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ADULT RACCOON SURVIVAL IN AN ENZOOTIC RABIES AREA OF PENNSYLVANIA

C. L. Brown[1,3], C. E. Rupprecht[1,4] and W. M. Tzilkowski[2]

[1] The Wistar Institute of Anatomy and Biology, Philadelphia, Pennsylvania 19104, USA
[2] School of Forest Resources, the Pennsylvania State University, University Park, Pennsylvania 16802, USA
[3] Present address: West Virginia Department of Natural Resources, French Creek, West Virginia 26218, USA
[4] To whom reprint requests should be sent

ABSTRACT: Twenty-one adult raccoons (*Procyon lotor*) were radio-marked on each of two areas in Centre County, Pennsylvania from 17 June to 23 August 1987. Raccoons on Area 1 were vaccinated with a commercial inactivated rabies virus vaccine administered intramuscularly, whereas on Area 2 raccoons were not vaccinated. Survival rates were estimated for three periods: pre-season (23 August to 23 October 1987), harvest season (24 October 1987 to 23 January 1988) and post-season (24 January to 26 March 1988). Kaplan-Meier survival rates (±SE) were 1.00 ± 0.00 for both areas during the pre- and post-season periods. Survival rates during the harvest period were 0.67 ± 0.11 and 0.69 ± 0.11 for Area 1 and Area 2, respectively. Survival rates between the two areas were not different ($P = 0.929$). During 23 August 1987 to 26 March 1988, rabies was not an apparent factor in raccoon survival. Conclusions regarding timing an oral rabies vaccination campaign based upon occurrence of rabies-related mortalities could not be presented because of the lack of obvious rabies mortality. However, our findings, combined with information about immunization, vaccine distribution, and peak periods of raccoon rabies, suggest a late winter or early spring vaccination period would be optimum for reducing the number of raccoons susceptible to rabies.

Key words: Raccoon, *Procyon lotor*, survival, survival rate estimates, rabies, oral vaccination, field study.

INTRODUCTION

Determination of the influence of rabies upon survival rates is necessary for understanding raccoon (*Procyon lotor*) population dynamics in epizootic and enzootic rabies areas. Harvest of furbearers, such as raccoons, is a population management tool, often rationalized as a disease control measure (Storm and Tzilkowski, 1982). However, the impact of disease upon wildlife populations, and the interaction of disease and harvest is poorly understood because ecological and epizootiological aspects of disease often are considered separately (Voight and Tinline, 1982).

The Mid-Atlantic region of the United States currently is experiencing a raccoon rabies epizootic (Jenkins and Winkler, 1987). Raccoons accounted for 77% of reported animal rabies cases from Virginia, Maryland, and Pennsylvania in 1987 (Centers for Disease Control, 1988). In Pennsylvania, reported raccoon rabies cases increased from 13 during 1900 to 1981, to >1,600 during 1982 to 1988 (Wampler and Kirkland, 1981; Centers for Disease Control, 1989). This epizootic has raised concern among public health and natural resource agencies because of human exposure to rabies, public fear, increased submissions to diagnostics laboratories, and wildlife and domestic animal losses. Because of economic, public health, and wildlife management implications due to this epizootic, potential wildlife rabies control methods are being re-evaluated.

Historically, attempts to control wildlife rabies were limited to reduction of vector populations through hunting, trapping, poisoning, gassing, and bounty programs (Lewis, 1975). However, population reduction has not proven effective for long term wildlife rabies control (Lewis, 1975; MacDonald, 1980). Oral wildlife immunization has been studied intensively as an alternative measure (e.g., Baer et al., 1971). Results from European field trials, where oral attenuated rabies vaccines were used,

indicate that local eradication of an epizootic rabies front may be feasible (Wandeler, 1988; Schnieder et al., 1988). Unfortunately, raccoons have been refractive to routine oral immunization using methodology and vaccines designed primarily for red fox (*Vulpes vulpes*) rabies control (Rupprecht et al., 1986). In response, an oral immunization program using recombinant subunit vaccine for raccoon rabies control currently is being developed (Rupprecht et al., 1986, 1987).

Development of an oral immunization program for raccoon rabies control requires consideration of many critical components. The primary components of an oral immunization program are development of a safe, efficacious vaccine and a method for distributing vaccine within an edible bait, although many other factors could contribute to the success or failure of an immunization program. Aside from the primary components, timing of vaccination campaigns is potentially the most important component in determining the success of an immunization program. Estimation of raccoon survival rates allows interpretation of the intensity and seasonal variation in mortality, both of which will critically affect the timing of vaccine delivery. Accurate timing of vaccine delivery is necessary for optimal immunization of susceptible animals to achieve maximum levels of immunity and a reduction in rabies transmission. Therefore, determination of survival rates, and the subsequent effect on vaccine delivery, is an additional step in the process of developing a feasible and effective methodology for the control of wildlife rabies.

