Wildlife Rabies in the United States: Recent History and Current Concepts

Author: McLEAN, ROBERT G.

Source: Journal of Wildlife Diseases, 6(4) : 229-235

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-6.4.229

Wildlife Rabies in the United States: Recent History and Current Concepts

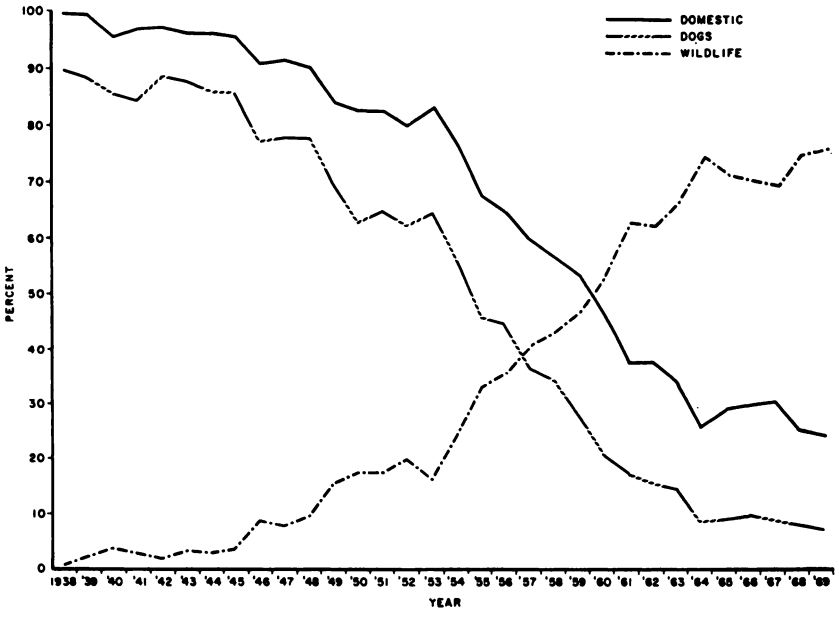
ROBERT G. McLEAN

National Communicable Disease Center, Atlanta, Georgia

History and Distribution

Wildlife rabies in the United States has a long and varied history. Animal rabies has been present at least since 1753 in Virginia,[17] and an epizootic of fox rabies occurred in Massachusetts in 1812.[15] Major epizootics in wildlife have been recognized throughout the country within the last 100 years.[15] Since statistical data on the incidence of rabies in the United States were first compiled in 1938 by the U.S. Department of Agriculture,[31] the total number of reported cases of rabies has declined more than 50 percent. Most of the decline occurred during the 1950's. Before this date, wildlife rabies was obscured by the prevalent dog rabies cases.[20] However, during the last 20 years, dog rabies and wildlife rabies have reversed their relative positions in the annual summaries of reported cases (Figure 1). Dog rabies has declined from over 60 percent to less than 10 percent of total rabies cases while

Figure 1
REPORTED INCIDENCE OF RABIES IN THE UNITED STATES, 1938-1969



wildlife rabies has increased a similar amount. This dramatic decline in dog rabies is even more significant when the substantial increase in the dog population in the United States during this period is considered.[16]

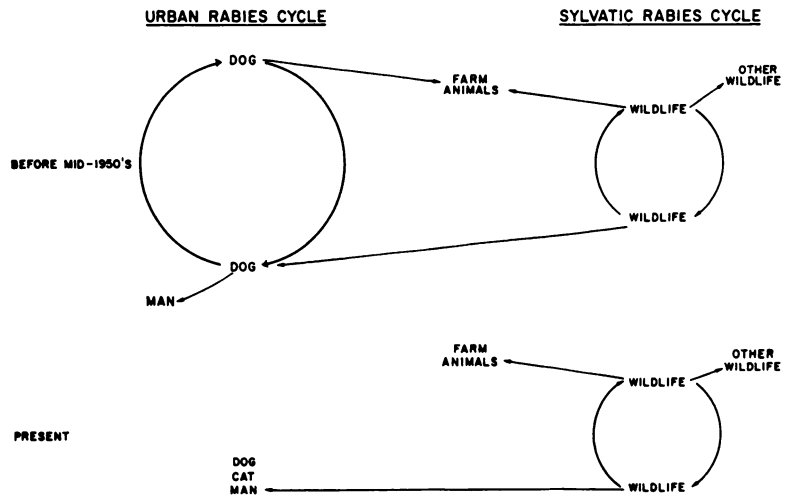
Since 1957, more cases of rabies have been reported in wildlife than in dogs, and in 1960, wildlife cases surpassed the total cases in domestic animals. During 1969, wildlife rabies comprised 76 percent of all rabies cases.

