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The Use of Wildlife to Monitor Zoonoses*

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Abstract

Wildlife are usually considered vectors, reservoirs or primary targets of infectious disease. A seldom considered epidemiological role which they can play involves their use as disease sentinels for the detection and monitoring of zoonoses. Their potential for such utilization has been demonstrated with the wild turkey *(Meleagris gallopava intermedia)* and St. Louis encephalitis in Texas and the white-tailed deer *(Odocoileus virginianus)* and California encephalitis in North America. The limitations and criteria which are important in the use of wild populations for "sentinel" duty are discussed.

As we use it, the phrase "wildlife zoonoses" usually connotates wildlife as either a reservoir of disease such as tularemia, a vector of a malady such as rabies, or the primary target of a disease such as botulism. Wildlife does play all of these epidemiological parts, but another role seldom considered involves their potential as sentinels for the detection and monitoring of zoonoses. This will be the subject of discussion in this presentation.

Rio Grande Turkey

Since 1958, the natural history of a wild turkey population has been studied at the Welder Wildlife Foundation, a 7800 acre refuge in south Texas. As part of this study, serum samples were collected annually from live trapped birds and tested for serologic evidence of disease exposure.[7] To determine arbovirus experience, sera were tested for their ability to neutralize $10^{1.5}$ to $10^{2.5}$ ID_{50} of various virus antigens in a metabolic inhibition test using HeLa cells. The population of turkeys studied normally gather in winter concentrations on lands of the Foundation and disperse in all directions each spring in a 30 mile radius to nest and raise their broods. Birds sampled in this study, therefore, occupy approximately 2800 square miles of range.

Serologic results indicated little or no exposure of these wild turkeys to three of the arbovirus studied (Table 1). Two exceptions were St. Louis encephalitis (SLE) and western encephalitis (WE).

Of particular interest was the serologic results for SLE from 963 turkey sera from 1963-1970 as represented in Figure 1. SLE reactors appeared for the first time in the 1965 sample when 20% of the birds were positive. This percentage of reactors increased to 27% by 1967 and then dropped. Among the serologic reactors detected in 1965 more immature

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TABLE 1. *A summary of serologic results of 963 wild turkeys at the Welder Foundation (1963-1970).*

Year	Sample Size	Serologic results (percent 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

than adult birds were involved; since immature birds constituted more than half of the sample 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 been bled in 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, and in the summer of 1966, a significant SLE outbreak occurred in the Corpus Christi area.[9] The serologic results of wild turkeys at Welder reflected the SLE activity in human populations of the area.

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 1965, therefore, had probably been exposed during the summer of 1964. In retrospect, serologic data of this turkey study predicted the SLE human outbreaks of 1965 and 1966.

A similar situation where wildlife serology predicted an arbovirus epidemic occurred with western encephalitis (WE) in Alberta, Canada. In 1966, WE occurred in snowshoe hares *(Lepus americanus)* prior to an outbreak in horses. In this instance, the disease in the wild host preceded the equine epidemic by 2 months.[10]

Serologic results from turkey sera at Welder suggested an increase of WE in 1966 and 1967 (Table 1). A similar serologic pattern (Figure 2) existed for other wild mammal populations on the Refuge during this period.[1,8]

White-tailed Deer

The number one big game species of North America is the white-tailed deer. To increase our knowledge of specific diseases in deer, numerous serologic studies have been done, (i.e., brucellosis,[8] leptospirosis,[6] arbovirus[8]). The serologic results for California encephalitis group (CE) viruses have been of particular interest. In one study, approximately 1300 deer sera from seven states and provinces were tested for CE neutralizing antibodies.[8] Thirty percent of the deer were serologic reactors and the reactor rate varied from zero in some sites such as New York State and Quebec to as high as 26 percent in Wisconsin and 30 percent in Texas (Table 2).

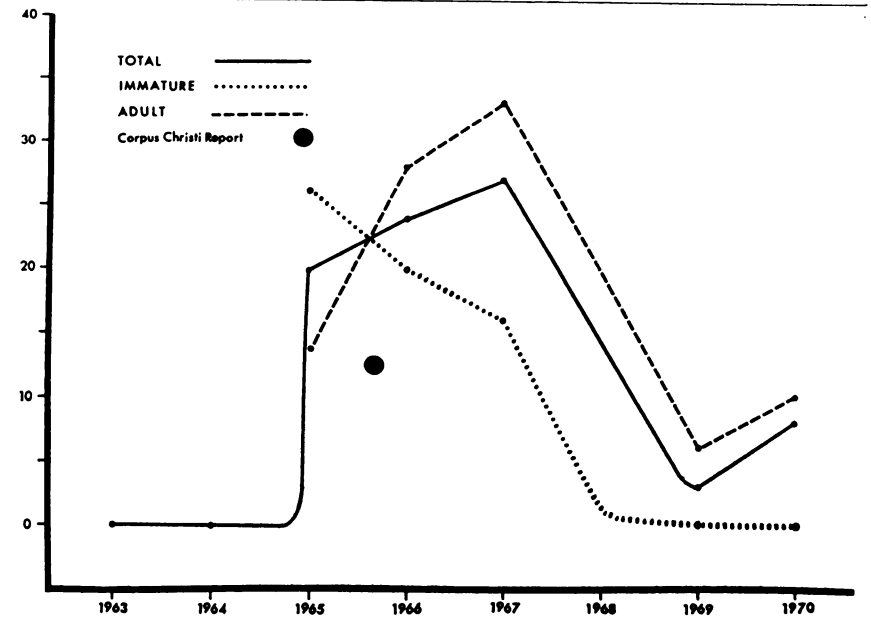

FIGURE 1. *The percent of serologic reactors to St. Louis encephalitis virus in a wild turkey population in South Texas (1963-1970).*

