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THE POPULATION DYNAMICS OF BRUCELLOSIS IN THE YELLOWSTONE NATIONAL PARK[1]

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Abstract. The role that pathogens play in structuring ecological communities needs to be examined from both a theoretical and empirical perspective. This paper provides a brief introduction to the ecology and epidemiology of brucellosis in Yellowstone National Park. Observed patterns of infection for brucellosis in bison in national parks in the United States suggest that *Brucella* has a relatively sharply defined threshold for establishment and that the proportion of the host population infected increases as a relatively simple function of population density. Unfortunately, the threshold population for *Brucella* establishment in bison is low, a herd of at least 200 individuals; this makes it very hard to eradicate *Brucella* from wild populations. Simple calculations based on observed prevalence and recruitment data for the Yellowstone bison herd suggest that unacceptably high levels of culling would be required to eradicate brucellosis in the park.

A simple mathematical model is used to describe aspects of the population dynamics of *Brucella*. The analyses suggest that an understanding of infectious disease dynamics is crucial to the management and conservation of wild and domestic ungulate species in and around national parks and other nature reserves. The consequences of disease control for the maintenance of biodiversity in other natural and captive communities need to be better understood.

Key words: bison; Brucella; *cattle; disease ecology; elk; epidemiology; pathogen; population dynamics; Yellowstone.*

INTRODUCTION

Ecologists are beginning to show interest in models for pathogens that infect more than one host species. This work develops from the earlier studies of Holt and Pickering (Holt and Pickering 1985, Begon et al. 1992) and Anderson and May (Anderson and May 1986) and has more recently been developed by Begon, Bowers, and their colleagues (Begon et al. 1992) and Hochberg and Holt (1990). Less attention has been paid to the role that pathogens play in structuring ecological communities (Anderson and May 1986, Dobson and Hudson 1986). This lacuna is unfortunate as pathogens that infect both wildlife and domestic animals will cause problems around the edge of parks and wildlife reserves where wild and domestic host species interact (Dobson and May 1986, Dobson and Miller 1989). The transmission of pathogens between host species with different evolutionary histories of exposure to pathogens will thus create a special class of problems that conservation biologists interested in disease will have to face.

Yellowstone National Park was the first national park in the United States. The park contains nearly a complete fauna for the Northern Rocky Mountain region. It is situated in the north-western corner of Wyoming, and the greater Yellowstone ecosystem extends into Montana and Wyoming. The presence of *Brucella* in bison in Yellowstone National Park is a cause of concern to the cattle owners in Montana, Wyoming, and Idaho. If bison or elk move out of the park in winter it is important to know if they can transmit *Brucella* to cattle. At present a hazing and removal operation is used in areas to the north and west of the park whenever bison herds cross the park boundaries and encroach on neighboring grazing lands.

In this paper we use a mixture of long-term ecological data and mathematical models to examine the epidemiology of brucellosis in the Greater Yellowstone ecosystem. The paper emphasizes not only how pathogens affect the ecology of wild species, but also how ecological considerations can help in the development of epidemiological models.

[1] For reprints of this Special Feature, see footnote 1, page 989.

History of Bison and Brucellosis in Yellowstone

The original herds of bison in the great plains east of the Rocky Mountains were reduced to a herd of ≈25 animals within Yellowstone National Park by the beginning of the present century. With protection, the wild bison increased and intermixed and interbred during summer with captive bison brought to the park in 1902. These bison formed two distinct wintering subpopulations; a third formed after 71 bison from the managed subpopulation were released in 1936 on historic, but vacant, winter range (Meagher 1973). The three subpopulations remained distinct in winter until the early 1980s (Meagher 1973, 1989). The subpopulations were managed variously, with population control a regular occurrence only on the northern range. Population regulation by human interference ceased in 1966 (Meagher 1973).

Detailed records of bison numbers exist for most of the last 90 yr (Fig. 1A). The size of the population has been increasing steadily for the last 20 yr; and total population size has varied over two orders of magnitude at different times this century. Similar data are available for elk; the estimated size of the Northern Yellowstone elk population between 1920 and the present is illustrated in Fig. 1B (Huston 1982, and Yellowstone park records). The major determinants of population size over this period have been management removals, hunting pressure, and variability in climate (Huston 1982). After removals ceased in 1968, elk numbers increased rapidly and the population now numbers around 18 000–20 000 elk (J. Mack, *personal communication*).

Brucellosis in bison and elk

Brucellosis is a disease of ungulates caused by bacteria in the genus *Brucella.* It has been present in Yellowstone National Park in Wyoming for over 75 yr (Thorne et al. 1991); it was first diagnosed serologically in the park in 1917 from two bison that aborted their fetuses (Mohler 1917). Since then, the bison and elk herds have been tested opportunistically, and occasionally systematically, for the presence of brucellosis (Meyer 1992, Meagher and Meyer 1994, Meyer and Meagher 1995). *Brucella abortus* is essentially a pathogen of the reproductive tract, but most transmission occurs directly by the licking of aborted foetuses and grazing contaminated forage.

