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Authors: Zarnke, Randall L., and Ballard, Warren B.

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SEROLOGIC SURVEY FOR SELECTED MICROBIAL PATHOGENS OF WOLVES IN ALASKA, 1975–1982

Randall L. Zarnke[1] and Warren B. Ballard[2]

ABSTRACT: Serum samples were collected from 116 wolves which were captured in southcentral Alaska during 1975 through 1982. Antibodies to the following infectious disease agents were found: infectious canine hepatitis virus—72 of 87 (81%), canine parvovirus type 2—0 of 55 (0%) through 1979 and 10 of 32 (31%) after 1979, *Francisella tularensis*—16 of 67 (25%), canine distemper virus—10 of 83 (12%), *Coxiella burnetti*—5 of 95 (5%), rabies virus—1 of 88 (1%), *Brucella* spp.—1 of 67 (1%), *Leptospira interrogans*—1 of 82 (1%). Apparently rabies, brucellosis, and leptospirosis were rare and had little effect on the wolf population. Conversely, the other five infections were comparatively common and may have had a negative impact on the health of specific individual wolves, but did not appear to influence the health of the population.

INTRODUCTION

A number of factors influence the population dynamics of wolves (*Canis lupus*) in North America (Mech, 1970). Food availability and human harvest appear to be the most important factors regulating their abundance, particularly in Alaska (Ballard et al., 1981, 1982b; Stephenson and James, 1982; Peterson et al., 1984). However, several investigators have reported that infectious diseases could have been important mortality factors in several populations of wolves in North America (Neiland, 1970; Choquette and Kuyt, 1974; Carbyn, 1982; Stephenson et al., 1982). Usually the occurrence of a particular life-threatening infection in a population of wolves is not known until it manifests itself through mortality. The objective of this study was to determine the prevalence of antibodies to selected canine infectious agents in a population of wolves in southcentral Alaska.

MATERIALS AND METHODS

The study was conducted in Game Management Unit 13 (GMU-13), an area of 61,600 km² located in southcentral Alaska (Fig. 1). Climate, vegetation, physiography, etc. have been described previously (Skoog, 1968; Bishop and Rausch, 1974). Wolf pack size ranged from 2–20 ($\bar{x}$ = 8) depending upon availability of prey, human harvests, and time of year (Ballard et al., 1981, 1982b). Population density ranged from 2.6 wolves/1,000 km² to 10.3/1,000 km². Moose (*Alces alces*) comprised approximately 80% of the prey biomass utilized by wolves (Ballard et al., 1982b). Caribou (*Rangifer tarandus*), beaver (*Castor canadensis*), muskrat (*Ondatra zibethica*), and snowshoe hare (*Lepus americanus*) also were utilized. Both red foxes (*Vulpes vulpes*) and coyotes (*Canis latrans*) are found throughout the study area. Either species could serve as a reservoir of infection for any of the diseases included in this survey.

Wolves were captured from 1975 through 1982 by means of tranquilizer darts fired from helicopters, using drugs and methods described previously (Ballard et al., 1982a). Blood samples were allowed to settle for 6–36 hr at ambient or refrigerated temperatures before centrifugation. Sera were separated by aspiration and frozen.

Serologic tests were performed at the National Veterinary Services Laboratory (United States Department of Agriculture, Ames, Iowa 50010, USA). Sera were tested for evidence of antibodies to:

(1) infectious canine hepatitis virus and canine distemper virus by serum neutralization test (Appel and Robson, 1973),
(2) *Francisella tularensis* by tube agglutination test (Owen, 1970),
(3) canine parvovirus type 2 by fluorescent neutralization test (King and Croghan, 1965),
(4) rabies virus by rapid fluorescent focus inhibition test (Smith et al., 1973),

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[1] Alaska Department of Fish and Game, 1300 College Road, Fairbanks, Alaska 99701, USA.
[2] Alaska Department of Fish and Game, P.O. Box 1148, Nome, Alaska 99762, USA.

