466	-4.466					
	3	0	0				3	0	0										
	4	0	0				4	0	0										
(BHA8) B.H.A.S	0	3.253	-3.253				1	2.332	-2.332								0	-2.719	2.719
	1	-3.253	3.253				2	-2.332	2.332								1	2.719	-2.719
							3	0.583	-0.583										
							4	0.442	-0.442										

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 11. PARAMETER ESTIMATES FOR SECOND ORDER EFFECTS IN MODELS FITTED ON DATA FROM JANUARY 1, 1964 THROUGH JULY 31, 1968.

MODEL	AGE	SEASON	RATIO OF THE LOG-LINEAR PARAMETERS (SECOND ORDER)					
			YEAR	SEASON	1	2	3	4
(ACRS) AS,X,C	1	1	-2.336	2.336				
		2	2.336	-2.336				
	2	1	-1.121	1.121				
		2	1.121	-1.121				
	3	1	2.19	-2.19				
		2	-2.19	2.19				
	4	1	0.96	-0.96				
		2	-0.96	0.96				
(ACTS) AS,C	1	1	-1.756	1.756				
		2	1.756	-1.756				
	2	1	0	0				
		2	0	0				
	3	1	0	0				
		2	0	0				
(ASCS) AS,X,H	1	1	-1.994	1.994				
		2	1.994	-1.994				
	2	1	-1.275	1.275				
		2	1.275	-1.275				
3	1	1.98	-1.98					
	2	-1.98	1.98					
4	1	0.957	-0.957					
	2	-0.957	0.957					
(BIDS) B,H,XS	1	1						
		2						
		3						
		4						
		5						
		6						
	2	1						
		2						
		3						
	3	1						
		2						
		3						
	4	1						
		2						
		3						
	5	1						
		2						
		3						
6	1							
	2							
	3							

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 11. PARAMETER ESTIMATES FOR SECOND ORDER EFFECTS OF MODELS FITTED ON DATA FROM JANUARY 1, 1964 THROUGH JULY 31, 1969 (cont.)

MODEL (ABYS) AB,AYS	AGE	SEASON	RAB FOR		YEAR	SEASON		RAB FOR	
			0	1		0	1	0	1
	1	1	-2.665	2.665	1	1	0.14	-0.14	
	2	1	2.665	-2.665	2	1	0.126	-0.126	
	3	1			3	1	1.311	-1.311	
	1	2	-0.902	0.902	4	1	-0.748	0.748	
	2	2	0.902	-0.902	5	1	1.33	-1.33	
	3	2			6	1	0.764	-0.764	
	1	3	1.112	-1.112	1	2	0.721	-0.721	
	2	3	-1.112	1.112	2	2	0.077	-0.077	
	3	3			3	2	0.966	-0.966	
	1	4	1.830	-1.830	4	3	0.817	-0.817	
	2	4	-1.830	1.830	5	3	0.109	-0.109	
	3	4			6	3	-1.108	1.108	
	1	3			1	3	2.349	-2.349	
	2	3			2	3	1.433	-1.433	
	3	3			3	3	0.962	-0.962	
	4	3			4	3	0	0	
	5	3			5	3	0	0	
	6	3			6	3	-2.782	2.782	
	1	4			1	4	-1.178	1.178	
	2	4			2	4	-0.976	0.976	
	3	4			3	4	-1.982	1.982	
	4	4			4	4	0.065	-0.065	
	5	4			5	4	-0.038	0.038	
	6	4			6	4	1.579	-1.579	

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 12. RESULTING MODELS AND P VALUES FOR THE STEPWISE MODEL SELECTION PROCESS ON THE DATA FROM JANUARY 1, 1988 THROUGH JULY 31, 1989 AND FROM JANUARY 1, 1984 THROUGH JULY 31, 1989.

