



**GEOGRAPHIC PATTERN OF SERUM ANTIBODY
PREVALENCE FOR BRUCELLA SPP. IN CARIBOU,
GRIZZLY BEARS, AND WOLVES FROM ALASKA, 1975–
1998**

Authors: Zarnke, Randall L., Ver Hoef, Jay M., and DeLong, Robert A.

Source: Journal of Wildlife Diseases, 42(3) : 570-577

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-42.3.570>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

GEOGRAPHIC PATTERN OF SERUM ANTIBODY PREVALENCE FOR *BRUCELLA* SPP. IN CARIBOU, GRIZZLY BEARS, AND WOLVES FROM ALASKA, 1975–1998

Randall L. Zarnke,^{1,3} Jay M. Ver Hoef,^{1,2} and Robert A. DeLong¹

¹ Alaska Department of Fish and Game, 1300 College Road, Fairbanks, Alaska 99701-1599, USA

² Current address: NOAA National Marine Mammal Lab, 7600 Sand Point Way NE, Bldg 4, Seattle, Washington 98115-6349, USA

³ Corresponding author (email: itrapp2@acsalaska.net)

ABSTRACT: Blood samples were collected from 2,635 caribou (*Rangifer tarandus*), 1,238 grizzly bears (*Ursus arctos*), and 930 wolves (*Canis lupus*) from throughout mainland Alaska during 1975–98. Sera were tested for evidence of exposure to *Brucella* spp. Serum antibody prevalences were highest in the northwestern region of the state. In any specific area, antibody prevalences for caribou and wolves were of a similar magnitude, whereas antibody prevalence for bears in these same areas were two to three times higher.

Key words: Alaska, *Brucella* spp., caribou, grizzly bear, wolf.

INTRODUCTION

Caribou (*Rangifer tarandus*) are widely distributed throughout mainland Alaska (Valkenburg, 1998). They live in herds that range in size from a few hundred animals to a few hundred thousand (Valkenburg et al., 1996). Size of individual herds can vary considerably due to the effects of predation, quantity and quality of available food, and weather (Adams and Dale, 1998). Infectious and parasitic diseases also play a role in population dynamics (Dieterich, 1980).

Wolves (*Canis lupus*) and grizzly bears (*Ursus arctos*) are the two primary terrestrial predator species in Alaska. Both species prey extensively on caribou where they are sympatric (Valkenburg et al., 1996). The predation process provides ample opportunity for transmission of diseases and parasites from caribou to predators (Neiland, 1970).

Brucellosis is a bacterial disease with a worldwide distribution (Tessaro, 1986). Several species comprise the genus *Brucella*, and each species has a preferred host range (Witter, 1981). Reindeer and caribou are the primary hosts for *Brucella suis* IV (Forbes, 1991). Infection localizes primarily in joints and the reproductive tract (Dieterich and Morton, 1987), but other tissues also can be infected (Tessaro and

Forbes, 1986). Clinical signs of disease include orchitis in males, abortion in females, and bursitis in both sexes (Forbes, 1991). Prevalence of antibody to *B. suis* IV in caribou herds varies from year to year, but temporal patterns were not reported for the period 1975–99 (Zarnke, 2000).

Isolations of *B. suis* IV have been reported from caribou, wolves, and grizzly bears in Alaska on numerous occasions (Neiland et al., 1968; Neiland, 1970, 1975); other *Brucella* spp. have not been isolated from these species. Unfortunately, the number of isolations has not been adequate to determine whether geographic patterns of infection exist. The objective of the current study was to use a large collection of sera that were available for caribou, wolves, and grizzly bears in Alaska to determine whether *Brucella* sp. antibody prevalences for these species were spatially dependent.

MATERIALS AND METHODS

Caribou, wolves, and grizzly bears were captured by personnel of the Alaska Department of Fish and Game, US Fish and Wildlife Service, and National Park Service. Several individual bears were captured more than once. For the purpose of this study, only the blood sample from the first capture was used. Blood samples were collected and stored at either ambient or refrigerated temperatures for 12–36 hr. Sera were removed and stored

temporarily at -15°C . Long-term storage was at -55°C for 1–10 yr until the time of testing.