The proportional (Figure 1) and actual increases (44 cases in 1938[21] to 2672 cases in 1969) in reported wildlife rabies probably represent a change in reporting emphasis. When the urban rabies cycle in dogs was being limited and now practically eliminated by control programs (principally vaccination[21]), public interest shifted to the sylvatic rabies cycle.[12] The sylvatic cycle has probably not changed much in the last 20 years except now it is most likely the source of infection for all cases of rabies in the United States (Figure 2). The urban dog rabies cycles were undoubtedly initiated by transfer of virus from the sylvatic cycles and, therefore, represented only extensions of rabies in wildlife to an incidental host in which the virus was quite virulent.[19]

The major hosts of reported wildlife rabies have changed during the last 17 years (Figure 3). During the 1950's, there was a shift from a predominance of fox rabies to skunk rabies by the early 1960's. Recently, the relative positions of these two species have stabilized at about 50 percent of total wildlife cases for skunks and 25-30 percent for foxes. The percentage of bat rabies cases has steadily increased since it was first recognized in 1953 (this parallels the increasing awareness of bat rabies) until the number of cases also has leveled off during the last 5 years. A few rabid raccoons were reported sporadically throughout the country until the 1960's when they emerged as a major host in Florida and later in Georgia. An epizootic of raccoon rabies that occurred in Florida in 1969 is discussed by Kappus, *et al.*[21]

The geographical distributional patterns of wildlife rabies have expanded during the last decade. Skunk rabies was reported principally in the upper mid-west, Texas, and Central California at the beginning of the decade, and gradual expansion in all

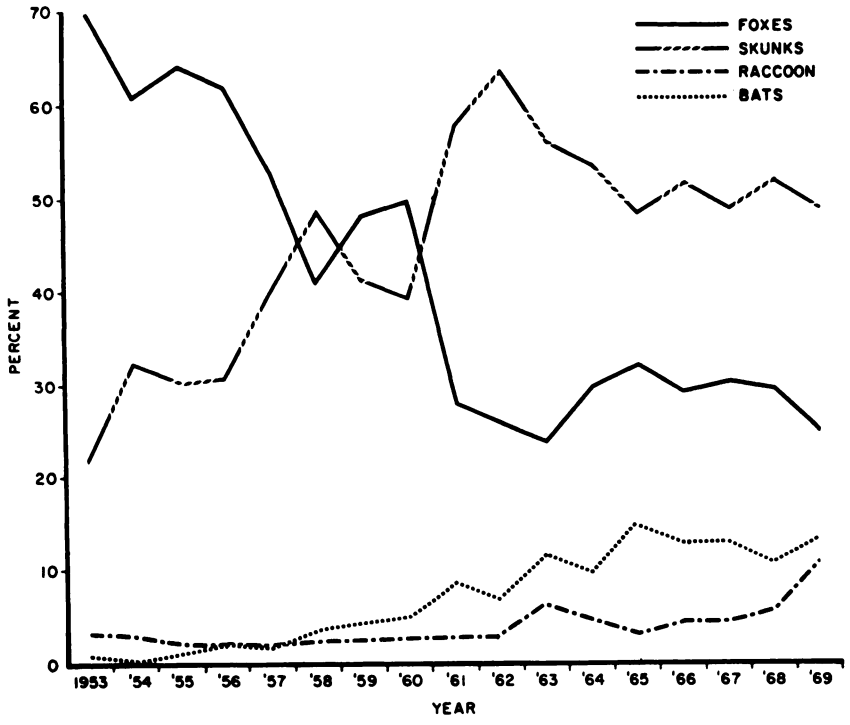
Figure 2
CHANGE IN THE RABIES CYCLES IN THE U.S. IN THE LAST 20 YEARS