<u>MODEL</u>	<u>1988-1989 model</u> <u>p value</u>	<u>1984-1989 model</u> <u>p value</u>
(BHXS)	(BHXS) B (0.13)	(BHXS) XS,BH (0.085)
(AHYS)	(AHYS) A,H (0.06)	(AHYS) H,A (0.09)
(BHYS)	(BHYS) B (0.10)	(BHYS) YS,BH (0.17)
(BCYS)	(BCYS) B (0.07)	(BCYS) B,C,YS (0.04)
(ABYS)	(ABYS) A,B (0.01)	(ABYS) A,B (0.02)
(BCAS)	(BCAS) B,A (0.04)	(BCAS) B,A (0.01)
(ACYS)	(ACYS) AS,C (0.05)	(ACYS) C,A (0.02)
(AHZS)	(AHZS) H,Z,A (0.01)	(AHZS) H,Z,A (0.01)
(ACXS)	(ACXS) A,C (0.02)	(ACXS) A,C (0.02)
(AHXS)	(AHXS) H,A (0.22)	(AHXS) H,A (0.22)
(BHAS)	(BHAS) B,H,A (0.35)	(BHAS) B,H,A (0.35)
(BCXY)	(BCXY) B,X (0.30)	(BCXY) X,B,Y (0.06)
(BHXY)	(BHXY) B (0.11)	(BHXY) BH,Y,X (0.27)
(ACXY)	(ACXY) A,C (0.25)	(ACXY) A,C (0.25)
(BCAY)	(BCAY) B,A (0.09)	(BCAY) B,A (0.09)
(ACHZ)	(ACHZ) A,H,Z (0.32)	(ACHZ) A,H,Z (0.32)
(ABXY)	(ABXY) A,B (0.09)	(ABXY) A,B (0.09)

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 13. CONDITIONAL TESTS FOR EACH TERM IN THE FINAL 17 SUBSET MODELS.

MODEL	CONDITIONAL TESTS			p Value
	TERM	GQI	df	
(BHX) B,H,X	B	58.92	1	0.0005
	H	24.7	1	0.0005
	X	6.31	15	0.0005
	S	5.89	12	0.0005
	X	4.33	12	0.0005
(BCAS) B,A,S,C	C	7.9	2	0.01 > p < .025
	S	42.34	3	0.0005
	A	21.7	1	0.0005
	B	53.85	1	0.0005
(ABX) A,B,X	X	26.13	3	0.0005
	B	53.82	1	0.0005
	A	18.41	1	0.0005
(ACY) A,C,Y	Y	16.59	3	.001 > p < .01
	X	37.3	3	0.0005
	C	28.85	2	0.0005
	A	36.72	1	0.0005
(BCAY) B,A,Y,C	C	7.52	2	.025 > p < .01
	A	28.11	1	0.0005
	Y	28.57	3	0.0005
	B	59.11	1	0.0005
(ACYS) A,S,C	C	28.11	2	0.0005
	A	61.47	4	0.0005
	S	55.81	6	0.0005
	AS	106.91	7	0.0005
(ACXS) A,S,X,C	C	23	2	0.0005
	X	33.22	3	0.0005
	AS	88.86	7	0.0005
	A	47.26	4	0.0005
	S	52.39	6	0.0005
(BCXY) B,X,Y	Y	17.49	3	0.0005
	X	36.99	3	0.0005
	B	76.3	1	0.0005
(ACHZ) A,H,Z	Z	152.28	4	0.0005
	H	65	1	0.0005
	A	27.55	1	0.0005
(AHS) A,S,X,H	H	62.28	1	0.0005
	X	25.95	3	0.0005
	A	35.41	4	0.0005
	S	44.96	6	0.0005
	AS	68.2	7	0.0005
(AHS) A,H,Y,S	S	26.41	3	0.0005
	Y	13.37	3	0.0005
	H	83.91	1	0.0005
	A	24.38	1	0.0005

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 13. CONDITIONAL TESTS FOR EACH TERM IN THE FINAL 17 SUBSET MODELS (cont.).

MODEL	CONDITIONAL TESTS			p value
	TERM	SSQ	df	
(BCY) B,C,Y,S	S	45.85	3	0.0005
	Y	13.85	3	.005 > p < .001
	C	8.67	2	.025 > p < .01
	B	86.6	1	0.0005
(BHY) B,H,S	S	42.28	3	0.0005
	H	28.73	1	0.0005
	B	84.17	1	0.0005
(BXY) B,H,X,Y	Y	18.99	3	0.0005
	X	24.81	3	0.0005
	H	25.48	1	0.0005
	B	50	1	0.0005
(ABY) A,S,B,Y,S	B	89.29	1	0.0005
	S	11.83	3	.025 > p < .01
	A,S	33.08	4	0.0005
	Y,S	32.29	12	.005 > p < .001
	A,S,Y,S	189.61	19	0.0005
(BHS) B,H,S,A	A	12.81	1	0.0005
	H	25.32	1	0.0005
	S	37.27	3	0.0005
	B	45.98	1	0.0005
(AHZ) A,H,Z,S	S	29.21	3	0.0005
	Z	159.86	4	0.0005
	H	89.18	1	0.0005
	A	21.65	1	0.0005

SEE TABLE 1 FOR EXPLANATION OF CODES.

TABLE 14. PROBABILITY VALUES FOR EACH TERM IN THE MODEL, ABXS.

