A COMPUTER MODEL OF WILDLIFE RABIES EPIZOOTICS AND AN ANALYSIS OF INCIDENCE PATTERNS

by

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INTRODUCTION

"Rabies is an acute infectious disease caused by a filterable virus" (Johnson 1959a:405). It is "primarily an infection of the canine race ... but may affect any of the warm blooded animals. Secondarily it is a disease of man" (Kelser 1955:250).

The history of rabies in domestic species is long and well documented. Kelser (1955:250) cited reports of rabies as early as the 13th century B. C. in Europe. Spangler (1966:1) called the disease "one of the oldest, one of the most dangerous, and one of the most stubborn plagues of both man and animal."

THE RABIES PROBLEM

The importance of rabies cannot be overemphasized. Acha (1966:140) reported 2407 human rabies deaths in the western hemisphere from 1954 to 1965. Sikes (1967:4) noted 1964 rabies deaths in the world from 1962 to 1963, but estimated that twice that number went unreported.

But the danger of human deaths is not as important as the economic consequences of rabies. Held et al. (1967:1017) reported that 30,000 persons annually receive the expensive immunization treatment, 200 in Virginia alone (Marx and Swink 1963:173). Livestock deaths, however, account for the greatest economic losses. During the first half of 1969 at least 269 farm animals were lost to rabies in the United States. Virginia lead the nation in cattle rabies for this period with 50 reported lossed (Baker 1969). These figures do not reflect the total impact of rabies upon livestock, however. Parker (1961:278) felt that many cases of livestock rabies are simply not reported due to the expense and difficulty of packaging and shipping the heads of large animals for laboratory examination. The total

economic loss in livestock, then, may be several times that indicated by laboratory confirmations.

Wildlife Rabies

Several authors (Merriam 1966; Friend 1968:82) have recognized the positive relationship between rabies deaths in domestic animals and rabies epizootics in local wildlife. Prior (1969:1) cited rabies as a public health problem, an agricultural problem, and a wildlife problem. The latter problem is becoming increasingly important.

The existence of rabies in North American wildlife prior to the arrival of the white man is uncertain. Verts (1967:149) thought that if it did exist it was probably prevalent in the members of the Canidae, due to their circumpolar distribution. The earliest records of animal rabies in the United States are from an epidemic in dogs in Virginia in 1753 (Johnson 1959a:406). Gier (1948:143) reported a fox rabies epizootic in Massachusetts in 1812 and Johnson (1959b:270) stated that the earliest reports of rabies in the spotted skunk were from California in 1826. Up until the last decade, before vaccine came into widespread use, rabies continued to be considered primarily a disease of domestic dogs. Wildlife rabies is currently a problem in Europe (Serakowa 1968) and in Vietnam (Baker 1967:7-8).

In recent years researchers have begun to recognize the increasing importance of wildlife rabies (Tierkel 1954:1; Steele 1967:264). Washburn (1966:4) noted the conversion of rabies from an urban to a rural problem. National statistics reveal that wildlife has accounted for the most rabies cases for at least nine consecutive years (Baker 1968:1) and the same trend continues (Baker 1969:1). The incidence of rabies in wildlife species

appears to have increased as vaccination programs have become more effective in combatting the disease in domestic dogs (Sikes and Tierkel 1960:1).

Tierkel (1954:1) stressed the importance of three wildlife-man rabies relationships. The first, wildlife transmission of rabies to man by direct contact, occurs infrequently. However, Washburn (1966:4) has pointed out the increased probability of this danger in the near future with the current increases in leisure time and increases in contact between the American public and wildlife species. The second relationship, rabies transmission from wildlife to domestic species to man, is of more immediate concern to public health workers. This route is well documented (Friend 1968; Serakowa 1968) and is the basis for the third significant aspect of wildlife rabies, eradication. Eradication of rabies in domestic animals and man ultimately depends upon the control of the disease in wildlife species.

Rabies Eradication

D. H. Spangler (1966:1) in his address to the 1966 National Rabies Symposium articulated the need for the complete eradication of rabies. He felt that eradication of any disease is superior to mere control. It is the "best protection" we can have, he said, and is now economically feasible. Through technology and research man has conquered a large number of once-serious diseases including brucellosis, foot-and-mouth disease, vesicular exanthema, glanders, yellow fever, typhoid, diptheria, and polio (Spangler 1966:1; Washburn 1966:5-6). If these diseases could be eradicated there is little reason why energies might not now be turned to the complete elimination of rabies. Through strict quarantine and control measures, 36 contries, islands, and territories are now reported as entirely free of rabies (Baker 1968:6). It seems probable that the United States

could be added to this list if public support can be mustered for a research and eradication program.

Such a program must be national in scope (Spangler 1966:1). Besides public education, most authorities feel that research is the first and most important step in an eradication scheme (Washburn 1966:5). Meyer (1955:2), in discussing the control of zoonoses, felt that eradication is "much less a problem of wiping out overt disease than of learning the usual habits of infective agents and then dealing with what are often ineradicable mammalian, bird, or arthropod resevoirs." With the investigation of wildlife vaccination techniques as suggested by Marx (1966:120) and Washburn (1966:5), these resevoirs may no longer remain inconquerable. Nelson (1966) listed the current needs in rabies research. These include more effective techniques for reducing the population of susceptible animals, figures for the proper degree of reduction, the study of a latent carrier state, and research concerning the effects of density stress.

A SYSTEMS APPROACH TO RABIES

The basis of an effective understanding of the epidemiology of any disease is a comprehensive view of the entire complex of factors affecting its dissemination. Rabies is no exception. Detailed knowledge of only a few factors, such as etiology or pathogenesis, is not enough. Ultimate effectiveness of control strategies will depend upon an ecological viewpoint, a holistic approach encompassing not only the factors affecting disease, but also their interrelationships. In short the study of disease must be the study of a system, a term defined by Watt (1968) as "an interlocking complex of processes characterized by many reciprocal cause-effect pathways." The status of our current knowledge of rabies is relatively

firm in the realm of domestic animals and man. Vaccination and control programs have been most effective in reducing rabies incidence in this sector. Wildlife rabies and its relationship to domestic rabies are, however, areas in which scientists are comparatively ignorant. Complete rabies control will depend upon the analysis of this disease system and its related processes as a unified whole.

System analysis according to Hayne (1969:120) must start with the construction of a model large and complex enough to adequately describe the system in question. The first step in the analysis of the disease system of rabies, then, should be the construction of a comprehensive model. Ecologists have long been utilizing the process of modeling in their studies. However, the resulting models have been, for the most part, mental abstractions of reality. These foster an intuitive understanding of the complex interactions of the natural world, but often lack the concreteness of precise quantification necessary for authoritative analysis.

In recent years the techniques of modeling and simulation by means of the electronic computer have been vastly improved. The tremendous bookkeeping capacity of the computer makes it the ideal device for simulation. Large files of information can be quickly examined for the status of many factors. The effects of status changes on variables of interest may easily be computed. This entire process may be repeated over many short periods. Computer modeling is often a more desirable method of analysis than is experimentation with actual components of a system in question, such as rabies. It is less expensive and nondestructive of actual system elements. Little risk to humans or to ecosystem components is involved as a result of improper or speculative decisions. The modeling process does

not take as long as would the experiments required to yield the same information, utilizing only minutes of computer time as opposed to days or years of experimentation. The effects of extreme situations, difficult to reproduce in reality, are easily simulated by computer. Of course it may not be possible to examine satisfactorily all facets of a system by computer simulation alone. Field experiments and verification of results go hand in hand with model building and are the foundation of improved data inputs.

Abstract models possess two inherent values. The first, and most obvious, is their predictive value. Hypothetical situations may be arranged and system performance may be observed for each set of situation specifications. The second value of modeling is perhaps the more important. The very act of designing a computer model of a system is a valuable discovery process. The necessity of simplifying and abstracting the processes and elements comprising a system and mathematically describing their interactions often leads to new insights of relationships never before considered. The elimination of extraneous complexity often reveals facts previously hidden to the experimenter.

It is with this second value in mind that the experimenter chose to approach the study of wildlife rabies by means of simulation. The objectives of this study were twofold. The first was to examine records of reported rabies incidences in ecologically divergent areas and thereby establish a graphical model of the temporal and spatial patterns, if any, which characterize wildlife epidemics. The second objective was to incorporate this information and the sum of current knowledge concerning wildlife rabies into a viable, computer-based simulation of the disease's dynamics. Such a model might provide a most useful tool for the decision-maker involved in the execution of optimum rabies research and control strategies.

LITERATURE REVIEW

Rabies is an encephalitic viral disease of endothermic animals (Johnson 1959a:405). The virus itself is double-walled and elongated (Matsumoto 1962:199). It is 250 to 400 millimicrons in diameter, having surface projections about 100 Angstrom units in length (Almeida et al. 1962:148-149). Johnson (1959a:415-416) stated that the virus is resistant to antibiotics, but is quickly destroyed by strong acids or bases, formalin, and sunlight.

Characteristic inclusion bodies, termed Negri bodies, are considered diagnostic (Rhodes and van Rooyen 1962:401).

WILDLIFE SPECIES AFFECTED

Verts (1967:145) stated that rabies is restricted to mammals under natural conditions. It is most commonly reported in members of the Mustelidae, Canidae, Procyonidae, Vespertilionidae, and Molossidae.

The disease is most commonly reported in the striped skunk (Mephitis mephitis) (Verts 1967). It does affect spotted skunk (Spilogale putorius) populations, but not to the same degree (Johnson 1959b;25).

The next most commonly reported wildlife family with rabies is the Canidae. Rausch (1958:247), in a study of Alaskan rabies, reported the disease in the red fox (Vulpes fulva), the arctic fox (Alopex lagopus), and the timber wolf (Canis lupus). The coyote (Canis latrans) is occasionally reported as infected in the western United States (Baker 1968). Prior (1969:31) reported the gray fox (Urocyon cinereoargenteus) as the species primarily affected in Virginia.

Together the skunks and the foxes have accounted for over 80 percent of the total wildlife rabies reports in the United States (Baker 1968:1).

Bat rabies is assuming increasing importance in the United States. First reported in 1953 (Venters et al. 1954:18), bat rabies has been confirmed in 25 of the 40 known species (Constantine 1965:252). Baer and Bales (1967) stated that rabies virus may be disseminated by hemataphagous, frugivorous, and insectivorous bats.

Other species which are occasionally reported as rabid are the ocelot (Felis pardalis), oppossum (Didelphis marsupialis), raccon (Procyon lotor), bobcat (Lynx rufus), badger (Taxidae taxus), woodchuck (Marmota monax), gopher (Citellus spp.), and coati (Nasua narica) (Baker 1968). Toro (1966: 131) reported the mongoose (Viverridae) as a major reservoir in Puerto Rico. Ballantyne and O'Donoghue (1954) reported rabies in caribou (Rangifer spp.) and weasels (Mustela erimina). In addition Baker (1967) has noted rabies in various species of deer, rabbits, and small rodents.

WILDLIFE RESERVOIRS

The exact nature of the wildlife reservoirs of rabies is relatively unknown. Johnson (1966:29), in discussing wildlife reservoirs, thought that the long-term wildlife host of rabies would be a species which did not ordinarily assume a role in epidemic outbreaks. Verts (1967:153-154) on the other hand, stated that reservoir species could be major participants in epizootics and might be expected to display active rabies symptoms.

The species considered to be the major rabies reservoirs in the United States are bats (all species), the striped skunk, red and gray foxes, and the ermine. These species act as hosts in distinct enzootic areas, the fox being the major vector in the East, the skunk in the central states, and both species serving as the reservoir in the West (Steele, personal communication 1970). Johnson (1959a:405) suggested that the "permanent hosts" of rabies are members of the Mustelidae.

Several researchers (Sanderson et al. 1967:92; Verts 1967:145) have considered and rejected the possibility of rabies being perpetuated by a multispecies complex in any one area. They stated that the ecological and behavioral barriers between species during interepizootic periods, the mechanical barriers to transmission, and the attenuation of virus after several passes through the same species rule out such a possibility.

Both Prior (1969:39) and Held et al. (1967) named the fox (red and gray) as the primary reservoir in the southeastern United States.

The striped skunk is considered the major rabies reservoir in California (Johnson 1966:28), and in the Midwest (Tierkel 1958: 445, 1959:196; Verts 1967:169).

Several authors (Sulkin 1962:496-497; Sikes 1967:2) have stated that one or more species of bats may serve as a wildlife rabies reservoir. Pawan (1936:401-422) found vampire bats to be asymptomatic rabies carriers in Central and South America. The bats were able to transmit the virus over extended time periods. Fredickson and Thomas (1965:497) attempted unsuccessfully to correlate fox rabies with the predominance of bat caves in Tennessee. The role of bats as a reservoir species is, however, controversial. Many authors hold the opinion that bat rabies, at least in North America, is independent of other wildlife species (Constantine 1967a:885; Friend 1968:92).

Raccoons, although susceptible to rabies, apparently do not serve as a reservoir for other species (Sikes 1967:2; Verts 1967:156; Friend 1968:90). The bite of a rabid raccoon will often fail to cause rabies in another animal and it is hypothesized that this species may denature or inactivate the virus in some way (Steele, personal communication 1970).

Rodents apparently do not serve as a rabies vector. Winkler (1966: 36) stated that rodents "are not particularly susceptible to rabies infection" and, when infected, do not normally show virus in their salivary glands. Tierkel (1959:198) found no evidence of rabies in over 1000 rodents collected in epizootic areas of New York and Georgia.

No arthropod vectors have been established. Bell et al. (1957:282) found that the virus lasted only a short time in ticks.

A number of authors (Marx 1966; Verts and Storm 1966) have held the opinion that wildlife rabies reports may be biased and unrepresentative of the actual degree of involvement of certain species as reservoirs. Johnson (1959b:271-274) stated that minks and weasels may be more important in rabies epizootiology than reports indicate. Weasels are one of the few species common to all endemic areas and may be the long-term rabies host, even though the frequency with which this species is reported is not high (Johnson 1966:29).

The discreteness of enzootic rabies areas of foxes versus skunks is still unexplained. Population levels of either species appear unrelated to the species which serves as a reservoir (Parker 1961:274; Parker and Wilsnack 1966:33-34; Friend 1968:95). Tentative explanations of the geographical predominance of one species rather than another include ecological or behavioral isolation of the species in question (Verts and Storm 1966:420; Verts 1967:156; Friend 1968:93) and variable species susceptibility to different virus strains or doses (Sikes and Tierkel 1960:272; Prior 1969:39). Sikes and Tierkel (1960) presented evidence that foxes infected with small doses of rabies virus would possess saliva of insufficient infectivity to affect skunks, but could transmit the disease to other

foxes. Sikes (1962:1046) stated that the greater infectivity of skunk saliva might completely overwhelm foxes and make them ineffective as carriers. Prior (1969:40) suggested that red foxes are less susceptible to rabies and produce a more virulent virus than do gray foxes. The saliva of a red fox would be more likely than that of a gray fox to initiate a rabies infection in a striped skunk, a relatively insusceptible species. For this reason the red fox is the more likely of the two species to be associated with the striped skunk in rabies epizootics.

RABIES SYMPTOMS IN WILDLIFE

Conventional disease texts recognize two types of rabies symptoms in animals. The "furious" condition is characterized by agitation and aggressiveness, as opposed to the relative inactivity of the "paralytic" condition (Kelser 1955:263). The symptoms are variable, however, and intermediate forms are recognized (Richards 1957:4). Different manifestations of rabies are related primarily to the host species (Verts 1967:147). Rabid skunks are often characterized by their extremely aggressive behavior (Richards 1957:4; Verts 1967:168). In early stages of symptoms, striped skunks are confused and lethargic. Their ability to expel musk is inhibited (Verts 1967:168-169). They are active at any hour and not nocturnal, as under normal conditions (Parker 1961:274).

Symptoms in bat species are more variable. Baer and Bales (1967) noted a wide variety of symptoms in Mexican freetail bats and found classification difficult.

LATENT SYMPTOMS

Verts (1967:148-149) recognized four possible courses of rabies in naturally infected animals. Besides death occuring several days after a

"normal" incubation period and the complete absence of symptoms or virus, he listed two chronic stages. The virus may become latent, only to be reactivated by external stimuli at a later date. Or the infected animal may display no symptoms, but produce infective saliva for a period of several months. Bell (1965) characterized chronic rabies in dogs as falling into one of four categories:

1) delayed onset of symptoms
2) clinical symptoms, followed by recovery
3) clinical disease of long duration
4) apparently healthy carrier state

He cited evidence of one or more of these categories being confirmed in experimental rats and rabbits, bats, skunks, foxes, and man. Taylor and Knowelden (1964:111) recognized three types of disease carriers - convalescent, healthy, and intermittent.

Asymptomatic carrier states have been demonstrated in bats (Pawan 1936; Sulkin et al. 1960; Sulkin 1962) and experimental animals (Fischman and Ward 1968:137). Johnson (1966:27) found that the virus of certain skunks produced asymptomatic infections in adult mice. Recovery from rabies has been recorded (Johnson 1959a:410; Merchant and Packer 1961:796), even in man (Kraut 1966:224), and might lead to the second carrier state cited by Bell (1965).

Reactivation of latent rabies infections is thought by some (Verts 1967:175-176) to be the key to rabies perpetuation in wildlife during interepizootic periods. The role of stress and trauma in precipitating disease symptoms is generally well documented (Turner 1960; Rhodes and van Rooyen 1962:81). Rabies-specific literature is also filled with evidence

suggesting the role of crowding and competition stress in activation of latent rabies infections (Bell 1965:171; Johnson 1965:823; Soave 1964: 268). Sulkin et al. (1960:613) found that rabies virus was demonstrable in aroused bats, but not in hibernating individuals. Moore and Raymond (1970:168) hypothesized that development of previously undemonstrated rabies symptoms in a naturally infected bat was the result of the stress of captivity.

INCUBATION PERIODS AND DURATION OF SYMPTOMS

Kelser (1955:262) stated that the length of rabies incubation periods in animals was related to the species of the virus donor and recipient, the location and extent of wounds, the quantity of virus transferred, and the virulence of that virus. Sikes (1962:1043) likewise found an inverse correlation between the quantity of virus injected and the length of the incubation period in experimental animals. Johnson (1959b:273), in referring to wildlife species, related length of virus incubation to the mode of transmission. As mentioned earlier, prolonged incubation may be the result of reactivation of latent virus (Koprowski 1952:963).

For purposes of modeling wildlife rabies epidemiology the current disease literature was searched in order to obtain reliable estimates of the limits and distributions of incubation and symptom duration in the species to be considered. The results are presented in Tables I and II.

Virus may appear in saliva before the actual display of clinical symptoms. Baer and Bales (1967:85) found rabies virus in the saliva of Mexican freetail bats up to 12 days prior to the onset of symptoms. Sikes (1962: 1043-1044) reported virus in skunk saliva up to 5 days before and 9 days after the appearance of symptoms.

Table I. Rabies incubation periods in selected animal species.

Species	Reference	Incubation Period (days) minimum	maximum	median
Dog	Johnson (1948)	10	180	21-56
Fox	Sikes (1962)	12	109	-
Fox	Parker & Wilsnack (1966)	14	57	25
Fox	Steele (1967)	-	395	-
Skunk	Parker & Wilsnack (1966)	14	172	42
Vampire bat	Pawan (1936)	9	38	-
Mexican freetail bat	Baer & Bales (1967)	14	181	-
Big brown bat	Moore & Raymond (1970)	-	209	-

Table II. Periods of clinical rabies symptoms in selected animal species.

Species	Reference	Duration of Symptoms (days) minimum	maximum	median
Dog	Johnson (1948)	-	10-12	-
Fox	Parker & Wilsnack (1966)	1	17	7
Skunk	Parker & Wilsnack (1966)	1	18	7
Mexican freetail bat	Baer & Bales (1967)	4	11	-

RABIES TRANSMISSION

Bite transmission has traditionally been assumed to be the major mode of rabies dissemination in mammalian species (Johnson 1959a:405; Verts 1967:145-146). However, recent evidence has suggested that this route is not completely responsible for the perpetuation of interepizootic rabies infections in wildlife populations (Verts 1967:171). Tierkel (1959:191) found that only 54 to 90 percent of laboratory animals dying from rabies carried rabies virus in their salivary glands. Other tissues have been found to be affected by the disease (Johnson 1966:29).

Besides bite transmission of rabies, urine transmission of the virus is a distinct possibility (Johnson 1959a:417), especially in bats (Steele, personal communication 1970).

Rabies transmission by ingestion of the virus has been demonstrated and could be a factor in natural populations. Johnson (1959a:415-416) stated that rabies virus infectivity is retained for 1 to 2 weeks in animal tissues exposed to air at room temperature. Kelser (1955:277) and Johnson (1959b:273) have reported evidence of rabies transfer from mother to young by milk. Serakowa (1969) confirmed food-borne rabies infections in experimental animals and suspected abrasions of the lips, gums, or intestinal tract to be the means of viral entry. However, Soave (1966: 45-46) and Fischman and Ward (1968:136) demonstrated infection from ingested virus in the absence of oral abrasions. Infection by means of oral routes required large doses of virus, as would be carried in the brains of wild species (Fischman and Ward 1968:136).

Aerosol transmission of rabies by means of urine and saliva droplets in bat caves has been reported and is considered a possible means of viral

transfer from bats to other wildlife (Constantine 1962:289; McLean, personal communication 1969). Constantine (1967b) was able to demonstrate rabies infection in a large number of wildlife species caged in bat caves, including the opossum, a species relatively resistant to the disease. Verts (1967:155), however, held that the requisite conditions for aerosol transmission occur too infrequently to make it a major source of wildlife infection. Winkler (1966) indicated that bats may contract rabies through aerosol transmission, but Baer and Bales (1967: 87) and Sulkin et al. (1960) indicated that the insectivorous species are relatively insusceptible to intranasal infection, except by large doses of virus.

The possibility of in utero transfer of rabies from a mother to her young has been confirmed by investigations (Remlinger 1919:378; Verts 1967:173-176). Konradi (1916:37-46) wrote that in utero transmission was possible, but that the rabies virus would be modified in the process. Sims et al. (1963:25) have demonstrated in utero rabies transfer in bats, as have Bell et al. (1965) in white mice.

Infectivity rates for natural transmission modes have not been determined, but it appears that not all individuals exposed to rabies contract infection. Sikes (1962:1046) found antibodies produced in only 1 of 12 skunks and 4 of 12 foxes experimentally infected. Verts (1967:172-173) noted seasonal differences in the sex ratios of rabid skunks, leading him to hypothesize the role of stress in increasing the susceptibility to infection. Tierkel (1959:193) stated that, in general, older animals were less susceptible to rabies infection than juveniles.

CYCLIC RABIES INCIDENCE

Schoening (1956:201) stated that rabies is a density dependent disease, the prevalence of which is directly related to the density of susceptible hosts. Numerous authors have related the seasonal peaks of rabies incidence to population peaks in foxes (Rausch 1958:255; Marx and Swink 1963:172; Friend 1968:71) and in skunks (Parker 1961:277-278; Verts 1967:169-176).

However, Verts (1967) wrote that other factors were involved in rabies peaks because, even though skunk populations were at a high point, the crowding effects of winter clumping were at a minimum. Serakowa (1968) noted a rabies peak in European foxes during April and May, coinciding with the increased stresses of estrus in females. Friend (1968: 82-93) demonstrated a peak incidence in New York foxes in late winter and early spring, a period of increased movement and food gathering. He also recorded a lesser peak in the fall while family dispersal was occurring. Johnston and Beauregard (1969:367-368) noted two rabies peaks in Ontario red foxes. One occurred in females during March, coinciding with parturition. The other involved juvenile males and took place in early fall, while this group was under the stress of dispersal. Sanderson et al. (1967:92) hypothesized the role of the stresses of lactation and pregnancy in the increased susceptibility of female striped skunks. Peaks in bat rabies have been associated with the fatigue and stress of migration (Constantine 1967a:873; Friend 1968:92; Prior 1969:42).

Johnston and Beauregard (1969:367) noted a three-year rabies cycle in Ontario foxes. Prior (1969:31) suggested that such cycles might be attributed to rabies immunity acquired by the young from their mother.

The degree of immunity would decrease with each successive litter, but several generations might pass before a population could regain a high degree of susceptibility.

In their discussion of disease cycles Taylor and Knowelden (1964: 265) suggested different explanations based on the pattern of incidence. "Periodic recurrence" of a disease, normally at intervals of more than one year, was attributed to the density of susceptible individuals. Seasonal factors of stress were presented as a possible explanation for disease peaks separated by approximately one year.