Before 1990, tests were conducted at the US Department of Agriculture's National Veterinary Services Laboratory in Ames, Iowa, USA. The preliminary test method was the buffered *Brucella* antigen (BBA) test (Angus and Barton, 1983). Samples testing positive were also tested by standard tube test (STT) (Alton and Jones, 1967). After 1990, sera were tested with the standard plate test (SPT) (US Department of Agriculture, undated) and card test (CAR) (Alton and Jones, 1967) at the University of Alaska's Institute of Arctic Biology, Fairbanks, Alaska, USA. All tests used *B. abortus* as antigen. Sera that caused agglutination in the SPT or STT at a serum dilution ($\geq 1:50$) were considered antibody positive. This is one dilution higher than the standard threshold titer (25) for both the SPT and STT (Alton and Jones, 1967). This higher positive threshold was selected to increase test specificity. The BBA and CAR tests were reported as either positive or negative.

If the BBA test result was negative, the sample was not tested using the STT. This result was interpreted as a *B. suis* IV negative. If positive by BBA, a sample was classified as positive only if the STT titer was ≥ 50 . The BBA-positive samples with STT titers < 50 were considered negative. Likewise, a CAR positive was classified as positive only if a titer of ≥ 50 was detected by SPT. Finally, a sample was considered positive if it tested negative on CAR but had an SPT titer ≥ 50 .

To aid in managing wildlife, the landmass of Alaska is divided into 26 Game Management Units (GMUs). These areas are based on major physiographic features such as mountain ranges and major river drainages. Hunting seasons and bag limits are based on the number and distribution of animals within a GMU. Several of the larger GMUs are further divided into subunits.

Statistical methods for disease mapping (Lawson, 2001; Lawson and Williams, 2001) were used to analyze the data. Because low sample sizes in some GMUs may have reduced the reliability of individual GMU prevalence estimates, a statistical model was developed to "smooth" prevalences (Lawson and Williams, 2001). In this model, sample sizes and rates from nearby GMUs affect estimated prevalences. This model also assumed that the underlying pattern of smoothed prevalence was shared by caribou, wolves, and bears. The estimated prevalence for each species was a multiplicative factor of the smoothed underlying rate, with an additional random component for each species. This model

allowed mapping of the smoothed antibody prevalence and comparison of the relative overall prevalences for each species. Details of the model are described below.

A Bayesian hierarchical model (Clayton and Kaldor, 1987; Devine et al., 1994; Bernardinelli et al., 1995; Waller et al., 1997; Xia et al., 1997) was used to estimate area-specific prevalences for all three species. Let N_{ij} be the number of samples from the i th area (for all GMUs listed in Table 1); $i = 1, 2, \dots, 26$, for the j th species; $j = 1$ (caribou), 2 (wolf), or 3 (grizzly bear). Let x_{ij} be the number of positives in the i th area. Assume that positives are binomially distributed,

$$x_{ij}|p_{ij}, N_{ij} \sim \text{Bin}(N_{ij}, p_{ij}),$$

where

$$\text{logit}(p_{ij}) = \mu + \alpha_j + b_i + \varepsilon_{ij}.$$

This is the usual logistic regression situation, except that b_i is a random effect that is spatially autocorrelated with its neighbors and ε_{ij} is an independent random effect. For the fixed effects, α_1 was assigned a value of 0. A normal distribution $\alpha_j \sim N(0, 10000)$ was used for $j = 2, 3$. An improper flat prior was given to μ . The independent random errors were given a normal distribution with a separate variance for each species, $\varepsilon_{ij} \sim N(0, \sigma_j^2)$, and the variance parameter was given a gamma distribution, $\sigma_j^2 \sim \text{Gam}(0.001, 0.001)$. The autocorrelation among the $\{b_i\}$ followed a conditional autoregressive model (see Cressie, 1993, p. 407). Any two GMUs that shared a border were defined as neighbors. A normally distributed conditional autoregressive model is defined where $b_i|\bar{b}_i, \phi, n_i$ is normally distributed $N(\bar{b}_i, \phi/n_i)$, where \bar{b}_i is the mean of the neighboring values for the i th GMU and n_i is the number of neighbors (Besag et al., 1991). The variance parameter was given a gamma distribution, $\phi \sim \text{Gam}(0.001, 0.001)$.

