7. SYNTHESIS - TESTING THE DISEASE-PREDATION HYPOTHESIS

There are two main hypotheses to explain the trend in bison abundance in WBNP (Gates et al. 1997). Carbyn et al. (1993) proposed what Gates et al. (1997) called the "habitat dispersion hypothesis" where bison in the Peace-Athabasca Delta are concentrated in large meadow complexes, with high spatial and temporal predictability, resulting in an increased predation risk relative to other areas of the park. The degree of bison concentration may be related to the construction of the W.A.C. Bennett dam in the late 1960s, which is thought to have resulted in a general drying trend, which has reduced the available habitat for bison. In contrast, the "disease-predation" hypothesis contends that the presence of tuberculosis and brucellosis (hereafter collectively referred to as “exotic pathogens”) reduces the productivity of the bison herd so that the population is regulated at low numbers by wolf predation (Messier 1989; Gates 1993, Gates et al. 1997). In an unpublished report to Parks Canada, Messier and Blyth (1995) proposed that the effect of exotic pathogens on the bison-wolf relationship is similar to the effect of bear predation on the moose-wolf relationship proposed by Messier (1994). Specifically, they proposed that in the absence of exotic pathogens, bison population growth rate would exceed predation rate at all densities and therefore bison abundance would be regulated by food competition at high numbers (e.g., line a in Figure 7.1). The presence of bovine tuberculosis and brucellosis would reduce productivity of the bison

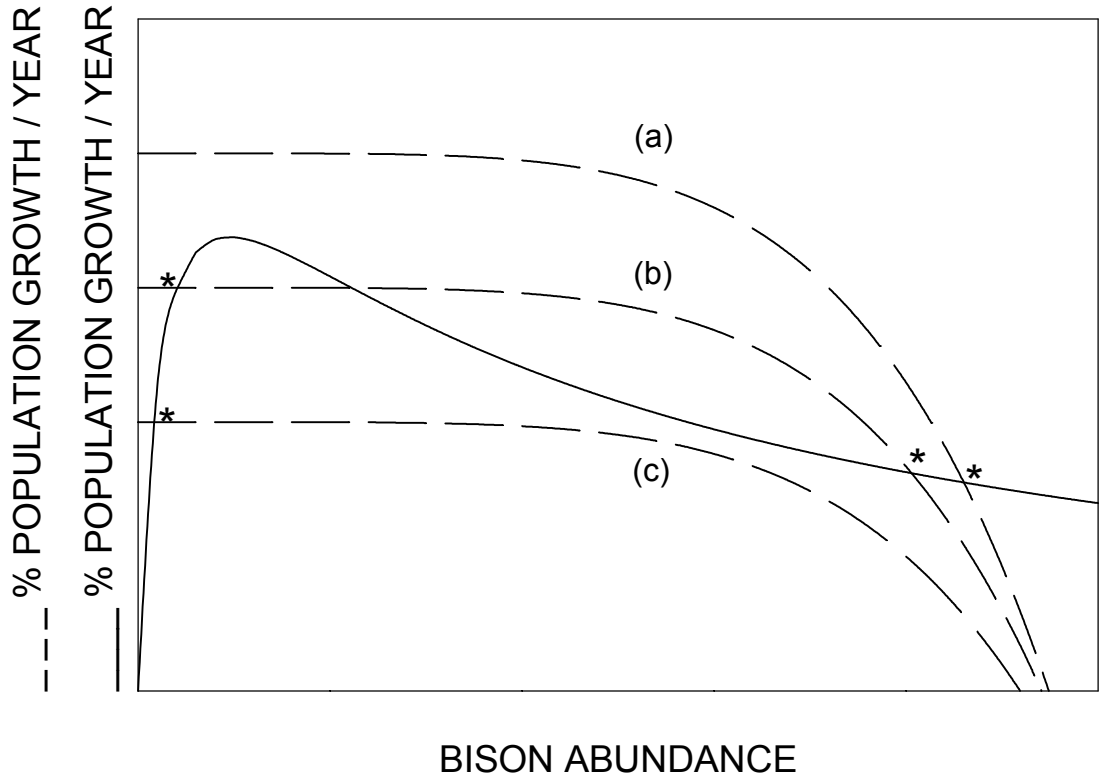

Figure 7.1. The disease-predation hypothesis. In the absence of brucellosis and tuberculosis, bison population growth rate (a) is hypothesized to exceed predation (solid line) at all densities. The presence of brucellosis and tuberculosis is hypothesized to create a multiple stable state system (b) or a single, low density equilibrium (c). Stable equilibria are indicated by an asterix.

population (through reduced reproduction and survival), and consequently bison abundance would be regulated by wolf predation at relatively low densities (e.g., line c in Figure 7.1).