Our objective was to determine survival rates of free-ranging raccoons vaccinated with a commercial inactivated rabies virus vaccine and unvaccinated raccoons in an enzootic rabies area of Pennsylvania.

MATERIALS AND METHODS

Two study areas were selected on State Game Lands in Centre County, Pennsylvania. Area 1 (40°59′N, 77°46′W) (440 ha), was located approximately 6 km northeast of Milesburg. Area 2 (40°50′N, 77°53′W) (370 ha), was located approximately 5 km northwest of State College and 20 km southwest of Area 1.

Centre County, as recently as 1982 and 1983 had no reported cases of raccoon rabies. In 1985 and 1986, Centre County had 21 and 29 reported raccoon rabies cases. Study areas were selected because they were considered to be on the leading edge of the Mid-Atlantic rabies epizootic based upon reported raccoon rabies cases.

Raccoons were captured during four 14-day sessions from 17 June to 23 August 1987 using live-traps (Model 207, Tomahawk Live Trap Company, Tomahawk, Wisconsin 54487, USA) set in semi-permanent locations, and baited with fresh fish. Captured raccoons were immobilized with a combination of 10 mg/kg ketamine hydrochloride (Bristol Laboratories, Syracuse, New York 13201, USA) and 1 mg/kg xylazine (Haver-Lockhart Laboratories, Shawnee, Kansas 66201, USA) administered intramuscularly (i.m.). Sex was determined and age was estimated by weight, penis extrusion, and teat characteristics, according to the criteria of Sanderson (1961). Raccoons were weighed, examined for physical abnormalities, and ear-tagged. A 3–5 ml blood sample was obtained by jugular venipuncture for determination of baseline rabies virus neutralizing antibody, as described by Rupprecht et al. (1986). Additional blood samples were collected if raccoons were recaptured ten or more days since the last blood collection. Raccoons captured on Area 1 were vaccinated i.m. with 1.0 ml of commercial inactivated rabies virus vaccine (Rabguard-TC, Norden Laboratories, Lincoln, Nebraska 68501, USA). Twenty-one adult raccoons on each area were fitted with a 150–152 Mhz transmitter attached to a collar (Lotek Engineering Inc., Aurora, Ontario, Canada L4G 4J9). Transmitters contained a time-delay mortality sensor activated after three hours of inactivity. Radio-marked raccoons were monitored at least on a weekly basis from a vehicle outfitted with an omnidirectional antenna and portable receiver (Advanced Telemetry Systems, Isanti, Minnesota 55040, USA). Locations of live raccoons were not determined. However, raccoons with a transmitter in mortality mode were located to determine if death had occurred.

Survival rates and survival distributions of adult raccoons were estimated using the Kaplan-Meier product limit estimator (Kaplan and Meier, 1958), and calculated with the Statistical Analysis System (SAS) procedures PHGLM and SURVFIT (SAS Institute, Inc., 1986). Estimates were calculated for pre-season, harvest season, and post-season. Each respective period consisted of 62 days (23 August to 23 October 1987),

93 days (24 October 1987 to 23 January 1988), and 62 days (24 January to 26 March 1988). Survival rates also were estimated for the entire study period, 23 August 1987 to 26 March 1988. Difference in survival rates, between areas, for the entire period was tested with the Wilcoxon-Gehan rank test using the SAS procedure SURVDIFF (SAS Institute, Inc., 1986).

Pollock et al. (1989) describe use of the Kaplan-Meier estimator for estimation of survival rates and distributions using radio-telemetry data. Survival estimates calculated from the Kaplan-Meier method are essentially probability estimates of an individual animal surviving during a specified time interval. Advantages of the estimator are that the assumption of a constant survival probability for all time intervals is not required and observations from animals with an unknown fate can be included in estimates. Observations of individuals with unknown fates are censored observations. Censored observations may result from transmitter failure, emigration, and survival beyond the end of the study period. Assumptions of the Kaplan-Meier estimator are (1) radio-marking does not affect survival, (2) radio-marked animals are representative of the population, (3) survival time for each animal is independent, and (4) the censoring mechanism is random and independent.

RESULTS

During 18 June 1987 to 26 March 1988, nine radio-marked adult raccoons were reported dead or recovered from Area 1 and six radio-marked adult raccoons were reported dead from Area 2. The difference in recorded mortality between males and females was not significant on Area 1 (Fisher's exact probability = 0.639) or on Area 2 (Fisher's exact probability = 0.166).