The statistical software package WinBUGS was used to obtain a sample from the posterior distribution for ϕ, b_i, α_j , and μ , and functions of these parameters. For example, the posterior distribution of

$$100 \times \exp(\mu + \alpha_i + b_j) / [1 + \exp(\mu + \alpha_i + b_j)]$$

provides an estimate of the prevalences (in %) in the j th GMU for the i th species. These values are known as "smoothed" rates. The mean of the sample from the posterior distribution was used to estimate the smoothed rates, and the standard deviation of the sample

TABLE 1. Serum antibody prevalence for *Brucella* sp. in caribou (*Rangifer tarandus*), wolf (*Canis lupus*), and grizzly bear (*Ursus arctos*) from Alaska.

GMU ^a	Caribou				Wolf				Grizzly bear						
	Sample size	No. positive ^b	Prevalence (%)	Smooth rate (%) ^c	SE ^d	Sample size	No. positive ^b	Prevalence (%)	Smooth rate (%) ^c	SE ^d	Sample size	No. positive ^b	Prevalence (%)	Smooth rate (%) ^c	SE ^d
7	13	0	0	1.3	1.38				2.8	3.04				5.6	5.35
8	3	0	0	1.3	0.40				3.0	1.08			6	6.0	1.51
9	337	0	0	1.9	0.49				4.4	1.49			15	8.8	1.98
11	19	0	0	0.9	1.15				2.0	2.60				4.1	4.62
12	77	0	0	0.5	0.40		0		1.1	0.91				2.4	1.83
13	214	1	1	1.1	0.32		1		2.5	0.84			8	5.1	1.30
14				1.0	0.93		0		2.3	2.03				4.8	3.87
15	22	0	0	1.2	0.93				2.7	2.13				5.4	3.89
16				1.0	0.72		0		2.2	1.60				4.6	3.06
17	87	0	0	0.7	0.55				1.7	1.28				3.4	2.49
18	3	0	0	0.6	0.37				1.3	0.84				2.7	1.65
19	44	0	0	0.8	0.64				1.9	1.46				4.0	2.82
20A	126	0	0	0.5	0.18				1.1	0.46				2.3	0.79
20B	3	0	0	0.7	0.34		3		1.5	0.64			2	3.3	1.52
20C	105	0	0	0.6	0.29		2		1.4	0.61				3.0	1.36
20D	97	0	0	0.5	0.36				1.1	0.83				2.4	1.66
20E	162	0	0	0.4	0.22		1		0.9	0.46				1.9	1.05
20F	1	1	100	3.7	4.26				7.8	7.59				14.5	11.01
21	53	5	9	2.9	1.25		0		6.3	2.70				12.5	4.79
22				2.9	1.01				6.4	2.51				12.5	3.52
23	541	38	7	5.6	0.79				12.1	3.00			19	22.5	2.44
24	20	0	0	3.2	1.38		4		7.0	2.66				13.9	5.04
25	103	5	5	3.8	1.52		0		8.4	3.74			40	16.1	5.62
26A	182	13	7	6.1	0.98		15		13.1	2.94			22	24.1	2.03
26B	327	8	2	2.7	0.69				6.0	2.04				11.8	2.82
26C	98	1	1	2.9	0.57		1		6.3	1.69			13	12.5	1.61

^a Game Management Unit.

^b Results of standard plate test and buffered *Brucella* antigen tests.

^c Estimated prevalence based on the observed prevalence, model effects, and effects from neighboring areas.