There are pronounced differences in the levels of pathology associated with brucellosis in elk, bison, cattle, and other hosts. Among experimental elk, ≈50% of infected cows aborted their first calf, and sometimes the second (Thorne et al. 1978); this may approximate the situation on the elk feed grounds in Wyoming. In contrast *B. abortus* in the Yellowstone bison does not appear to cause any discernible pathology (Meyer and Meagher 1995). Indeed, as the bison herd is growing at close to its maximal rate, the impact of the pathogen on fecundity would seem to be minimal. In contrast, brucellosis produces abortion in both elk and bison in the herds present on the National Elk Refuge adjacent to Grand Teton National Park to the south of Yellowstone. The winter feed ground for elk produce very high densities of hosts at the crucial time of year when *Brucella*-induced abortions occur. Infected animals are likely to have been exposed to high dosages of the pathogen and this may significantly increase the observed pathology (Meyer and Meagher 1995). In moose, brucellosis apparently is almost always fatal for the infected animal (Moore 1947). Efforts to detect differences in causative strains of *B. abortus* biovar 1 have so far proved futile (Meyer and Meagher 1995). It would appear that virulence differs depending upon the host species and the intensity of exposure.

The serological tests used to determine the presence of *Brucella abortus* in elk and bison were originally developed for cattle. The tests are fairly reliable when applied to cattle. Unfortunately, they do not extrapolate readily to bison and elk. In particular, the tests tend to give large numbers of false positives and negatives. Thus serological tests on blood may indicate the presence of *Brucella* antibodies in bison, but it is only possible to positively identify an animal as infected if positive cultures are grown from tissue collected from that animal (Meyer 1992). Estimates of the incidence of *Brucella* should thus be treated with extreme caution unless accompanied by data from tissue culture.

A number of sets of data are available that describe the possible levels of *Brucella* infections in the Yellowstone bison herd; both serological and culture data have been collected from the bison at different times between 1917 and 1992 (Meyer 1992, Meyer and Meagher 1995). These historical data are complemented by data obtained opportunistically during two recent removals of bison on the boundary of the park in the winters of 1988–1989 (Pac and Frey 1991) and 1991–1992 (Meagher and Meyer 1994, Meyer and Meagher 1995). In the 1991–1992 survey, tissue from the same individuals were analyzed using both serological tests and culture results. These data provide an important opportunity to determine the sensitivity and specificity of the serology test and to compare matching rates between the serology and culture results.

The observed prevalence of sero-reactors in different historical surveys in Yellowstone is illustrated in Fig. 2. These surveys suggest that sero-prevalence has varied between ≈20 and 65–70%. Culture tests indicate a prevalence of ≈10%. This suggests that estimates of

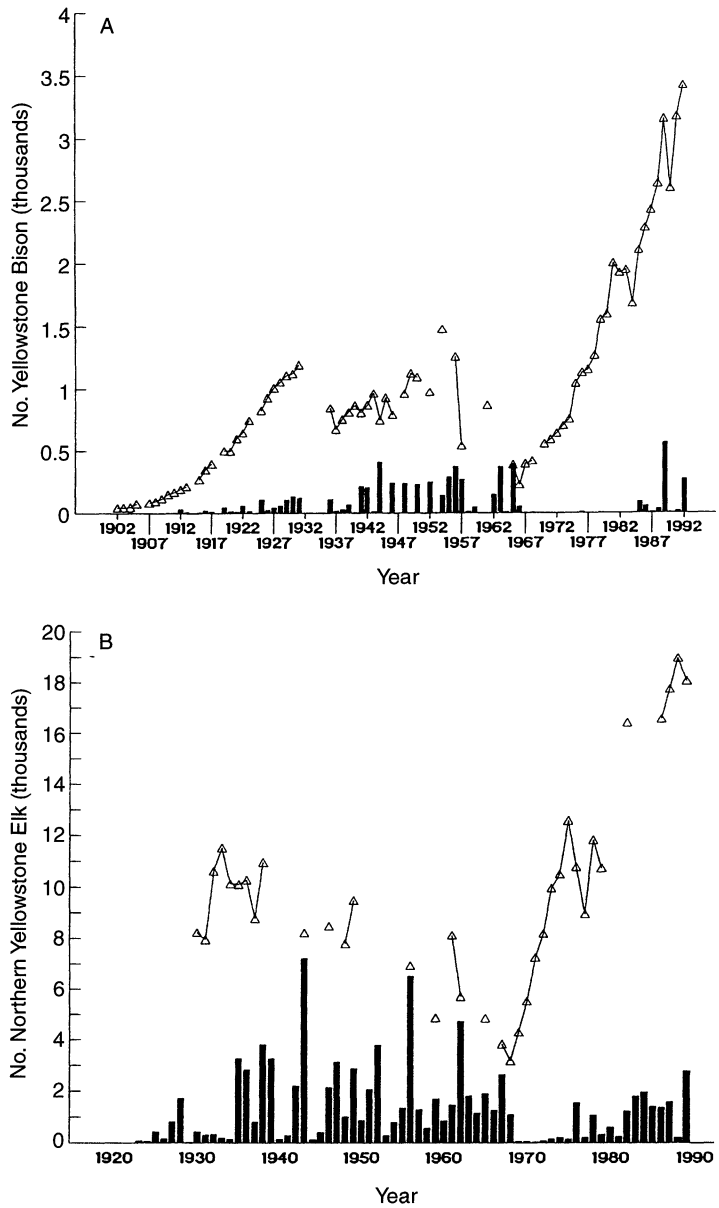

FIG. 1. (A) The size of the Yellowstone bison population since 1915 (after Meagher 1973, 1989). (B) The size of the Northern Yellowstone elk population since 1920 (after Houston 1982, and Park records). The open triangles give total population size; the vertical bars indicate the numbers of animals removed by culling.