directions occurred. The westward spread in the Great Plains stopped temporarily at the Continental Divide in the Mountain States, but has recently extended into Arizona and across the mountains to Utah.[25] Fox rabies has been present in relatively stable foci in the Appalachian region with temporary extensions into adjacent areas and in central Texas. The skunk and fox species zones have been virtually independent with some overlap along the Ohio River. Until recently, no important cycles in skunks existed where fox rabies was predominant, and vice versa. However, skunk rabies has become epizootic in northeastern Tennessee,[25] and there appear to be two independent cycles or a fox-skunk cycle in northeastern New York and central Missouri. During 1969, Clinton County, New York, reported 39 cases of fox rabies and 17 cases of skunk rabies, which is similar to what occurred in 1964,[14] and Cole County, Missouri, reported 12 skunk cases and 15 fox cases. Whether these localized outbreaks which occurred in two species simultaneously represent a change in the host species isolation where the virus seems to circulate only within a single species in a specific area[14,20,25] cannot be determined yet. Shifts of the major host species have been reported in several areas of New York[14] and Texas.[20] Changes in the distributional patterns of raccoon rabies[21] and bat rabies[3] are reviewed elsewhere. Different reasons for the distinct geographic host species zones have been given.[19,27,32,36] However, the reliability and discreteness of these geographic zones is based only upon

Figure 3

INCIDENCE OF WILDLIFE RABIES BY SPECIES, UNITED STATES, 1953 - 1969



a few studies[32,36,37,39] and the recorded distribution as determined by the county rabies reporting system. The reported data have been shown to lack quantitative and possibly even qualitative sensitivity on wildlife rabies.[23,29,37]

Ecology

The disease ecology of wildlife rabies has remained obscure because of the superficial and fragmentary approaches to the study of the disease. From the public health viewpoint, only the epizootiology of wildlife rabies is considered because of the greater public interest involved. The putting out of fires is necessary, but has little effect upon the underlying smoldering of rabies virus activity.

Rabies most likely exists naturally in wild animal species in well-defined enzootic foci like many of the other zoonoses.[1,2,13] Numerous authors have discussed their view that rabies virus is not maintained in wildlife by an acute, lethal cycle but rather by a balanced host-parasite system utilizing one or more reservoir mechanisms.[12,18,20,29,30,34,36] In the enzootic state, a slow, continuous circulation of the virus within the maintaining host population must occur by subclinical, mild and chronic, or lethal but prolonged infections. The proportion of infected animals of a particular host population that must possess this perpetuating type of infection to maintain an enzootic focus may vary according to the host species, biocenose, or virus infectivity and pathogenicity.

Changes in virus infectivity and pathogenicity for a particular host population may occur under certain conditions such as: overcrowding, nutritional deficiencies, other physically deteriorating conditions such as from other disease, reproductive and migratory activity, and others. Intra-specific serial passage and inter-specific transfer of virus may also result in changes. After these changes, rabies virus infections may be expressed more often in a lethal (acute, clinical) form by termination of chronic or prolonged infections or during initial infections in individual animals[4,9,33,34,36] or as an epizootic in animal populations.[6,7,8,9,15,24] The amplification in the form of an epizootic is more a result of all of the conditions of the natural host population than just increased physical contact due to higher population density[9,11,14,27] or the introduction of virus into a virgin population. Animals are more susceptible to pathogens while under stressful conditions[6,8,9,10,33] as a result primarily of depression of the active defense mechanisms.[5,7,34,28,38] Thus, animal populations that reach an imbalance with their natural habitat and an expansion of competition among animals results are more likely to experience epizootics. The enzootic form of wildlife rabies maintains the virus in definite foci for prolonged periods, whereas the lethal or epizootic form aids in amplification, dispersion, and inter-specific transfer of the virus. The epizootic also serves as a natural control mechanism on population size of many wildlife species.[8,24]

The host species that maintain the enzootic foci by intra-specific reservoir mechanisms are most likely the same as the major hosts of wildlife rabies reported from those areas, and the hosts that support epizootics. It has also been suggested that other host species serve as reservoirs of virus for these major hosts.[18,20,29,30] However, not enough information is available to support or exclude any of the possible reservoir mechanisms.

