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SEROLOGICAL SURVEY FOR MICROBIAL PATHOGENS

By

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Progress Report
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PROGRESS REPORT (RESEARCH)

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SUMMARY

Preliminary analysis of the data revealed a dramatic increase in prevalence of parainfluenza III virus antibody in the Delta Bison (Bison bison) Herd during the period 1975-83. Another obvious pattern involved the increase in prevalence of canine parvovirus antibody in wolves (Canis lupus) from the Nelchina Basin during the same time period.

Key words: Alaska, bison, canine parvovirus, parainfluenza III, serology, wolf.

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BACKGROUND

Alaska's ruminant wildlife species are largely free of serious infectious diseases, especially those diseases commonly associated with domestic livestock. Expansion of the agricultural industry has been proposed and may involve dramatic increases in movement of livestock into the state, as well as grazing of such animals in areas previously inhabited solely by wildlife. Such practices would increase the potential for introduction and spread of diseases in the wildlife species. In an effort to document the status of wildlife in relation to specific diseases, a serologic survey has been conducted on a continuing basis. All of the disease agents included in this survey have been detected in various species of North American wildlife by means of isolation or by serologic tests (Abdulla et al. 1962, Howe et al. 1966, Thorsen and Henderson 1971, Parks and England 1974, Barrett and Chalmers 1975, Thorsen et al. 1977, Couvillion et al. 1981).

OBJECTIVE

The objective of this study was to determine the prevalence of antibody to selected disease agents in wildlife species found in Alaska.

PROCEDURES

The primary activity on this project during the last year was entering data into computer storage. The process has been time consuming and is not yet completed. In fact, it will be an ongoing process with both specimen data and test result data being added as they become available. At present, the system contains information on approximately 4,000 samples and 18,000 serologic tests.

Preliminary data analysis was another major activity during the past year. The lack of adequate time for use of the only

available computer prohibited in-depth analysis. The analysis process will continue over the next several months, and a more definitive report can be expected for the next reporting period.

Examples of the type of analyses that have been conducted include the interaction between bison (Bison bison) and parainfluenza III virus and between wolves (Canis lupus) and canine parvovirus.

Blood samples were collected from bison and wolves at locations indicated in Fig. 1. Most bison samples were taken from animals killed by hunters. The remaining bison samples and all wolf samples were collected by Alaska Department of Fish and Game personnel during studies which entailed capture of free-ranging animals.

All sera were gathered during the period 1975-83. Blood samples were allowed to settle for 12-36 hours at ambient or refrigerated temperatures. Sera were separated from clots by aspiration and frozen. Bison sera were tested for the presence of parainfluenza III virus (PI3) antibody by means of the hemagglutination-inhibition test. A titer of 8 or greater was considered to represent evidence of past PI3 infection in the animal in question. Wolf sera were tested for the presence of canine parvovirus (CPV) antibody by means of the serum neutralization test. A titer of 20 or greater was considered indicative of past infection. All serologic tests were performed at the National Veterinary Services Laboratory (U.S. Department of Agriculture, Ames, Iowa).

RESULTS AND DISCUSSION

Certainly the most significant finding during the preliminary data analysis phase was the huge and sudden increase in prevalence of antibody to PI3 in the Delta Bison Herd (Table 1). Previous reports documented the serologic evidence for the presence of this virus in the bison (Zarnke 1983, 1984). However, the complexities of dealing with large data bases precluded anything more than cursory analysis. With the aid of the computer, it now becomes clear that PI3 was essentially absent prior to 1977 (Table 1). Since that time, serologic prevalence has risen dramatically to a peak of 83% in 1983. PI3 is 1 member of a group of 3 viruses that are often grouped together for discussion purposes and referred to as the "bovine respiratory viruses." As the name implies, these viruses often localize in the respiratory tract. However, they can also be found in the genital and/or gastrointestinal tracts. Wherever those viruses localize, they rarely cause disease that is

severe enough to cause death. Symptoms are often most severe when the infection occurs in conjunction with other disease agents.