Brucella prevalence based on sero-reactors give a significant overestimate of the true level of infection.

Relationship Between Population Size and Brucellosis Serology

The data on population size and *Brucella* sero-prevalence can be combined to examine the relationship between population size and brucellosis sero-prevalence. The data in Fig. 2 illustrate the proportion of samples that are sero-positive at different times during this century plotted against population size for the Yellowstone bison herd. Brucellosis is present whenever the herd size exceeds ≈200 animals and sero-prevalence rises slowly as buffalo numbers exceed this threshold for establishment. Data from similar surveys of bison populations in other national parks in the United States and Canada are also included in this figure (Moore 1947, Choquette et al. 1961, Tessaro 1986, 1989); they suggest a similar trend underlies the data for each herd. The pattern complements those observed

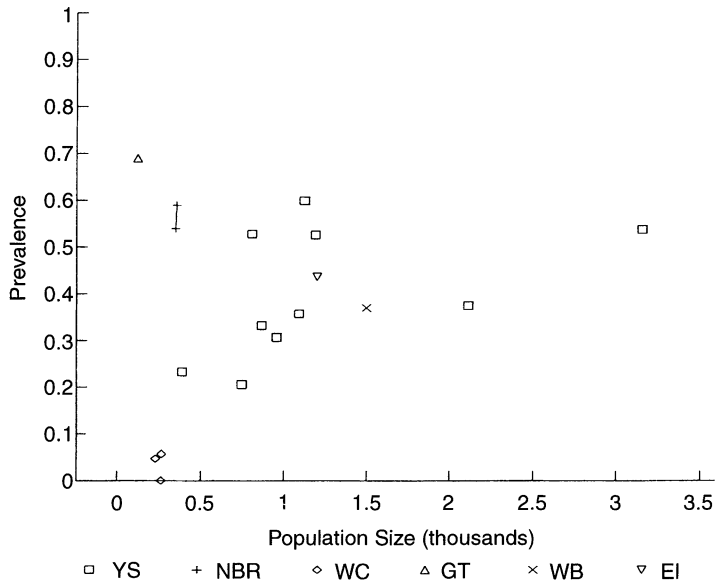

FIG. 2. The relationship between sero-prevalence of *Brucella* and herd size for bison populations in Yellowstone and other national parks. The data used to produce this figure were obtained from bison herds in Yellowstone (YS), the National Bison Range (NBR), Wind Cave National Park (WC), Grand Teton National Park (GT), Wood Buffalo National Park, Canada (WB), and Elk Island National Park, Canada (EI) (after Moore 1947, Choquette et al. 1961, Tessaro 1986, 1989, Tessaro et al. 1990, Pac and Frey 1991, Meyer 1992, and A. Cain [Grand Teton National Park], N. J. Hayes [Wind Cave National Park], and S. Malcolm [National Bison Range]).

in studies of measles in human populations where populations of ≈500 000 people are required to continuously sustain infections with measles (Bartlett 1960, Black 1966). Sustained infections of brucellosis require bison herds in excess of 200–300 animals. Once a herd drops below this number brucellosis tends not to be present.

The major exception to the observed general trend in the relationship between sero-reactors and population size is the case of bison in Grand Teton National Park, which is immediately to the south of Yellowstone. The bison in this park were introduced 20 yr ago from a stock of uninfected individuals. They have acquired brucellosis from elk on the adjacent National Elk refuge at Jackson, Wyoming. Brucellosis is endemic in the elk in the Jackson herd due to the concentration of elk on the winter feed grounds (Boyce 1990). Enhanced *Brucella* transmission from elk occurs when bison mix with elk on these feed grounds, where they are likely to acquire a high level of exposure to the infective stages of *B. abortus*.

THE WINTER REMOVALS OF 1988–1989 AND 1991–1992

In two recent winters movements of bison from the park into the areas north of Gardiner and west of West Yellowstone have led to a large removal operation, under the authority of Montana Fish, Wildlife, and Parks. These removals are controversial, but deemed necessary to prevent transmission of *Brucella* to domestic livestock. The removals provide important data on the epidemiological status of the bison herd. Where bison are removed lethally, it is important that all of the potential information be gathered and analyzed from as many different perspectives as possible. The two large removals in the winters of 1988–1989 (Pac and Frey 1991) and 1991–1992 (Aune and Schladweiler 1993) provided a wealth of epidemiological data. In 1988–1989 the only test to be undertaken was the serology test. In contrast, in 1991–1992, ≈500 animals provided serum for a serology test and a subsample of these also provided tissue that could be examined using a culture test for brucellosis. This allows positive identification of infected carcasses that harbor the organism. These additional data allow evaluation of the specificity and sensitivity of the serology test and more accurate classification of animals into infected and exposed individuals.