Control

Wildlife rabies control programs have utilized population reduction methods (increasing mortality) almost exclusively to combat wildlife rabies. The general lack of success of this approach has been due to improper or incomplete application of control techniques, insufficient knowledge and use of the biology of the target

species and the disease ecology of wildlife rabies, and attempts to coordinate control efforts with only the generally insensitive reporting of rabies cases. Until effective immunization of the wildlife hosts can be achieved, management of the host population at suitable densities by a combination of a population reduction method to increase mortality (trapping and poisoning) applied prior to the breeding season and a chemosterilant given to the survivors or a chemosterilant program alone appear to be the best approaches.[22]

Conclusion

Knowledge of the natural history (disease ecology) of wildlife rabies is quite fragmentary, because most studies have emphasized information from small groups of animals or only for specific parameters of all the various parameters involved in the disease ecology of rabies.

Abdussalam[1] states that "the absence of accurate knowledge concerning the ecology of wild reservoir hosts has been the most important reason for failures or hesitation to deal effectively with such important diseases as rabies, plague, tularaemia, yellow fever, and many other arthropod-borne diseases". Improvement of the present reporting of rabies cases and establishment of a wildlife rabies surveillance or monitoring system will be forthcoming in the near future. However, these systems are limited and can at best only describe epizootiological trends. To understand the ecology of wildlife rabies, in-depth and comprehensive investigations, in which basic ecological data on host populations and the interrelationships with their biotic and abiotic environments are collected, are required in each of the distinct foci to unravel the complexities of the disease system. Only a cooperative multi-discipline, team approach can accomplish the broad ecological objectives of such studies.

Literature Cited

1. ABDUSSALAM, M. 1959. Significance of ecological studies of wild animal reservoirs of zoonoses. Bull. Wld. Hlth. Org. 21: 179-186.
2. AUDY, J. R. 1958. The localization of disease with special reference to the zoonoses. Trans. Royal. Soc. Trop. Med. Hyg. 52 (4): 308-334.
3. BAER, G. M., and D. B. ADAMS. "A review of the incidence of rabies in bats in the United States, 1953-1965." Accepted for publication, Public Health Reports, 1970.
4. BURNS, K. F., C. F. FARINACCI, T. G. MURNANE, and D. F. SHELTON. 1956. Insectivorous bats naturally infected with rabies in Southwestern United States. Am. J. Pub. Hlth. 46: 1089-1097.
5. BURNS, K. F., D. F. SHELTON, J. M. LUKEMAN, and E. W. GROGAN. 1960. Cortisone and ACTH impairment of response to rabies vaccine. Pub. Hlth. Rep. 75 (5): 441-445.
6. CHITTY, D. 1960. Population processes in the vole and their relevance to general theory. Can. J. Zool. 38 (1): 99-113.
7. CHRISTIAN, J. J. 1963. Endocrine adaptive mechanisms and the physiologic regulation of population growth. *In* "Physiological Mammalogy" Vol. I. Eds. W. Mayer and R. Van Gelder. Academic Press. pp. 189-353.
8. CHRISTIAN, J. J., and D. E. DAVIS. 1964. Endocrines, behavior, and population. Sci. 146: 1550-1560.
9. DAVIS, D. E. 1960. Role of density in populations of mammalian plague reservoirs. Bull. Wld. Hlth. Org. 23: 417-418.

10. DAVIS, D. E., and C. P. READ. 1958. Effect of behavior on development of resistance in trichinosis. Proc. Soc. Exp. Biol. and Med. 99: 269-272.

11. DAVIS, D. E., and J. E. WOOD. 1959. Ecology of foxes and rabies control. Pub. Hlth. Rep. 74 (2): 115-118.

12. FENJE, P. 1968. Advances in rabies research. Can. J. Pub. Health 59 (6): 217-228.

13. FERRIS, D. H., and R. H. ANDREWS. 1967. Parameters of a natural focus of *Leptospira pomona* in skunks and opossums. Bull. Wildl. Dis. Assn. 3 (1): 2-10.

14. FRIEND, M. 1968. History and epidemiology of rabies in wildlife in New York. N.Y. Fish and Game Jour. 15 (1): 71-97.

15. GIER, H. T. 1948. Rabies in the Wild. J. Wildl. Mgmt. 12 (2): 142-153.

16. HELD, J. R., E. S. TIERKEL, and J. H. STEELE. 1967. Rabies in man and animals in the United States, 1946-65. Pub. Hlth. Rep. 82 (11): 1009-1018.