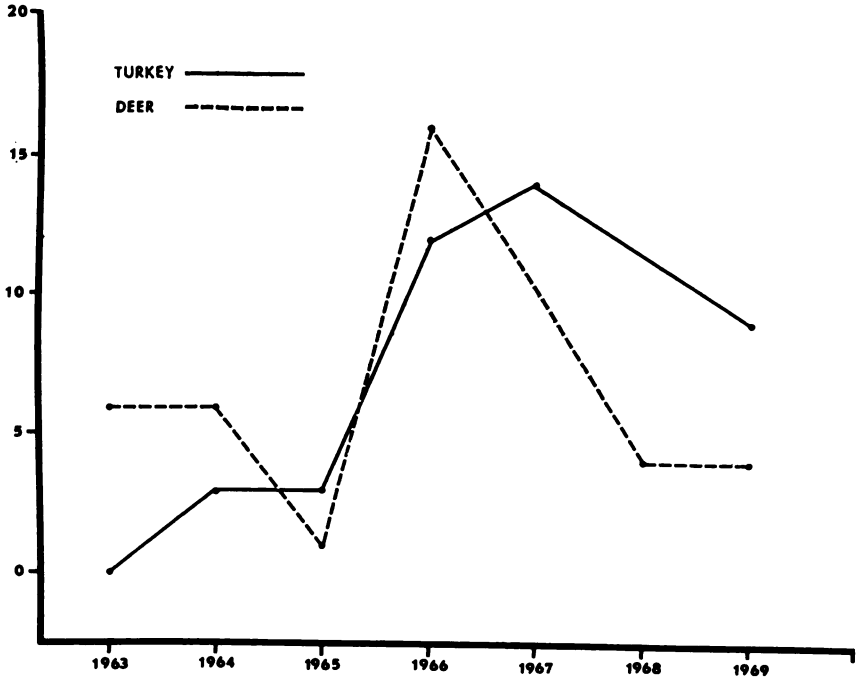

FIGURE 2. *The percent of serologic reactors to Western encephalitis virus in a wild turkey and a deer population in South Texas (1963-1969).*

TABLE 2. *A summary of serologic results of 1314 white-tailed deer from 7 states or provinces for selected arboviruses.*

Serum Source	Sample Size	Serologic results (percent 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

The largest number of serologic reactors occurred in areas where CE was reported to be endemic, Wisconsin[5] and Texas[1]. 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.[2]

These deer sera were also tested for antibodies to other arboviruses, which for the most part were negative (Table 1). Four percent of the sera did react to WE. All but 2 percent of these reactors were from west of the Mississippi, areas where WE might be expected to occur.

The deer sera from Quebec and their complete lack of serologic reactors is interesting but not totally unexpected. These serum samples were from deer on Anticosti Island, located 50 miles from the mainland. This is, therefore, removed from other deer and livestock and presents a unique isolated deer herd, possible with limited exposure to arbovirus activity.

The restriction of serologic reactors to certain viruses in appropriate geographic areas, the occurrence of reactors in high numbers in epidemic years and the absence of reactors in interepidemic 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 were detecting antibody against the specific antigens.

From these serologic results as well as experimental data,[2] it appears that white-tailed deer are sensitive indicators of the presence of some arbovirus infections. Because of their large population (20 million), ubiquitous distribution (Panama to Canada and coast to coast), nonmigratory behavior, ease and accuracy of sexing and aging specimens, and the fact that 2.5 million deer are harvested annually by hunters thus providing a ready supply of sera, this wild species could serve as a valuable indicator species for the activities of selected arbovirus—a wild sentinel.

The prevalence of specific serologic reactors in one wild species does not necessarily indicate disease prevalence in other populations. Serologic studies of SLE in white-tailed deer at the Welder Foundation[6] did not reflect the activity in wild turkeys or human populations in the area. A similar lack of correlation existed in Wisconsin where deer and cattle sera were tested against 13 leptospira serotypes from three distinct geographic areas of the state.[6] When reactor rates for leptospires from the respective study areas were compared, a direct relationship between deer and cattle reactors was not apparent. In deer, the prevalence of leptospirosis appeared to be related to population density of deer; in cattle, a number of factors including population density and herd management were important. Despite the fact that reactors to leptospires are common in deer, their detection and reactor rate does not necessarily reflect the status of leptospirosis in other species.

Conclusions

From the examples cited it would appear that serologic studies of wildlife can under the proper circumstances be utilized to monitor and even predict human disease outbreaks. Domestic and laboratory sentinels have been used to monitor arbovirus activity for some time.[7] Appropriate wildlife sentinels would 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 would be very difficult to interpret; 2) be present in good numbers and readily accessible so that test sera can be obtained easily periodically, such as wild turkeys and deer; 3) contain individuals which are easily bled, aged, and sexed; 4) be susceptible and respond serologically, yet not be adversely affected by the disease under study.

The disease to be monitored should 1) be readily transmitted to both the selected wild monitoring populations and human populations; 2) produce a sublethal 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. 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, chemical pollutants, etc.

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