AGE-PREVALENCE PROFILES FOR 1988–1989 AND 1991–1992

A large proportion of the samples were taken from animals for which age and sex could be determined. This allows us to examine the change in prevalence of both sero-reactor animals and culture-positive animals with age. These data are represented in Fig. 3. Both sets of data show an increase in exposure to brucellosis with age and both suggest that *Brucella* exhibits a higher level of exposure in males than in females. The slower rate of increase in the second survey may reflect the lowered force of infection due to the impact of the previous cull on bison numbers (approximately one-sixth of the herd was removed in the 1988–1989 cull). However, the difference may also reflect sampling from a different section of the population, so no major in-

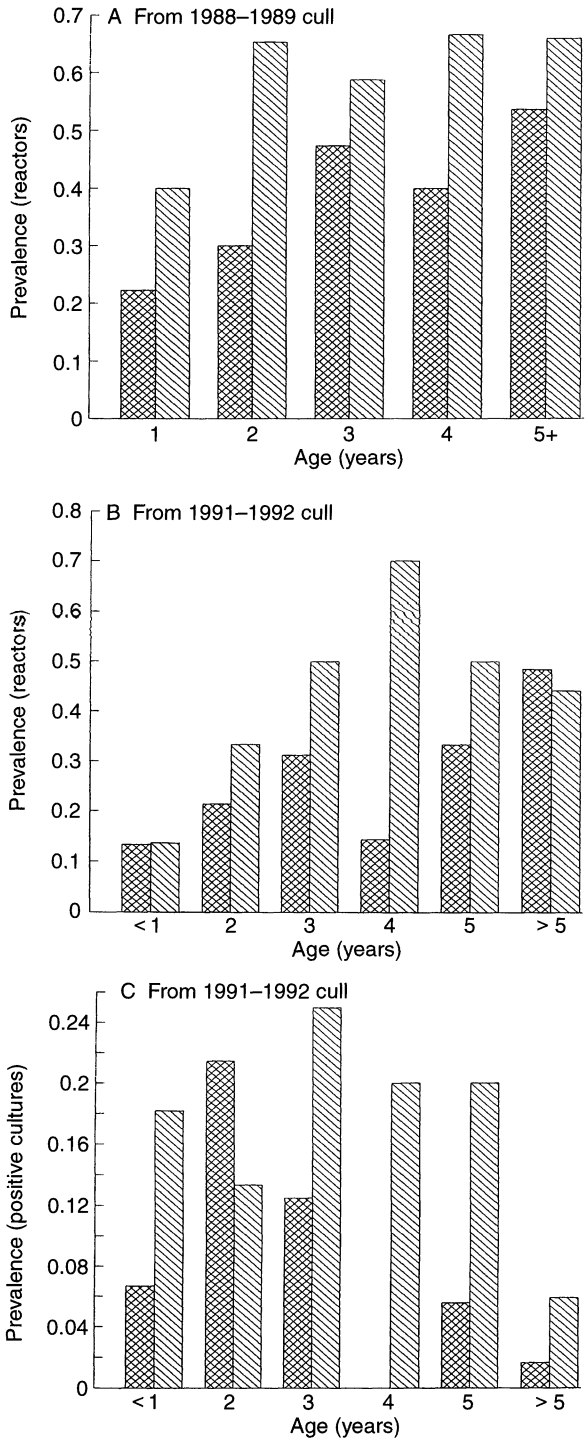

FIG. 3. (A) The relationship between age and sero-prevalence for male and female bison culled in the winter 1988–1989. (Data from Pac and Frey 1991.) (B) Age–prevalence profiles for male and female bison collected on the Northern boundary of Yellowstone National Park in the winter of 1991–1992. The relationship between age and culture prevalence for male and female bison culled in the winter 1991–1992. (Data from Aune and Schladweiler 1993.) In each age class data for females are given on the left, males on the right.

TABLE 1. A comparison of the specificity and sensitivity of the serology test using data from the 1991–1992 winter control operation. The data are arranged so that the individuals that were positive (+ve) or negative (−ve) in both tests are along the leading diagonal of the array. Those that were positive in one and negative in the other form the off-diagonal elements. The culture test provides a highly accurate test of whether an animal is infected. This has allowed us to calculate the sensitivity, specificity, positive predictive value, and matching rate between the two tests.

	Culture test		
Serology test	−ve	+ve	Total
−ve	112	2	114
+ve	80	24	104
Total	192	26	218

Notes: Sensitivity (% true positives) = 92%; specificity (% true negatives) = 58%. Matching rate = 62%. Positive predictive value = 23%.

sights may be obtained from these data as to the efficacy of culling as a control technique.

"TRUE" RELATIONSHIP BETWEEN PREVALENCE AND POPULATION SIZE

The culture profiles are only available for the 1991–1992 winter (Fig. 3C). These data suggest that the prevalence of infection with brucellosis is much lower than is suggested by the serology profiles. Furthermore, the culture test data show no increasing trend with age. Instead, the majority of the infected animals are aged 1, 2, and 3 yr. This clustering of culture-positive animals in the younger age classes suggests that animals that test sero-positive are either infected animals or individuals that have been infected and recovered, or are resistant animals that have merely been exposed to *Brucella.*

Table 1 compares the sensitivity and specificity of the two tests, assuming that the culture test is 100% accurate. Sensitivity is an estimate of the percentage of infected animals actually identified by the serology test, while specificity is the percentage of negative uninfected animals accurately identified by the test. This analysis suggests that although the serology test fairly accurately identifies infected animals (92%) it gives a large number of false positives. The matching rate between the two tests is only 64% with the seroculture test giving three times as many false positives as true positives. This indicates that the proportion of real positives to sero-positive animals is on the order of 23%. This suggests that the true prevalence of infected bison in Yellowstone is more on the order 10–15%, rather