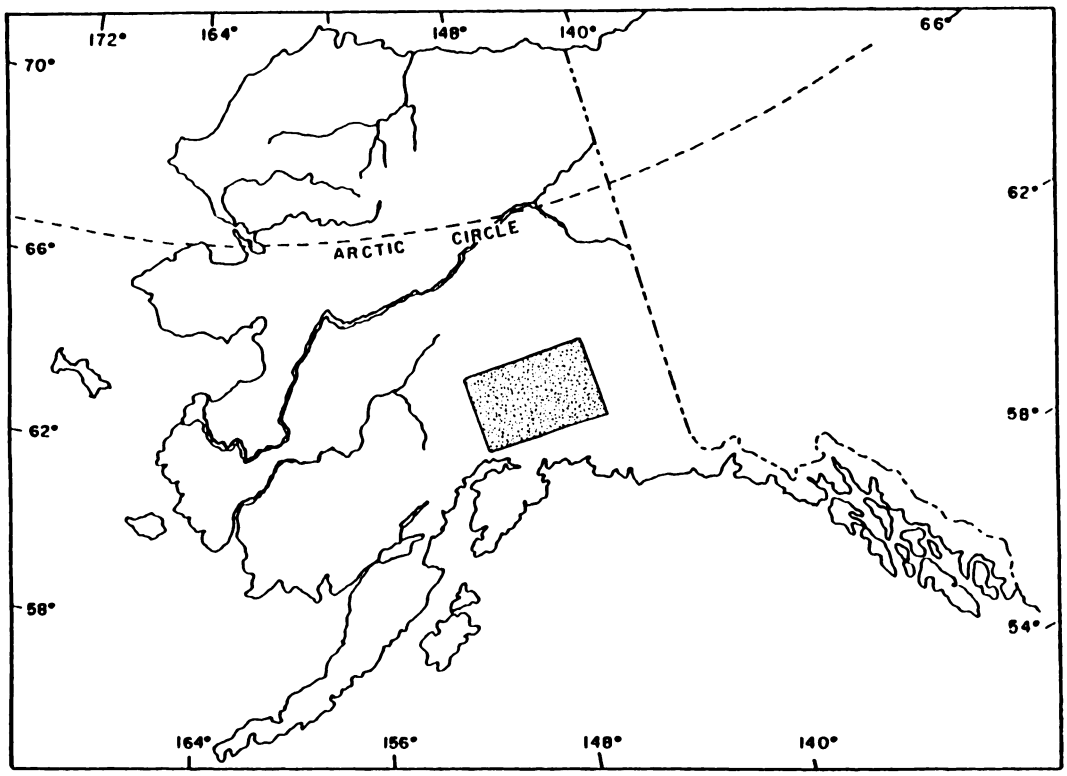

FIGURE 1. Game Management Unit 13 study area in southcentral Alaska from which serum samples were collected from wolves for serologic survey. The study area is shown by the stippled rectangular box.

(6) *Brucella* spp. by buffered acidified plate antigen test (Angus and Barton, 1984),
(7) *Leptospira* spp. by microscopic agglutination test (Cole et al., 1973), and
(8) *Coxiella burnetti* by complement fixation test (Erickson et al., 1975).

The following *Leptospira interrogans* serovarieties were included in the tests: *pomona, ballum, canicola, icterohemorrhagiae, wolffi, grippotyphosa, hardjo, autumnalis, bataviae, tarassovi, australis*, and *pyrogenes*. Minimum titers for all test results (except the *Brucella* plate test) were based upon natural or experimental infection of domestic dogs. Sera which met or exceeded these titers (see Table 1 for minimum values) were considered to indicate evidence of previous exposure to the agent in question. Hereafter, these samples may be referred to as "positive." All other samples may be referred to as "negative." The *Brucella* plate agglutination test was read as either positive or negative. Differences in serologic prevalence based upon sex, age, and proximity to towns and roads were tested for significance by means of the chi-square test (Johnson, 1980).