Although mortality may be low, morbidity within a population may be high. Symptoms may be severe enough to incapacitate an animal for several days. This decreases the animal's weight gain, makes it more susceptible to predation, and may influence the ability of a female to bear and/or raise young.

There continues to be no evidence of respiratory infections in this closely observed herd. However, the pattern of increasing PI3 antibody prevalence indicated in Table 1 is cause for increasing concern. In an attempt to verify that the observed serologic reactions were indeed due to PI3 infection, we attempted to isolate virus via nasal swabs collected from hunter-killed animals. To date, 6 swab samples from the 1984 season have been negative for virus content. An additional 21 samples are currently being processed.

There are several possibilities as to the source of this virus for the Delta Bison Herd. The most tenable hypothesis seems to be that the virus was introduced to the area via domestic cattle. The agriculture industry experienced significant growth near Delta Junction during the 2nd half of the 1970's. Introduction of domestic animal diseases into the area's wildlife may be one of the results of this expansion. If this hypothesis is true, the results of this study represent additional arguments supporting stringent enforcement of existing domestic animal health regulations.

A 2nd interesting discovery during the initial data analysis phase was the apparent introduction of CPV into the wolf population of the Nelchina Basin (Table 2).

Canine parvoviral disease emerged in 1978 (Appel et al. 1978, Pollock et al. 1980). It is believed to be a variant of feline panleukopenia virus (Craige 1979, Flower et al. 1980). The host range of CPV appears to include most canids (Eugster et al. 1978, Fletcher et al. 1979, Evermann et al. 1980, Mann et al. 1980) as well as raccoons (Nettles et al. 1980). In domestic dogs, cases of CPV are most prevalent in puppies less than 6 months old, and symptoms seem to be most severe in this age group as well (Appel et al. 1978). Dogs shed virus for at least 3 weeks (Pollock et al. 1980). Outbreaks of CPV in domestic dogs have occurred throughout the United States. Serum antibody prevalence ranges from 20% to 50% in dogs and varies among different geographic areas (Anonymous 1980, Kramer et al. 1980). Vaccines are available for dogs but provide immunity of only short duration (Appel et al. 1980). Treatment is primarily symptomatic.

There are 2 clinical forms of the disease in dogs. The first is a highly contagious enteritis (Appel et al. 1978, Black et al. 1979, Merickel et al. 1980). The second causes a myocarditis in puppies less than 6 months old (Kramer et al. 1980, Lenghaus et al. 1980). Little information is available on the occurrence or symptoms of this disease in wild, free-ranging animals. Serum antibody prevalence in free-ranging coyotes from Texas, Utah, and Idaho was zero prior to 1979 and increased to 70% at all 3 locations by 1982 (Thomas et al. 1984). As can be seen in Table 2, results from the Nelchina Basin concur with those of the Thomas study, i.e., CPV was introduced into the wild canid populations during 1979 or 1980.

Domestic dogs are believed to be the source of CPV for wild canids. Symptoms of the disease in wild, free-ranging canids are unknown. Based upon studies of captive wild species (Evermann et al. 1980), it can be assumed that symptoms are severe enough to cause mortality at least in younger animals. Such a situation could have significant management implications. Laboratory-based experimental exposures could provide the data to more accurately evaluate the impact of CPV on wolf populations.