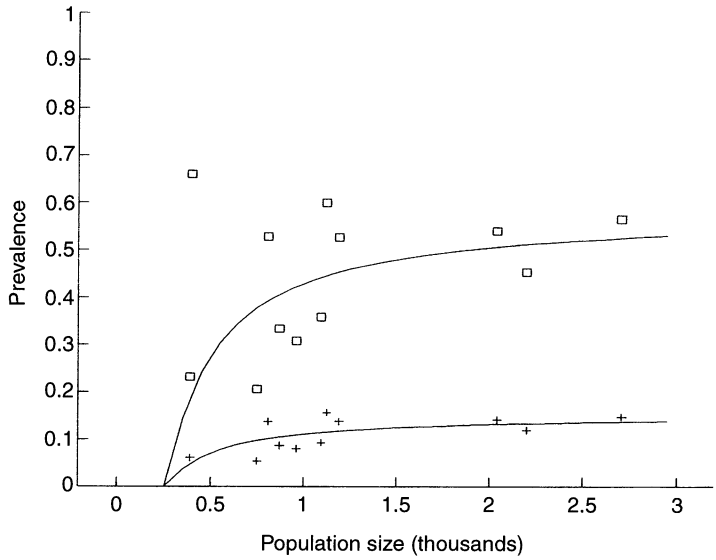

FIG. 4. The relationship between bison population density and sero-prevalence and culture prevalence. The square symbols give the observed data for sero-prevalence, the crosses give the corrected estimates for infected animals calculated using the data presented in Table 1. The curve gives the least-squares fit for the relationship between prevalence and population density for the SIR model presented in *Mathematical models for brucellosis in Yellowstone.*

than the 40–60% suggested by the serology samples. When this correction factor is used to calibrate the historical serology data we again predict a threshold herd size for brucellosis establishment on the order of 200 bison, but prevalence of infectives only rises to ≈10–15% as herd size increases (Fig. 4).

None of the females examined in either year showed any evidence of reproductive tract lesions that might reflect abortion due to brucellosis (Meyer and Meagher 1995). This suggests the pathogen is relatively mild toward bison. Furthermore, the absence of any tendency for prevalence to decrease with age for either culture or serology data suggests that there is no significant mortality associated with brucellosis in bison.

SURVEYS OF *BRUCELLA* PREVALENCE IN ELK

Some serology data are also available for elk carcasses collected in the Northern Yellowstone ecosystem (Table 2). Less than 1% of elk tested positive for brucellosis. There are insufficient data to produce an age–prevalence relationship for elk, but the very low incidence suggests that *Brucella* may not be endemic in the elk population. Instead, the infection seems to have been either acquired serendipitously from interactions with other sources of infection, or alternatively, the large natural concentrations of elk that occur in winter may maintain low levels of infections. This contrasts with the case for elk in the National Elk Refuge where brucellosis is endemic in the elk that use the winter feed grounds adjacent to Grand Teton National Park (Boyce 1990). Infected elk on the Northern range of Yellowstone are likely to acquire their infections from either infected bison or elk infected on the Wyoming feed grounds, or when aggregated together in large herds in winter.

TABLE 2. Results of two surveys in Montana for brucellosis in elk between 1960 and 1968 and in the winter of 1991–1992. The 1960–1968 data are from W. J. Barmore, Jr. (personal communication). The data from 1991–1992 were supplied by Keith Aune of Montana Fish, Wildlife, and Parks Department (Aune and Schladweiler 1993).

Species	Sample size	% positive
1960–1968 (W. J. Barmore, Jr., personal communication)		
Elk	988	1.32
Winter 1991–1992 (Aune and Schladweiler 1993)		
Elk	3268	0.67
Mule deer	575	0.00
White-tailed deer	708	0.14
Bighorn sheep	225	0.88

Although the prevalence of brucellosis in elk is low, there is a large population of elk on the Northern range (Houston 1982). The potential of infected elk as a threat to cattle may be examined by comparing the numbers of infected elk with the numbers of infected bison. These estimates can be made by multiplying the prevalence of infection in each species by the number of individuals estimated to be in each population. This simple calculation suggests that the numbers of infected elk (200–500) on the Northern range of Yellowstone are similar to, or greater than, the numbers of infected bison (350–450). This implies that elk are as great a threat to cattle as are bison.

MATHEMATICAL MODELS FOR BRUCELLOSIS IN YELLOWSTONE

A simple model for *Brucella* in bison, elk, or cattle can be constructed using the standard SIR (susceptible–infected–exposed and resistant) framework for an in-

fectious disease (Anderson and May 1991). This framework assumes that the host population can be divided into individuals that are susceptible, *S,* infected, *I,* and exposed and resistant, *R.* Models of this type have been widely applied to increase our understanding of infectious diseases of both humans (Anderson and May 1991) and wildlife (Grenfell and Dobson 1995).

Initially, let us consider a model for the dynamics of brucellosis in a single host population. The model assumes that calves born to uninfected mothers are brucellosis free. Susceptible individuals, *S,* may acquire infection from contact with infectious individuals, *I,* or a proportion of calves born to infected mothers will also be infected. There is thus some vertical transmission in the model. Here we also assume that infected mothers may exhibit some loss of fecundity, that transmission and pathogenicity of *Brucella* are low, and that transmission can be described by a simple mass action term β. This transmission rate can be either directly dependent upon the density of infected and susceptible animals, or upon the relative density of infected and susceptible individuals (Antonovics et al. 1995, Meyer and Meagher 1995, DeLeo and Dobson 1996).