17. JOHNSON, H. N. 1959. Rabies. Pp. 405-431 *In* T. M. Rivers and F. L. Horsfall (eds). *Viral and Rickettsial Infections of Man.* 3rd Ed. J. B. Lippincott Co., Philadelphia.

18. JOHNSON, H. N. 1959. The role of the spotted skunk in rabies. 63rd Ann. Proc. of the U.S. Livestock Sanitary Assn. 8 pp.

19. JOHNSON, H. N. 1965. Rabies virus. pp. 814-840. F. L. Horsfall and I. Tamm (Eds.) *In* Viral and Rickettsial Infections of Man. 4th Ed. 1282 pp.

20. JOHNSON, H. N. 1966. Sporadic cases of rabies in wildlife: relation to rabies in domestic animals and character of virus. Proc. Natl. Rabies Symposium, Atlanta. pp. 25-30.

21. KAPPUS, K. D., R. G. McLEAN, and H. A. TREVINO. An emerging wildlife rabies vector. To be published.

22. LORD, R. D. 1970. Vertebrate ecology in public health. Pub. Hlth. Rep. 85 (2): 105-111.

23. McLEAN, R. G., K. K. KAPPUS, and H. A. TREVINO. Comparison of reported rabies with wildlife serology in two Florida counties. To be published.

24. MYERS, K., I. D. MARSHALL, and F. FENNER. 1954. Observations of two succeeding epizootics in Australian wild rabbits on the Riverine plain of southeastern Australia, 1951-1953. J. Hyg. 52 (3): 337-360.

25. NATIONAL COMMUNICABLE DISEASE CENTER. 1968. Annual Rabies Surveillance Report.

26. PARKER, R. L. 1969. Epidemiology of rabies. Arch. Environ. Health 19: 857-861.

27. PARKER, R. L., J. W. KELLY, E. L. CHEATUM, and D. J. DEAN. 1957. Fox population densities in relation to rabies, N.Y. Fish and Game Jour. 4 (2): 219-228.

28. PLUMMER, G., P. H. CLEVELAND, and G. STEVENS. 1967. *Herpes simplex* virus and paralysis of rabbits. J. Exp. Med. 58: 415.

29. SCATTERDAY, J. E., N. J. SCHNEIDER, W. L. JENNINGS, and A. L. LEWIS. 1960. Sporadic animal rabies in Florida. Public Health Rept. 75 (10): 945-953.

30. SCHNEIDER, N. J., J. E. SCATTERDAY, A. L. LEWIS, W. L. JENNINGS, H. A. VENTERS, and A. V. HARDY. 1957. Rabies in bats in Florida. Amer. J. Pub. Hlth. 47 (8): 983-989.

31. SCHOLTENS, R. G., and E. S. TIERKEL. 1963. Incidence of animal rabies in the United States. J. Amer. Vet. Med. Assoc. 143 (1): 52-56.

32. SIKES, R. K. 1962. Pathogenesis of rabies in wildlife. I. Comparative effect of varying doses of rabies virus inoculated into foxes and skunks. Amer. J. Vet. Res. 23: 1041-1947.

33. SOAVE, O. A. 1964. Reactivation of rabies virus in a guinea pig due to stress of crowding. Am. J. Vet. Res. 25: 268-269.

34. SOAVE, O. A., H. N. JOHNSON, and K. NAKAMURA. 1961. Reactivation of rabies virus infection with adrenocorticotropic hormones. Sci. 133 (3461): 1360-61.

35. TIERKEL, E. S. 1958. Part IV. Recent developments in the epidemiology of rabies. N.Y. Acad. Sci. Ann. 70 (3): 445-448.

36. VERTS, B. J. 1967. *The biology of the striped skunk.* Univ. Illinois Press, Urbana and Chicago, 218 pp.

37. VERTS, B. J., and G. L. STORM. 1966. A local study of prevalence of rabies among foxes and striped skunks. J. Wildl. Mgmt. 30 (2): 419-421.

38. VESSEY, S. H. 1964. Effects of grouping on levels of circulating antibodies in mice. Proc. Soc. Exp. Biol. and Med. 115: 252-255.

39. WOOD, J. E., and D. E. DAVIS. 1959. The prevalence of rabies in populations of foxes in the southern states. J. Amer. Vet. Med. Assn. 135: 121-124.