^d Standard errors of the estimated prevalence (smooth rates).

gives the standard error of the smoothed rates (Besag et al., 1991; Besag and Kooperberg, 1995). The posterior sample was obtained using Markov Chain Monte Carlo methods, with a “burn-in” of 4,000 iterations. The sample was drawn from the next 50,000 iterations.

RESULTS

For GMUs with samples sizes >1 , observed antibody prevalences ranged from 0% to 9% for caribou, from 0% to 25% for wolves, and from 0% to 24% for bears. Prevalences for all three species were highest in the northern portion of the state (Table 1). Within any particular GMU, the relative magnitude of observed prevalences for caribou and wolves were similar. Prevalences for bears were often higher than for the other two species. The model predicts that antibody prevalence for wolves would be approximately three times higher than for caribou. For bears, antibody prevalence would be approximately 11 times higher than for caribou. The spatial model produced estimates of *Brucella* sp. antibody prevalence for caribou, wolves, and grizzly bears. Graphic representations of these estimates are shown in Figure 1.

DISCUSSION

For *Brucella* sp. antibody testing, individual samples are often tested by four or more serologic test methods. The University of Alaska laboratory, which provided diagnostic support for this survey, conducts serologic tests specifically for *B. suis* IV. Laboratory personnel have selected BBA and SPT as the most convenient and reliable serologic methods for assessing previous exposure to this agent. Testing the current collection of sera by means of additional methods may have produced slightly different results for a few samples. However, the pattern reported here is believed to accurately reflect actual prevalences in the respective geographic areas.

Estimates produced by the spatial

model (Fig. 1) confirm that antibody prevalences for all three species are highest in the northwest portion of the state. In some cases, the raw rates for an individual species may provide a somewhat biased picture of geographic distribution. Animals captured on the boundary of GMU “A” may actually spend most of their time in adjacent GMU “B.” In addition, only a few animals of this species may have been captured in GMU “A.” Therefore, these few animals have a large influence on the overall prevalence attributed to GMU “A.” The best examples of this phenomenon in the current study are 1) the 100% prevalence (1/1) for caribou in GMU 20F and 2) the 40% prevalence (2/5) for bears in GMU 25 (Table 1).

Therefore, the model estimates of antibody prevalence provide a better overall representation of the geographic distribution of *Brucella* sp. exposure. Multiple samples from a few animals may have exerted a small bias on the reported prevalence.

For most GMUs, antibody prevalences for bears were higher than prevalences for caribou and wolves (Table 1). Bears and wolves are exposed to *B. suis* IV while preying on infected caribou (Neiland, 1975; Neiland and Miller, 1981). Hypothetically, antibody prevalence should be directly related to the number of caribou killed by wolves and bears. However, for GMU 20E in eastern Alaska, an average wolf killed approximately four times as many caribou (all ages) as an average grizzly during 1994–97 (Boertje and Gardner, 2000). Thus, the hypothesized direct relationship between kill rate and antibody prevalence was not valid for this geographic area. Comparable data was not available for other areas.

Transmission of *Brucella* spp. is typically higher during the calving period (Forbes, 1991). Perhaps, bears have a higher level of predation during the period surrounding calving. Hypothetically, higher kill totals at this time of year could account for higher antibody prevalences in bears.