RESULTS AND DISCUSSION

Infectious canine hepatitis (ICH)

Transmission of ICH normally occurs via direct contact with contaminated saliva, urine or feces (Cabasso, 1981). Signs may include rhinitis, ataxia, anorexia, blood in feces, ocular keratitis, and occasionally convulsions leading to paralysis and death (Cabasso, 1981). The antibody prevalence of 81% (Table 1) in this current study falls between the level of 13% reported for wolves from northern Canada (Choquette and Kuyt, 1974) and 95% for wolves from three areas of Alaska (including GMU-13) (Stephenson et al., 1982). The uniformly high annual preva-

TABLE 1. Serum antibody prevalence for seven microbial pathogens in wolves (*Canis lupus*) collected from Game Management Unit 13 of southcentral Alaska between 1975–1982.

Disease agent	1975	1976	1977	1978	1979	1980	1981	1982	Total
Infectious canine hepatitis virus SN 20[a]	2/2[b]	9/13	13/13	18/25	2/2	11/14	10/11	7/7	72/87 (81%)
Canine parvovirus type 2 SN 16	0/2	0/13	0/13	0/25	0/2	1/14	5/11	4/7	10/32 (31%)[c]
Francisella tularensis TAT 20	0/1	1/10	4/8	4/13	0/2	2/14	5/11	0/8	16/67 (25%)
Canine distemper virus SN 20	1/2	2/13	1/13	2/24	0/1	2/12	0/11	2/7	10/83 (12%)
Coxiella burnetti CF 20	0/2	0/11	1/16	3/30	0/10	1/9	0/9	0/8	5/95 (5%)
Rabies virus RFFIT 10	0/2	0/13	0/13	1/25	0/2	0/14	0/11	0/8	1/88 (1%)
Brucella sp. BAPA (±)	0/1	0/10	0/8	0/13	0/2	0/14	1/11	0/8	1/67 (1%)
Leptospira sp. MAT 100	0/2	0/11	0/12	0/23	0/2	0/14	1/11	0/7	1/82 (1%)

[a] Name of test: SN = serum neutralization test; TAT = tube agglutination test; RFFIT = rapid fluorescent focus inhibition test; BAPA = buffered acidified plate antigen test; MAT = microscopic agglutination test; CF = complement fixation test. Numbers indicate minimum titer necessary to be considered as evidence of past infection. (±) indicates that the test is read as simply either positive or negative.

[b] Number positive/number tested.

[c] Total includes only years 1980–1982.

lences reported here suggest that ICH is enzootic in the wolf population in GMU-13. Forty-two percent of the positive wolves (26 of 62) were <1 yr of age, indicating that wolves are exposed commonly to ICH as pups. Antibody prevalences showed no sex-specificity.

Stephenson et al. (1982) speculated that higher ICH prevalences in wild canid populations resulted from frequent contact with domestic dogs and/or their excreta. If this hypothesis is correct, our results would indicate a very high degree of such contact. Stephenson et al. (1982) also made an alternate hypothesis that ICH or some related virus might be enzootic. We agree that ICH is enzootic in populations of wolves throughout Alaska, rather than being periodically introduced by domestic dogs. The occurrence of positive specimens during all years of the current study supports this hypothesis of the enzootic nature of ICH. In addition, we found no significant difference ($P > 0.10$) in prevalence of ICH antibody between wolf packs whose territories were >30 km from towns or roads, and packs whose territories were <30 km from such human activity centers. In summary, we suggest that ease and frequency of contact between dogs and wolves play no direct role in the epizootiology of ICH. The significance of ICH as a mortality factor in wolves is unknown.