SPATIAL RELATIONSHIPS IN WILDLIFE RABIES

It has been demonstrated that rabies incidences are not spread homogeneously over large land areas. They appear to be, in fact, clustered about certain foci (Verts 1967:164; Serakowa 1968). Little is known about the ecological reasons for the clustered patterns of incidence which result.

The movements of rabid wildlife are also relatively unstudied. Gier (1948:150) thought that rabid foxes probably do not travel far after developing rabies symptoms. He concluded that rapid spread of the disease would not be the result of only a few infected individuals. Rabid skunks may travel up to three quarters of a mile or farther (Parker 1961:275). Storm and Verts (1966:705-708) stated that the movements of rabid skunks were not significantly different from those of noninfected individuals of similar age and sex.

WILDLIFE RABIES REPORTING

National rabies statistics are compiled from animal incidence reports issued by individual state health departments. The researcher

using such reports as a basis for determining patterns of rabies incidence should be aware of the biases to which they are subject.

Meyer (1955:4) stated that rural disease reporting depends largely upon the initiative of the individual owners of affected animals. Constantine (1967a:885) felt that reporting frequency with respect to wildlife rabies is proportional to human population density. Prior (1969: 34) disagreed, asserting that the influence of individual health officers would mask the effect of population density on reporting frequency. Wildlife rabies reporting is excellent in suddenly prominent rabies areas (Parker 1961:278), but drops as the disease becomes "old hat". Marx (1966:119) cited local news media as playing an important role in rabies reporting. Parker (1961:278) discussed reporting bias with respect to species. He stated that skunks, due to their unpleasant odor, were less likely than other wildlife species to be submitted for laboratory examination. Wildlife are not submitted unless rather positive symptoms have been demonstrated. The result is a relatively higher percentage of positive laboratory confirmations in wildlife species than in domestic animals (Prior 1969:36-37).

WILDLIFE RABIES CONTROL

According to Meyer (1955:23), rabies control in rural areas may be accomplished by two means - supervised killing of wildlife to reduce animal-to-animal contacts and preinfection vaccination of domestic animals. With respect to wildlife population reduction, Schnurrenberger et al. (1964:165) stated that "arithmetic reductions" in the density of wildlife should result in a "geometric reduction of contact probability." Assuming that this principle does hold and that population reduction is

helpful, a certain knowledge of the proper degree of reduction is necessary (Tierkel 1954:1). Population reduction may not be as effective in the control of wildlife rabies as was once thought. Marx (1966) indicated that Virginia's fox trapping program appears ineffective and Nelson (1966) warned of instances where trapping has been shown to stimulate reproduction.

Rabies control options other than trapping are open to control agencies. They include population reduction by means of controlled poisons, chemosterilants, and wildlife rabies vaccination (McLean, personal communication 1969). All of these areas are relatively unexplored, however.

Rabies control requires intensive application of the selected techniques. The economic requirements of wildlife rabies control, especially in wilderness areas, may make it impractical at the present time (Rausch 1958:258-259). Unwise control measures applied without thorough knowledge of their consequences may adversely affect delicate natural communities. Constantine (1967b:29) has warned of the severe ecological repercussions which may result from the indiscriminate destruction of bat caves as reservoirs of rabies vectors.

MODELS AND SIMULATION

The exploration of computer modeling and its application to research in industry and the sciences is a relatively new field, having been possible only with the recent advent of computer systems of suitable storage capacity. In the few decades of the development of computer simulation techniques applications to a number of fields have been conceived. These include urban planning (Wilson 1968; Forrester 1969), industrial engineering (Pritsker and Kiviat 1969; Pritsker and Burgess 1970), medicine (Meier et al. 1964; Priban 1968), general ecology (Garfinkel 1962; Watt

1968), forestry (O'Regan et al. 1965; Newnham 1967), and fisheries management (Southward 1968). Orcutt et al. (1961) used an IBM 704 computer to model the social and economic structure of American commerce in minute detail. Waggoner and Horsfall (1969) have written a FORTRAN IV simulator of plant disease. The simulation of the management of white-tailed deer herds by means of computer has recently been explored (Hayne 1969; Riffe 1970). Davis (1967) constructed a general mathematical model of deer herd management by the use of dynamic linear programming techniques. Watt (1968) discussed a wide variety of ecological computer models of population dynamics, dispersal, predation, and disease. A comprehensive review of modern computer simulation techniques is available in references by Churchman et al. (1957) and Naylor et al. (1966).

The earliest recorded attempts at modeling disease were made by William Farr in 1840 as cited by Bailey (1957:7-8). Farr successfully fitted a normal curve to data concerning the quarterly smallpox death rate in England. In 1866 he used the same curve-fitting technique to predict the course of a rinderpest outbreak in cattle, but met little success. The observed and predicted curves were bell-shaped, but displayed little agreement. The basis of most future disease modeling work was provided by Hamer (1906). He demonstrated that, in general, the course of an epidemic depended upon the number of susceptible individuals in a population and the contact rate between infectives and susceptibles. The reduction of susceptibles by disease and slow subsequent increases in the size of this group were presented as a possible explanation for the cyclic disease occurrence. Bailey (1957:8) cited the work of Soper

(1929) who attempted to explain recurrent epidemics. Soper's analysis was deterministic in nature, not allowing for variations due to chance. Bartlett (1957) used stochastic processes for the same purpose and achieved results agreeing more closely to actual data.

Stochastic techniques offer an added degree of complexity to disease simulation. Bailey (1957:10) named McKendrick (1926) and Greenwood (1931) as early pioneers in stochastic epidemic modeling. Bartlett (1957) presented a stochastic model of measles. Watt (1968:333-340) has written a FORTRAN program which describes the general disease model of Kermack and McKendrick (1927). His program allows for additional factors including:

1) immigration and emigration
2) variable infectivity rates due to weather
3) up to four species of animals
4) stochastic assumptions

Recent mathematical models for measuring the rate of spread of hypothetical epidemics to fixed groups of individuals have been constructed by Haskey (1957) and Becker (1968). A complete review of historical models used in the science of epidemiology is presented by Bailey (1957) and Bartlett (1960).

Rabies reservoirs have been demonstrated in several wildlife species, notably skunks, foxes, and bats. Rabies incubation periods and symptoms are highly variable, but it has been hypothesized that a latent form of the disease is responsible for its interepizootic maintenance. The bite route is the major mode of virus transmission, but other methods are possible under conditions of crowding and stress. Wildlife rabies control

has been studied little and consists of stopgap, emergency measures. Models of other infectious diseases have been successfully constructed and have been most valuable in determining research and control strategies. A similar model of rabies epidemiology might also serve to indicate priorities for such strategies.

METHODS

Two distinct approaches were undertaken to describe wildlife rabies epizootics. The first method used was to analyze health department records of confirmed rabies reports in several counties where the disease had reached epidemic proportions. The objective of this approach was to describe temporal and spatial relationships between rabies incidences in wildlife populations. A comparison of these patterns for similarities or differences between ecologically dissimilar areas was intended to more closely pinpoint the factors affecting the spread of rabies.

A second approach to the study of wildlife rabies was to construct a stochastic model of the disease, a simulation adapted to the electronic digital computer. Even above the predictive value of such a model would be its value as an important heuristic device for rabies study. With this heuristic objective in mind, a simulation scheme was designed to be as general in nature as possible. It was constructed so that a minimum of reprogramming would be necessary in expanding the simulation to accommodate more wildlife species, other possible transmission modes, modified incubation periods, or iteratively changing probabilities.

ANALYSIS OF RABIES REPORTS

Complete listings of positive laboratory rabies reports were obtained from three Virginia counties (Bedford, Nelson, and Washington) selected for their high rabies incidence. These reports were collected by staff of the Virginia Department of Public Health under the supervision of Mr. John R. Beck, Bureau of Sport Fisheries and

Wildlife, Division of Wildlife Services. They included the date and place at which the rabid animal was found, the name of the person submitting the specimen, and the species of the animal infected. In addition, data from Imperial County, California, were obtained from the National Communicable Disease Center for analysis as a high rabies area of a vastly different ecological nature. Table III summarizes the content of these reports.

The initial step in the analysis of the patterns of reported rabies incidences for each county was the plotting of each rabies occurrence on a map and visual examination of the spatial arrangement of reports.

Secondly, time patterns were analyzed on a traditional epidemiologic basis, the cumulative percentage of the total rabies cases reported per week being graphed by county.

Finally the time and space relationships between reports were analyzed simultaneously by county, in an attempt to demonstrate significant patterns of "subsequent" and "precedent" reports as first explored by Schnurrenberger (1968). The times (days) and distances (miles) from each report to every other report occurring at a later date were recorded, then punched on IBM cards. By means of a FORTRAN IV program (Appendix I) the relationship of every possible pair of reports was assigned a category based on the separation between them in time and distance. After every possible combination of two reports had been classified, the number of combinations falling within the spatial and temporal separation limits of each category were tabulated for each county. The resulting figures

Table III. Summary of rabies laboratory reports used for analyses of time and space relationships of incidences in wildlife species.

County	State	Inclusive Dates	No. Positive Reports
Bedford	Virginia	9-29-66 to 7-31-68	79
Nelson	Virginia	1-26-67 to 1-7-69	29
Washington	Virginia	1-9-67 to 4-10-69	86
Imperial	California	5-5-69 to 4-3-70	48

were used in the construction of two graphs for the display of the patterns of time and distance separations between reports for the four areas studied. These graphs assumed the form of frequency distributions, the dependent variables being the percentage of the total tabulations for each county falling into each time or distance separation class.

COMPUTER SIMULATION OF RABIES EPIDEMIOLOGY

The simulation language selected for the modeling of rabies epidemiology was GASP II (Pritsker and Kiviat 1969). The investigator had recently completed coursework with one of the authors of this language and was more familiar with this simulation technique than any other. At the time of this study the required GASP subroutines were operational in the V.P.I. computing system and easily accessible.

Description of GASP Simulation Language

GASP II is a generalized activity simulating program written in FORTRAN IV. Originally designed and documented with reference to engineering applications, it is non-specific enough to be useful in the modeling of a wide variety of systems, e.g., rabies epidemiology. GASP II consists of approximately 20 subprograms which serve to advance time during the simulation, store and retrieve required information, monitor simulation progress, collect and report measurements of system performance, and generate random variables. A complete description of the program and its concepts is presented by Pritsker and Kiviat (1969). The following description will serve only to familiarize the reader with GASP

procedures sifficiently for understanding the rabies simulation.

Definitions and General Approach to Simulation with GASP

By simulation the programmer seeks to model a system or some part of reality which is of interest. The system may be viewed as being composed of entities and processes. Entities are any physical parts of a system, such as people, animals, or machinery. Processes are viewed best as the activities in which entities may engage and the means by which entities may interact. Each entity of a system is characterized by a set of attributes. Attributes are characteristics of an entity which may be quantified. Table IV illustrates an example of an elevator system. It may be seen that each entity of the system is characterized by a set of values (attributes). The attributes of any entity need not describe it completely. They need only to be detailed enough to yield a simulation sufficiently realistic to meet the programmer's requirements.

An entity may be said to be in a particular state when its attributes have specific numerical values. The action of an entity moving from one state to another is referred to as an activity. An event takes place at a point in time and either starts or ends an activity. Events, like entities, have attributes. In fact, each event must be characterized by at least one attribute, the point in time at which it is to occur. Other event attributes may define the type of event and the attributes of entities affected by this event. If "Elevator starts to travel" is an event which may take place in the system described in Table IV, it may have attributes such as those listed in Table V.

Table IV. Definition of an elevator system in a building and its entities and attributes (from Pritsker and Kiviat 1969).

Entities	Attributes
Building	Number of floors. Distance between floors.
Elevator	Passenger capacity. Speed. Minimum stopping time at a floor. Current floor location.
Floor	Number of people waiting for elevator. Probability that people will want to go to other floors given that they are on this floor.
People	Arrival rate at building. Preference for floors. Arrival rate of people on floor waiting to go up or down.

Table V. Possible attributes of a simulation event (from Pritsker and Kiviat 1969).

Attribute	Possible Value
Time at which event is to occur	10.3 hours
Event type	9
Floor at which elevator currently resides	3
Direction of movement of elevator	0=up, 1=down

GASP Simulation Procedures

The concept of the event is the basis of GASP II programming. In order to write a simulation program, the GASP programmer is only required to supply sufficient subprograms to describe system state changes which occur at event times and the future events which may be generated as a result of the occurrence of events. The only other programming required is a main program and an event selection subroutine called EVNTS. The main program serves to initialize non-GASP variables and call the GASP executive routines which control the simulation. The event selection subroutine is merely a computed GO TO statement which examines the attribute identifying the type of each event as it is pulled out of the filing array. In addition, it channels program execution to the subroutine effecting the proper changes in the system state called for by that event.

Fig. 1 illustrates the logical flow of a generalized GASP simulation. After the main program has initialized variables describing the system in question, control is transferred to subroutine GASP. Here additional GASP variables are initialized and the attributes of events which will start the simulation are read and stored chronologically in the array NSET. Time of event occurrence is always the first attribute and a code for type of event is always the second.

To start the simulation, GASP searches the chronological event file in NSET for the earliest scheduled event. The numerical values for the attributes (1 through n) of this earliest event are stored in the dummy variables ATRIB (1) through ATRIB (n).

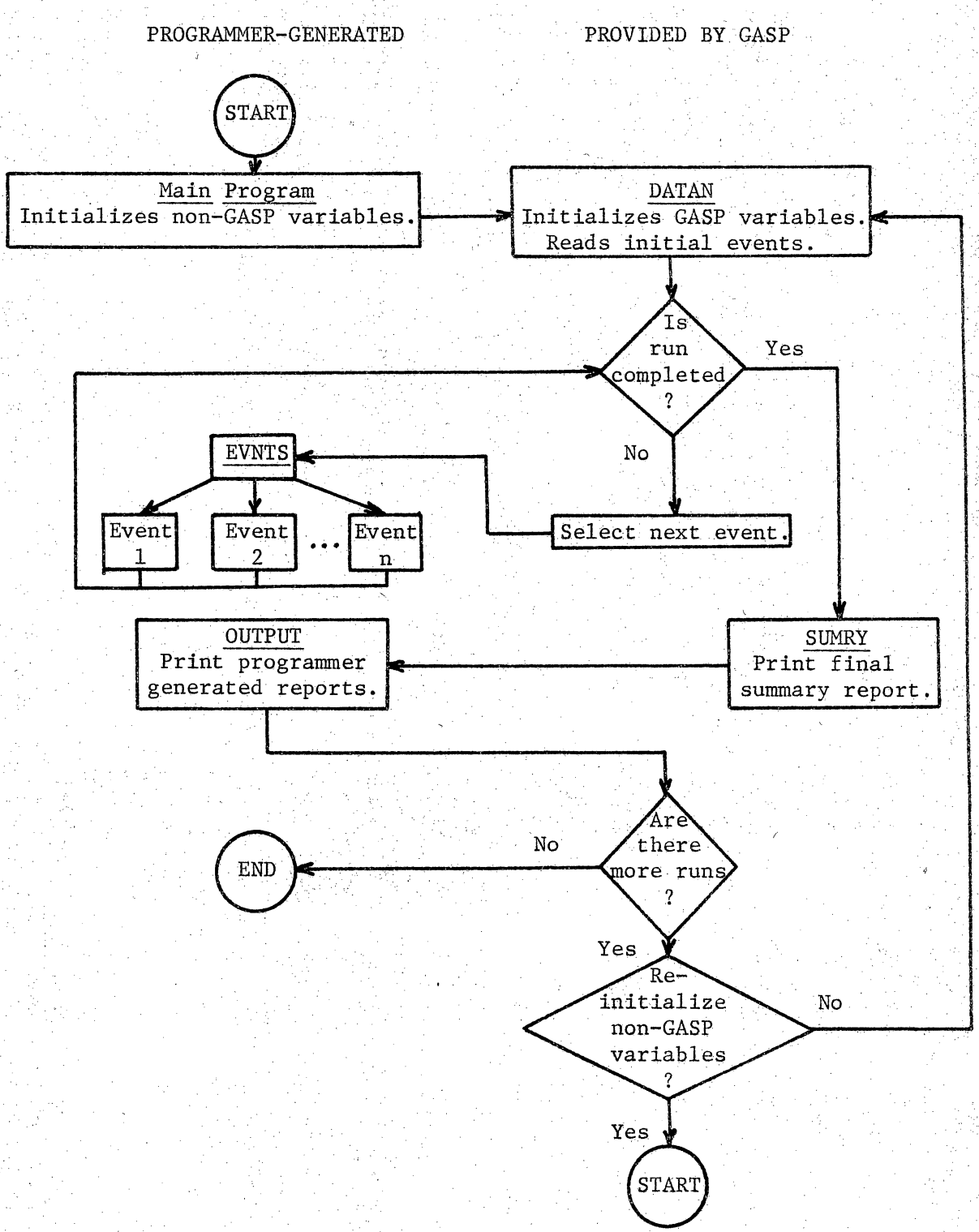

Fig. 1. Generalized GASP simulation program (from Pritsker and Kiviat, 1969).

Control is then transferred to subroutine EVNTS which examines the second attribute of the scheduled event and transfers execution to the proper programmer-written event subroutine. During execution of the event routine, new events may be scheduled by changing the values stored in ATRIB and calling subroutine FILEM, which stores the values of ATRIB as new event attributes in NSET.

Simulation of Rabies Epizootics--Program Description

In the GASP simulation of wildlife rabies five major types of system state changes, or events, are recognized:

1) Contact between an infected animal and another animal, of any species, from the population
2) End of virus incubation period and the display of symptoms in a rabid animal
3) The death of a rabid animal
4) Contact between a predator and the body of a rabid animal
5) Reproduction - the introduction of new susceptibles to the population each spring season.

The array NSET is used to keep a chronological file of these events and the attributes which serve to describe them. These are as follows:

Attribute	Description
1	Event time
2	Event type code
3	Species code of animal initially causing the event

4	Species code of animal which infected the animal involved
5	Code for symptoms displayed by animal involved
6	Time period animal involved lives after displaying symptoms
7	Time animal involved is scheduled to die

The only exception to these definitions occurs in contact events between predators and the bodies of rabid animals. When such contacts occur, attributes 1 through 4 remain unchanged. However attribute 5 is redefined as the point in time after which the body is decomposed and unpalatable. Attributes 6 and 7 are not used in describing these contact events.

Description of Rabies Simulation Subprograms

The following are verbal and graphical descriptions of the programmer-generated subroutines in the GASP rabies simulation. Complete source listings of these subroutines are given in Appendix II. A listing of variable definitions is provided in Appendix III.

Main Program (see Fig. 2):

1) Identifies codes for card reader and printer.

2) Reads and initializes system state variables.

3) Calls GASP executive routine.

Subroutine EVNTS (IX, NSET) (see Fig. 3):

1) Examines code of the event to take place next and

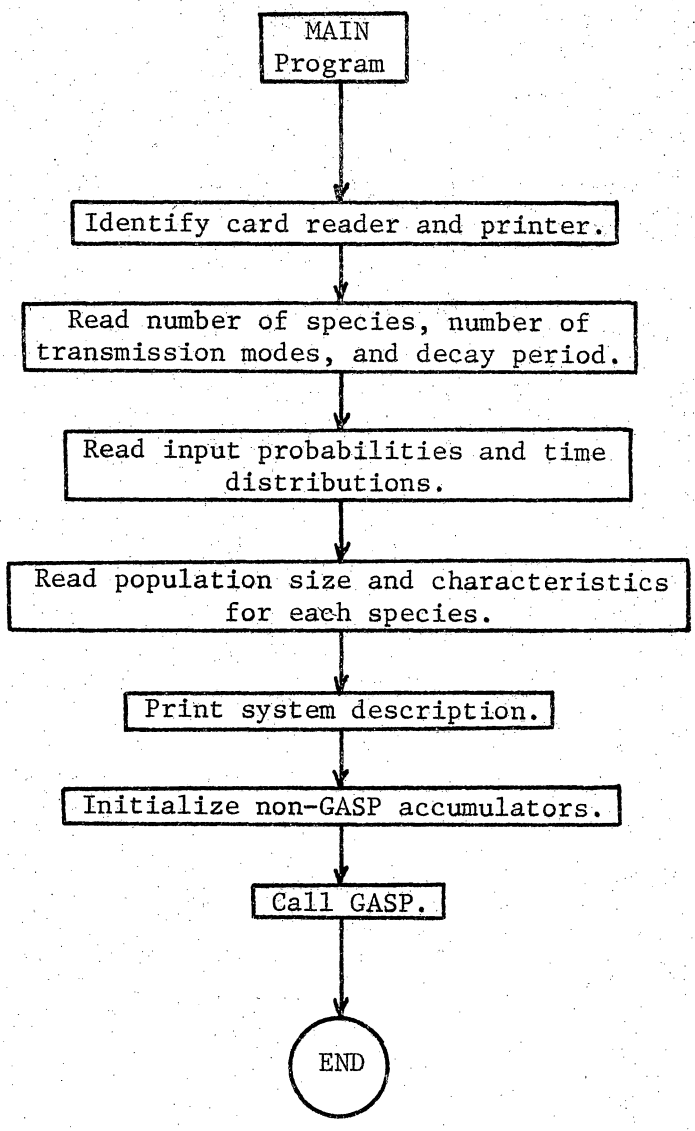

Fig. 2. Main program.

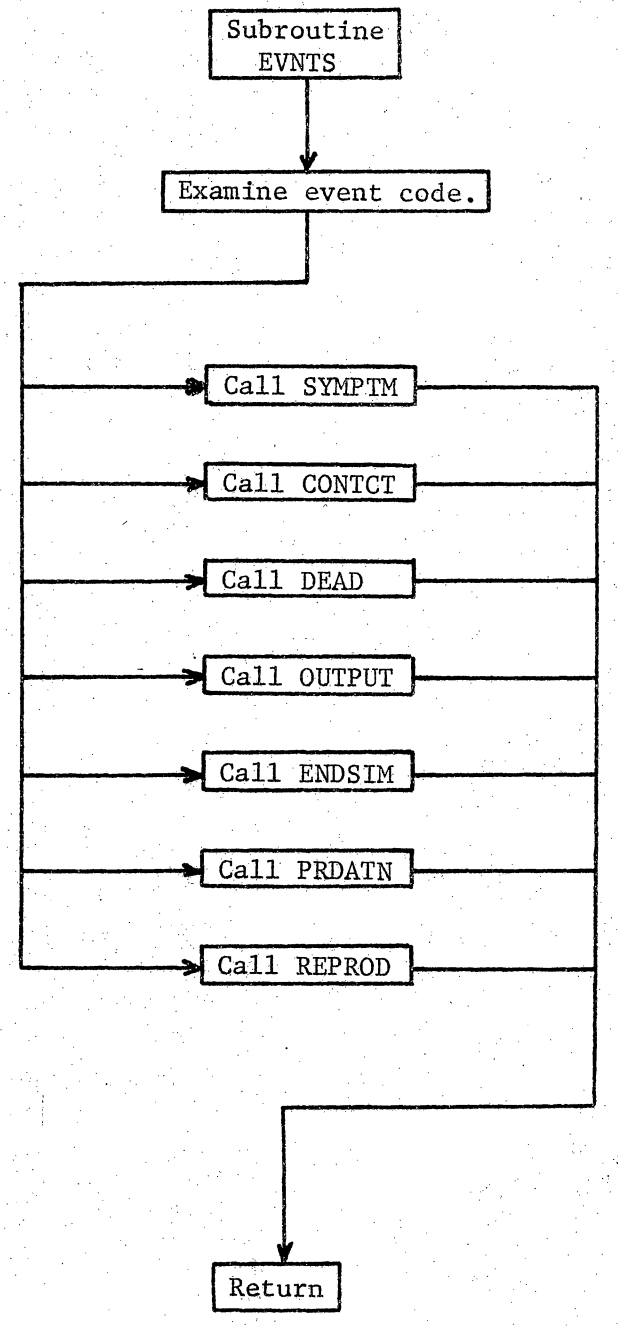

Fig. 3. Subroutine EVNTS.

transfers control to the proper subprogram.

Subroutine SYMPTM (NSET) (see Fig. 4):

1) Makes changes in probabilities and time distributions on the basis of population reductions.
2) Determines the type of symptoms which the animal in question displays.
3) Schedules the animal's death if active symptoms are displayed.
4) Schedules a contact between this animal and a member of each species in the community. The time of the occurrence of this event is sampled from a uniform distribution for the length of time between contacts of the two species in question.

Subroutine CONTCT (NSET) (see Fig. 5):

1) Makes changes in probabilities and time distributions on the basis of population reductions.
2) Makes a new determination of symptoms if the rabid animal has not yet shown active symptoms. If active symptoms are displayed, the death of this animal is scheduled.
3) Schedules a new contact between the rabid animal and another member of the same species it is contacting at the moment.
4) Determines transmission mode used in transferring virus to present contact.
5) Determines if present contact is susceptible to infection.