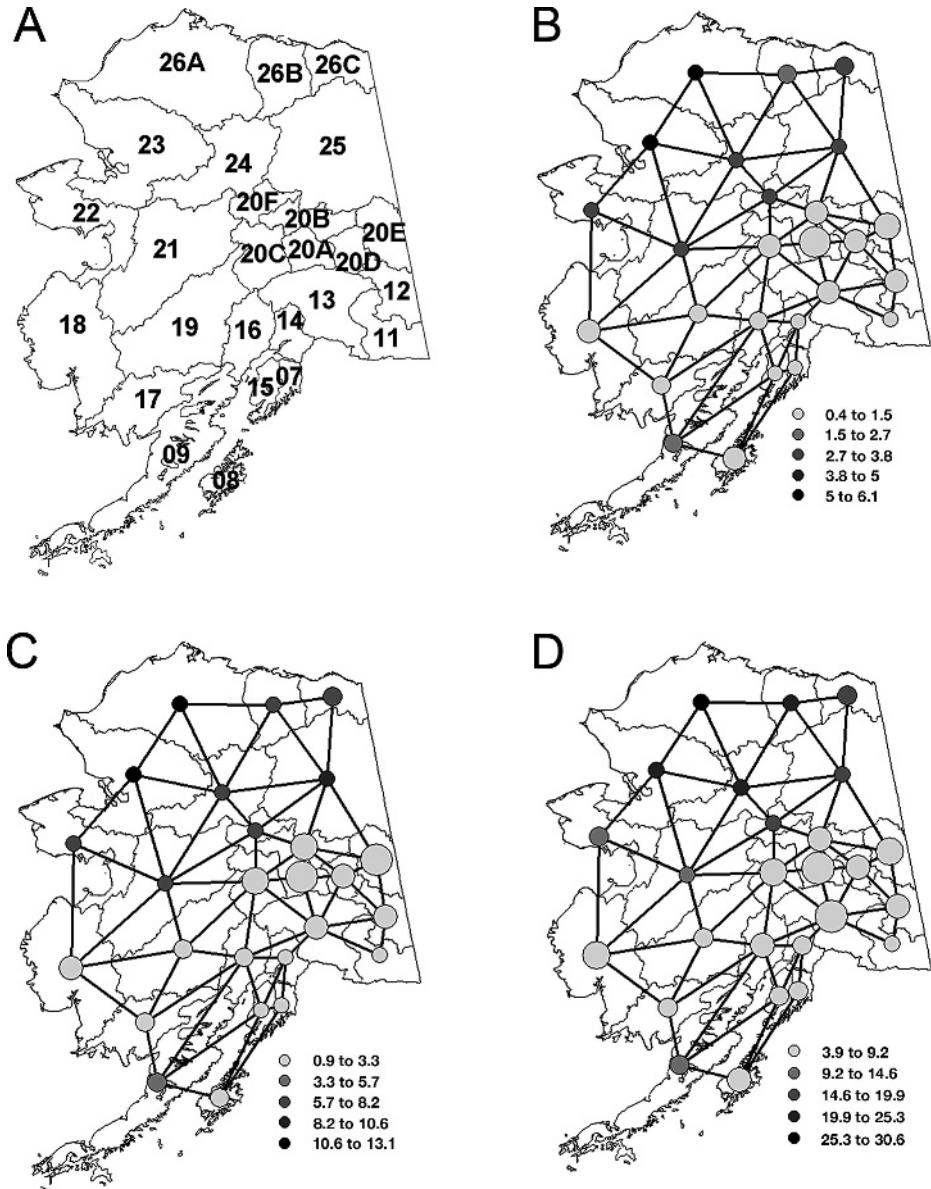


FIGURE 1. Location of Game Management Units in Alaska and model estimates of *Brucella* sp. serum antibody prevalence in caribou (*Rangifer tarandus*), wolves (*Canis lupus*), and grizzly bears (*Ursus arctos*). (A) Game Management Unit boundaries. (B) Antibody prevalence (%) for caribou. (C) Antibody prevalence (%) for wolves. (D) Antibody prevalence (%) for grizzly bears. Note that the scale is different for each species. Circle size is inversely proportional to standard errors. Lines connecting circles represent neighboring populations used in the spatial statistical model.

However, this hypothesis is not supported by data. The average wolf killed twice the number of caribou calves as the average bear during the summer months in GMU 20E (Boertje and Gardner, 2000). Thus, even when bears are out of hibernation,

wolves have a higher kill total and presumably higher opportunity for exposure to *B. suis* IV.

Perhaps bears scavenge more aborted fetuses, placentae, and cubs that die as a result of brucellosis after birth. These

would be prime sources of *B. suis* IV exposure for scavengers. There is no data to address this latter hypothesis.