Canine parvovirus type 2 (CPV)

Since first being reported in domestic dogs in 1978, CPV has occurred worldwide (Appel et al., 1978; Pollock et al., 1980). It appears capable of infecting all wild canids (Eugster et al., 1978; Fletcher et al., 1979; Evermann et al., 1980; Mann

et al., 1980). In domestic dogs, the disease may be manifested as an enteritis or a myocarditis (Appel et al., 1978; Atwell and Kelly, 1980). Published reports on the occurrence and/or signs of CPV in free-ranging canids are limited in number, but increasing. Antibodies in free-ranging coyotes from Texas, Utah, and Idaho were not detected prior to 1979, but increased to about 70% by 1982 (Thomas et al., 1984). Antibody prevalences for free-ranging red foxes and coyotes in Ontario were equally high during 1980–1981 (Barker et al., 1983). Our results (Table 1) basically concur with these two recent studies. Results of all three studies reflect the introduction of CPV into a wild canid population during 1979 or 1980, followed by increases to over 50% prevalence by 1981–1982. We found no sex-specific ($P > 0.05$) or age-specific ($P > 0.05$) differences in CPV antibody prevalences, nor was there any significant difference ($P > 0.05$) in prevalence between packs > or <30 km from towns and roads.

Based upon studies of CPV infection in captive wild species (Evermann et al., 1980), the disease may be severe enough to cause mortality at least in young wolves. If this assumption is correct, CPV could have significant implications for management of wolf populations, particularly at high population densities. On the other hand, no dramatic decrease in either productivity or survival of wolves due to CPV has been observed in GMU-13 (Ballard et al., 1981, 1982b).

Tularemia

Tularemia is an acute, febrile, plaguelike disease of wild lagomorphs and rodents caused by the bacterium *Francisella tularensis*. Snowshoe hares are the primary reservoir of tularemia in Alaska (Dieterich, 1981). The disease is transmitted usually among hares by ticks, particularly when the population density of hares is high. Transmission to predators occurs usually as a result of their preying on infected hares. Historically serum antibody prevalence has been low in red fox and domestic dog populations in Alaska (Zarnke, 1983).

Populations of hares peaked about 1980–1981 in most of Interior Alaska (Zarnke, unpubl. data). The overall 25% prevalence of antibody to *Francisella tularensis* in wolves (Table 1) may reflect a high prevalence of infection in the hare population which increased during at least the first 5 yr of the study period. Antibody prevalence was neither sex-specific nor age-specific. The impact of tularemia infection on individual wolves is unknown, but we hypothesize that most healthy adults would recover from an uncomplicated bout with the disease.

Canine distemper virus (CDV)

Signs of canine distemper in wild canids may include oral icterus and ulceration, swollen feet, anorexia, ataxia, dyspnea, and neurologic abnormalities (Monson and Stone, 1976). Transmission occurs via direct contact or aerosol droplet (Budd, 1981). Serologic evidence of CDV in wolves has been reported previously from northern Canada (Choquette and Kuyt, 1974) and from two areas of Alaska including GMU-13 (Stephenson et al., 1982). The 12% prevalence reported here (Table 1) does not differ significantly ($P > 0.05$) from data presented in earlier studies. Low serologic prevalence of an infection in a host population suggests that: (1) the host species is resistant to infection, (2) infection usually results in death of the host, or (3) exposure of the host to the disease agent is uncommon. For the purpose of the present situation, there is ample evidence to reject the first hypothesis (Elton, 1931; Budd, 1981; Stephenson et al., 1982). In addition, no cases of CDV-induced mortality were confirmed in any of the more than 150 wolves radio-collared during the study. Thus, although the sec-

ond hypothesis cannot be formally rejected, we suspect that the third hypothesis most accurately reflects the situation in populations of wolves.

The presence of CDV seropositive wolves during 6 of 8 yr of this study supports the hypothesis of Trainer and Knowlton (1968), and Choquette and Kuyt (1974) who maintain that CDV is enzootic in free-ranging canid populations rather than being introduced sporadically from domestic dogs as suggested by Elton (1931) and Stephenson et al. (1982). If the latter hypothesis was correct we would have anticipated a high prevalence in the population, followed by a gradual decline to very low levels, followed by another peak. Such was not the case (Table 1).