MODEL	SEASON	BEHAVE	BITED	AGE	BAR POS		TOTAL	PROB
					0	1		
ABXS, A,BX,B	1	1	0	1	6.1	8.9	7	0.13
				2	18.5	4.5	20	0.23
			1	1	2	2	4	0.5
				2	4.1	7.9	12	0.06
		2	0	1	3.6	2.4	6	0.4
				2	8.5	10.5	19	0.55
			1	1	2.9	4.1	7	0.50
				2	2.8	7.2	10	0.72
		5	0	1	0	0	0	0
				2	1.6	2.4	4	0.6
			1	1	1.4	2.6	4	0.65
				2	2.2	7.8	10	0.78
	6	0	1	2.2	0.8	3	0.27	
			2	13.5	0.5	23	0.41	
		1	1	1.9	3.1	5	0.62	
			2	2.8	8.2	11	0.75	
	2	1	0	1	16.5	1.5	18	0.08
				2	13.6	2.4	16	0.16
			1	1	4.3	2.7	7	0.39
				2	3.7	4.3	8	0.54
		2	0	1	12.1	4.9	17	0.29
				2	36.9	28.1	65	0.43
			1	1	2.7	2.3	5	0.46
				2	13	21	34	0.62
		5	0	1	0	0	0	0
				2	6.6	6.4	13	0.38
			1	1	0.5	0.5	1	0.5
				2	7.8	17.2	25	0.69
	6	0	1	6.1	1.9	10	0.19	
			2	18.1	7.9	26	0.3	
		1	1	5.6	5.4	11	0.40	
			2	8.5	15.5	24	0.65	
	3	1	0	1	48.6	2.4	51	0.05
				2	22	2	24	0.08
			1	1	8.3	2.7	11	0.25
				2	9.2	5.8	15	0.39
		2	0	1	24	5	29	0.17
				2	23.6	9.4	33	0.28
			1	1	6.2	2.8	9	0.31
				2	15.2	12.8	28	0.46
		5	0	1	0	0	0	0
				2	4	2	6	0.33
			1	1	1.2	0.8	2	0.4
				2	11.8	13.4	25	0.54
	6	0	1	11.8	1.4	13	0.11	
			2	9.8	2.2	12	0.18	
		1	1	8	4	12	0.33	
			2	11.7	11.3	23	0.40	
4	1	0	1	23	1	24	0.04	
			2	16.7	1.3	17	0.08	
		1	1	1.5	0.5	2	0.25	
			2	17.9	10.1	28	0.38	
	2	0	1	14.3	2.7	17	0.16	
			2	26	9	34	0.26	
		1	1	7.8	3.2	11	0.29	
			2	18.4	12.8	29	0.43	
	5	0	1	4	1	5	0.2	
			2	4.8	2.2	7	0.31	
		1	1	3.2	1.8	5	0.36	
			2	13.2	13.8	27	0.51	
6	0	1	2.7	8.3	3	0.1		
		2	14.9	3.1	18	0.17		
	1	1	5.5	2.5	8	0.31		
		2	16	14	30	0.47		

SEE TABLE 1 FOR EXPLANATION OF CODES.

VITA

The author was born on October 22, 1956, in Steubenville, Ohio. Her parents moved to Charleston, West Virginia, in 1967, where they still remain, and she still calls home. She attended West Virginia University, Morgantown, WV, where she received her Bachelor of Science degree in Animal Science, magna cum laude, in 3 years. In 1982, she received her Doctor of Veterinary Medicine degree from The Ohio State University, School of Veterinary Medicine, Columbus, Ohio. From 1982 to June 1985, she practiced small animal medicine and surgery in two hospitals in Charleston, WV. At the same time, she organized a pet-facilitated therapy program with the local obedience club and established pet visitations to several local nursing homes, while supervising the medical care of the animals in the program. In June 1985, she became the State Veterinarian for the West Virginia Racing Commission at the newly opened Tri-State Greyhound Racetrack in Charleston, WV., where she supervised drug testing of the racing greyhounds, as well as physical exams, and emergency care for injured animals. In the fall of 1987, she entered Virginia Polytechnic Institute and State University to pursue a PhD in veterinary medical science at the Virginia Maryland Regional College of Veterinary Medicine, with an emphasis in public health and epidemiology. She has continued her interest in the human-animal bond by establishing a support group for pet owners who have lost their pets due to illness or who have pets with terminal illnesses. She is a member of the Delta Society, the Conference of Public Health Veterinarians, and the Association of Teachers of Veterinary Preventive Medicine and Public Health.

A handwritten signature in black ink, reading "Mary E. Trueman", written over a horizontal dashed line.