Serum antibody prevalence for many agents in many host species is directly related to age (Zarnke and Evans, 1989; Zarnke et al., 1997, 2001). The average age of the grizzly bears sampled in this survey is probably higher than the average age of the entire population. Cubs (<1 yr old) are only occasionally captured during normal field studies. Thus, the cub cohort is underrepresented in the sample. Conversely, the average age of the wolves sampled is probably similar to the age profile of wolf population. Wolf pups (<1 yr old) are routinely caught during field studies. Perhaps, the deviation from the perceived normal age distribution for bears was partially responsible for the higher than expected antibody prevalence for *B. suis* IV.

Historically, brucellosis has been considered to be present in caribou herds throughout Alaska (Neiland et al., 1968). The observed serum antibody prevalence for caribou from the southern half of the state is essentially 0% (Table 1). One interpretation of these data is that the disease is absent from this region. Observed prevalences for bears from all regions (including the southern half of the state) are higher than prevalences for caribou. These data indicate that bears are being exposed to *Brucella* sp. in the southern portion of the state. Presumably, the source of that exposure would be infected caribou. No other species serve as an effective large-scale reservoir for transmission to predators and scavengers. Perhaps the disease is indeed present in most (if not all) caribou herds, but at very low levels in the southern portion of the state. Perhaps sampling intensity was simply incapable of detecting this very low frequency of infection in these southerly herds. A second potential explanation is that bears are exposed to *Brucella* sp. by scavenging on infected marine mammal carcasses (Zarnke et al., 2006) that wash

up on the beach. A third alternative explanation would be that the disease does not occur in caribou herds from the southern portion of the state, and the positive serologic test results for bears and wolves from this region are incorrect. There is a fourth potential explanation for the presence of positive bears in regions of the state where no positive caribou were found. Perhaps, positive bears make long-range movements from the northwestern region to other areas of the state. However, movements of this magnitude are inconsistent with data collected during long-term studies. Juvenile female bears typically establish home ranges near their mother's home range. Juvenile male bears may establish home ranges <75 mi from their mothers. Movement >100 mi has been observed once during radiotelemetry studies of >500 bears (Reynolds, pers. comm.).

Numerous free-ranging, semidomestic reindeer herds live in GMU 22 on the Seward Peninsula (Fig. 1). Brucellosis is enzootic in these herds (Dieterich and Morton, 1987). The Western Arctic caribou herd has a large home range, covering portions of GMUs 21, 22, 23, and 26A (Fig. 1). During the winter, the Western Arctic Herd migrates to the southwestern portion of its range. At that time, there is often opportunity for contact between Seward Peninsula reindeer and caribou from the Western Arctic Herd. Reindeer may have been the original reservoir for transmission of brucellosis to other arctic species (Davydov, 1965). Alternatively, perhaps the disease has always been enzootic in free-ranging caribou (Huntley et al., 1963). The current study provides no evidence to confirm or refute either theory.

Brucellosis has two major impacts on caribou: 1) abortion in pregnant females and 2) lameness in both sexes (Forbes, 1991). The lameness can result in animals becoming more susceptible to predation. In addition to the deaths of individual animals, presumably both conditions (es-

pecially abortion) have a negative impact on population dynamics of the herd. Presumably, the impact of abortion on wolf and bear population dynamics is similar (Neiland and Miller, 1981). However, populations of all three species can thrive even when the disease is present. Thus, mortality caused by *B. suis* IV may be compensatory, rather than additive.

A vaccine is available that significantly reduces the prevalence of the disease in semidomestic reindeer (Dieterich and Morton, 1987). Theoretically, the vaccine could have a similar positive benefit for caribou herds. However, human control or eradication of the disease in free-ranging caribou is not presently feasible. The large number of animals (~1,000,000) spread over a vast and remote landscape precludes efficient intervention. Therefore, management implications of the vaccine for caribou are minimal. Wildlife biologists are limited to advising hunters regarding potential risk of exposure and how to avoid contracting the disease. In addition, translocation of caribou from the northern part of Alaska should be avoided.

