Wildlife can be used as sources for detecting diseases and predicting epidemics. Arboviruses are discussed and examples are cited in support. Potentially such a system could be expanded to non-infectious maladies as well.

Wildlife as Monitors of Disease

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Introduction

When discussing wildlife and disease, the usual relationships which come to mind involve specific morbidity and/or mortality such as is induced by botulism in waterfowl,[2] or a wildlife vector such as the fox and its role as a transmitter of rabies,[3] or a wildlife reservoir such as the rabbit or muskrat for tularemia.[3] These associations certainly exist, but wildlife also can be utilized as sentinels for the detection of diseases and the prediction of epidemics. I would cite several examples in points:

A Wild Avain Population

Since 1958, the natural history of a wild turkey population (*Meleagris gallopava intermedia*) has been studied at the Welder Wildlife Foundation, a 7,800-acre refuge in south Texas. As part of this study, turkeys were live-trapped with conventional cannon nets or the Texas drop net. Birds were weighed, sexed, aged, and marked with appropriate bands for population movement, behavior and survival studies. In addition, serum samples were collected from these live-trapped birds and tested for serologic evidence of virus exposure in a metabolic inhibition test.[6] The population of turkeys studied, normally gathers in large numbers during the winter on lands of the Foundation and disperses each spring over an area with a 30-mile radius to nest and raise its broods. Birds sampled in this study, therefore, occupy approximately 2,800 square miles of range.

Serologic results (Table 1) indicated little or no exposure of the 963 wild turkeys to most of the arboviruses tested for.

Of particular interest, however, was the serologic results for St. Louis encephalitis (SLE) as represented in Figure 1. SLE reactors first appeared in the 1965 sample at which time 20% of the birds were positive. This percentage of reactors increased to 27% in 1967 and then decreased abruptly. Among the serologic reactors detected in 1965 more than half were in immature birds; the 1965 results suggest a primary exposure. In 1966 and 1967 the percentage of reactors increased in the adult portion of the population, probably because of additional opportunity for exposure. There was little serologic evidence of SLE infection in wild turkeys after 1968. All 11 SLE reactors in 1969 and 1970 were adults: seven of these had also been bled during 1966 or 1967 at which time they were SLE positive. There was no evidence of clinical SLE in turkeys during the study.

SLE has occurred periodically in Texas and in 1965 human cases were reported in Corpus Christi, 30 miles from the Welder Foundation; in the summer of 1966, a significant SLE epidemic occurred in the Corpus Christi area.[9]

All turkeys were bled during the winter, January and February, and recorded as that year; therefore, the serologic results reported for a specific year actually indicates virus activity of the previous year(s). Turkey reactors reported in 1963, therefore, had probably been exposed during the summer of 1964. In retrospect, serologic data of this turkey study predicted the SLE epidemic of 1965 and 1966 in man.

A similar situation in which wildlife serology predicted an arbovirus epidemic occurred with western encephalitis (WE) in Alberta, Canada.[10] In 1966, WE occurred in snowshoe hares (*Lepus americanus*) prior to an outbreak in horses and man. In this instance, the disease in the wild host preceeded the equine and human epidemic by two months.

A Wild Mammalian Population

The major big game species of North America is the white-tailed deer (*Odocoileus virginianus*). Because of its lofty status, management of this species is intense and many states require that all hunting season kills are examined for biological data such as weight, sex, age, condition of animal, and reproductive success. In addition, many special controlled hunts are held where similar biological data are

Figure 1—The Per cent of Serologic Reactors to St. Louis Encephalitis Virus in a Wild Turkey Population in South Texas (1963-1970)

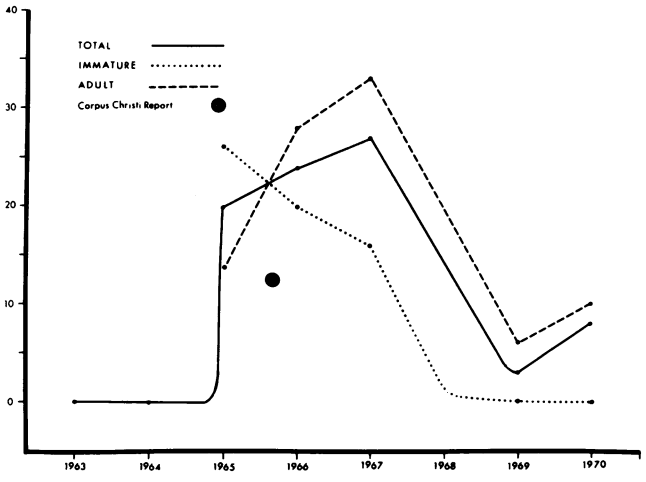

Table 1—A Summary of Serologic Results of 963 Wild Turkeys at the Welder Foundation (1963-1970)

Year	Sample size	Serologic results* (per cent positive) EE	WE	CE	SLE	VE
1963	33	0	0	0	0	0
1964	64	0	3	0	0	3
1965	88	0	3	0	20	1
1966	324	0	12	1	24	0
1967	222	2	14	-	27	-
1968	12	0	0	0	0	0
1969	112	0	9	0	3	0
1970	108	1	9	1	8	3

*EE = Eastern encephalitis; WE = Western encephalitis; CE = California encephalitis; SLE = St. Louis encephalitis; VE = Venezualian encephalitis.