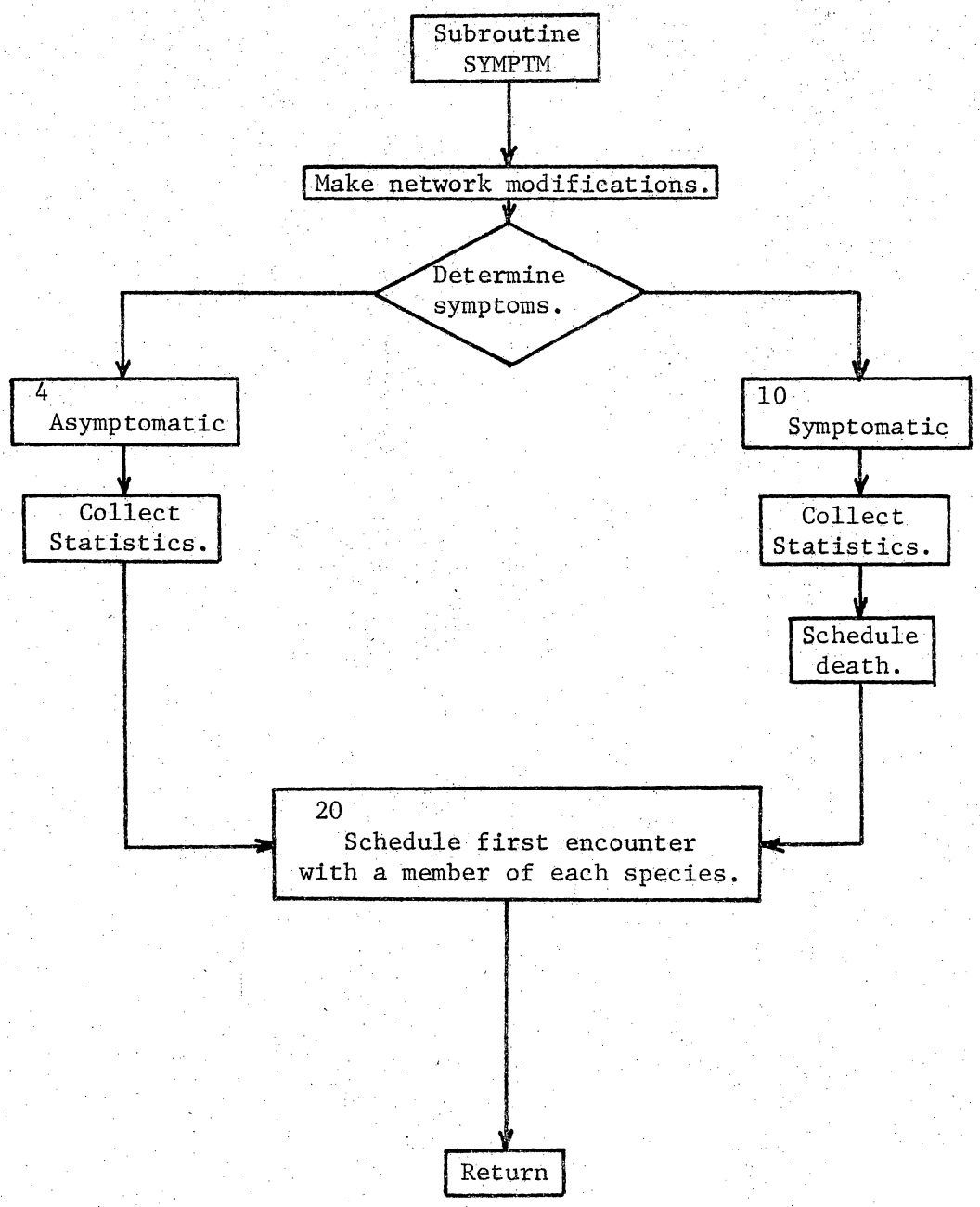

Fig. 4. Subroutine SYMPTM.

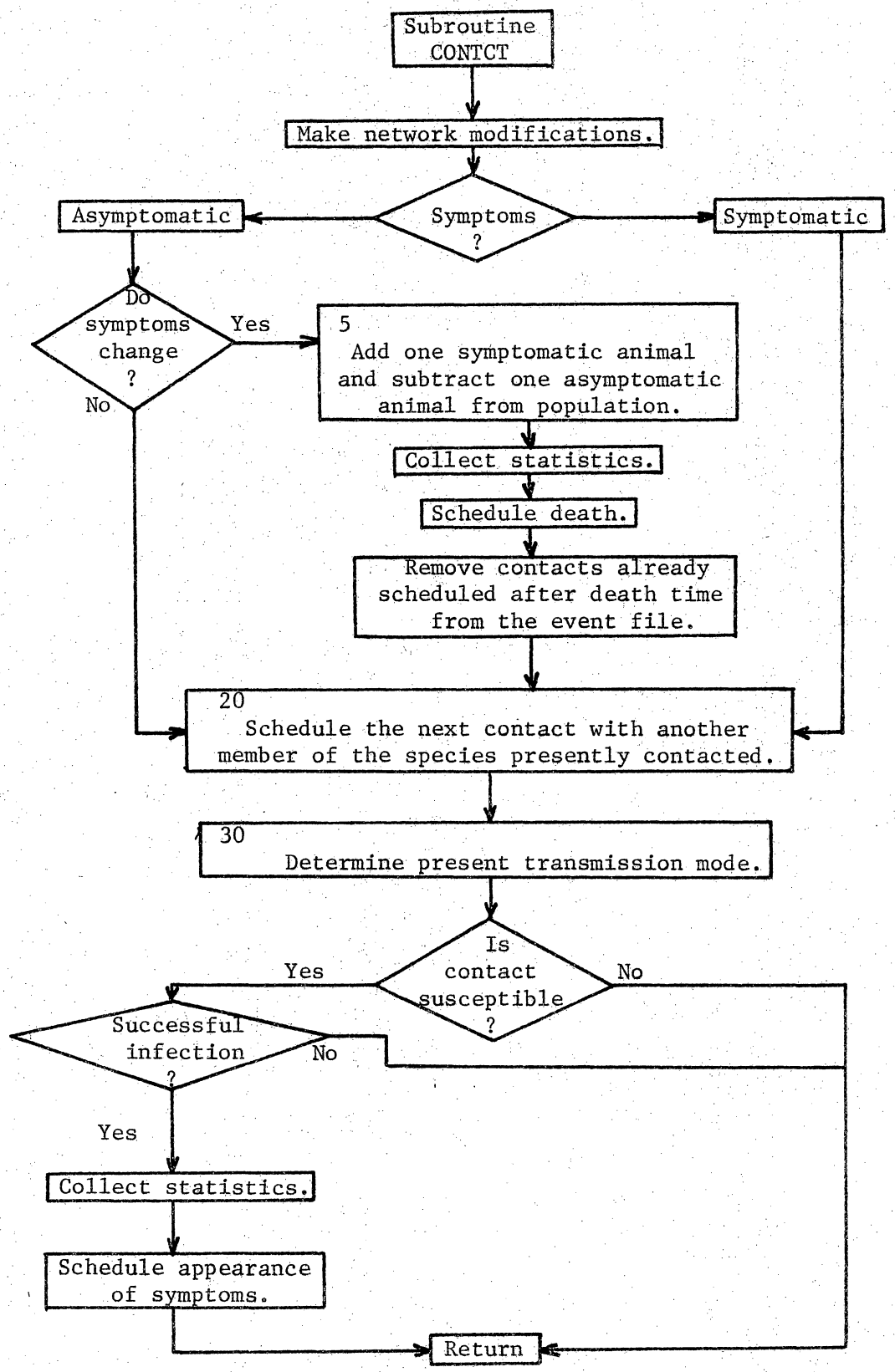

Fig. 5. Subroutine CONTCT.

If not, control is transferred back to GASP before the contact is scheduled to become rabid.

6) Determines if infection succeeds or fails in a susceptible contact. If it fails, a return is made to GASP. If not, the system description accumulators are modified accordingly and the end of the virus incubation period is scheduled for the contacted animal.

Subroutine DEAD (NSET) (see Fig. 6):

1) Modifies system state description accumulators to simulate one less individual in the population.
2) Modifies probabilities and time distribtions on the basis of population reductions.
3) Schedules an encounter by a member of each species with the dead body.

Subroutine PRDATN (NSET) (see Fig. 7):

1) Modifies probabilities and time distributions on the basis of population reductions.
2) Schedules next contact with the body by another member of the species presently involved.
3) Determines if the individual currently involved eats the flesh of the dead animal. If not, a return to GASP is made.
4) Determines if a scavenger, having eaten the flesh, is susceptible.
5) Determines if a susceptible scavenger will contact

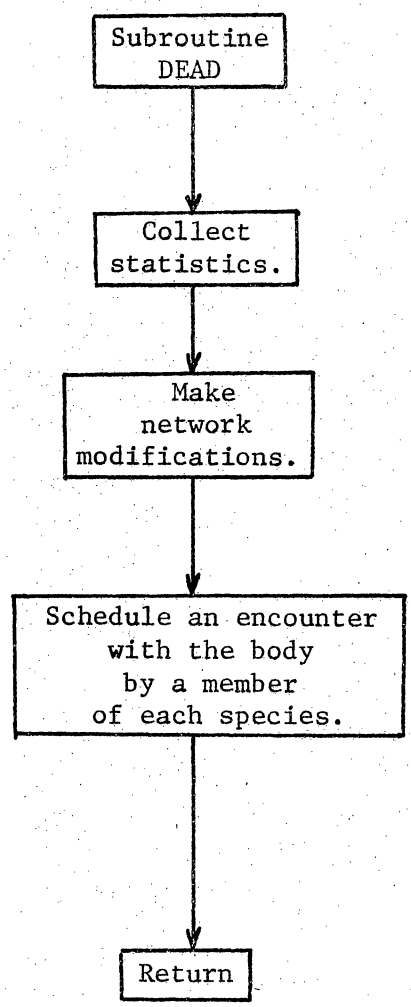

Fig. 6. Subroutine DEAD.

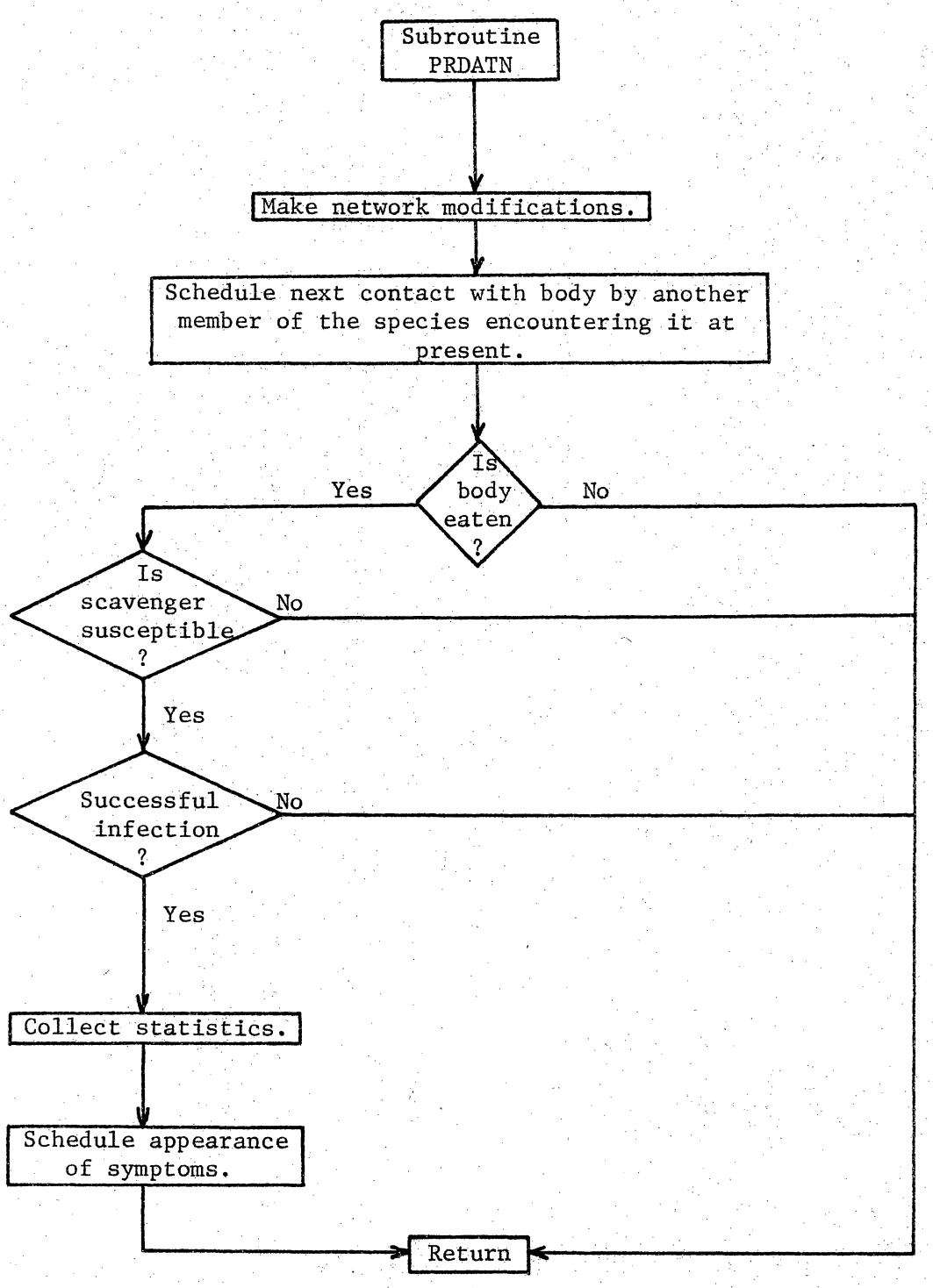

Fig. 7. Subroutine PRDATN.

rabies by eating infected flesh. If it will not, a return to GASP is made.

6) Suitably modifies the system state description accumulators and schedules the end of virus incubation in the newly infected animal if the infection does succeed.

Subroutine REPROD (NSET) (see Fig. 8):

1) Schedules next spring's reproduction.

2) Increases the population and the number of susceptible individuals in each species by a randomly distributed percentage of the existing population.

Subroutine OUTPUT (NSET) (see Fig. 9):

1) Schedules next weekly check of the epidemic.

2) Files weekly statistics concerning morbidity and mortality.

Subroutine ENDSIM (NSET) (see Fig. 10):

1) Resets accumulators at the end of each run of 100 simulated weeks.

2) Calls for a final report summarizing all runs made.

Six other subroutines were written by the investigator. Subroutine GASP (NSET) and ERROR (J, NSET) were modified in order to suppress unnecessary printing of the filing array, NSET. XMOVE (KCOL, JQ, NSET) is a modification of the standard GASP subroutine RMOVE (KCOL, JQ, NSET) which removes entries from the event file without entering them into the dummy array ATRIB. Subroutine BETAXF and the functions PGAMMA (TK, NPRNT, JP, ISEED) and BETA (J) were adapted from another simulation program, GERT III (Prisker and

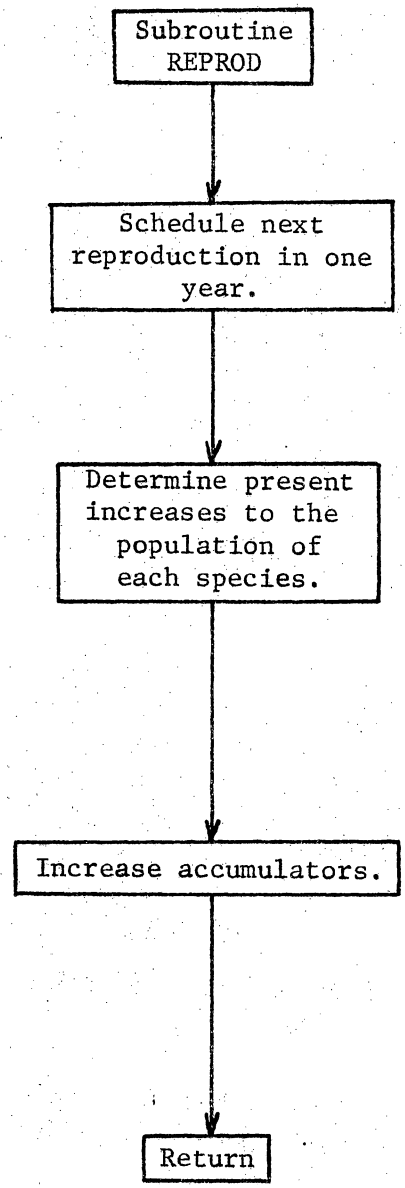

Fig. 8. Subroutine REPROD.

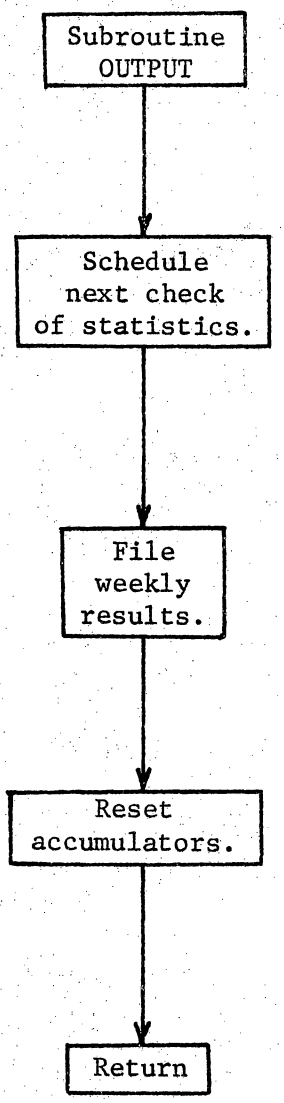

Fig. 9. Subroutine OUTPUT.

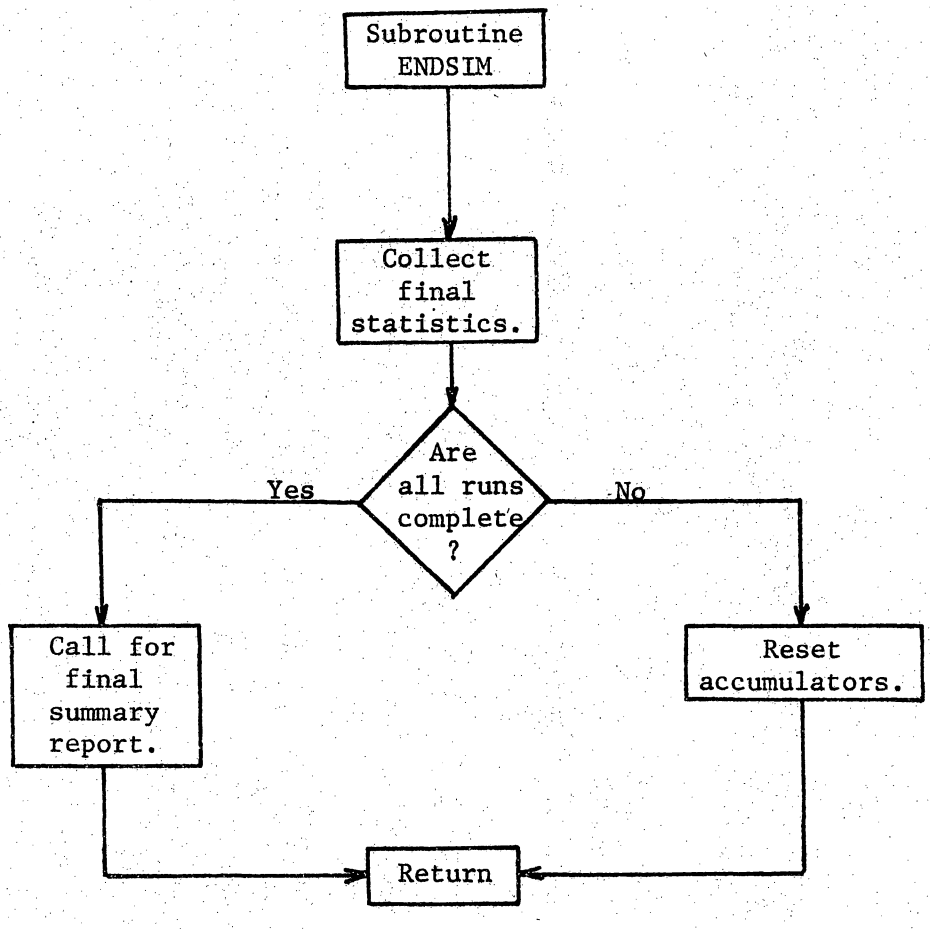

Fig. 10. Subroutine ENDSIM.

Burgess 1970), in order to generate random variables for incubation periods from the beta distribution, as suggested by the experimental findings of Parker and Wilsnack (1966) and Baer and Bales (1967). In addition the array SUMA was redimensioned on all GASP common cards to allow storage of 3000 variables.

Data Input

Data input specifications for GASP and non-GASP variables are outlined in detail in Appendix IV.

The first variables required as input data refer to the basic structure of an initial wildlife community before a rabies outbreak. They are the number of species under consideration, the number of possible transmission modes, the number of individuals in each species, and minimum and maximum increase factors for reproduction (minimum and maximum percentage population increase divided by 100).

The next set of variables required as input is the true basis for the stochastic nature of this simulation. It is a list of probabilities and time distribution parameters which characterize wildlife rabies epidemiology in the hypothetical community initially established. The time distributions describe, for each combination of two species, the minimum and maximum times which may elapse between interspecies contacts and minimum and maximum times required for a member of one species to discover the body of a member of another. In simulation, random numbers from uniform distributions having these characteristics are used in the determination of actual individual time lapses between events.

The probabilities read as input data are:

1) The probabilities of members of each species developing rabies if contacted by a member of any species which is displaying specified symptoms.
2) The probabilities of infected members of each species developing active symptoms, as opposed to a latent infection.
3) The probabilities of each possible transmission mode being utilized by rabid members of each species in contacting other species given the symptoms of the rabid animal.
4) The probabilities of members of each species eating the body of any other species if it is encountered.
5) The probabilities of members of each species contracting rabies by eating the flesh of a rabid animal.
6) The minimum probabilities of members of each species developing active symptoms. Use of the minimum establishes a lower limit on any reduction of the probability of active symptoms due to reductions in population, hence crowding stress.

In determining the consequences of contact events between individual animals, the symptoms displayed by rabid individuals, and the transmission modes used to disseminate rabies virus, these probabilities are compared to random numbers between zero and one. The random numbers are generated anew for each contact. Their relationship to the probability in question (greater than,

less than, or equal to) determines the subsequent course of program execution.

Parameters (mean, minimum, maximum, and standard deviation) of the distribution of length of incubation periods and the duration of clinical symptoms for each species are read in as GASP parameter values. Three other species variables are required for the simulation of the application of rabies control measures to the hypothetical wildlife community. These are the proportion of each species population immunized by wildlife vaccination devices, the proportion by which natural reproduction may be reduced by the use of chemosterilants, and the proportion of each population removed by trapping.

Dynamic Modification

In an attempt to lend an added degree of reality to the simulation of wildlife rabies epidemiology the program allows for the modification of specific probabilities and time distributions on the basis of population changes. Each time animals die or are added to the community, appropriate changes are effected upon the distributions of time lapses between contacts and between discoveries of dead animals.

In addition the hypothesized effects of density stress upon latent infections are simulated by two means. Population decreases exert a reducing influence on the probabilities of active rabies symptoms being displayed by members of each species, down to a minimum value. Population increases on the other hand increase the probabilities of active symptoms. Therefore the probability of

an individual displaying active rabies symptoms after some period of incubation is dynamic in nature. Once an animal has initially been labeled asymptomatic, however, it is continually under surveillance and the program periodically allows for new determiniations of whether the animal is symptomatic or asymptomatic. Once an individual assumes active symptoms, the death of that animal is scheduled and no reversion to an asymptomatic condition is possible.

RESULTS

ANALYSIS OF RABIES REPORTS

In general the analysis of spatial and temporal relationships between reported wildlife rabies incidences disclosed few patterns common to any of the four counties under study.

Report Site Mapping

Visual analysis of the report sites plotted on maps of the respective counties demonstrated the only real similarity of report patterns between ecologically divergent communities. Reports were, in general, concentrated near human population centers, giving the impression of infection foci. This effect was most distinct in Bedford and Nelson counties. These areas are largely mountainous and dense human populations are restricted to valley areas, as are the reports of rabid wildlife. The clustering effect in rabies reports was least apparent in Washington County. This county, elongated in shape, coincides with a wide, flat river valley. As a result the human population of this area is more evenly distributed and centers of population are less distinct. Likewise rabies reports were more evenly spread over the entire area of this county. Rabies reporting in any area appears to be a function of the number human observers present. Human population density is largely related to the physiography of each county, being concentrated in valleys. Rabies reporting is best in settled valley areas, resulting in apparent concentrations of infection. It seems likely, however, that in reality the distribution of rabid wildlife follows a much more uniform pattern than indicated by health department reports.