No pups had serologic evidence of previous CDV infection. This finding was consistent with earlier studies (Choquette and Kuyt, 1974; Stephenson et al., 1982). Perhaps maternal antibody was protective even at concentrations below that which we selected as a minimum threshold. If correct, such a situation would differ from that described for domestic dogs (Gorham, 1966; Gillespie and Carmichael, 1968), where pups are susceptible within a couple of months of birth and virtually all members of a population have protective antibody by the time they reach 1 yr of age. Alternatively, perhaps other factors such as cellular immunity play a role in protecting wolf pups from CDV infection.

Clinical CDV was reported in domestic dogs during February and March 1979, in the vicinity of Glennallen (Tobey, pers. comm.), the major human settlement within the study area. Based upon results of serologic tests (Table 1), the prevalence of CDV did not increase in the wolf population during this period. We found no significant difference ($P > 0.05$) in prevalence between packs whose territories were greater or less than 30 km from towns and roads. Therefore, it appears that the outbreak in domestic dogs was just one of several which occurred over a period of decades, and was not a direct source of infection for wolves.

There was no evidence ($P > 0.05$) of sex-specificity for CDV seroprevalence. Most packs involved in the study were highly productive (Ballard et al., 1981, 1982b). Therefore, we conclude that although CDV may have temporarily incapacitated individual animals, it was not a major mortality factor.

Q fever

This disease is caused by the rickettsium *Coxiella burnetti* (Randhawa et al., 1977), which usually localizes in the respiratory tract. Although the disease is usually mild in domestic species, abortions can occur in domestic sheep and goats (Bell, 1981). Death is rare (Bell, 1981). *Coxiella burnetti* is shed in milk, feces, placental fluids, and placental tissues (Enright et al., 1969).

Q fever has a broad host range, including many species of wild and domestic birds and mammals. Caribou are the most common host in Alaska, with serologic prevalences averaging 10% over a 4-yr period in the Delta Herd south of Fairbanks (Hopla, 1975). In addition, several species of rodents have been identified frequently as hosts of the disease agent (Hopla, 1965). Scavengers and predators such as grizzly bears (*Ursus arctos*), wolverines (*Gulo gulo*), arctic foxes (*Alopex lagopus*) and red foxes have been implicated also in Alaska by means of serologic surveys (Hopla, 1966; Zarnke, 1983) with annual prevalences reaching as high as 38% in an arctic fox population near Prudhoe Bay (Zarnke, 1983). The course or ultimate resolution of infection in wild carnivores is largely unknown. The 5% prevalence reported here may be the first report of antibodies to the Q fever agent in wolves (Table 1). Presumably, wolves are exposed when feeding on infected rodents, ungu-

lates or scavengers such as foxes. The significance of Q fever to the wolf population is unknown.

Rabies virus

Within Alaska, rabies virus is distributed almost exclusively along coastal regions in southwestern and northern portions of the state where red and arctic fox are the primary hosts (Ritter, 1981). For most mammalian species, rabies is usually fatal. When wolves become involved in rabies epizootics, entire packs may be lost (Chapman, 1978). Thus, rabies could be a limiting factor, especially at high wolf population densities. However, cases in either wild or domestic canids from the Interior are rare (Ritter, 1981). The low antibody prevalence in the current study (Table 1) fits this pattern. The single seropositive animal represents an anomaly. We might speculate on long-range dispersal of wolves or other canids from enzootic areas hundreds of kilometers to the west of the study area; movements greater than 700 km have been recorded for wolves in Alaska (Ballard et al., 1983). Other possibilities include that (a) the animal had survived infection or (b) the results were non-specific.

Brucellosis

Brucella suis biotype 4 is the causative agent of brucellosis in Alaskan wildlife species (Neiland et al., 1968). Caribou are the primary reservoir of this disease (Neiland et al., 1968). Carnivores are infected most commonly by preying or scavenging on infected caribou (Neiland, 1970). The 1% antibody prevalence reported here (Table 1) was lower than the 30% prevalence for wolves in northern Alaska (Neiland, 1970; 1975) or 11% for wolves in Siberia (Pinigin and Zabrodin, 1970). However, this was not surprising for several reasons. Antibody prevalence in the Nelchina caribou herd, which lives in GMU-13, has been relatively low (less than 5%) since at least 1962 (Neiland et al., 1968; Zarnke and Neiland, 1980), compared with caribou herds in these other areas, where prevalence has reached 30% (Neiland et al., 1968). In addition, moose was the most important prey item for most packs in GMU-13 during the current study (Ballard et al., 1980, 1981), and brucellosis is rare in moose (Neiland et al., 1968). Consequently, wolves in GMU-13 would have less exposure to brucellosis than wolves in northern Alaska which prey heavily on caribou.