Table 2—A Summary of Serologic Results of 1314 White-Tailed Deer from 7 States or Provinces

Serum source	Sample size	Serologic results* (per cent positive) EE	WE	CE	SLE	VE
Quebec	103	0	0	0	0	0
Wyoming	23	0	4	0	4	0
New York	122	0	0	0	0	0
Iowa	28	0	0	21	-	0
Nebraska	8	0	62	75	-	-
Wisconsin	512	0	2	26	5	0
Texas	518	0	7	50	3	7

*EE = Eastern encephalitis; WE = Western encephalitis; CE = California encephalitis; SLE = St. Louis encephalitis; VE = Venezualian encephalitis.

collected. The integration of serum collections into the activities of check stations where biological data are obtained on deer, has proven to be simple and productive. Serologic results from such collections have increased our knowledge on prevalence of specific diseases, i.e., brucellosis, leptospirosis, arboviruses.[4,7,8] In addition, serologic results for California encephalitis group (CE) viruses have provided additional information. In one study[8] approximately 1,300 deer sera from seven states and provinces were tested for CE neutralizing antibodies. Thirty per cent of the deer were serologic reactors and the reactor rate varied from zero in some sites such as New York and Quebec to as high as 26 per cent in Wisconsin and 50 per cent in Texas. (Table 2)

The largest number of serologic reactors occurred in areas where CE was reported to be endemic, Wisconsin and Texas. There has as yet been no association of overt disease in deer with the viruses of CE, but a detectable antibody response results from experimental challenge.[1]

These deer sera were also tested for antibodies to other arboviruses, and no evidence to most of them was found. (Table 2) Four per cent of the sera did react to WE and all but two of these reactors were from west of the Mississippi, areas where WE might be expected to occur.

The discovery that the deer sera from Quebec had no serologic reactors is interesting, but not totally unexpected. These serum samples were from deer on Anticostic Island, located 50 miles from the mainland. This area is, therefore, isolated from other deer herds, and may have limited exposure to nidi of arbovirus activity.

The restriction of serologic reactors to certain viruses in appropriate geographic areas, the occurrence of reactors in high numbers in epidemic years, the limiting of reactors to a single antigen, the complete lack of reactors in an isolated island population, all add credence to the fact that the methods used detected antibody against the specific antigens.

From these serologic results as well as from experimental data, it appears that deer are sensitive indicators of the presence of some arbovirus infections. Because of their large population (20 million), ubiquitous distribution (Panama to Alaska and coast to coast), non-migratory behavior, ease and accuracy of sexing and aging specimens, and the fact that 2.5 million deer are harvested annually by hunters in the United States, an opportunity is afforded for obtaining large numbers of sera. This wild species can serve as a valuable indicator species for the activities of selected arbovirus—a wild sentinel.

The detection of specific serologic reactors in one wild species does not necessarily indicate presence of infection in other populations. For example serologic studies of SLE in white-tailed deer at the Welder Foundation, the study site described for the wild turkey earlier, did not reflect the activity in wild turkeys or human population in the area.

We also have evidence that there is considerable host specificity among the California group viruses. The domestic rabbit cannot detect Jamestown Canyon virus which is probably the principal California virus affecting deer, but it is a good sentinel for LaCrosse virus, another member of the group. Deer, however, will develop antibodies to the LaCrosse as well as the Jamestown Canyon virus.

Conclusions

From the examples cited it would appear that serologic studies of wildlife can under the proper circumstances be utilized to monitor and sometimes even predict arbovirus outbreaks. Domestic and laboratory sentinels have been used to monitor arbovirus activity for some time.[5] Appropriate wildlife sentinels have certain logistical advantages such as little or no maintenance problems as well as a more natural population distribution, movement, behavior and density. To utilize wild populations for "sentinel" duty certain specific conditions must exist.

The wild population should: 1) have a known limited home range so that the area being monitored can be defined—results from migratory population could be very difficult to interpret; 2) be present in good numbers and readily accessible so that test sera can be obtained periodically—such as we described for wild turkeys and deer; 3) contain individuals which are easily bled, aged and sexed; 4) be susceptible and respond serologically, yet not be decimated by the disease under study.

The disease to be monitored should: 1) be similarly transmitted to both the selected wild monitoring populations and human populations; 2) produce a sub-lethal disease in the sentinel; and 3) stimulate a detectable serologic response.

When the above predisposing factors are properly integrated such as with the white-tailed deer and California encephalitis, a "natural" monitoring system can be in effect on a local, national, or even continental basis. Although only arboviruses have been discussed, the potential for such a system is unlimited and could be expanded to include not only additional infectious diseases, but noninfectious maladies as well, such as radioactivity, pesticides, heavy metals, and other pollutants.

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Section Election Deadlines

As established by the Executive Board, deadlines for the 1973, APHA, section election procedures are:

April 2—Committee on Nominations to be appointed by each section;

May 1—Committee's nominations to be appointed by each section;

Aug. 8—Section's nominations by petition due at APHA headquarters.

Along with maintaining this schedule, each section must provide, in writing, the following information on each nominee for each office: full name, academic degrees, current position, and current and past APHA activities.

Nominations by petition must be approved by the section's Committee on Nominations. Only information submitted by these committees will appear on the ballots. Ballots will be mailed on Aug. 15, and counted on Sept. 15.