Examination of the patterns of reports in order of occurrence yielded no significant patterns except in Washington County. Here the rabies epidemic appeared to occur in waves, moving up the central valley from southwest to northeast. In early 1967 most reports were concentrated in the vicinity of the town of Bristol. By mid and late summer of that year rabies incidences predominated near Abingdon. In early 1968 the focus of infection had shifted to the area of Damascus and Glade Spring.

Cumulative Time Patterns

Cumulative curves of percentage of total reports in each county per week were constructed in an attempt to demonstrate pattern characteristics common to all four study areas (see Fig. 11). By visual examination of these graphs the experimenter was able to detect little similarity in incidence patterns between any two counties.

Overall Temporal and Spatial Relationships

The analysis of the time and space relationships between each rabies report and every other report was conducted by computer for three counties. The graphical patterns of the percentages of the total possible combinations in each county falling into specific time or distance separation classes are shown in Figs. 12 and 13. Again no pattern characteristic of all counties can be described. The distribution of time separations was essentially uniform for Nelson County. However, Imperial County displayed a pattern of most combinations being separated by only a few weeks, while Washington County report combinations were most often separated by 8 to 15 weeks. The distance separation patterns for Nelson and Imperial

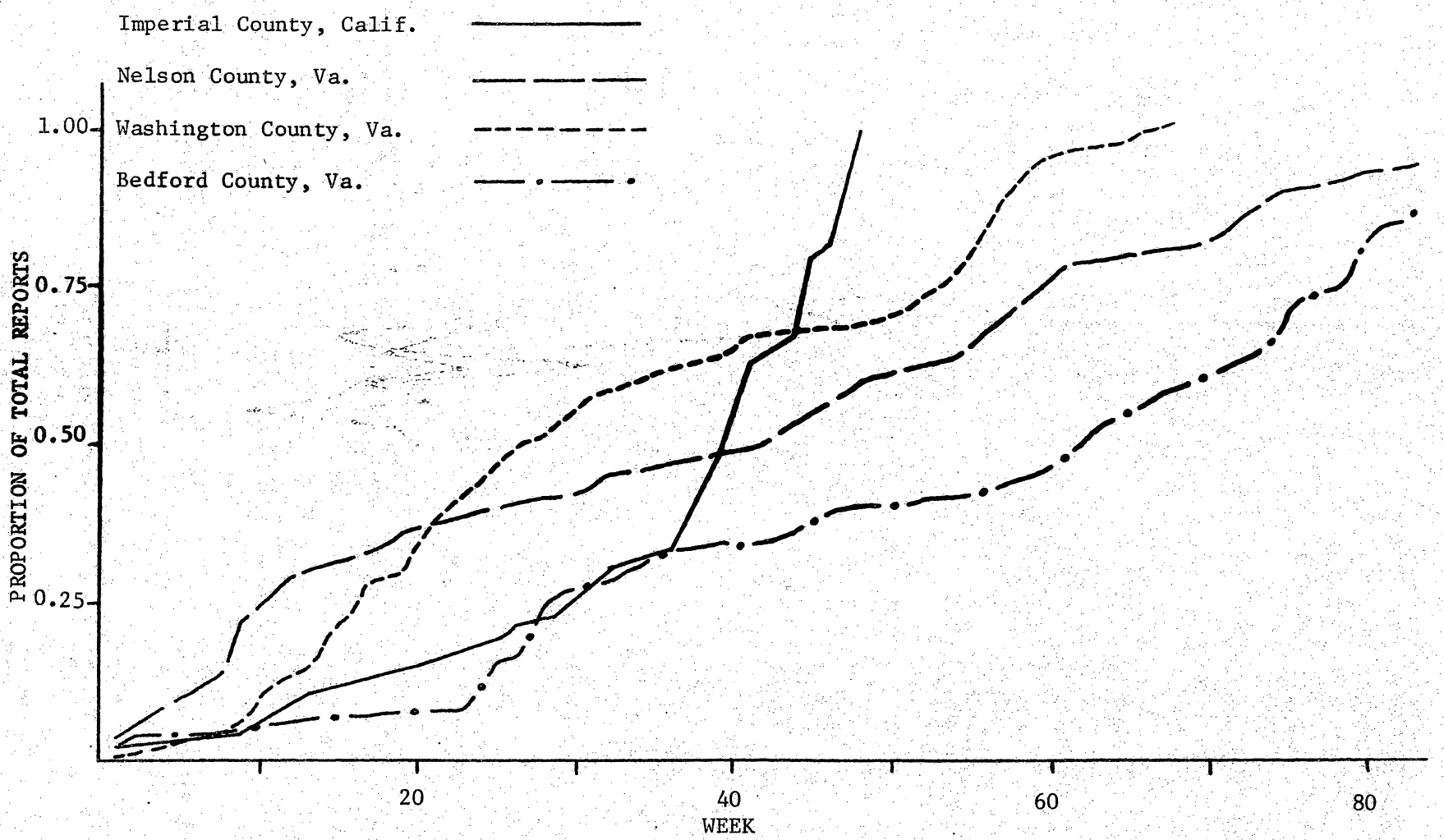

Fig. 11 Cumulative rabies reports by week from four counties studied.

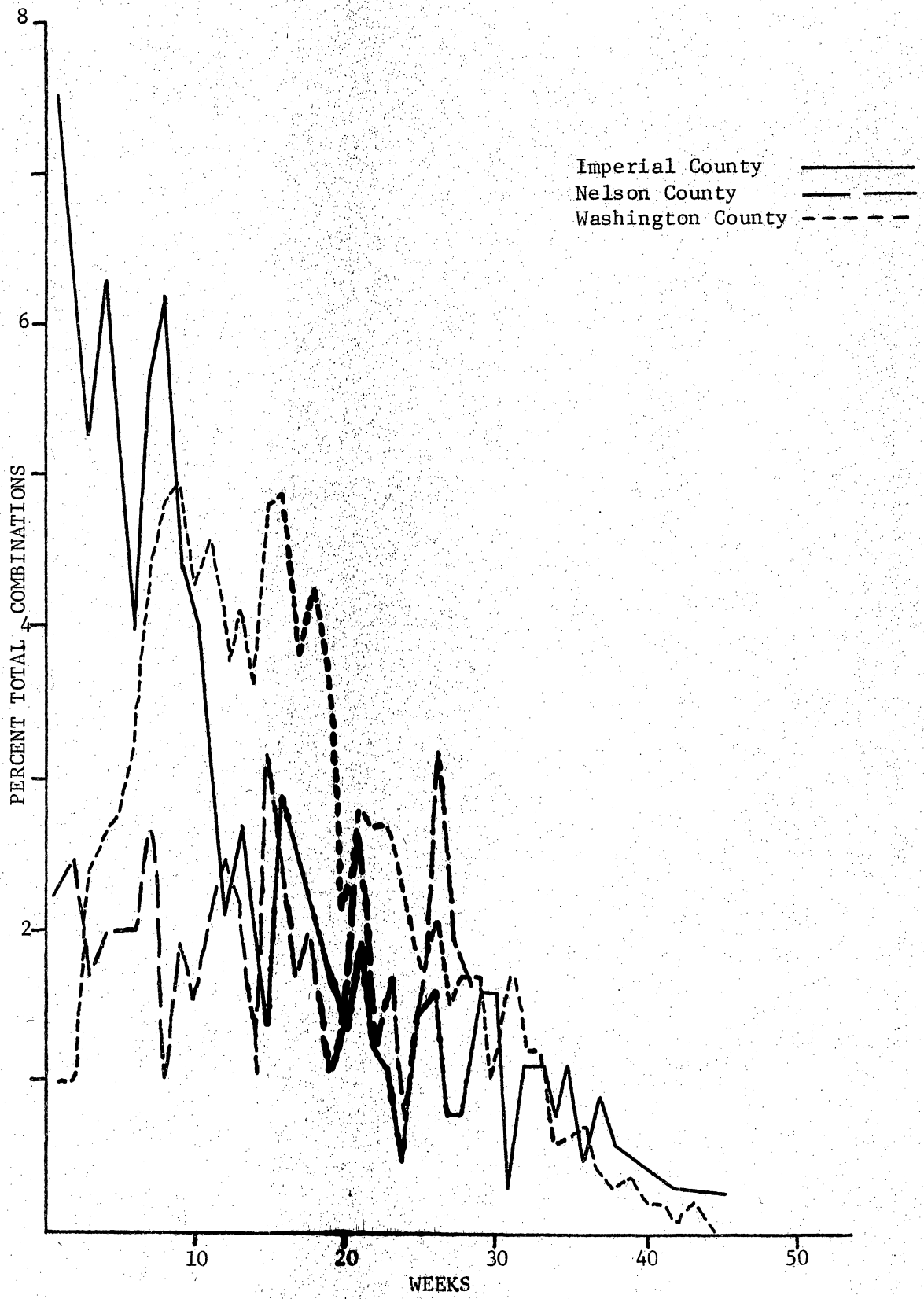

Fig. 12 Temporal separations between each possible combination of rabies reports, by county.

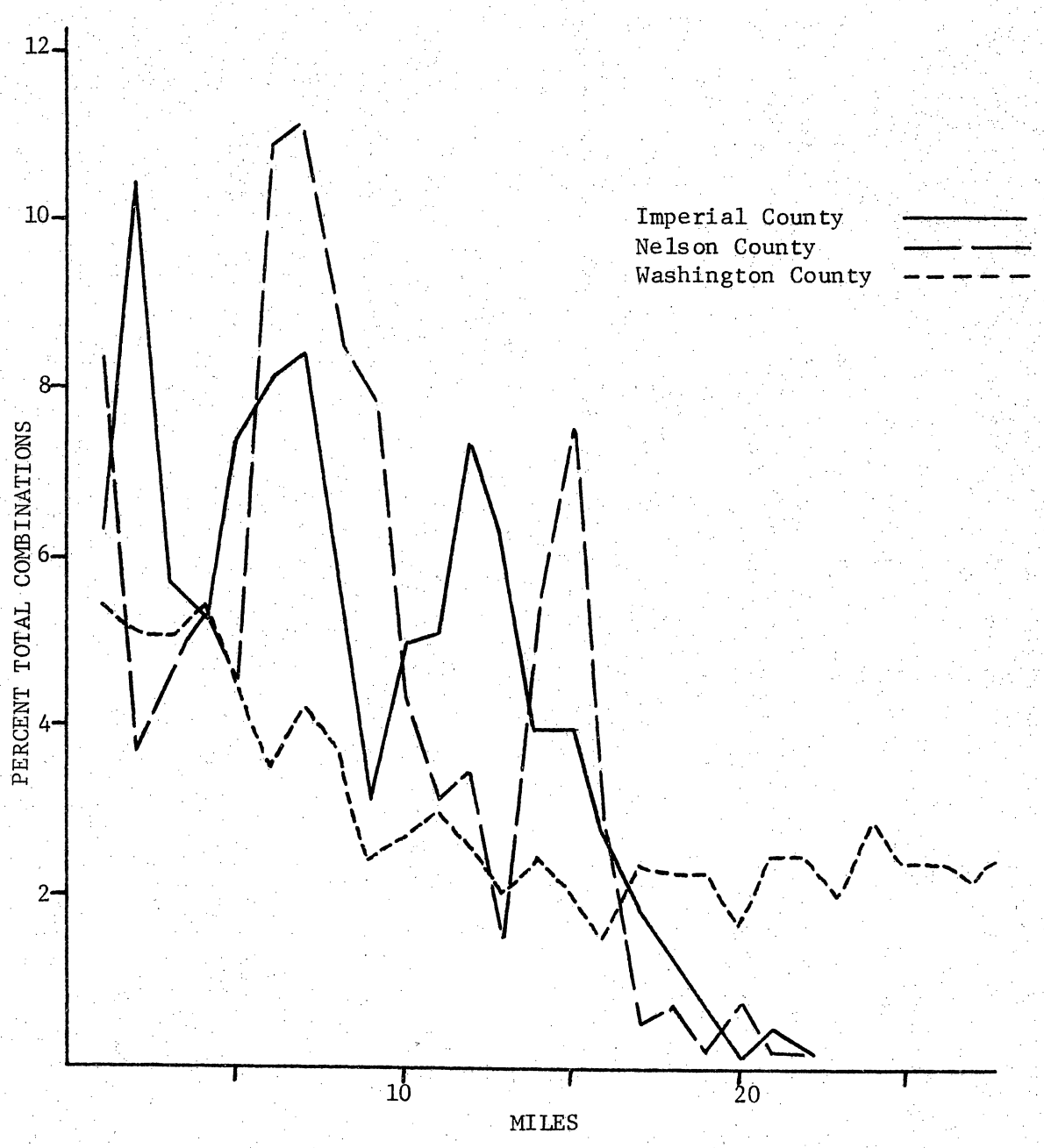

Fig. 13 Spatial separations between each possible combination of rabies reports, by county.

counties are, at best, erratic. Washington County reports display a relatively uniform distribution, slightly more report combinations falling into the 1-8 mile classes than into the greater separation classes.

COMPUTER SIMULATION OF RABIES EPIDEMIOLOGY

In order to demonstrate the use of the final model of wildlife rabies epidemiology to simulate the effects of varying control strategies, four simulation runs were performed. The following control procedures were simulated for a rabies epidemic in a complex of three species - skunks (species 1), foxes (species 2), and domestic dogs (species 3).

1) no control
2) 25% reduction of fox population
3) use of chemosterilants to reduce annual fox reproduction by 25%
4) vaccination of 25% of fox population

Each strategy was simulated for three 100 week periods, each under the influence of a different random number seed. The results of the three runs were then averaged for comparison with the results of other strategies. All initial population parameters not related to control strategies were kept constant for all runs. Three possible modes of rabies transmission were recognized. They were bite, saliva contact with wounds, and aerosol transfer. Two symptom conditions were possible: active symptoms and an asymptomatic carrier state. The input data used to characterize the sample epidemics is summarized in Tables VI through XI.

Table VI. Initial population parameters for sample simulation.

Species	Skunk	Fox	Dog
Initial population	600	300	300
Minimum reproductive increase (percent of adults present at the breeding season)	100	100	75
Maximum reproductive increase	300	200	130
Initial probability of displaying active rabies symptoms	0.70	1.00	0.98
Minimum probability of displaying active rabies symptoms	0.30	0.30	0.30
Probability of contracting rabies by eating the flesh of a rabid animal	0.05	0.05	0.05

Table VII. Time in days required for rabid members of species (I) to come into contact with a member of species (J).

J=	1		2		3	
I=	min.	max.	min.	max.	min.	max.
1	0	2	1	15	1	7
2	1	15	0	5	1	20
3	1	3	1	20	0	2

Table VIII. Time in days required for a member of species (J) to find the body of a rabid member of species (I).

J=	1		2		3	
I=	min.	max.	min.	max.	min.	max.
1	0	5	1	10	2	9
2	1	5	1	10	2	9
3	1	5	2	9	1	2

Table IX. Probabilities of susceptible members of species (J) contracting rabies if attacked by a rabid member of species (I) displaying symptoms (K).

K=	1			2		
J=	1	2	3	1	2	3
I=						
1	0.70	0.80	0.80	0.50	0.40	0.50
2	0.75	0.90	0.90	0.50	0.30	0.40
3	0.50	0.90	0.90	0.50	0.30	0.40

Table X. Probabilities of transmission mode (L) being used by a member of species (I), showing symptoms (K), in contacting members of species (J).

K=		1			2		
L=		1	2	3	1	2	3
I	J						
1	1	0.90	0.10	0.00	0.30	0.10	0.00
1	2	0.98	0.02	0.00	0.90	0.10	0.00
1	3	0.98	0.02	0.00	0.90	0.10	0.00
2	1	1.00	0.00	0.00	0.98	0.02	0.00
2	2	0.90	0.10	0.00	0.30	0.70	0.00
2	3	0.90	0.10	0.00	0.30	0.70	0.00
3	1	1.00	0.00	0.00	0.98	0.02	0.00
3	2	0.90	0.10	0.00	0.30	0.70	0.00
3	3	0.90	0.10	0.00	0.30	0.70	0.00

Table XI. Probabilities that species (I) will be eaten by species (J).

J= / I=	1	2	3
1	0.00	0.50	0.70
2	0.00	0.05	0.10
3	0.00	0.10	0.05

The first event for each run was the end of virus incubation in a skunk (species 1). The attributes of this event were read in as data. Reproduction in each run first took place on day 230.

Uncontrolled Epidemic

Fig. 14 displays the number of animals dying from rabies each week under a situation of no control. There are two peak periods of death in both skunks and foxes. The first is relatively extended (about 10 weeks in duration) for both species, but the second is of short duration and displays higher weekly mortality. The dog population however reaches only one mortality peak.

Fig. 15 demonstrates, by species, the average number of animals becoming infected with rabies each week. Double peaks, corresponding with mortality peaks, will be noted for skunks and foxes, while the dog population displays only one peak of infection.

Fig. 16 shows the relative abundance of rabies carriers in the fox population with respect to symptoms. Dual peaks are again evident. It should be noted that the number of asymptomatic carriers remains relatively static after 70 weeks of simulation, indicating that population reductions have reduced the probability of animals displaying active rabies symptoms, thereby resulting in a reservoir of five or six asymptomatic foxes.

Rabies Control by Trapping

Fig. 17 shows the pattern of weekly deaths in each of the three wildlife species under a simulated control strategy of reducing the fox population by trapping. Twenty five percent of the foxes were removed before the initiation of the epidemic. Slight reductions

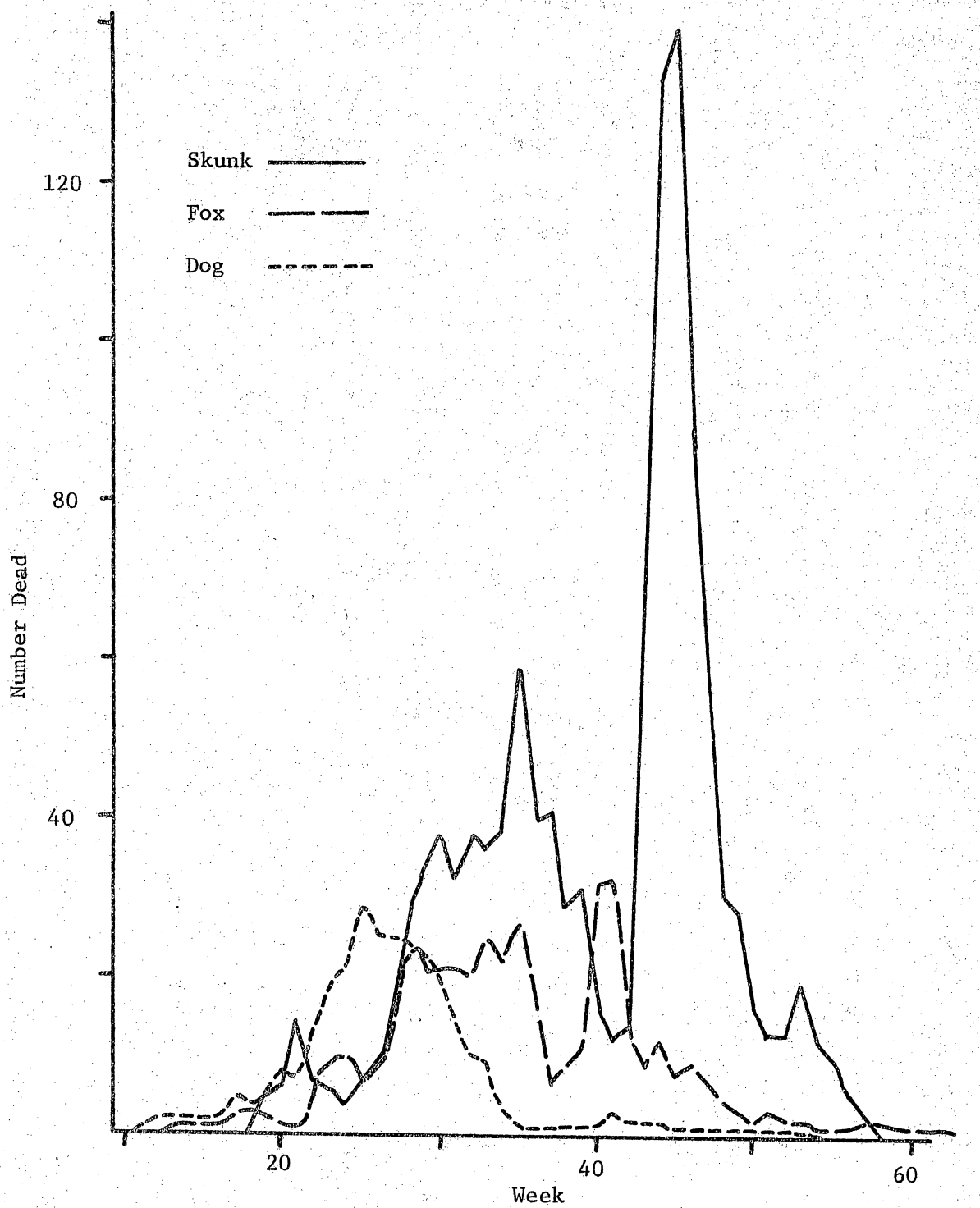

Fig. 14. Number of animals dying per week under a strategy of no control.

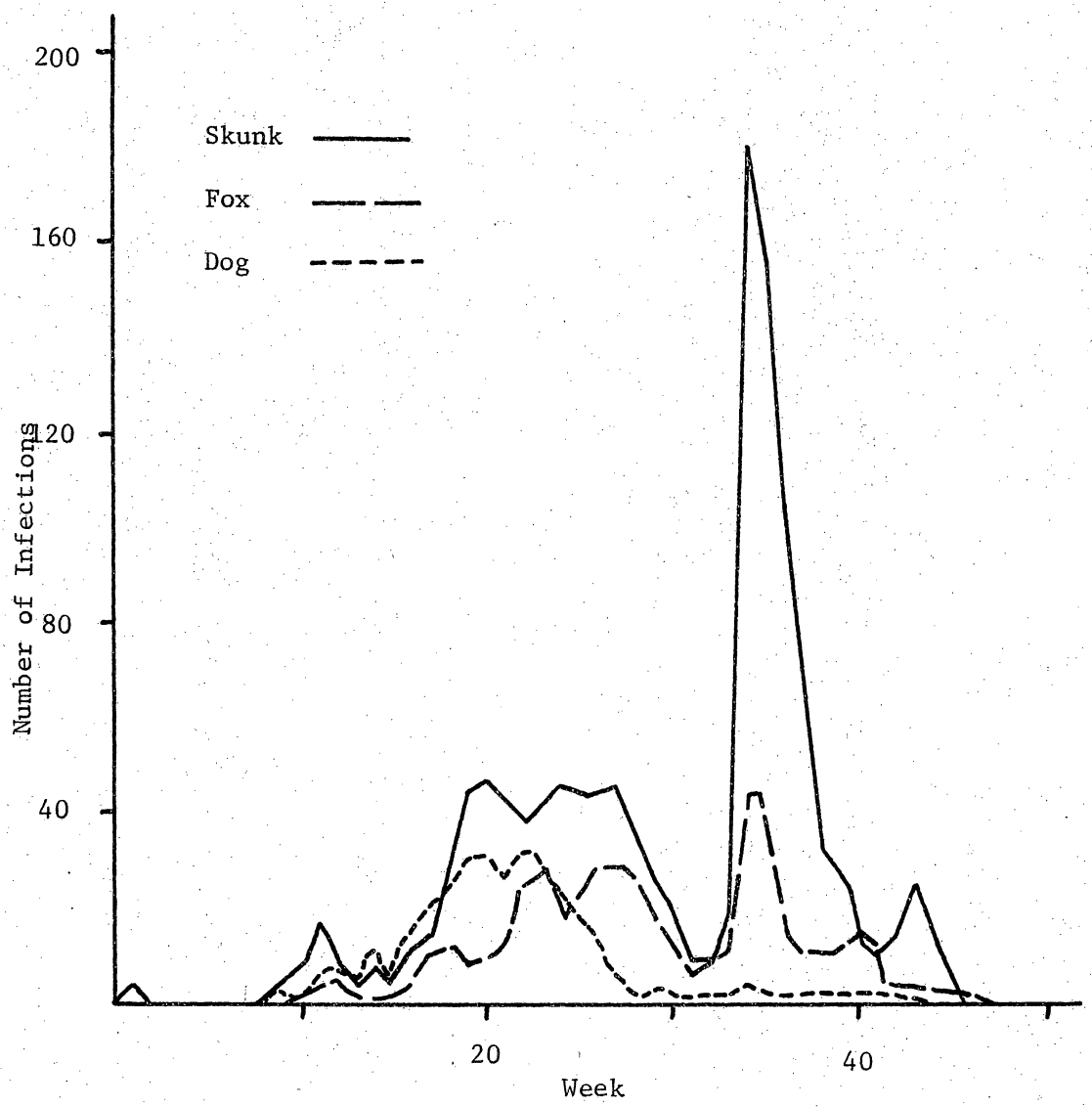

Fig. 15. Number of animals infected per week under a strategy of no control.