Infected individuals are assumed to maintain the infection for ≈1–2 yr, when they recover and enter a resistant or immune class of hosts. The model can be expressed algebraically using three coupled differential equations:

$$dS/dt = (a - \phi N)[S + R + I\rho(1 - e)] - bS + \delta R - \lambda(I) \quad (1)$$

$$dI/dt = \lambda(I)S + I(a - \phi N)e\rho - (\alpha + b + \nu)I \quad (2)$$

$$dR/dt = \nu I - (b + \delta)R \quad (3)$$

$$N = S + I + R. \quad (4)$$

The parameters used in the model are host birth rate, a (bison 0.26, elk 0.25); host death rate, b (bison 0.1, elk 0.15); ϕ, density-dependent reduction in host births; β, transmission rate of *B. abortus*; ϵ, proportion of infected females that produce infected offspring; ρ, reduction of fecundity in infected individuals; ν, recovery rate of infected individuals ($1/\nu \rightarrow 2$ yr); α, virulence (increase in mortality rate of infected hosts); δ, rate of loss of resistance. Estimates of their magnitude were made from studies of bison and elk demography (Meagher 1973, Houston 1982) and published epidemiological studies on brucellosis (Nicoletti 1980, Witter 1981). We have assumed that the bison herd will equilibrate at ≈4500 individuals (thus $\phi = 0.00004$), that elk may equilibrate at 25000 individuals (thus $\phi = 0.000004$); and that a high proportion of infected females will produce infected calves ($\epsilon = 0.9$) and that 50% of infected females fail to produce a calf ($\rho = 0.5$). There is very little evidence for increased mortality of bison or elk infected with *Brucella.* We have therefore set $\alpha = 0.005$ for each species. We also assume that an infected animal may transmit the disease for ≈2 yr ($\nu = 0.5$). As with any epidemiological study, estimating the transmission rate poses the greatest problems. We have assumed that the rate at which animals transfer from the susceptible to the infected class, the "force of infection" $\lambda(I)$, can take one of two forms that correspond to the two main types of transmission. When the probability of an animal becoming infected is a function of the density of infected individuals in the population then $\lambda(I) = \beta I$. In contrast, frequency-dependent transmission will occur when transmission is a function of the proportion of individuals infected, in this case we use $\lambda(I) = \beta I/N$. Because the threshold for establishment seems to be a herd size of between 200 and 500 individuals we can coarsely estimate transmission rate in the density-dependent case by deriving an expression for the threshold for establishment.

$$H_T = \frac{\alpha + b + \nu - \rho \cdot a \cdot \epsilon}{\beta}.$$

Substitution of our initial parameter estimates into this equation suggests that β in the density-dependent case is in the range 0.001–0.0005. Alternatively, DeLeo and Dobson (1996) have recently demonstrated that the transmission rates in models for different pathogens will scale allometrically with the body mass of the host species. Their work suggests that where transmission is density dependent, a good estimate of the minimum transmission rate necessary for the pathogen to establish, β_{min} is $\beta_{min} = 0.0247w^{0.44}$; in contrast, in the case of frequency-dependent transmission $\beta_{min} = 0.4w^{-0.26}$, where w is the mass (in kilograms) of the host species. When we rescale these transmission rates to obtain estimates for transmission rates for bison herds on the entire Northern range we obtain values of $\beta \approx 0.002$ for density-dependent transmission, and $\beta \approx 2.0$ for frequency-dependent transmission. The two different versions of the model may then be used to run numerical simulations for brucellosis in the bison population (Fig. 5). Both simulations broadly capture the essential features of the interaction, however the frequency-dependent case produces levels of prevalence that more closely resemble those observed in the serological surveys. In contrast to the basic SIR model where disease prevalence is dependent upon population density, in the frequency-dependent transmission case, the proportion of the population infected is the same for all population sizes.

The dynamics of the model are very stable for the broad range of parameter values that correspond to brucellosis in wild and domestic ungulates. The pronounced stability of the interaction is primarily due to the long period of time for which animals are infected with the pathogen.

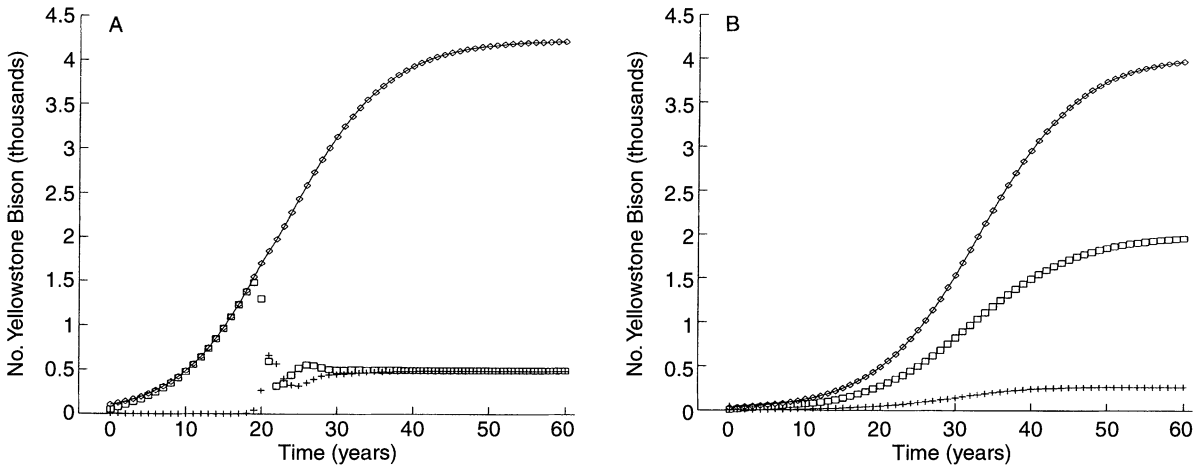

FIG. 5. Dynamics of model for bison in Yellowstone when *Brucella* is introduced at a population size of ≈100. The figure illustrates the total size of the bison population over a period of 50 yr, assuming no removal and an ecological carrying capacity in the park (in the absence of winter roads) of ≈4500 bison. In both graphs the total population is illustrated by the diamonds joined by a solid line, the numbers of susceptibles are shown as squares and the number of infecteds by the crosses. (A) corresponds to frequency-dependent transmission $\lambda(I) = \beta I/N$, (B) illustrates the case for density-dependent transmission $\lambda(I) = \beta I$.