Observations of three raccoons from Area 1 were not included in the analyses because death occurred prior to 23 August 1987, the date the last raccoon was radio-marked. Also, observations of three raccoons from Area 2 were censored because of signal loss prior to the end of the study. During 23 August 1987 to 26 March 1988, 92% (n = 12) of adult raccoon mortality was due to harvest; 75% of harvest mortality occurred during 18 to 27 November 1987.

Survival rates (±SE) were 1.00 ± 0.00 for both areas during the pre- and post-season periods. Survival rates during the harvest period were 0.67 ± 0.11 and 0.69 ± 0.11 for Area 1 and Area 2, respectively. Because mortality of radio-marked raccoons occurred only during the harvest season, survival for the entire period (23 August 1987 to 26 March 1988) was equivalent to that of the harvest period. Survival rates did not differ between the 2 study areas from 23 August 1987 to 26 March 1988 (Wilcoxon-Gehan χ^2 = 0.01, 1 df, P = 0.929). Of the 15 dead raccoons, four were trapped, seven were shot, one was killed by an automobile, and the cause of death for three raccoons was undetermined. Two recovered raccoons were submitted to a diagnostics laboratory for rabies testing; both were negative for rabies virus.

Paired serum samples, pre- and post-vaccination available for eight raccoons from Area 1 demonstrated seroconversion for seven of the eight (88%) raccoons (Brown and Rupprecht, 1990). Baseline geometric mean titres for rabies virus neutralizing antibody were 0.11 and 0.08 IU/ml for Areas 1 and 2, respectively, whereas the post-vaccination geometric mean titre for the seven raccoons exhibiting seroconversion was 2.37 IU/ml.

DISCUSSION

Johnson (1970) reported a majority of raccoon mortality is from human activity, such as, hunting, trapping and automobile-raccoon collisions. Other sources of mortality included predation, starvation and disease. Although Johnson (1970) suggested distemper as the only disease that had a major role in regulating raccoon populations, rabies has been recognized as an important mortality factor in the southeastern United States since 1960 (Kappus et al., 1970), and more recently in the Mid-Atlantic region. Although we expected rabies to be a major mortality factor, it was not an apparent factor in raccoon survival during our study. We attributed the lack of rabies mortality to at least three possible factors: (1) rabies was present, but im-

munization or natural resistance prevented obvious transmission: (2) rabies was present, but undetected; or (3) rabies was not present.

Vaccinated raccoons on Area 1 may have been refractive to rabies, as exhibited by seroconversion of some vaccinates. In addition, virus neutralizing antibody levels from raccoons on Area 2 ranged from 0.01–6.00 IU/ml in 1985 (C. E. Rupprecht, unpublished data); however, baseline titers were seronegative (all ≤0.22 IU/ml) in 1987. Therefore, surviving raccoons may have developed natural immunity as suggested by McLean (1975), but with waning antibody titers. Raccoon rabies was present in Centre County during 1987, and cases were reported within 5 km of both areas (Bureau of Animal Industry, Summerdale Laboratory, Summerdale, Pennsylvania 17093, USA).

Although definite conclusions regarding the timing of vaccination campaigns based on survival estimates and immunization were not possible because of the lack of rabies mortality; the process of induction allows presentation of a potential strategy for timing vaccine delivery. If immunization is effective for at least 6 mo, and population densities are subsequently reduced during a fall harvest, an early spring or late winter vaccination period would result in an effective period of 9 mo or more. A reduced probability of rabies transmission should result because of a reduction in the number of susceptible animals. A vaccination strategy exploiting the spring parturition and lactation periods of raccoons may also result in maximal levels of immunity through passive antibody transfer to kits (Rupprecht and Keiny, 1988). Additionally, aerial distribution of vaccine-laden baits (Johnston et al., 1988) would be facilitated prior to spring leafout. A spring vaccination period is further warranted if peak submission periods for rabies diagnostic testing are considered. Submissions of raccoons for rabies testing peak in spring and early fall (Jenkins and Winkler, 1987). If incubation time for rabies is considered, then peak submissions may be associated with previous periods of high raccoon activity, such as during the breeding season in late winter (Sanderson and Nalbandov, 1973) and during fall dispersal (Urban, 1970). An early spring or late winter rabies vaccination campaign would therefore be optimum for effectively reducing the number of susceptible raccoons through a combination of immunization and harvest.

More information is needed regarding the interactions between mortality factors and immunization in areas where rabies occurs. Further study using similar techniques and analyses should be undertaken for raccoons in other areas and other furbearer populations (e.g., fox and skunk) preparatory to, or in conjunction with oral rabies vaccine trials.

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