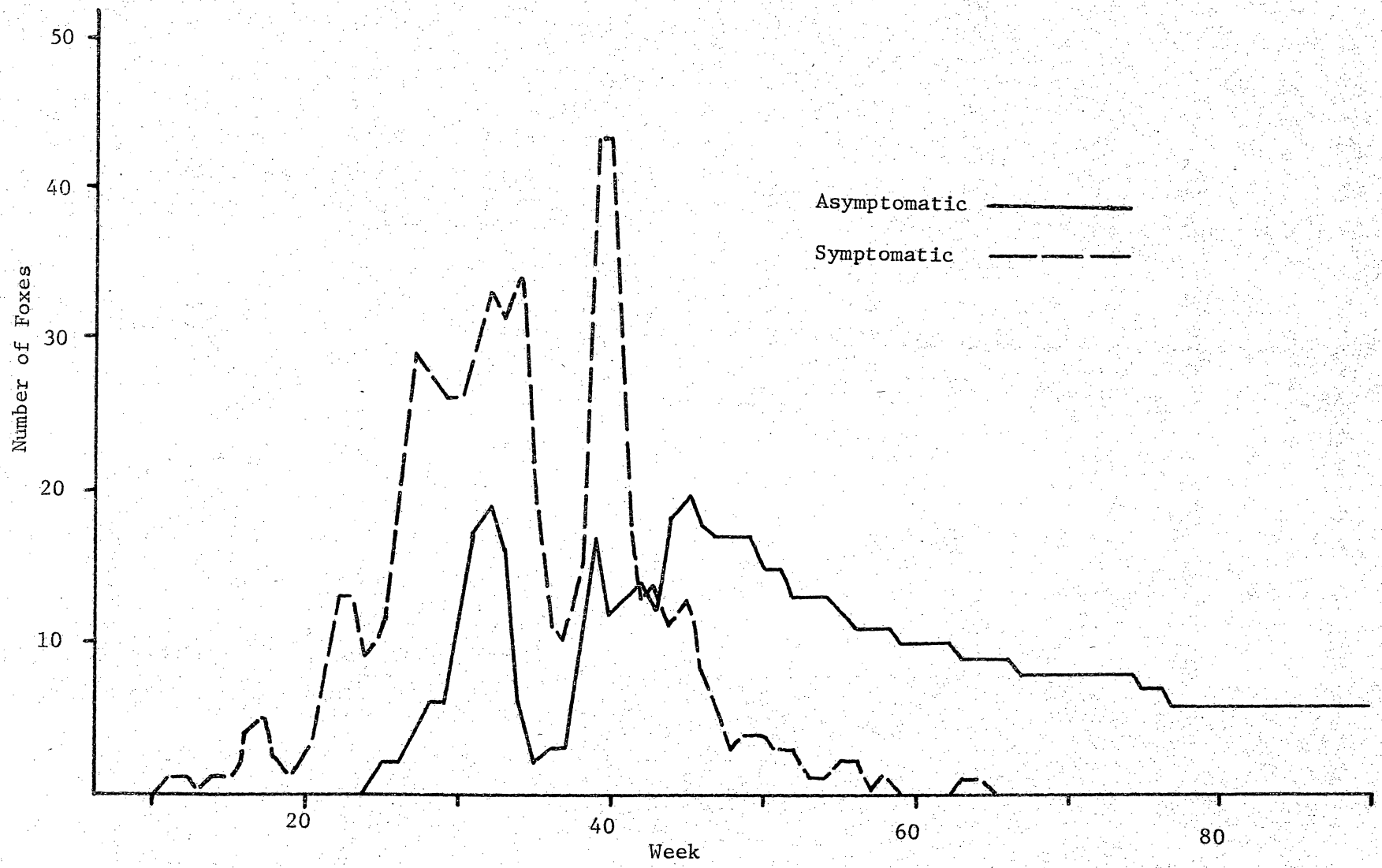

Fig. 16. Number of infective foxes present per week under a no-control strategy.

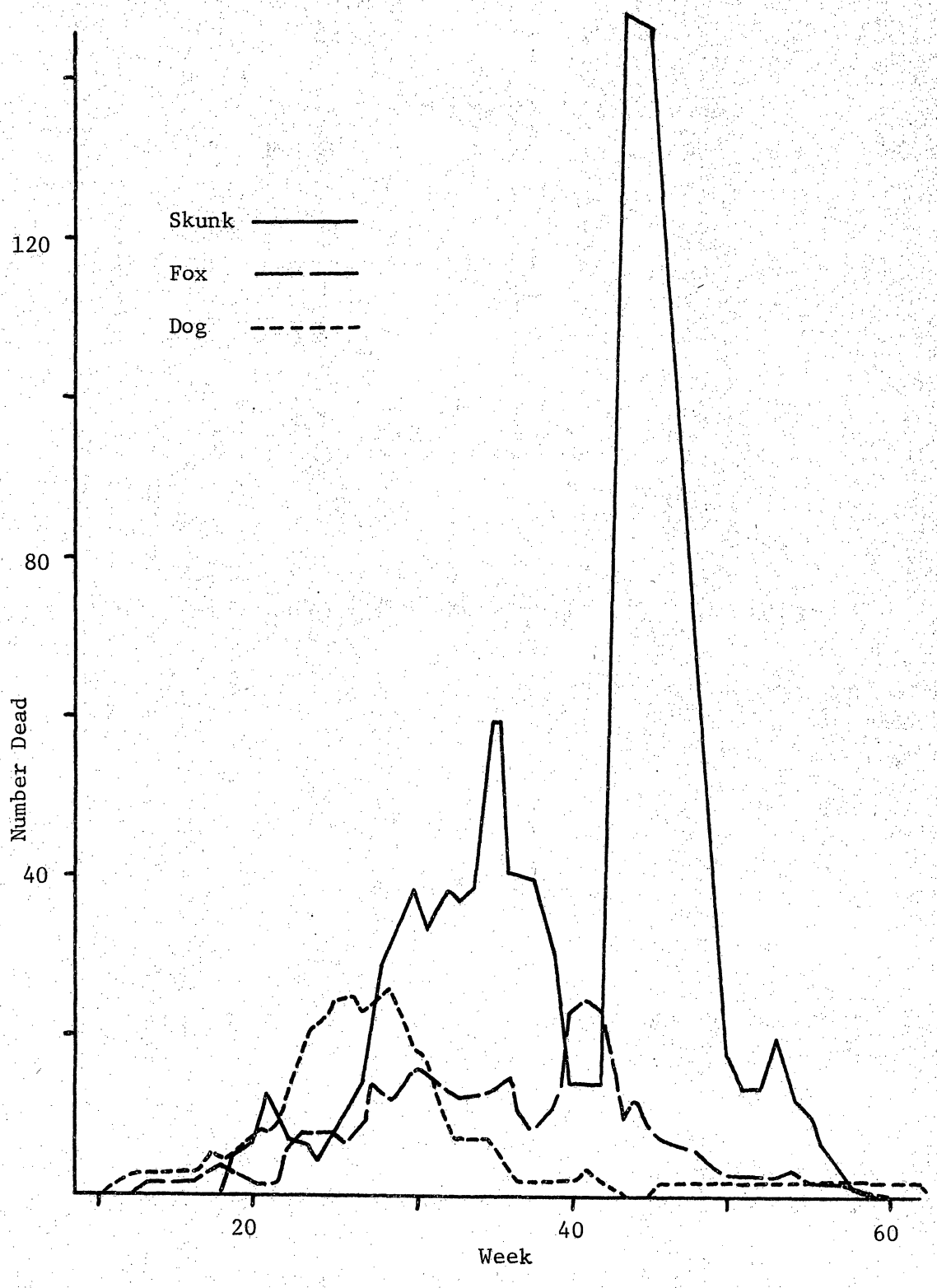

Fig. 17. Number of animals dying per week under a strategy of fox trapping.

in the severity of the two peaks of fox deaths were detected under this control strategy. However it is important to note that control of fox populations caused no apparent reduction in the weekly deaths of other species. In fact, the results indicate a slight increase in the severity of the epidemic in the skunk population.

Fig. 18 shows a similar reduction in the weekly number of infections in foxes, but little or no change in the number of infections in other species.

Fig. 19 indicates that fox population reduction by trapping slightly decreased the number of symptomatic rabies carriers of that species present. However it appears that reduced population levels caused a higher prevalence of asymptomatic carriers in foxes throughout the epidemic. The reservoir of asymptomatic foxes remaining after 100 weeks of simulation was slightly higher than that remaining under a no control strategy.

Rabies Control Utilizing Chemosterilants

Fig. 20 graphically displays the number of animals dying of rabies each week under a control strategy of fox sterilization. The reproductive capacity of the fox population was assumed to have been reduced by 25 percent, for demonstration purposes. It should be noted that the results of simulation under this strategy are identical to those under no rabies control for the first 32 weeks. The effects of the reduction of reproductive success are not active until reproduction takes place at that time. Comparison of Fig. 20 with Fig. 14 demonstrates a significant decrease in the severity of the fox rabies peak at week 40. However it appears that the course

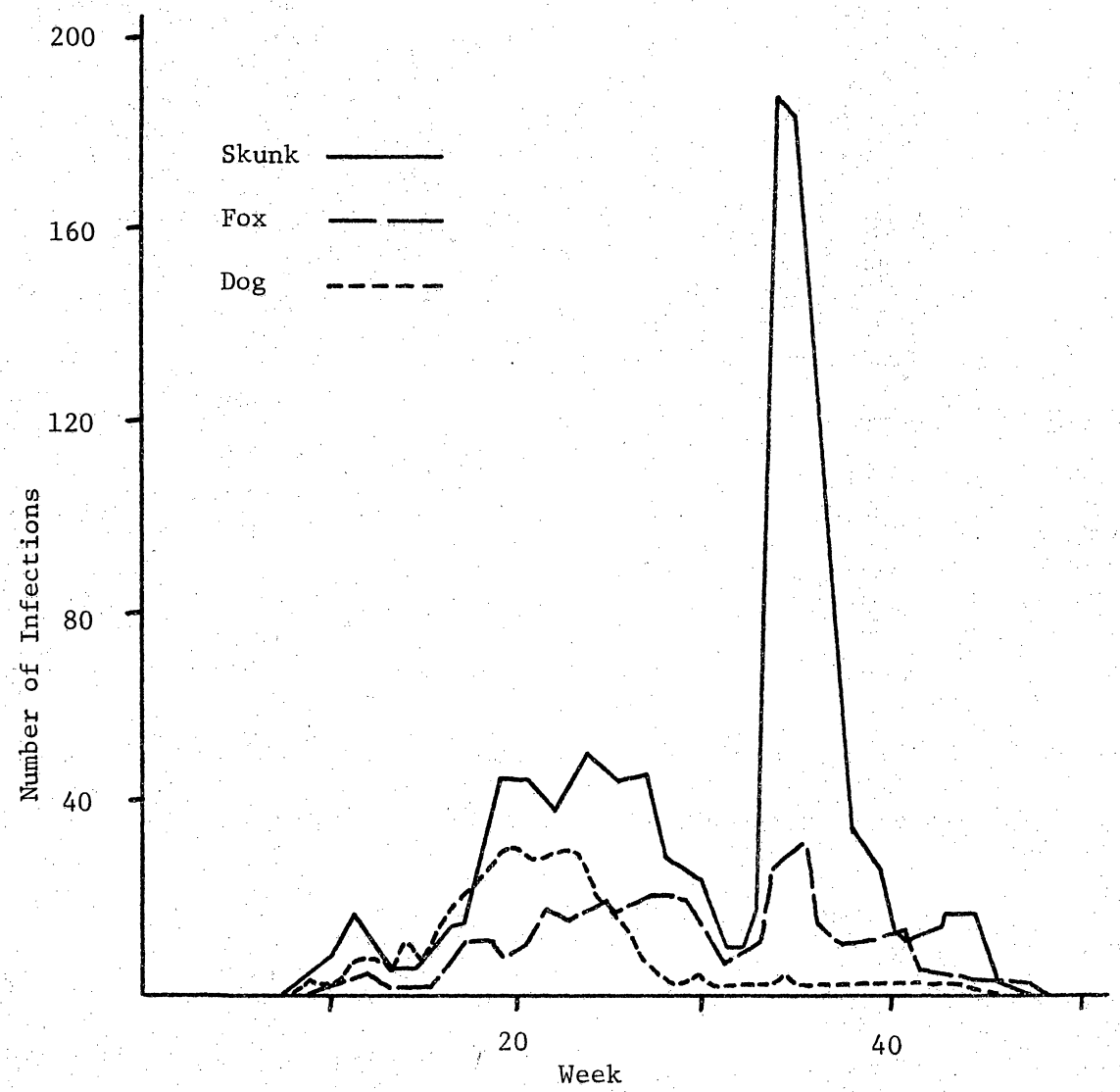

Fig. 18. Number of animals infected per week under a strategy of fox trapping.

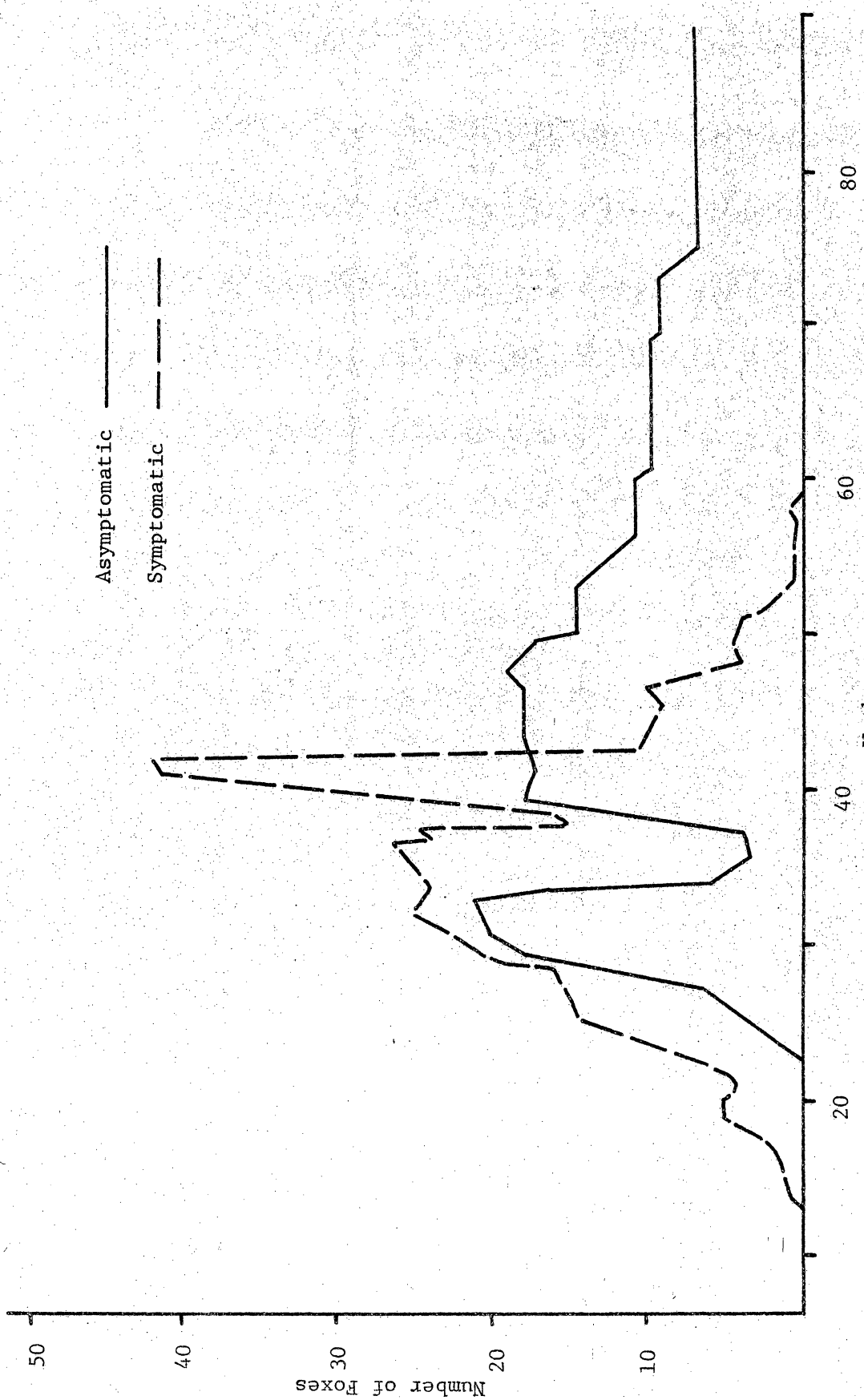

Fig. 19. Number of infective foxes present per week under a strategy of fox trapping.

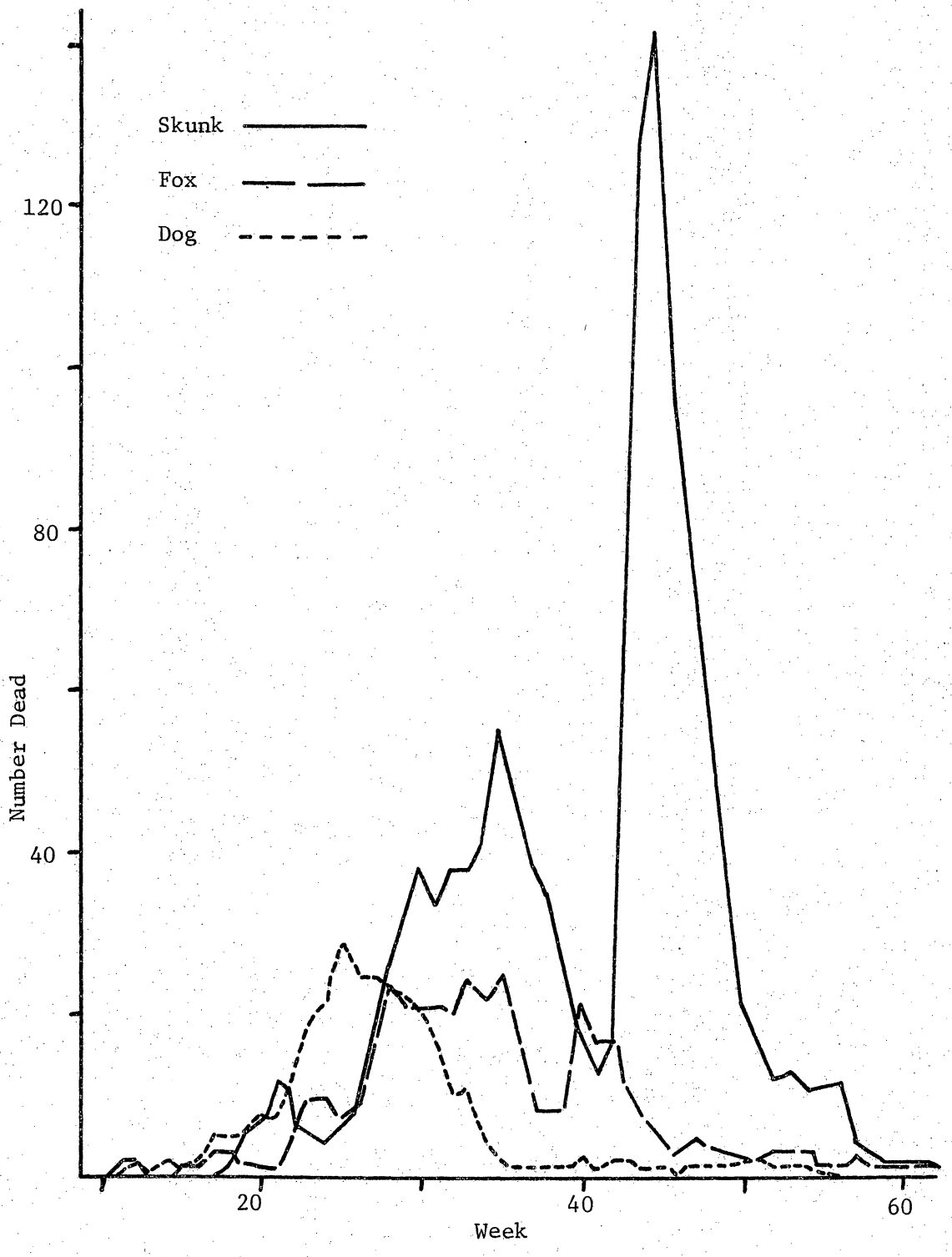

Fig. 20. Number of animals dying per week under a strategy of fox sterilization.

of the epidemic in skunk and dog populations is little affected by fox sterilization.

A corresponding decrease in weekly fox infections may be observed during weeks 34 and 35 (see Fig. 21). The epidemic in the other two species remains relatively unaffected.

Comparison of Fig. 22 with Fig. 16 shows the effects of fox sterilization on the number of infective foxes present each week. The strategy of sterilization reduced peak numbers of both symptomatic and asymptomatic carriers in the fox population. At low population levels, from week 80 to week 100, a decrease in the reservoir of asymptomatic foxes may be detected (three or four carriers).

Rabies Control by Vaccination

The simulated use of wildlife vaccinating devices to immunize 25 percent of the fox population before the start of the 100 week simulation period reduced the severity of the initial peak of fox deaths (see Fig. 23). However it is important to note that this strategy caused a sharp increase in fox deaths during the second peak at week 40. The probable explanation is that more foxes survived to breed because of immunization and thereby introduced a larger number of susceptible young to the population than normal.

Fig. 24 reflects the increased severity of the epidemic in foxes after the first breeding, with a record of almost 18 more fox infections than normal at week 34.

Fig. 25 displays the numbers of symptomatic and asymptomatic carriers in the fox population throughout the epidemic. Significantly more symptomatic foxes are present at week 39 under a

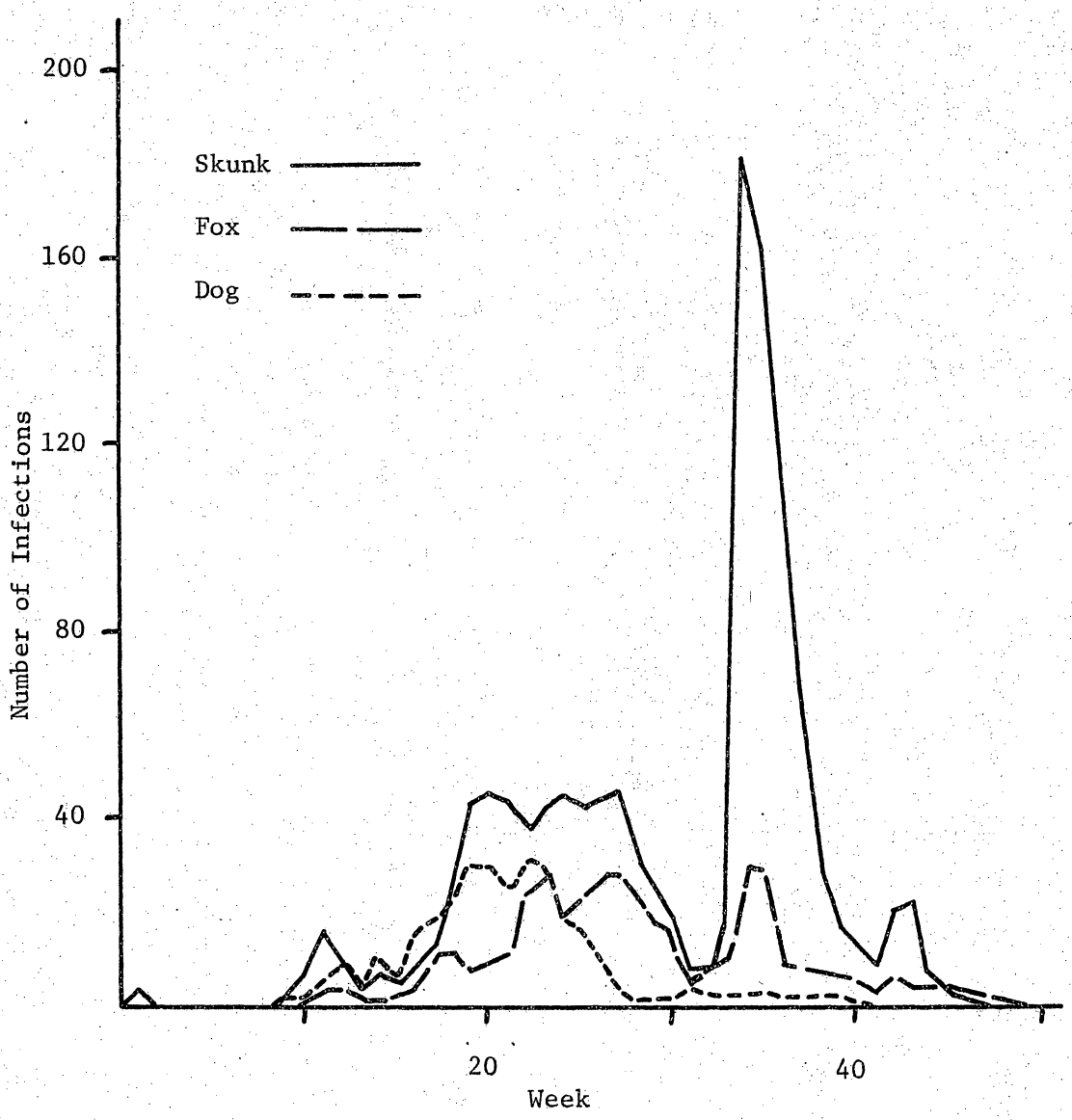

Fig. 21. Number of animals infected per week under a strategy of fox sterilization.