ACKNOWLEDGMENTS

The authors thank the many agency field personnel who collected blood samples that made this study possible. This study was supported by Federal Aid in Wildlife Restoration.

LITERATURE CITED

- ADAMS, L. G., AND B. W. DALE. 1998. Reproductive performance of female Alaskan caribou. *Journal of Wildlife Management* 62: 1184–1195.
- ALTON, G. G., AND L. M. JONES. 1967. Laboratory techniques in brucellosis. World Health Organization, Geneva, Switzerland, 92 pp.
- ANGUS, R. D., AND C. E. BARTON. 1983. The production and evaluation of a buffered plate antigen for use in a presumptive test for brucellosis. Third international symposium on brucellosis, Algiers, Algeria, 1983. *Developments in Biological Standardization* 56: 349–356.
- BERNARDINELLI, L., D. CLAYTON, AND C. MONTOMOLI. 1995. Bayesian estimates of disease maps: How important are priors? *Statistics in Medicine* 14: 2411–2431.
- BESAG, J., AND C. L. KOOPERBERG. 1995. On conditional and intrinsic autoregressions. *Biometrika* 82: 733–746.
- , J. YORK, AND A. MOLLIE. 1991. Bayesian image restoration, with two applications in spatial statistics. *Annals of the Institute of Statistical Mathematics* 43: 1–59 (with discussion).
- BOERTJE, R. D., AND C. L. GARDNER. 2000. The Fortymile caribou herd: Novel proposed management and relevant biology, 1992–1997. *In* Eighth North American Caribou Workshop, Whitehorse, Yukon, Canada, 20–24 April 1998. *Rangifer Special Issue* 12: 17–37.
- CLAYTON, D., AND J. KALDOR. 1987. Empirical Bayes estimates of age-standardized relative risks for use in disease mapping. *Biometrics* 43: 671–681.
- CRESSIE, N. A. C. 1993. *Statistics for spatial data*, revised edition. John Wiley & Sons, New York, New York, 900 pp.
- DAVYDOV, N. N. 1965. La brucellose du renne dans le Grand Nord. *Bulletin de Office International des Epizooties* 63: 1005–1014.
- DEVINE, O. J., M. E. HALLORAN, AND T. A. LOUIS. 1994. Empirical Bayes methods for stabilizing incidence rates prior to mapping. *Epidemiology* 5: 622–630.
- DIETERICH, R. A. 1980. Current status of reindeer/caribou diseases in Alaska. *In* Proceedings of the second international reindeer/caribou symposium, Røros, Norway, 1979. E. Reimers, E. Gaare, R. A., and S. Skjenneberg (eds.). Direktoratet for vilt og ferskvannsfisk, Trondheim, Norway, pp. 438–441.
- , AND J. K. MORTON. 1987. Effects of live *Brucella abortus* strain 19 vaccine on reindeer later challenge exposed with *Brucella suis* type 4. *Rangifer* 7: 33–36.
- FORBES, L. B. 1991. Isolates of *Brucella suis* biovar 4 from animals and humans in Canada, 1982–1990. *Canadian Veterinary Journal* 32: 686–688.
- HUNTLEY, B. E., R. N. PHILIP, AND J. E. MAYNARD. 1963. Survey of brucellosis in Alaska. *Journal of Infectious Diseases* 112: 100–106.
- LAWSON, A. B. 2001. *Statistical methods in spatial epidemiology*. John Wiley & Sons, Chichester, UK, 277 pp.
- , AND F. L. R. WILLIAMS. 2001. *An introductory guide to disease mapping*. John Wiley & Sons, Chichester, UK, 133 pp.
- NEILAND, K. A. 1970. Rangiferine brucellosis in Alaskan canids. *Journal of Wildlife Diseases* 6: 136–139.
- . 1975. Further observations on rangiferine brucellosis in Alaskan carnivores. *Journal of Wildlife Diseases* 11: 45–53.
- , AND L. G. MILLER. 1981. Experimental *Brucella suis* type 4 infections in domestic and wild Alaskan carnivores. *Journal of Wildlife Diseases* 17: 183–189.
- , J. A. KING, B. E. HUNTLEY, AND R. O. SKOOG.