Reducing this time period to a value that corresponds to weeks rather than years can produce epidemic outbreaks in the model (Anderson and May 1991). There is no evidence that this occurs with brucellosis. Introduction of the pathogen into a growing herd of bison results in a steady spread of the pathogen through the population, with ≈10% of the population being infected at any time and a further 30% of the population exhibiting a positive serology test reflecting past exposure to the pathogen (Fig. 5). The dynamics of the model are most sensitive to the equilibrium population density at which the herd would equilibrate $(a - b)/\Phi$, the transmission rate β, and the pathological impact of the pathogen on host fecundity ρ, and mortality α.

Two-species Brucellosis Model

The model can be readily extended to include a second species of host:

$$dS_i/dt = (a_i - \phi_i N_i)[S_i + R_i + I_i\rho_i(1 - e_i)] + \delta_i R_i - b_i S_i - \beta_{ii} S_i I_i - \beta_{ij} S_i I_j \quad (6)$$

$$= \beta_{ii} S_i I_i + \beta_{ij} S_i I_j + I_i(a_i - \phi_i N_i)e_i\rho_i - (\alpha_i + b_i + \nu_i)I_i \quad (7)$$

$$dR_i/dt = \nu_i I_i - (b_i + \delta_i)R_i. \quad (8)$$

Here the β_{ii} terms represent within-species transmission and the β_{ij} terms reflect between-species transmission. As with any epidemiological model the main empirical problem is determining how to quantify transmission rates. This problem is greatly compounded in multihost models. We have attempted to reduce this inherent complexity by assuming that transmission has both an ecological and a physiological component, but that between-species transmission is dominated by the ecological component. Rates of transmission between species will thus be determined by the amount of range overlap and by the tendency of the different species to aggregate together while foraging and resting.

Range overlap for bison and elk in Yellowstone was quantified using data provided by the Yellowstone GIS (Geographic Information System) laboratory. The analysis suggests that only ≈7% of the elk winter range is occupied by bison. This implies that low levels of range overlap between the two species may be responsible for the low levels of prevalence observed in elk. Obviously, this result could be considerably biased if the species tend to aggregate together on the parts of their range that they both jointly occupy. Table 3 provides estimates of the density of bison and elk on their winter ranges in Yellowstone and in the National Elk Range in Jackson. Although overall elk and bison densities in Yellowstone are about the same, the density of elk in Jackson is a factor of 10 higher than in Yellowstone. Furthermore, the elk are highly aggregated in winter on feed grounds in the elk refuge. As the Jackson elk maintain endemic brucellosis infections at high levels of prevalence, this implies that the Yellowstone elk would have to increase considerably before they too could support *Brucella* at the levels of prevalence observed on the National Elk Refuge. As it is, elk are only likely to transmit *Brucella* when they are temporarily aggregated together in large herds during the winter.

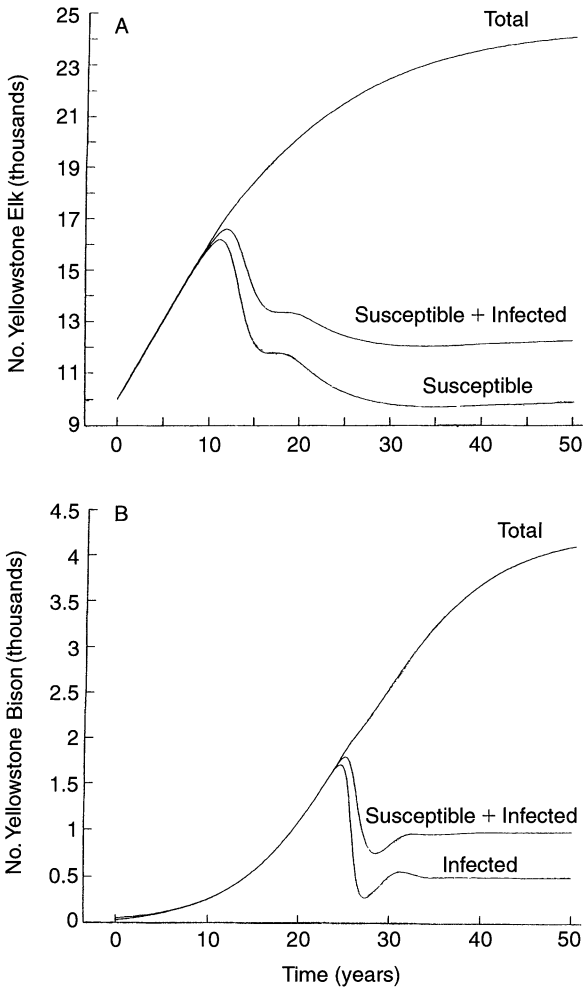

FIG. 6. Dynamics of model for basic SIR model for brucellosis in bison and elk in Yellowstone. (A) illustrates the total elk population and the number of susceptibles (lower line) and susceptibles plus infected (middle line). Similar data are presented in (B) for the bison population.