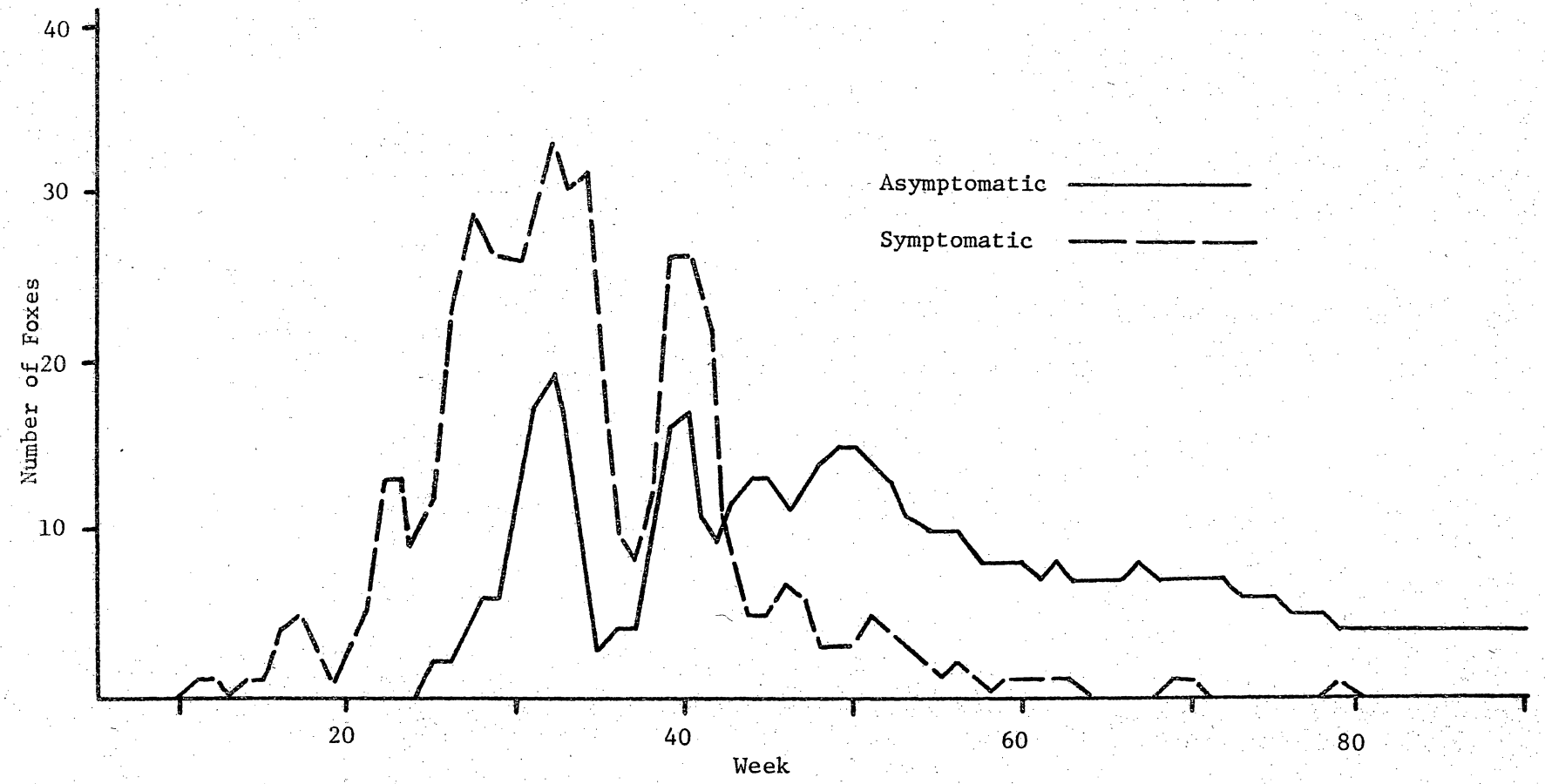

Fig. 22. Number of infective foxes present per week under a strategy of fox sterilization.

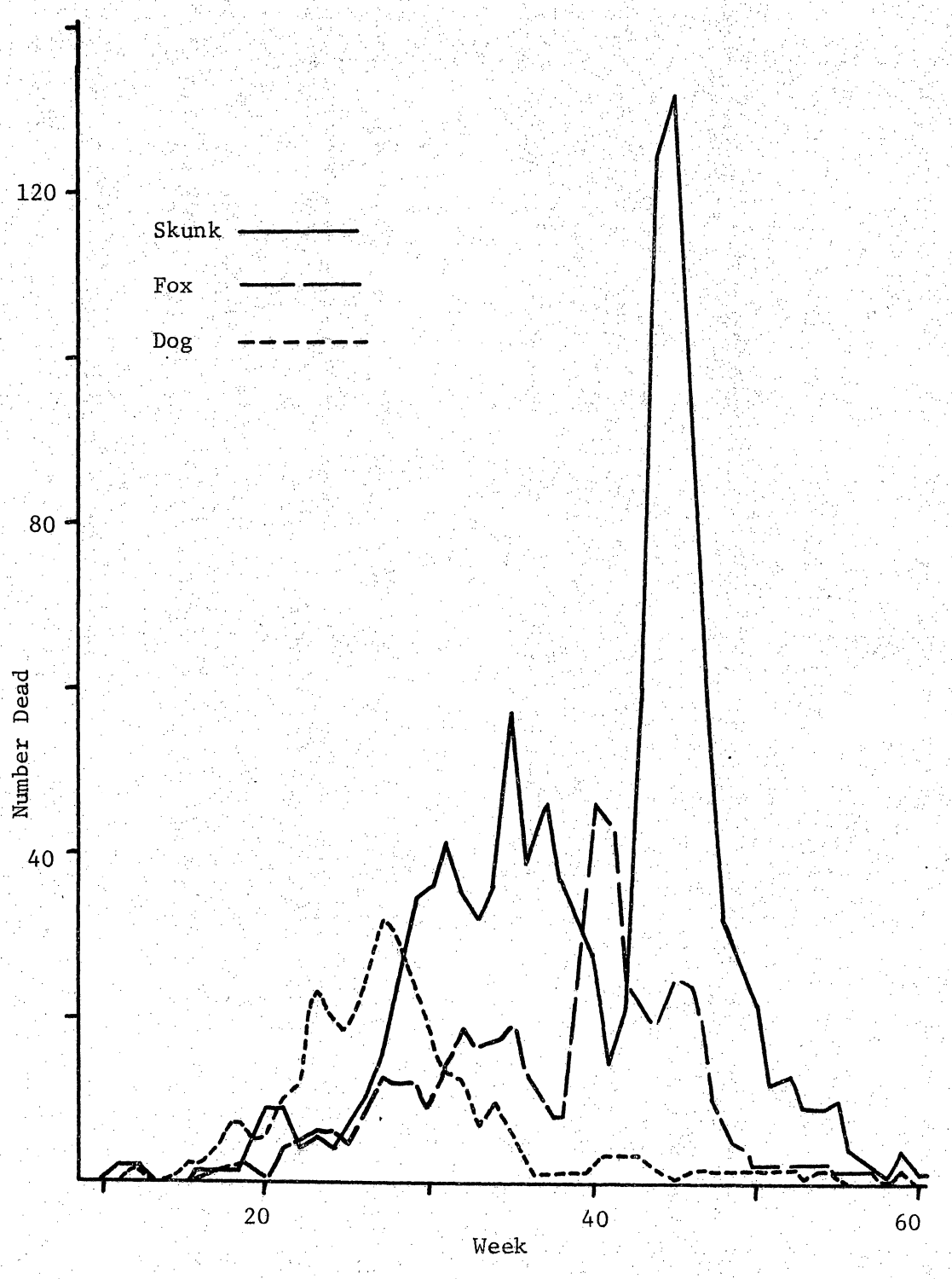

Fig. 23. Number of animals dying per week under a strategy of fox vaccination.

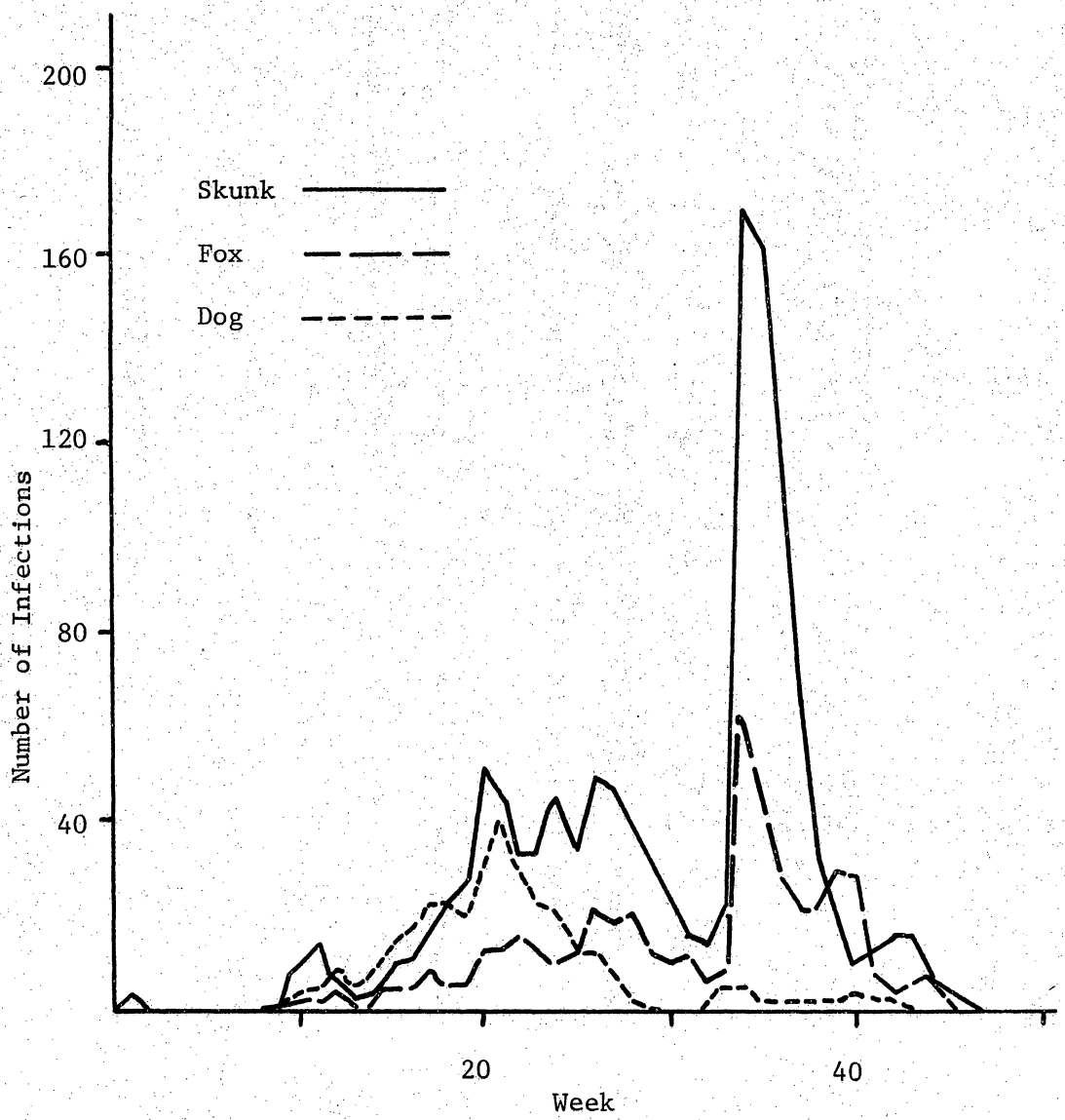

Fig. 24. Number of animals infected per week under a strategy of fox vaccination.

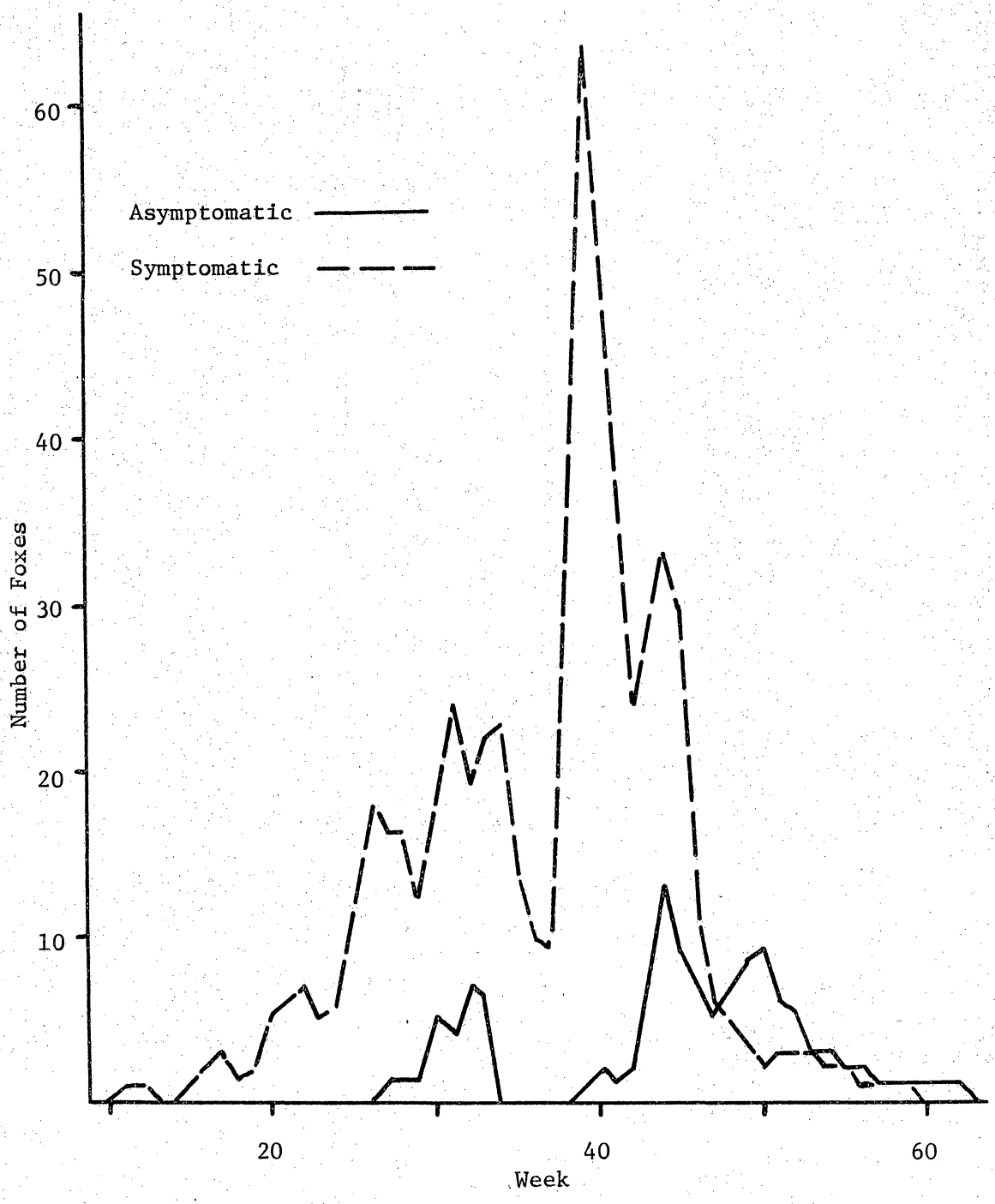

Fig. 25. Number of infective foxes present per week under a strategy of fox vaccination.

vaccination strategy than are present with no control. However no carriers, symptomatic or asymptomatic, remain after 62 weeks of simulation. The reservoir of asymptomatic infectives noted under a no control situation is no longer present.

DISCUSSION

ANALYSIS OF RABIES REPORTS

The results of the analyses of rabies report patterns indicate that wildlife rabies outbreaks display little similarity in either time or space relationships between individual rabies cases. The patterns of rabies reports appear to be most closely related to the patterns of human population in each county, as suggested by Constantine (1967a:885), and to the physiography of the region. The pattern of human population is itself closely correlated to physiography, so that the latter may indeed be the prime factor in determining the reported incidence of rabies in a given area. The differences noted between Washington County and Nelson County point up an excellent example of this relationship. The former is cut longitudinally by a long valley and has a relatively dispersed human population engaged in livestock raising. The latter is wooded and mountainous, with distinct centers of human population. Rabies incidences were widespread and followed a wavelike pattern in Washington County, while they were confined to a relatively small portion of Nelson County near foci of human population.

All four curves in Fig. 11 appear completely dissimilar. However the rabies reports represented were gathered over arbitrary time periods and with varying diligence in each county. The starting point of reporting relative to the severity of the epidemics may also be variable. By shifting the Bedford County curve approximately 17 weeks to the left a relatively close match may be achieved with Washington County. Successful analysis of rabies report data in

the future will depend upon a more uniform reporting system than was available at the time of this study.

The biases to which wildlife rabies reports are subject are many and largely unmeasured. Many authorities (Parker 1961; Beck, personal communication 1970) feel that rabies reporting in rural areas is unreliable and provides a weak basis for detailed analysis. In view of the high variability of results obtained in this study it would appear that any future analysis of rabies patterns by similar techniques would be fruitless until more accurate reporting systems are devised.

COMPUTER SIMULATION OF RABIES EPIDEMIOLOGY

Model Testing

The wildlife rabies epidemiology model, as presented, is yet untested. Before any amount of reliance may be placed upon results obtained by use of this model, considerable time will have to be spent in its validation. The results of rabies modeling by computer will have to be carefully compared to detailed field data concerning rabies epidemics in wild populations. As noted earlier such data are not yet available.

Even after the accuracy of the model has been confirmed its sensitivity to control measures will have to be compared with that of similar wildlife populations. If modeled changes in rabies incidence due to control do not agree with actual changes in nature the model will have to be suitably modified. The untested nature of the present rabies model points out the major difficulty encountered in its construction, the lack of adequate information concerning wildlife rabies.

Information Gaps

At the present time the requisite information for testing and validation of a wildlife rabies model is unavailable. The time distributions of viral incubation periods and periods of clinical symptoms are relatively well established for most species. However, nearly all of the probabilities used for demonstration purposes were little more than best estimates gleaned from the implications of current rabies literature. For many of these probabilities, laboratory experimentation has failed to yield even mean values. An outstanding example is the phenomenon of density stress and its relationship to the probabilities of infection success and of active symptoms appearing versus an asymptomatic carrier state. Selye (1956) has documented the general implications of stress phenomena, but little rabies-specific work has been attempted yet. For simplicity, straight line relationships between population density and related probabilities were assumed. However it seems likely that curvilinear relationships would be more realistic.

The other major gap in our knowledge of rabies epidemiology is the behavior of rabid animals and their rates of contact with other species. Detailed telemetry studies of animals under the influence of hallucinogens may yield suitable information in this area.

Sample Simulation Results

The results of the four sample simulations performed using the GASP-based model of rabies epidemiology demonstrate the general utility of such a procedure in combating disease. By making appropriate changes in the values of PCTRED, PCTTRP, and PCTVAC, the investigator

is enabled to simulate the effect of the varying control strategies of sterilization, trapping, and vaccination upon the course of a hypothetical rabies epidemic. He may thereby compare the effects of several alternate control strategies and select that one which best achieves his goals. The model may be used to aid persons responsible for rabies control in deciding upon optimum procedures before embarking upon expensive control programs. The risk of making improper or ineffective decisions can thereby be reduced.

For example, the results of the four sample simulations previously presented indicate several important relationships. First, control measures applied to a single species (foxes) appear to have no effect on the course of a rabies epidemic in any other species. The rabies model was constructed with interspecies contact as an integral part, but control measures do not appear to cause significant rabies reductions for species other than those to which they are applied. These results indicate that control measures must be applied to all susceptible wildlife populations in order to obtain complete rabies eradication.

Secondly, of the three control measures tested, only vaccination of wild foxes effectively eliminated the reservoir of asymptomatic carriers in that species. Fox trapping, by reducing both population levels and the probability of displaying active symptoms, actually allowed a greater number of asymptomatic carriers to remain. An increase in the severity of fox rabies peak near week 40 was noted under the fox vaccination strategy. The probable cause is increased reproduction, as a larger number of immune breeders lived to reproduce. If vaccination was undertaken on a continuous basis and the influx

of new susceptibles due to reproduction could be immunized it seems reasonable to assume that this increase in the secondary fox rabies peak would be eliminated. The role of reproduction in rabies perpetuation within wildlife populations may be more important than previously thought. The increase in population density associated with reproduction could conceivably stimulate the appearance of active symptoms in previously asymptomatic carriers, resulting in a new outbreak of rabies among the susceptible young.

The Expense of Simulation

The GASP-based computer model of wildlife rabies epidemiology presented was designed with the specific goal of including as many of the significant factors affecting the disease as possible. In addition it has been constructed to be as adaptable as possible to new advances of our knowledge of rabies. These two factors, combined with the stochastic nature of the model, make its use in the simulation of rabies practical only for those having access to relatively large and efficient computers. Each of the four sample simulations was executed on an IBM 360 model 65. Storage requirements were approximately 205K and program execution lasted 40 to 50 minutes in each case. A program of this magnitude would cost approximately 170 dollars per run at standard processing rates.

Expansion of the Model

The rabies epidemiology model, as presented, may accomodate a complex of up to four wildlife species. Storage limitations of the computing system upon which the program was run for this study necessitated this maximum. The expansion of program capacity, up

to 10 species, is possible on larger computer systems by appropriate changes of array sizes in all DIMENSION and COMMON statements. No other modifications are necessary.

The use of this model is not strictly limited to the simulation of rabies in wildlife species. Man or livestock might be other species easily included. A most helpful addition to the model would be an index of the economic losses due to rabies under each control strategy. For every head of cattle lost to rabies an average dollar value could be added to the total economic loss. For every man contacted by a rabid animal the expense of a series of antirabies innoculations could be added to this total.

Finally the computer model of rabies can be modified to fit other diseases of interest very rapidly. In most cases only revised data input will be required to adequately simulate another disease system. The use of a discrete-time simulator, such as GASP, can be a significant addition to the tools of the epidemiologist. A lengthy review of epidemiological literature disclosed that the majority of models used in the study of disease patterns have been strictly concerned with population phenomena. The typical model has been a simple algorithm which relates the number of animals dying at any time to the number dead at the previous check. The GASP-based simulator, on the other hand, assumes a more detailed view of a disease system, modifying accumulators at every point in time when changes are made in that system. The basic units of this model are individual animals, not entire populations. System variables are examined only at weekly intervals for simplicity in data presentation.

The linkage of this disease simulator to the CALCOMP plotter would be one method by which the investigator could keep an even more detailed record of epidemic patterns.

The use of computer simulation in the study of natural resource problems is only now emerging as a new and useful tool. The wise manager of animal populations will recognize its potential and will use simulation in the study of his own problems. Computer simulation cannot make the proper management decisions, but it can aid the population manager in making them.