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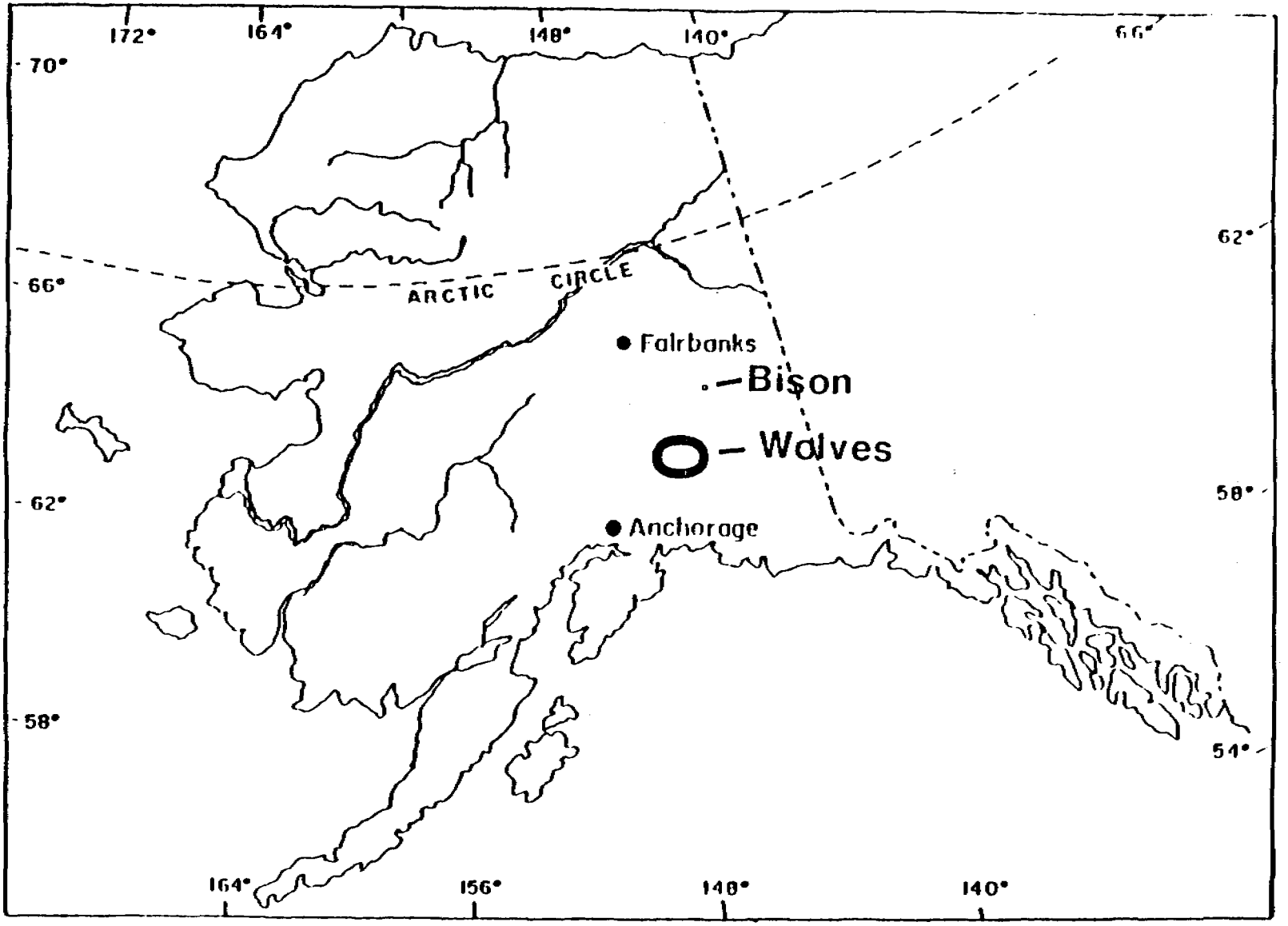

Figure 1. Location of collection sites for bison and wolf sera, 1975-1983.

Table 1. Prevalence of parainfluenza III virus antibody in serum from bison near Delta Junction, Alaska, 1975-83.

Year specimens collected	Prevalence[a]	
1975	0/1	(0%)
1976	0/11	(0%)
1977	1/13	(8%)
1978	13/30	(43%)
1979	4/6	(67%)
1980	5/8	(63%)
1981	23/45	(51%)
1982	17/54	(32%)
1983	38/46	(83%)

[a] Prevalence = number positive/number tested.

Table 2. Prevalence of canine parvovirus antibody in serum from wolves collected in Game Management Unit 13, Alaska, 1975-82.

Year specimens collected	Prevalence[a]
1975	0/2 (0%)
1976	0/13 (0%)
1977	0/13 (0%)
1978	0/25 (0%)
1979	0/2 (0%)
1980	1/13 (8%)
1981	5/11 (45%)
1982	4/7 (57%)

[a] Prevalence = number positive/number tested.