1968. The diseases and parasites of Alaskan wildlife populations, part I. Bulletin of the Wildlife Disease Association 4: 27–36.
- TESSARO, S. V. 1986. The existing and potential importance of brucellosis and tuberculosis in Canadian wildlife: A review. Canadian Veterinary Journal 27: 119–124.
- , AND L. B. FORBES. 1986. *Brucella suis* biotype 4: A case of granulomatous nephritis in a barren ground caribou (*Rangifer tarandus groenlandicus* L.) with a review of the distribution of rangiferine brucellosis in Canada. Journal of Wildlife Diseases 22: 479–483.
- US DEPARTMENT OF AGRICULTURE. Undated. National Animal Disease Laboratory Diagnostic Reagents Manuals 65D, E, F. National Animal Disease Laboratory, Ames, Iowa, 57 pp.
- VALKENBURG, P. 1998. Herd size, distribution, harvest, management issues, and research priorities relevant to caribou herds in Alaska. Seventh North American caribou conference, Thunder Bay, Ontario, Canada, 19–21 August 1996. Rangifer 10: 125–129.
- , J. L. DAVIS, J. M. VER HOEF, R. D. BOERTJE, M. E. MCNAY, R. M. EAGAN, D. J. REED, C. L. GARDNER, AND R. W. TOBEY. 1996. Population decline in the Delta caribou herd with reference to other Alaskan herds. Sixth North American caribou workshop, Prince George, British Columbia, Canada, 1–4 March 1994. Rangifer 9: 53–62.
- WALLER, L. A., B. P. CARLIN, H. XIA, AND A. E. GELFAND. 1997. Hierarchical spatio-temporal mapping of disease rates. Journal of the American Statistical Association 92: 607–617.
- WITTER, J. F. 1981. Brucellosis. In Infectious diseases of wild animals. 2nd Edition, J. W. Davis, L. H. Karstad and D. O. Trainer (eds.). Iowa State University Press, Ames, Iowa, pp. 280–287.
- XIA, H., B. CARLIN, AND L. A. WALLER. 1997. Hierarchical models for mapping Ohio lung cancer rates. Environmetrics 8: 107–120.
- ZARNKE, R. L. 2000. Alaska Wildlife Serologic Survey, 1975–2000. Alaska Department of Fish and Game. Federal Aid in Wildlife Restoration. Research Final Report. Grants W-24-5 and W-27-1 through W-27-4. Study 18.71. Juneau, Alaska, 28 pp.
- , AND M. B. EVANS. 1989. Serologic survey for infectious canine hepatitis virus in grizzly bears (*Ursus arctos*) from Alaska, 1973 to 1987. Journal of Wildlife Diseases 25: 568–573.
- , R. GAMBLE, R. A. HECKERT, AND J. VER HOEF. 1997. Serologic survey for *Trichinella* spp. in grizzly bears from Alaska. Journal of Wildlife Diseases 33: 474–479.
- , J. P. DUBEY, J. M. VER HOEF, M. E. MCNAY, AND O. C. H. KWOK. 2001. Serologic survey for *Toxoplasma gondii* in lynx from Interior Alaska. Journal of Wildlife Diseases 37: 36–38.
- , J. SALIKI, A. MACMILLAN, S. BREW, AND J. M. VER HOEF. 2006. Serologic survey for *Brucella* spp. bacteria phocid herpesvirus-1, phocid herpesvirus-2, and phocine distemper virus in harbor seals (*Phoca vitulina richardsii*) from Alaska, 1976–1999. Journal of Wildlife Diseases 42: 290–300.

Received for publication 14 January 2002.