SUMMARY

The modeling of wildlife rabies epidemiology was approached by means of two different procedures. Initial analysis involved the search for patterns of rabies incidences common to ecologically divergent areas. Rabies report data collected by the public health departments of three Virginia counties from 1966 to 1969 were examined for possible similarities of time and distance separation between incidences. In addition data collected from 1969 to 1970 in Imperial County, California were analyzed. The results of these analyses were widely divergent, leading the investigator to believe that patterns of rabies occurrence are largely a function of the topography of each local area. No definite pattern of time or distance separations could be detected as characteristic of rabies outbreaks.

A detailed computer model of wildlife rabies epidemiology was written in FORTRAN IV for an IBM model 65 system. Based on a general activity simulating program, GASP II, this model was presented as an aid to wildlife disease control decision making. Four sample runs simulating an uncontrolled rabies epidemic and three separate management strategies of trapping, sterilization, and vaccination were summarized for demonstration purposes. Preliminary examination of the results have indicated the utility of the model in grouping many previously separate and detailed processes into a unified whole. The investigator is thereby allowed to examine not only the processes, but the results of their interactions as well. Initial analysis has indicated that reproductive capacity

of wildlife populations involved in a rabies outbreak may be of more importance in the course of the epidemic than previously thought. Likewise the effect of interspecies contact may be less significant than once supposed. Suggestions concerning future use and testing of the model were presented.

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APPENDIX

APPENDIX I. PROGRAM FOR ANALYSIS OF RABIES REPORTS.

```
      DIMENSION DIST(90,90),NDAYS(90,90),NOBS(200,30),NTSEP(
     1NDSEP(30)
      DATA NTSEP/200*0/,NDSEP/30*0/,NOBS/6000*0/,NTOBS/0/
      READ(5,1000) N
C
C*****READ AND PRINT THE DISTANCES AND TIMES BETWEEN INCIDEN
C
      DO 10 I=1,N
   10 READ(5,100)(DIST(I,M),M=1,N)
      DO 20 I=1,N
   20 READ(5,200)(NDAYS(I,M),M=1,N)
C
C*****CALCULATE THE NUMBER OF OBSERVATIONS BY CLASS.
C
      DO 30 I=1,N
      DO 30 M=1,N
      MILES=DIST(I,M)+.9999
      IF (MILES .EQ. 0) GO TO 30
      IF (MILES .GT. 30) MILES=30
      ITIME=NDAYS(I,M)/7+1
      IF (ITIME .GT. 200) ITIME=200
      NTSEP(ITIME)=NTSEP(ITIME)+1
      NDSEP(MILES)=NDSEP(MILES)+1
      NTOBS=NTOBS+1
      NOBS(ITIME,MILES)=NOBS(ITIME,MILES)+1
   30 CONTINUE
C
C*****PRINT TABLE.
C
      WRITE(6,300) (I,I=1,30)
      DO 40 ITIME=1,45
   40 WRITE(6,400)ITIME,(NOBS(ITIME,MILES),MILES=1,30),NTSEP
      WRITE(6,900) (NDSEP(MILES),MILES=1,30),NTOBS
C
C*****FORMAT STATEMENTS.
C
  100 FORMAT(26F3.1/26F3.1/26F3.1/8F3.1)
  200 FORMAT(26I3/26I3/26I3/8I3)
  300 FORMAT(1H1,55X,'DISTANCE IN MILES'/,1X,'WEEK',30I4,'TO
  400 FORMAT(1X,32I4)
  500 FORMAT(1X,I2,2X,29F4.1)
  600 FORMAT(1X,I2,2X,29I4)
  700 FORMAT(1H1,55X,'ARRAY DIST'/,5X,29I4/)
  800 FORMAT(1H1,55X,'ARRAY NDAYS'/,5X,29I4/)
  900 FORMAT('0','TOTAL',I3,30I4)
 1000 FORMAT(I3)
      CALL EXIT
      END
```

APPENDIX II. CASP II WILDLIFE RABIES SIMULATOR.

```
C*****MAIN PROGRAM
      DIMENSION NSET(12,1500)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NOQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP,NTMODE,TPOP,TPOPI,DECAY,TIND(4),TINDI(4),
     1TSIND(4),TSINDI(4),PCTRED(4),PCTVAC(4),PCTTRP(4),
     2XMAXIN(4),XMININ(4),TCONTP(4,4,2),TCONT(4,4,2),
     3PSYMPP(4),PSYMPT(4),PSYMPM(4),PGD(4,4,2),PTRANS
     4(4,2,4,4),TFINDP(4,4,2),TFIND(4,4,2),PFAT(4,4),
     5PGDFAT(4),ADD(4),NAI(4),NEXT
      COMMON XASM(4),XDASM(4),XSM(4),XDSM(4),XDINF(4),
      COMMON XASM(4),XDASM(4),XSM(4),XDSM(4),XDINF(4),
C
C*****IDENTIFY CARD READER AND PRINTER.
C
      NCRDR=5
      NPRNT=6
C
C*****READ NUMBER OF SPECIES, NUMBER OF POSSIBLE TRANSMIS-
C*****SION MODES,AND DECAY PERIOD.
C
      READ(NCRDR,1000) NSP,NTMODE,DECAY
 1000 FORMAT(2I3,F3.0)
C
C*****READ INPUT PROBABILITIES AND TIME DISTRIBUTIONS.
C***** I = DONOR SPECIES.
C***** J = RECIPIENT SPECIES.
C***** K = SYMPTOM TYPE.
C***** L = TRANSMISSION MODE.
C
C*****READ MINIMUM AND MAXIMUM TIME BETWEEN CONTACT, BY
C*****SPECIES.
C
      DO 20 I=1,NSP
   20 READ(NCRDR,1004)((TCONTP(I,J,M),M=1,2),J=1,NSP)
 1004 FORMAT(20F4.0)
C
C*****READ THE PROBABILITY OF A SUSCEPTIBLE MEMBER OF
C*****SPECIES (J) CONTRACTING RABIES IF ATTACKED BY A
C*****MEMBER OF SPECIES (I), SHOWING SYMPTOMS (K)
C
      DO 30 J=1,NSP
   30 READ(NCRDR,1003)((PGD(J,I,K),I=1,NSP),K=1,2)
```

APPENDIX II (CONTINUED).

```
 1003 FORMAT(20F4.2)
C
C*****READ THE PROBABILITY OF ACTIVE SYMPTOMS IN AN IN-
C*****FECTED MEMBER OF SPECIES (I).
C
      READ(NCRDR,1005)(PSYMPP(I),I=1,NSP)
 1005 FORMAT(10F4.2)
C
C*****READ THE PROBABILITY OF TRANSMISSION MODE (L) BEING
C*****USED BY SPECIES (I), SHOWING SYMPTOMS (K), IN
C*****CONTACTING SPECIES (J).
C
      DO 40 I=1,NSP
      DO 40 J=1,NSP
   40 READ(NCRDR,1006)((PTRANS(I,K,J,L),L=1,NTMODE),K=1,2)
 1006 FORMAT(8F4.2)
C
C*****READ THE MINIMUM AND MAXIMUM TIME FOR THE BODY OF A
C*****MEMBER OF SPECIES (I) TO BE FOUND BY A MEMBER OF
C*****SPECIES (J).
C
      DO 45 I=1,NSP
   45 READ(NCRDR,1004)((TFINDP(I,J,M),M=1,2),J=1,NSP)
C
C*****READ THE PROBABILITY OF SPECIES (I) BEING EATEN BY
C*****SPECIES(J).
C
      DO 50 I=1,NSP
   50 READ(NCRDR,1005)(PEAT(J,I),J=1,NSP)
C
C*****READ THE PROBABILITY OF A SUSCEPTIBLE MEMBER OF
C*****SPECIES (J) CONTRACTING RABIES BY EATING THE FLESH
C*****OF A RABID ANIMAL.
C
      READ(NCRDR,1005)(PGOEAT(J),J=1,NSP)
C
C*****READ POPULATION SIZE,MINIMUM PROBABILITY OF ACTIVE
C*****SYMPTOMS, MINIMUM AND MAXIMUM ANNUAL INCREASE FAC-
C*****TORS, PERCENT REPRODUCTIVE REDUCTION,PERCENT
C*****VACCINATION,AND PERCENT OF THE POPULATION TRAPPED,
C*****BY SPECIES.
C
   92 DO 100 I=1,NSP
  100 READ(NCRDR,1001) TINDI(I),PSYMPM(I),XMININ(I),XMAXIN
     1(I),PCTRED(I),PCTVAC(I),PCTTRP(I)
 1001 FORMAT(F8.0,6F5.2)
C
C*****CALCULATE TOTAL POPULATION DENSITY.
C
```

APPENDIX II (CONTINUED).

```
      TPOPI=0.
      TPOP=0.
      DO 10 I=1,NSP
      TIND(I)=TINDI(I)*(1.0-PCTTRP(I))
      TSIND(I)=TIND(I)*(1.0-PCTVAC(I))
      TSINDI(I)=TSIND(I)
      TPOPI=TPOPI+TIND(I)
   10 TPOP=TPOP+TIND(I)
C
C*****PRINT INPUT VALUES.
C
      DO 110 I=1,NSP
  110 WRITE(NPRNT,2000) I,TINDI(I),PSYMPM(I),XMININ(I),
     1XMAXIN(I),PCTREC(I),PCTVAC(I),PCTTRP(I)
 2000 FORMAT(1H ,I3,F8.0,6(5X,F5.2))
      WRITE(NPRNT,2001) NTMODE
 2001 FORMAT(1H ,I3)
      DO 120 I=1,NSP
  120 WRITE(NPRNT,2004)((TCONTP(I,J,M),M=1,2),J=1,NSP)
 2004 FORMAT(1H ,20(2X,F4.0))
      DO 130 J=1,NSP
  130 WRITE(NPRNT,2003)((PGO(J,I,K),I=1,NSP),K=1,2)
 2003 FORMAT(1H ,20(2X,F4.2))
      WRITE(NPRNT,2005)(PSYMPP(I),I=1,NSP)
 2005 FORMAT(1H ,10(2X,F4.2))
      DO 140 I=1,NSP
      DO 140 J=1,NSP
  140 WRITE(NPRNT,2006)((PTRANS(I,K,J,L),L=1,NTMODE),K=1,2)
 2006 FORMAT(1H ,8(2X,F4.2))
      DO 145 I=1,NSP
  145 WRITE(NPRNT,2004)((TFINDP(I,J,M),M=1,2),J=1,NSP)
      DO 150 I=1,NSP
  150 WRITE(NPRNT,2005)(PEAT(J,I),J=1,NSP)
      WRITE(NPRNT,2005)(PGOEAT(J),J=1,NSP)
C
C*****INITIALIZE ACCUMULATORS.
C
      DO 60 I=1,NSP
      NAI(I)=0
      XASM(I)=0.
      XDASM(I)=0.
      XSM(I)=0.
      XDSM(I)=0.
      XDINF(I)=0.
      XFAIL(I)=0.
      XEATIN(I)=0.
      XATS(I)=0.
      XDEAD(I)=0.
   60 XTDEAD(I)=0.
```

APPENDIX II (CONTINUED).

```
      DO 70 J=1,NTMODE
   70 N(J)=0
      CALL GASP(NSET)
C
C*****CHECK FOR CONTINUATION OF PROGRAM.
C
   90 READ(NCRDR,1002) NEXT
 1002 FORMAT(I1)
      IF (NEXT-0) 91,91,92
   91 CALL EXIT
      END
```

APPENDIX II (CONTINUED).

```
      SUBROUTINE EVNTS(IX,NSET)
      DIMENSION NSET(12,1)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NOQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP,NTMODE,TPOP,TPOPI,DECAY,TIND(4),TINDI(4),
     1TSIND(4),TSINDI(4),PCTRED(4),PCTVAC(4),PCTTRP(4),
     2XMAXIN(4),XMININ(4),TCONTP(4,4,2),TCONT(4,4,2),
     3PSYMPP(4),PSYMPT(4),PSYMPM(4),PGO(4,4,2),PTRANS
     4(4,2,4,4),TFINDP(4,4,2),TFIND(4,4,2),PEAT(4,4),
     5PGOEAT(4),ADD(4),NAI(4),NEXT
      COMMON XASM(4),XDASM(4),XSM(4),XDSM(4),XDINF(4),
     1XFAIL(4),XEATIN(4),XDEAD(4),XTDEAD(4),XATS(4),N(4)
      GO TO (1,2,3,4,5,6,7,8), IX
    1 CALL SYMPTM(NSET)
      RETURN
    2 CALL CONTCT(NSET)
      RETURN
    3 CALL DEAD(NSET)
      RETURN
    4 CALL OUTPUT(NSET)
      RETURN
    5 CALL ENDSIM(NSET)
      RETURN
    6 CALL PRDATN(NSET)
      RETURN
    7 CALL REPROD(NSET)
      RETURN
    8 CALL BETAXF
      RETURN
      END
```

APPENDIX II (CONTINUED).

```
      SUBROUTINE SYMPTM(NSET)
      DIMENSION NSET(12,1)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NOQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KCL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP,NTMODE,TPOP,TPOPI,DECAY,TIND(4),TINDI(4),
     1TSIND(4),TSINDI(4),PCTRED(4),PCTVAC(4),PCTTRP(4),
     2XMAXIN(4),XMININ(4),TCONTP(4,4,2),TCONT(4,4,2),
     3PSYMPP(4),PSYMPT(4),PSYMPM(4),PGO(4,4,2),PTRANS
     4(4,2,4,4),TFINDP(4,4,2),TFIND(4,4,2),PEAT(4,4),
     5PGOEAT(4),ADD(4),NAI(4),NEXT
      COMMON XASM(4),XDASM(4),XSM(4),XDSM(4),XDINF(4),
     1XFAIL(4),XEATIN(4),XDEAD(4),XTDEAD(4),XATS(4),N(4)
C
C*****MODIFY PROBABILITIES ON THE BASIS OF POPULATION
C*****CHANGES.
C
      IF (TPOP) 2,2,1
    2 CALL ENDSIM(NSET)
      RETURN
    1 NSPR=ATRIB(3)
      IF (TIND(NSPR)) 100,100,120
  120 DO 130 I=1,NSP
      PSYMPT(I)=PSYMPP(I)*TIND(I)/TINDI(I)
      IF (PSYMPT(I) .LE. PSYMPM(I)) PSYMPT(I)=PSYMPM(I)
      IF (PSYMPT(I) .GT. 1. ) PSYMPT(I)=1.
  130 CONTINUE
      DO 110 I=1,NSP
      DO 110 J=1,NSP
      IF (TIND(J)) 110,110,3
    3 DO 110 K=1,2
      TFIND(I,J,K)=TFINDP(I,J,K)/(TIND(J)/TINDI(J))
      TCONT(I,J,K)=TCONTP(I,J,K)/(TIND(J)/TINDI(J))
  110 CONTINUE
C
C*****DETERMINE SYMPTOMS.
C
      ASM=DRAND(ISEED)
      IF (ASM-PSYMPT(NSPR)) 10,10,4
C
C*****INDIVIDUAL IS ASYMPTOMATIC.
C
    4 NSYM=2
      CALL TMST(XASM(NSPR),TNOW,NSP+NSPR,NSET)
```

APPENDIX II (CONTINUED).

```
      XASM(NSPR)=XASM(NSPR)+1.
      XDASM(NSPR)=XDASM(NSPR)+1.
      NAI(NSPR)=NAI(NSPR)+1
      GO TO 20
C
C*****INDIVIDUAL SHOWS ACTIVE SYMPTOMS.
C
   10 NSYM=1
      CALL TMST(XSM(NSPR),TNOW,NSPR,NSET)
      XSM(NSPR)=XSM(NSPR)+1.
      XDSM(NSPR)=XDSM(NSPR)+1.
C
C*****SCHEDULE DEATH.
C
      ATRIB(1)=TNOW+RNORM(NSP+NSPR)
      ATRIB(2)=3.
      ATRIB(3)=NSPR
      DO 40 I=4,6
   40 ATRIB(I)=0.
      ATRIB(7)=ATRIB(1)
      CALL FILEM(1,NSET)
C
C*****SCHEDULE FIRST CONTACT WITH EACH SPECIES.
C
   20 DO 30 I=1,NSP
      IF (TIND(I)) 30,30,22
   22 ATRIB(1)=TNOW+UNFRM(TCONT(NSPR,I,1),TCONT(NSPR,I,2))
      IF (NSYM-1) 5,5,21
    5 IF (ATRIB(1)-ATRIB(7)) 21,30,30
   21 ATRIB(2)=2.
      ATRIB(3)=I
      ATRIB(4)=NSPR
      ATRIB(5)=NSYM
      ATRIB(6)=0.
      IF (NSYM .GT. 1) ATRIB(6)=NAI(NSPR)
      CALL FILEM(1,NSET)
   30 CONTINUE
  100 RETURN
      END
```

APPENDIX II (CONTINUED).

```
      SUBROUTINE CONTCT(NSET)
      DIMENSION NSET(12,1)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NCO,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP,NTMODE,TPOP,TPOPI,DECAY,TIND(4),TINDI(4),
     1TSIND(4),TSINDI(4),PCTRED(4),PCTVAC(4),PCTTRP(4),
     2XMAXIN(4),XMININ(4),TCONTP(4,4,2),TCONT(4,4,2),
     3PSYMPP(4),PSYMPT(4),PSYMPM(4),PGD(4,4,2),PTRANS
     4(4,2,4,4),TFINDP(4,4,2),TFIND(4,4,2),PEAT(4,4),
     5PGOEAT(4),ACD(4),NAI(4),NEXT
      COMMON XASM(4),XDASM(4),XSM(4),XOSM(4),XDINF(4),
     1XFAIL(4),XEATIN(4),XDEAD(4),XTDEAD(4),XATS(4),N(4)
C
C*****MODIFY PROBABILITIES ON THE BASIS OF POPULATION
C*****CHANGES.
C
      IF (TPOP) 13,13,12
   13 CALL ENDSIM(NSET)
      RETURN
   12 NSPR=ATRIB(3)
      NSPD=ATRIB(4)
      IF (TIND(NSPR)) 100,100,120
  120 DO 130 I=1,NSP
      PSYMPT(I)=PSYMPP(I)*TIND(I)/TINDI(I)
      IF (PSYMPT(I) .LE. PSYMPM(I)) PSYMPT(I)=PSYMPM(I)
      IF (PSYMPT(I) .GT. 1.) PSYMPT(I)=1.
  130 CONTINUE
      DO 110 I=1,NSP
      DO 110 J=1,NSP
      IF (TIND(J)) 110,110,14
   14 DO 110 K=1,2
      TFIND(I,J,K)=TFINDP(I,J,K)/(TIND(J)/TINDI(J))
      TCONT(I,J,K)=TCONTP(I,J,K)/(TIND(J)/TINDI(J))
  110 CONTINUE
      NSYM=ATRIB(5)
C
C*****DETERMINE IF ANIMAL HAS STARTED TO SHOW SYMPTOMS,
C*****DUE TO STRESS.
C
      IF (ATRIB(5)-1.) 20,20,4
    4 ASM=DRAND(ISEED)
      IF (ASM-PSYMPT(NSPD)) 5,5,20
C
```

APPENDIX II (CONTINUED).

```
C*****ACTIVE SYMPTOMS ARE NOW DISPLAYED.
C
     5 ATRIB(5)=1.
       NSYM=1
       CALL TMST(XSM(NSPD),TNOW,NSPD,NSET)
       CALL TMST(XASM(NSPD),TNOW,NSP+NSPD,NSET)
       XSM(NSPD)=XSM(NSPD)+1.
       XASM(NSPD)=XASM(NSPD)-1.
       XATS(NSPD)=XATS(NSPD)+1.
C
C*****DETERMINE TIME OF DEATH.
C
       DTIME=TNOW+RNORM(NSP+NSPD)
C
C*****SEARCH NSET AND REMOVE ALL CONTACTS SCHEDULED FOR
C*****THIS ANIMAL LATER THAN DTIME.
C
       NVAL=ATRIB(6)*SCALE
       NDTIME=DTIME*SCALE
       DO 50 I=1,ID
       IF (NSET(6,I)-NVAL) 50,2,50
     2 IF (NSET(1,I)-NDTIME) 1,3,3
     1 NSET(5,I)=1.*SCALE
       NSET(6,I)=0
       NSET(7,I)=NDTIME
       GO TO 50
     3 CALL XMOVE(I,1,NSET)
    50 CONTINUE
C
C*****SCHEDULE DEATH.
C
    60 ATRIB(1)=DTIME
       ATRIB(2)=3.
       ATRIB(3)=NSPD
       DO 61 I=4,6
    61 ATRIB(I)=0.
       ATRIB(7)=ATRIB(1)
       CALL FILEM(1,NSET)
C
C*****SCHEDULE NEXT CONTACT WITH SPECIES (NSPR).
C
    20 ATRIB(1)=TNOW+UNFRM(TCONT(NSPD,NSPR,1),
      1TCONT(NSPD,NSPR,2))
       IF (NSYM-1) 6,6,21
     6 IF (ATRIB(1)-ATRIB(7)) 21,30,30
    21 ATRIB(2)=2.
       ATRIB(3)=NSPR
       ATRIB(4)=NSPD
       IF (ATRIB(5) .LE. 1.) ATRIB(6)=0.
```

APPENDIX II (CONTINUED).

```
      CALL FILEM(1,NSET)
C
C*****DETERMINE TRANSMISSION MODE.
C
   30 TRANS=DRAND(ISEED)
      XN=0.
      DO 10 I=1,NTMODE
      XN=XN+PTRANS(NSPD,NSYM,NSPR,I)
      IF (TRANS-XN) 7,7,10
   10 CONTINUE
    7 NTRANM=I
C
C*****IS CONTACTED INDIVIDUAL SUSCEPTIBLE?
C*****IF NOT, RETURN.
C
      IF (TSIND(NSPR)) 100,100,8
    8 SUS=DRAND(ISEED)
      PROSUS=TSIND(NSPR)/TIND(NSPR)
      IF (SUS-PROSUS) 9,9,100
C
C*****DOES INFECTION SUCCEED OR FAIL?
C*****IF FAILURE, RETURN
C
    9 GO=DRAND(ISEED)
      IF (GO-PGO(NSPR,NSPD,NSYM)) 11,11,101
   11 TSIND(NSPR)=TSIND(NSPR)-1.
      XDINF(NSPR)=XDINF(NSPR)+1.
      N(NTRANM)=N(NTRANM)+1
C
C*****SCHEDULE APPEARANCE OF SYMPTOMS.
C
      ATRIB(1)=TNOW+BETA(NSPR)
      ATRIB(2)=1.
      ATRIB(3)=NSPR
      DO 40 I=4,7
   40 ATRIB(I)=0.
      CALL FILEM(1,NSET)
      GO TO 100
  101 XFAIL(NSPR)=XFAIL(NSPR)+1.
  100 RETURN
      END
```

APPENDIX II (CONTINUED)

```
      SUBROUTINE DEAD(NSET)
      DIMENSION NSET(12,1)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NOQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP,NTMODE,TPOP,TPOPI,DECAY,TIND(4),TINDI(4),
     1TSIND(4),TSINDI(4),PCTREQ(4),PCTVAC(4),PCTTRP(4),
     2XMAXIN(4),XMININ(4),TCONTP(4,4,2),TCONT(4,4,2),
     3PSYMPP(4),PSYMPT(4),PSYMPM(4),PGO(4,4,2),PTRANS
     4(4,2,4,4),TFINDP(4,4,2),TFIND(4,4,2),PEAT(4,4),
     5PGOEAT(4),ADD(4),NAI(4),NEXT
      COMMON XASM(4),XDASM(4),XSM(4),XDSM(4),XDINF(4),
     1XFAIL(4),XEATIN(4),XDEAD(4),XTDEAD(4),XATS(4),N(4)
C
C*****MODIFY ACCUMULATORS.
C
      IF (TPOP) 2,2,6
    2 CALL ENDSIM(NSET)
      RETURN
    6 NSPD=ATRIB(3)
      IF (TIND(NSPD)) 100,100,1
    1 XTDEAD(NSPD)=XTDEAD(NSPD)+1.
      XDEAD(NSPD)=XDEAD(NSPD)+1.
      TPOP=TPOP-1.
      TIND(NSPD)=TIND(NSPD)-1.
      CALL TMST(XSM(NSPD),TNOW,NSPD,NSET)
      XSM(NSPD)=XSM(NSPD)-1.
C
C*****MODIFY PROBABILITIES ON THE BASIS OF POPULATION
C*****CHANGES.
C
  120 DO 130 I=1,NSP
      PSYMPT(I)=PSYMPP(I)*TIND(I)/TINDI(I)
      IF (PSYMPT(I) .LE. PSYMPM(I)) PSYMPT(I)=PSYMPM(I)
      IF (PSYMPT(I) .GT. 1.) PSYMPT(I)=1.
  130 CONTINUE
      DO 110 I=1,NSP
      DO 110 J=1,NSP
      IF (TIND(J)) 110,110,3
    3 DO 110 K=1,2
      TFIND(I,J,K)=TFINDP(I,J,K)/(TIND(J)/TINDI(J))
      TCONT(I,J,K)=TCONTP(I,J,K)/(TIND(J)/TINDI(J))
  110 CONTINUE
C
```