Neiland (1975) predicted and later (Neiland and Miller, 1981) provided evidence to support the possibility of reproductive failure in wolves as a result of brucellosis infection. The continued high productivity of wolves in GMU-13 (Ballard et al., 1981, 1982b) fits the pattern of low *Brucella* spp. prevalence in this population.

Leptospirosis

Leptospirosis is found in wildlife species throughout North America (Shotts, 1981). Carnivores may be infected via exposure to contaminated urine or by feeding on infected prey (Reilly et al., 1970). Signs may include chronic kidney infections, hepatitis, and/or abortions (Shotts, 1981). The disease is common in red foxes (Clark et al., 1960), gray foxes (*Urocyon cinereoargenteus*) (Clark et al., 1961), and coyotes (Marler et al., 1979; Drewek et al., 1981) in the contiguous United States. We have no data on prevalences of infection in small or medium-sized mammals in GMU-13, but moose, black bear (*Ursus americanus*), and grizzly bear populations are seropositive in the 3–6% range (Zarnke, unpubl. data). Therefore, the virtual absence of detectable antibody in the GMU-13 wolf population (Table 1) cannot be explained at this time.

CONCLUSIONS

We concluded that rabies, brucellosis, and leptospirosis were rare and posed little or no threat to the health of the wolf population in GMU-13 during 1975–1982. Conversely, CPV, ICH, CDV, Q fever, and tularemia were relatively prevalent, but likewise had no apparent effect upon wolf abundance. Historically, wolves in Alaska have likely been exposed to CDV, ICH, Q fever, and tularemia. Certainly, all four infections are capable of causing significant mortality, particularly in high density populations. Apparently these host/parasite relationships in this moderately dense population of wolves have evolved to the point where large-scale die-offs are relatively rare.

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BOOK REVIEW . . .

The Coccidian Parasites (Protozoa, Apicomplexa) of Artiodactyla, N. D. Levine and V. Ivens. University of Illinois Press, 54 East Gregory Drive, Champaign, Illinois, USA. 1986. 265 pp. $19.95 U.S.

This monograph is an update of a volume published in the same series in 1970 by the same authors. The number of named species has more than doubled since the 1970 version. Species that have been named in the interim or for which new information is available have been added. It also contains information on genera that recently have been included in the coccidia such as *Sarcocystis*, *Toxoplasma*, *Besnoitia*, and *Hammondia*. New data on *Cryptosporidium* is presented on a variety of host species. The listing of species is very complete.

A brief discussion and summary follow the listing of species encountered in each genus of artiodactylid. Data on each species include synonyms, type-hosts, other hosts, location in gut, oocyst morphology, description of merogony and gametogony, prepatent and patent period, pathogenicity, immunology, cross transmission studies and results from cultivation. It should be remembered that for the majority of these species, we do not know the complete life cycle. This reference would make a good starting point for people interested in doing research on coccidia of these mammals.

The largest deficit of the book is that of the illustrations. All are line drawings and they have all been copied from other sources. Some of these did not reproduce well. More detracting, however, is the deletion of figures included in the 1970 volume. This problem is magnified by the earlier volume being out of print and thus not readily available. It is hoped that when the Illinois Biological Monographs rodent coccidia volume is redone, that economics and editorial policy will not allow this mistake to be repeated.

This book is an essential reference for persons interested in coccidian parasites.

Ellis C. Greiner, Department of Infectious Diseases, College of Veterinary Medicine, University of Florida, Gainesville, Florida 32610, USA.