TABLE 3. Estimates of the average density of bison and elk in 1970 and 1990 in Yellowstone National Park and on the National Elk Refuge in Jackson, Wyoming. The data for bison and elk numbers were taken from Meagher (1973, 1993) and Houston (1985); the data on range size were taken from Boyce (1989).

	Bison (no./ha)			Elk (no./ha)	
Time of estimate	Lamar/ Black-tail	Pelican	Firehole/ Madison	Northern Range	National Elk Refuge
1970	0.02	0.01	0.06	0.11	0.90
1990	0.11	0.06	0.37	0.18	1.05

The dynamics of this two-species model are again very stable (Fig. 6). Introduction of a *Brucella*-type pathogen into the bison herds leads to similar levels of prevalence of infected and exposed animals as in the single species case. Levels of infection in the second host species (elk) are substantially lower and infections in elk tend to die out if transmission from bison is reduced. A significant increase in the size of the elk population is required before it can sustain *Brucella* infections at the levels observed in the winter-fed elk from areas south of Yellowstone.

CONTROL OF BRUCELLOSIS

The removal of animals crossing the boundaries of the park is the present policy for bison in the Yellowstone ecosystem. The historical records that detail the relationship among stock, recruitment, and removals, and the relationship between population size and prevalence can be combined to examine the relationship between culling intensity and resultant prevalence (Fig. 7). This analysis suggests one would need to almost eradicate the bison before one could produce significant reduction in prevalence. More significantly the levels of removal required to eradicate *Brucella* may be sufficient to also drive the bison to extinction. Similar results are obtained for the frequency-dependent transmission model where the proportion of hosts infected remains unchanged by random removal of hosts from the population. Although it may be possible to modify

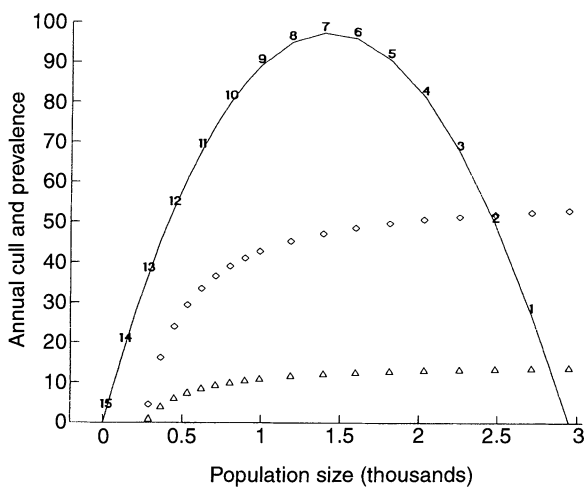

FIG. 7. The relationship among population density, *Brucella* prevalence, and the annual cull required to reduce the bison population to a fixed level. The recruitment curve for the bison population has been obtained by fitting a simple logistic curve to the recruitment data for the bison population depicted in Fig. 1. The figures above the harvest curve give the annual percentage harvest rate necessary to reduce the bison population level to a specified size. △ give the number of infected individuals in the population at this density; ◇ give the numbers reacting to a serology test.

the removal level if only infected animals could be identified and removed, there is no guarantee that *Brucella* would not re-establish unless the bison population were kept at a very low level (<200 individuals). This is unlikely to be acceptable on ecological, ethical, or aesthetic grounds.

Although a *Brucella* vaccine is available for cattle, it has only been used with limited success against elk (Thorne et al. 1981, Herriges et al. 1989) and marginal success against bison (Davis et al. 1991). As brucellosis is likely to have a basic reproductive rate of around unity in Yellowstone, slightly more than 50% of the bison would have to be effectively inoculated if the *Brucella* is to be eradicated by vaccination (Anderson and May 1982, 1985). The low vaccine efficacy in bison and the logistics of having to treat this many animals suggest that it is unlikely that this level of vaccination coverage will be achieved. As neither culling nor vaccination present a viable option for the control of brucellosis in Yellowstone, the only remaining option for ensuring that cattle in the surrounding area remain brucellosis free is to instigate a cordon sanitaire or buffer zone around the park. As *Brucella* is a pathogen of the reproductive system this could most readily be done by only allowing heifers and bullocks to be raised within some suitable distance of the park's boundaries, that corresponds to the maximum distance an infected elk or bison could wander without detection. Any intact cattle entering the buffer zone would require a mandatory vaccination for *Brucella* and significant restrictions should be placed on the movements of cattle leaving the zone.

Conclusions

This paper has described one particular example of a problem that pathogens cause in wildlife communities. These epidemiological problems are likely to become more frequent around nature reserves. The analyses presented here suggest that the best approach to brucellosis control would be to create a cordon sanitaire or buffer zone around the park. This could easily be done by only allowing vaccinated or sterile cattle in areas around the park. There are two alternatives to pay for this program; government subsidies could pay for the brucellosis vaccination scheme in cattle, or, present levels of subsidies could be reduced, or removed, from ranchers who continue to ranch cow–calf herds in this area. A complete transformation to either heifers and steers for an area within 30–80 km around the park should insure that brucellosis is contained within the area of the park.

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