APPENDIX II (CONTINUED).

```
C*****SCHEDULE FIRST ENCOUNTER WITH BODY BY EACH SPECIES.
C
      DO 10 I=1,NSP
      IF (TIND(I)) 10,10,4
    4 ATRIB(1)=TNOW+UNFRM(TFIND(NSPD,I,1),TFIND(NSPD,I,2))
      ATRIB(2)=6.
      ATRIB(3)=I
      ATRIB(4)=NSPD
      ATRIB(5)=TNOW+DECAY
      IF (ATRIB(1)-ATRIB(5))5,10,10
    5 CALL FILEM(1,NSET)
   10 CONTINUE
  100 RETURN
      END
```

APPENDIX II (CONTINUED).

```
      SUBROUTINE PRDATN(NSET)
      DIMENSION NSET(12,1)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NOQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP,NTMODE,TPOP,TPOPI,DECAY,TIND(4),TINDI(4),
     1TSIND(4),TSINDI(4),PCTRED(4),PCTVAC(4),PCTTRP(4),
     2XMAXIN(4),XMININ(4),TCONTP(4,4,2),TCONT(4,4,2),
     3PSYMPP(4),PSYMPT(4),PSYMPM(4),PGO(4,4,2),PTRANS
     4(4,2,4,4),TFINDP(4,4,2),TFIND(4,4,2),PEAT(4,4),
     5PGOEAT(4),ADD(4),NAI(4),NEXT
      COMMON XASM(4),XDASM(4),XSM(4),XDSM(4),XDINF(4),
     1XFAIL(4),XEATIN(4),XDEAD(4),XTDEAD(4),XATS(4),N(4)
C
C*****MODIFY PROBABILITIES ON THE BASIS OF POPULATION
C*****CHANGES.
C
      IF (TPOP) 2,2,9
    2 CALL ENDSIM(NSET)
      RETURN
    9 NSPR=ATRIB(3)
      NSPD=ATRIB(4)
      IF (TIND(NSPR)) 1000,1000,120
  120 DO 130 I=1,NSP
      PSYMPT(I)=PSYMPP(I)*TIND(I)/TINDI(I)
      IF (PSYMPT(I) .LE. PSYMPM(I)) PSYMPT(I)=PSYMPM(I)
      IF (PSYMPT(I) .GT. 1.) PSYMPT(I)=1.
  130 CONTINUE
      DO 110 I=1,NSP
      DO 110 J=1,NSP
      IF (TIND(J)) 110,110,3
    3 DO 110 K=1,2
      TFIND(I,J,K)=TFINDP(I,J,K)/(TIND(J)/TINDI(J))
      TCONT(I,J,K)=TCONTP(I,J,K)/(TIND(J)/TINDI(J))
  110 CONTINUE
C
C*****SCHEDULE NEXT CONTACT FOR A MEMBER OF SPECIES (NSPR)
C*****WITH THE BODY.
C
      ATRIB(1)=TNOW+UNFRM(TFIND(NSPD,NSPR,1),
     1TFIND(NSPD,NSPR,2))
      IF (ATRIB(1)-ATRIB(5)) 4,10,10
    4 ATRIB(2)=6.
      CALL FILEM(1,NSET)
```

APPENDIX II (CONTINUED).

```
C
C*****IS BODY EATEN?
C*****IF NOT, RETURN.
C
   10 EAT=DRAND(ISEED)
      IF (EAT-PEAT(NSPR,NSPD)) 5,5,1000
C
C*****IS SCAVENGER SUSCEPTIBLE TO INFECTION?
C*****IF NOT, RETURN.
C
    5 IF (TSIND(NSPR)) 1000,1000,6
    6 SUS=DRAND(ISEED)
      PROSUS=TSIND(NSPR)/TIND(NSPR)
      IF (SUS-PROSUS) 7,7,1000
C
C*****DOES INFECTION SUCCEED OR FAIL?
C*****IF FAILURE, RETURN
C
    7 GO=DRAND(ISEED)
      IF (GO-PGOEAT(NSPR)) 8,8,1000
    8 TSIND(NSPR)=TSIND(NSPR)-1.
      XEATIN(NSPR)=XEATIN(NSPR)+1.
      XDINF(NSPR)=XDINF(NSPR)+1
C
C*****SCHEDULE APPEARANCE OF SYMPTOMS
C
      ATRIB(1)=TNOW+BETA(NSPR)
      ATRIB(2)=1.
      ATRIB(3)=NSPR
      DO 20 I=4,7
   20 ATRIB(I)=0.
      CALL FILEM(1,NSFT)
 1000 RETURN
      END
```

APPENDIX II (CONTINUED).

```
      SUBROUTINE REPROD(NSET)
      DIMENSION NSET(12,1)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NDQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP,NTMODE,TPOP,TPOPI,DECAY,TIND(4),TINDI(4),
     1TSIND(4),TSINDI(4),PCTRED(4),PCTVAC(4),PCTTRP(4),
     2XMAXIN(4),XMININ(4),TCONTP(4,4,2),TCONT(4,4,2),
     3PSYMPP(4),PSYMPT(4),PSYMPM(4),PGO(4,4,2),PTRANS
     4(4,2,4,4),TFINDP(4,4,2),TFIND(4,4,2),PEAT(4,4),
     5PGOEAT(4),ADD(4),NAI(4),NEXT
      COMMON XASM(4),XDASM(4),XSM(4),XDSM(4),XDINF(4),
     1XFAIL(4),XEATIN(4),XDEAD(4),XTDEAD(4),XATS(4),N(4)
C
C*****SCHEDULE NEXT REPRODUCTION.
C
      ATRIB(1)=TNOW+365.
      ATRIB(2)=7.
      CALL FILEM(1,NSET)
C
C*****INCREASE POPULATION AND ACCUMULATORS.
C
      XINCR=ORAND(ISEED)
      DO 10 I=1,NSP
      ADD(I)=TIND(I)*(XMININ(I)+XINCR*(XMAXIN(I)-XMININ(I)
     1))*(1.0-PCTRED(I))
      TIND(I)=TIND(I)+ADD(I)
      TSIND(I)=TSIND(I)+ADD(I)
   10 TPOP=TPOP+ADD(I)
      RETURN
      END
```

APPENDIX II (CONTINUED).

```
      SUBROUTINE OUTPUT(NSET)
      DIMENSION NSET(12,1)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NOQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP,NTMODE,TPOP,TPOPI,DECAY,TIND(4),TINDI(4),
     1TSIND(4),TSINDI(4),PCTRED(4),PCTVAC(4),PCTTRP(4),
     2XMAXIN(4),XMININ(4),TCONTP(4,4,2),TCONT(4,4,2),
     3PSYMPP(4),PSYMPT(4),PSYMPM(4),PGO(4,4,2),PTRANS
     4(4,2,4,4),TFINDP(4,4,2),TFIND(4,4,2),PEAT(4,4),
     5PGOEAT(4),ADD(4),NAI(4),NEXT
      COMMON XASM(4),XDASM(4),XSM(4),XDSM(4),XDINF(4),
     1XFAIL(4),XEATIN(4),XDEAD(4),XTDEAD(4),XATS(4),N(4)
      ATRIB(1)=TNOW+7.
      CALL FILEM(1,NSET)
      ND=TNOW/7.
      XD=ND
      IF (TNOW/7.-XD) 11,11,10
   10 ND=ND+1
   11 DO 20 I=1,NSP
      M=(I-1)*1000
      CALL COLCT(XDEAD(I),M+ND,NSET)
      XDEAD(I)=0.
      CALL COLCT(XDASM(I),M+100+ND,NSET)
      XDASM(I)=0.
      CALL COLCT(XDSM(I),M+200+ND,NSET)
      XDSM(I)=0.
      CALL COLCT(XDINF(I),M+300+ND,NSET)
      XDINF(I)=0.
      CALL COLCT(XFAIL(I),M+400+ND,NSET)
      XFAIL(I)=0.
      CALL COLCT(XEATIN(I),M+500+ND,NSET)
      XEATIN(I)=0.
      CALL COLCT(XATS(I),M+600+ND,NSET)
      XATS(I)=0.
      CALL COLCT(XASM(I),M+700+ND,NSET)
      CALL COLCT(XSM(I),M+800+ND,NSET)
   20 CALL COLCT(XTDEAD(I),M+900+ND,NSET)
      RETURN
      END
```

APPENDIX II (CONTINUED).

```
      SUBROUTINE ENDSIM(NSET)
      DIMENSION NSET(12,1)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NOQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP,NTMODE,TPOP,TPOPI,DECAY,TIND(4),TINDI(4),
     1TSIND(4),TSINDI(4),PCTRED(4),PCTVAC(4),PCTTRP(4),
     2XMAXIN(4),XMININ(4),TCONTP(4,4,2),TCONT(4,4,2),
     3PSYMPP(4),PSYMPT(4),PSYMPM(4),PGO(4,4,2),PTRANS
     4(4,2,4,4),TFINDP(4,4,2),TFIND(4,4,2),PEAT(4,4),
     5PGOEAT(4),ADD(4),NAI(4),NEXT
      COMMON XASM(4),XCASM(4),XSM(4),XDSM(4),XDINF(4),
     1XFAIL(4),XEATIN(4),XDEAD(4),XTDEAD(4),XATS(4),N(4)
      MSTOP=-1
      CALL OUTPUT(NSET)
      DO 20 I=1,NSP
      CALL TMST(XSM(I),TNOW,I,NSET)
   20 CALL TMST(XASM(I),TNOW,NSP+I,NSET)
      IF (NRUNS-1) 2,2,1
    2 NORPT=0
      RETURN
    1 DO 10 I=1,NSP
      XTDEAD(I)=0.
      XSM(I)=0.
      XASM(I)=0.
      NAI(I)=0
      TIND(I)=TINDI(I)*(1.0-PCTTRP(I))
   10 TSIND(I)=TSINDI(I)
      TPOP=TPOPI
      RETURN
      END
```

APPENDIX II (CONTINUED).

```
      SUBROUTINE BETAXF
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NDQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NFP,VNQ
     3(100),KOF,KLE,KGL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      COMMON NSP
      DO 10 J=1,NSP
      BMEAN=(PARAM(J,1)-PARAM(J,2))/(PARAM(J,3)-PARAM(J,2))
      BVAR=PARAM(J,4)/(PARAM(J,3)-PARAM(J,2))**2
      PARAM(J,1)=BMEAN*(BMEAN*(1.0-BMEAN)/BVAR-1.0)
   10 PARAM(J,4)=PARAM(J,1)*((1.0-BMEAN)/BMEAN)
      RETURN
      END
```

APPENDIX II (CONTINUED)

```
      FUNCTION PGAMMA(TK,NPRNT,JP,ISEED)
   50 IF (TK) 60,60,70
   60 WRITE(NPRNT,61) JP
   61 FORMAT(/38HSTANDARD FIX TAKEN:   K  WAS SET = 1.0)
      TK=1.0
   70 GAMMA = 1.0
      K1 = TK
      TK1 = K1
      R = DRAND(ISEED)
      IF (R -(TK - TK1)) 90,90,100
   90 K1 = K1 + 1
  100 DO 110 I = 1,K1
      R = DRAND(ISEED)
  110 GAMMA = GAMMA * R
      PGAMMA = -ALOG(GAMMA)
      RETURN
      END
```

APPENDIX II (CONTINUED).

```
      FUNCTION BETA(J)
C
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NOQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
C
  200 IF(PARAM(J,1))10,10,20
   10 WRITE(NPRNT,11) J
   11 FORMAT(/39H STANDARD FIX TAKEN:   K  WAS SET = 1.0)
      PARAM(J,1)=1.0
   20 IF(PARAM(J,4))110,110,30
  110 WRITE(NPRNT,11) J
      PARAM(J,4)=1.0
   30 P = PGAMMA(PARAM(J,1),NPRNT,J,ISEED)
      BETA = P/(P + PGAMMA(PARAM(J,4),NPRNT,J,ISEED))
      BETA = BETA * (PARAM(J,3) - PARAM(J,2)) + PARAM(J,2)
  300 RETURN
      END
```

APPENDIX II (CONTINUED).

```
      SUBROUTINE XMOVE (KCOL,JQ,NSET)
      DIMENSION NSET(12,1)
      COMMON ID,IM,INIT,JEVNT,JMNIT,MFA,MSTOP,MX,MXC,NCLCT
     1,NHIST,NOQ,NORPT,NOT,NPRMS,NRUN,NRUNS,NSTAT,OUT,
     2SCALE,ISEED,TNOW,TBEG,TFIN,MXX,NPRNT,NCRDR,NEP,VNQ
     3(100),KOF,KLE,KOL
      COMMON ATRIB(10),ENQ(100),INN(100),JCELS(10,32),
     1KRANK(100),JCLR,MAXNQ(100),MFE(100),MLC(100),MLE
     2(100),NCELS(10),NQ(100),PARAM(40,4),QTIME(100),
     3SSUMA(30,5),SUMA(3000,5),NAME(6),NPROJ,MON,NDAY,NYR
      IF (KCOL) 16,16,2
   16 CALL ERROR(97,NSET)
    2 XTRIB=NSET(1,KCOL)
      DO 32 I=1,IM
   32 NSET(I,KCOL) = 0
      JL = NSET(MX,KCOL)
      JK= NSET(MXX,KCOL)
      IF (JL-KOL) 33,34,33
   33 IF (JK-KLE) 35,36,35
   35 NSET(MX,JK) = JL
      NSET(MXX,JL) = JK
      GO TO 37
   36 NSET(MXX,JL) = KLE
      MFE(JQ) = JL
      GO TO 37
   34 IF (JK-KLE) 38,39,38
   38 NSET(MX,JK) = KOL
      MLE(JQ) = JK
      GO TO 37
   39 MFE(JQ) = 0
      MLE(JQ) = 0
C****UPDATE POINTERS
   37 NSET(MX,KCOL) =MFA
      NSET(MXX,KCOL) = KLE
      IF (MFA-KOF) 234,235,235
  234 NSET(MXX,MFA) = KCOL
  235 MFA= KCOL
C****UPDATING FILE STATISTICS
      XNQ = NQ(JQ)
      IF (JQ -1)  16, 301, 302
  301 TIME=XTRIB
  302 ENQ(JQ)=ENQ(JQ)+XNQ*(TIME-QTIME(JQ))
      VNQ(JQ)=VNQ(JQ)+XNQ*XNQ*(TIME-QTIME(JQ))
      QTIME(JQ)=TIME
      NQ(JQ) = NQ(JQ)-1
      RETURN
      END
```

Appendix III. Important rabies model variables and accumulators.

Variable	Definition
ADD (I)	Annual increment to the population of species (I).
ASM	Random number between 0.0 and 1.0 used to determine rabies symptoms.
DECAY	Number of days required for flesh to attain an unpalatable condition.
EAT	Random number between 0.0 and 1.0 used to determine if a scavenger eats a body it has encountered.
GO	Random number between 0.0 and 1.0 used to determine if rabies infection will succeed in a susceptible animal.
N (J)	Number of times transmission mode (J) is used to transmit rabies.
NAI (I)	A number used to identify individual asymptomatic rabies carriers of species (I). The value of NAI is stored in ATRIB (6).
NEXT	Check for program continuation. If NEXT is less or equal to zero stop execution.
NSP	Number of species under consideration.
NSPD	Code for the species designation of a rabies virus donor.
NSPR	Code for the species designation of a rabies virus receiver.
NSYM	Code for symptom type.
NTMODE	Number of possible transmission modes.
PCTTRED (I)	Percentage reduction in the reproductive capacity of species (I) by use of chemosterilants.
PCTTRP (I)	Percentage reduction in species (I) by trapping.
PCTVAC (I)	Percentage of species (I) vaccinated against rabies.

Appendix III. (continued).

Variable	Definition
PEAT (J, I)	Probability of a member of species (J) eating the flesh of a dead member of species (I).
PGO (J, I, K)	Probability of rabies infection succeeding in a susceptible member of species (J) contacted by a rabid member of species (I), showing symptoms (K).
PGOEAT (J)	Probability of rabies infection succeeding in a susceptible member of species (J) after eating the flesh of a rabid animal.
PROSUS	Proportion of a species population which is susceptible to rabies.
PSYMPM (I)	Minimum probability of active rabies symptoms appearing in members of species (I).
PSYMPP (I)	Initial probability of active rabies symptoms appearing in members of species (I).
PTRANS (I,K,J,L)	Probability of a rabid member of species (I) displaying symptoms (K), using transmission mode (L) in contacting a member of species (J).
SUS	Random number between 0.0 and 1.0 used to determine if a contacted individual is susceptible to infection.
TCONT (I,J,M)	If M=1, minimum time (days) between contacts for a rabid member of species (I) and members of species (J). If M=2, this is the maximum time between contacts.
TCONTP (I,J,M)	Initial values for minimum and maximum times between contacts for rabid members of species (I) and members of species (J).
TFIND (I,J,M)	If M=1, minimum time (days) required for the body of a rabid member of species (I) to be found by a member of species (J). If M=2, this is the maximum time required.

Appendix III. (continued).

Variable	Definition
TFINDP (I,J,M)	Initial values for minimum and maximum times required for the body of a rabid member of species (I) to be found by a member of species (J).
TIND (I)	Current number of animals present in population of species (I).
TINDI (I)	Initial population of species (I).
TPOP	Total population size.
TPOPI	Initial size of total population.
TRANS	Random number between 0.0 and 1.0 used to determine the transmission mode used in each contact between a rabid animal and another animal.
TSIND (I)	Current number of susceptible members of species (I).
TSINDI	Initial susceptible population of species (I).
XASM (I)	Current number of asymptomatic carriers of species (I).
XATS (I)	Number of weekly changes by infected members of species (I) from an asymptomatic to a symptomatic state.
XDASM (I)	Number of new asymptomatic carriers of species (I) added per week.
XDEAD (I)	Number of weekly deaths in species (I).
XDINF (I)	Number of weekly infections in species (I).
XDSM (I)	Number of new symptomatic members of species (I) added per week.
XEATIN (I)	Number of members of species (I) contracting rabies by ingestion each week.

Appendix III. (continued).

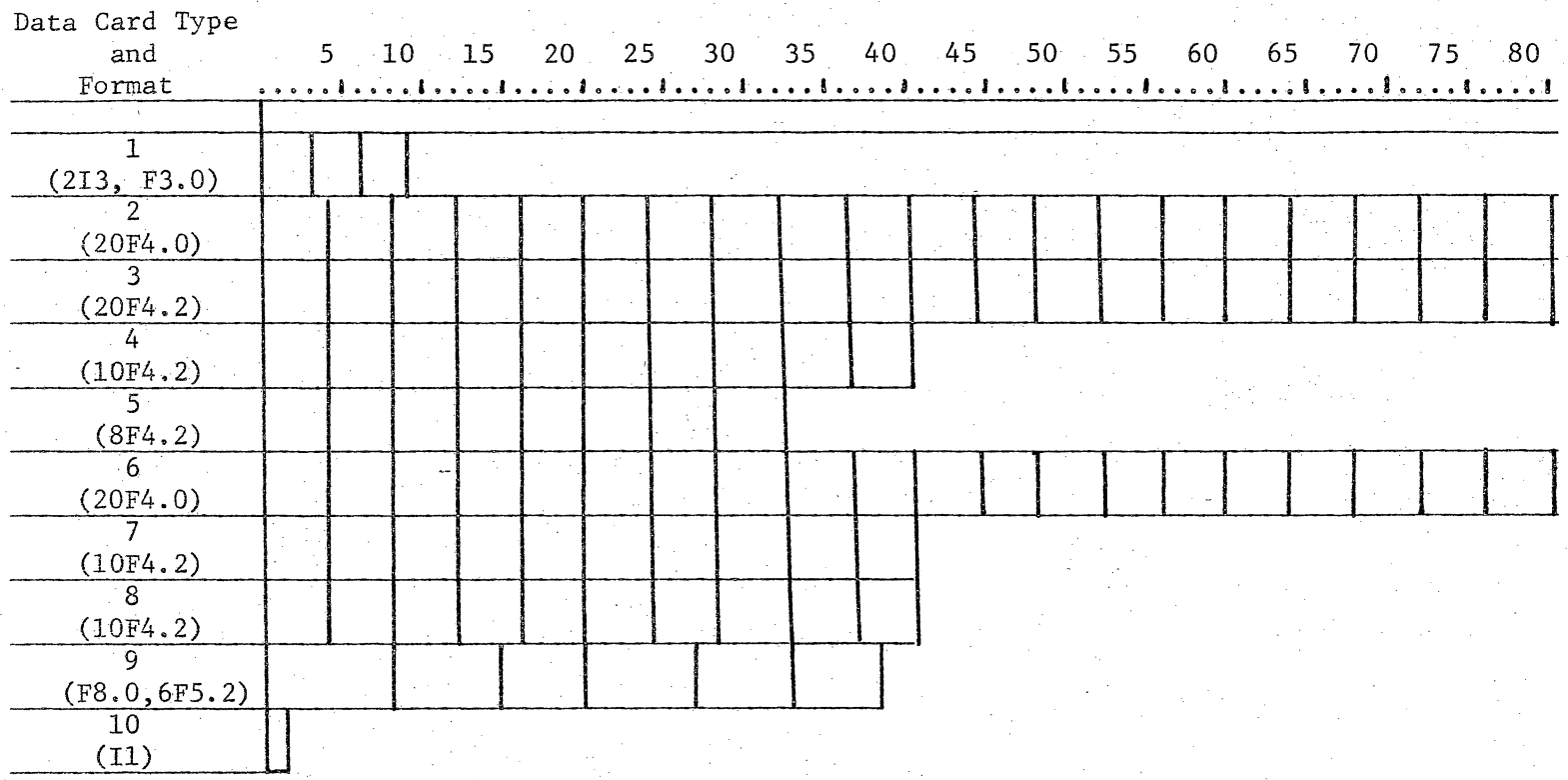
Variable	Definition
XFAIL (I)	Number of susceptible members of species (I) failing to contract rabies per week.
XINCR	Random number between 0.0 and 1.0 used to determine the quality of each breeding season.
XMAXIN (I)	Maximum factor of increase for species (I).
XMININ (I)	Minimum factor of increase for species (I).
XSM (I)	Current number of symptomatic carriers of species (I).
XTDEAD (I)	Total number of individuals of species (I) which have died.

Appendix IV. Data input specifications for non-GASP variables.

Data Card Type	No. Required[1]	Variables Read
1	1	NSP, NTMODE, DECAY
2	n	TCONTP(I,J,M)
3	n	PGO(J,I,K)
4	1	PSYMPP(I)
5	n^2	PTRANS(I,K,J,L)
6	n	TFINFP(I,J,M)
7	n	PEAT(J,I)
8	1	PGOEAT(J)
9	n	TINDI(I), PSYMPM(I), XMININ(I), XMAXIN(I), PCTRED(I), PCTVAC(I), PCTTRP(I)
10	1	NEXT

[1]where n= number of species to be considered.

Non-GASP input data is followed by the requisite number of GASP data cards, as outlined in Pritsker and Kiviat (1969). The first n parameter cards are used to specify the mean value, minimum, maximum, and standard deviation of virus incubation periods in species 1 through n. The second n parameter cards are used to specify similar parameters for the duration of clinical rabies symptoms in each species.



Data Card Formats

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A COMPUTER MODEL OF WILDLIFE RABIES EPIZOOTICS AND AN ANALYSIS OF INCIDENCE PATTERNS

by

Charles Willard Smart

ABSTRACT

A two-part study was undertaken to investigate and model the characteristics of rabies epizootics in wildlife populations. Initial analysis of temporal and spatial relationships between reported rabies cases in four ecologically divergent counties was conducted. No single pattern of report incidence was found to be characteristic of all counties. Concentration of rabies reports near foci of human population was the only common trend, confirming previous studies of the dependence of rabies reporting upon human population density. Topography partly determines the pattern of human habitation, thereby influencing rabies report data.

A computer model of wildlife rabies epizootics was written for an IBM 360/65-360/50. Based on a generalized simulation language, GASP II, this model may be used to simulate the effects of proposed rabies control strategies on present and future epizootics. Demonstrations of techniques for simulating control by population reduction, vaccination, and sterilization were presented. Statements of model limitations and suggestions for future improvements were included in the discussion of project results.