The purpose of this chapter is to evaluate the potential role of tuberculosis and brucellosis in the decline of bison abundance by testing predictions of the disease-predation hypothesis. First, Carbyn et al. (1993: 240; 1998) discounted the role of exotic pathogens in the decline of bison abundance as the decline in numbers of bison south of the Peace River has not been mirrored by a decline in numbers north of the Peace, despite the presence of exotic pathogens in both areas (Carbyn et al. 1993: 240; Carbyn et al. 1998). Carbyn et al. (1993, 1998) discussed the relative population trajectories in "Areas I and II" (north and south of the Peace River respectively). Classifying bison numbers relative to the Peace River resulted in three bison populations (Hay Camp, Little Buffalo, Nyarling River) and part of a fourth and fifth (Garden River, Delta) being included in Area I, while Area II contains both the Delta population and the portion of the Garden River population south of the Peace River (Figure 7.2). Although there is some demographic continuity among these five populations, their population dynamics are driven by intrinsic factors (chapter 3). Combining them into "Areas I and II" may obscure population trends of individual populations. Therefore, we believe there has not been an adequate test of the hypothesis that population declines are most pronounced in the Peace-Athabasca Delta relative to other populations. Following Carbyn et al. (1993: 240; 1998), the disease-predation hypothesis would be falsified if:

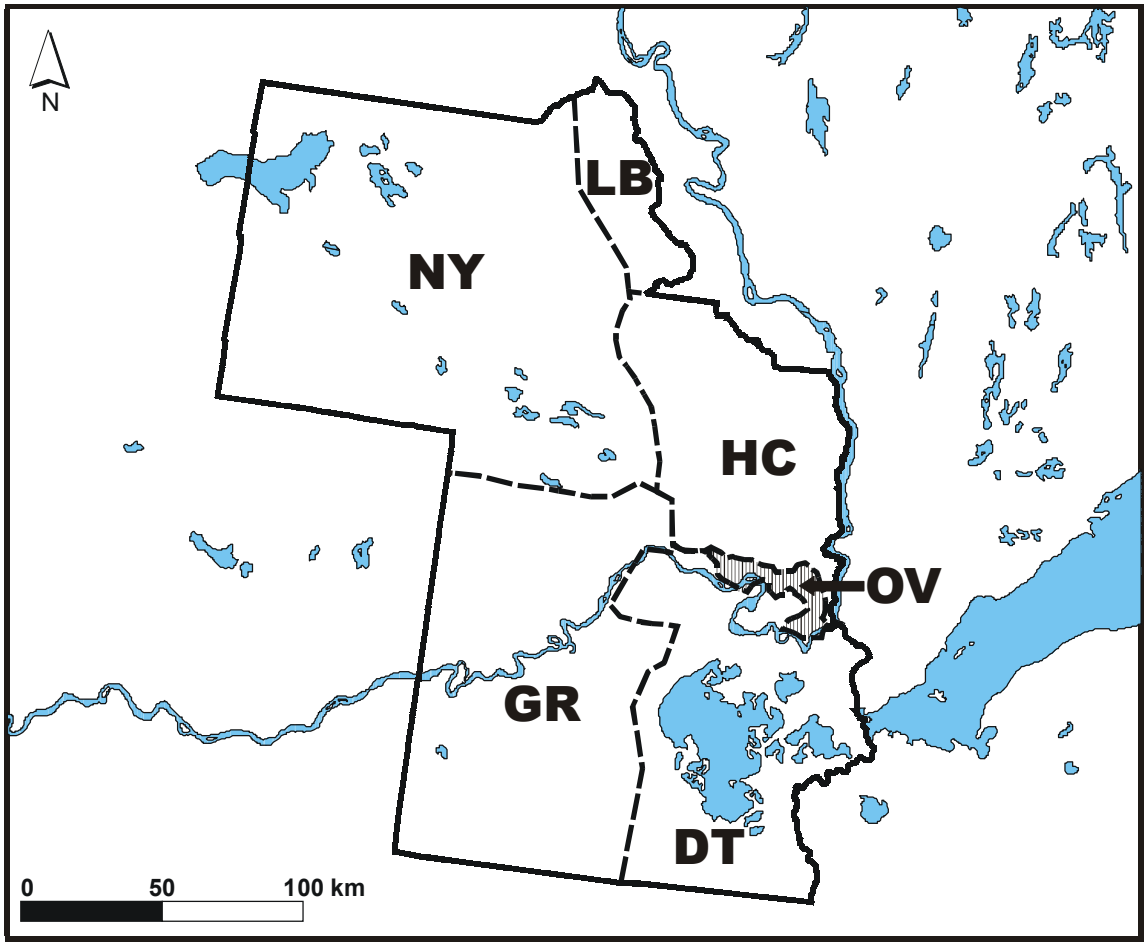

Figure 7.2 Zones to *a posteriori* classify observations of bison during population surveys in Wood Buffalo National Park (1981-1999). Letters indicate the following zones: LB, Little Buffalo; NY, Nyarling River; HC, Hay Camp; OV, Overlap; GR, Garden River; and DT, Delta. Bison in overlap area may be associated with either HC or DT.

H_1: the decline in bison abundance in the Peace-Athabasca Delta is unique within the WBNP metapopulation.

The disease-predation hypothesis predicts that in the presence of exotic pathogen, bison will be regulated at low abundance by wolf predation. Further, this hypothesis predicts that in the absence of exotic pathogens, bison populations will be regulated at high densities by food competition and predation will be a limiting but not regulating factor (Messier 1989; Gates 1993; Gates et al. 1997). However, the disease-predation interaction has the potential to be much more complex. Predation has been traditionally viewed as being a function of the numerical and behavioural (e.g., functional) response of a predator to prey density (Solomon 1949). Messier (1996) reviewed how the proportion of the prey population killed by predators (e.g., predation rate) varies with prey density with various combinations of the simplest forms of each of these responses (linear, hyperbolic, and sigmoidal). When satiation in both killing rate (functional response) and predator abundance occurs, predation rate can be positively and then negatively density-dependent as prey density increases, creating the potential for multiple stable equilibrium states (e.g., line b in Figure 7.1; Holling 1959, 1965; Messier 1994, 1996). Specifically, if predation rate exceeds the growth potential of the prey population at intermediate prey densities (known as a "predator-pit"), prey populations can stabilize at low, predator-regulated densities or high, food-regulated densities. A high-density prey population that is reduced in density by factors other than predation into this predator-pit will shift to a low-density equilibrium.

As wolf numbers and killing rate per wolf satiate at high at high moose density

(Messier 1994; Messier and Joly 2000), I believe that the dual relationship between prey density and predation rate would occur for bison. Thus, in order to examine whether exotic pathogens and predation interacted to cause the decline in bison abundance, it is necessary to determine if bison growth potential exceeds predation at all densities in the absence of exotic pathogens. The following predictions can be made based on the disease-predation hypothesis:

H_2: the maximum predation rate by wolves does not exceed the intrinsic growth rate for bison populations without exotic pathogens; and

H_3: the maximum predation rate by wolves on bison exceeds the intrinsic growth rate for bison populations with exotic pathogens.

Falsification of H_2 would suggest that it may not be possible to distinguish between the disease-predation hypothesis and a more simple predation-only model as the cause of the decline. Falsification of H_3 leads to rejection of the disease-predation hypothesis. However, H_3 itself cannot be used to distinguish between single and multiple stable state systems. Although a predator-pit in the presence of exotic pathogens does not preclude the disease-predation hypothesis, it is critical to evaluate the probability that a high-density bison population with exotic pathogens will shift to a low-density equilibrium due to stochastic variability in survival and reproduction. This is particularly important in light of potential mass mortalities of bison associated with anthrax (Gates et al. 1995; Dragon and Elkin 2001) and drowning (Carbyn et al. 1993). In this chapter, I use stochastic population simulations to evaluate the disease-predation hypothesis.

7.1 Methods

7.1.1 Population trends in WBNP

Bison surveys have taken place in WBNP since the creation of the park (Tessaro 1987). Since the early 1970s survey methods have been relatively standard, and are reviewed by Carbyn et al. (1993: 92-95, 1998). Briefly, aerial surveys were flown annually in February and March. Areas identified as "primary ranges" (principally the Peace-Athabasca Delta and between the Slave River and Pine Lake Road) were systematically searched for bison, resulting in a total coverage for those areas. Flights over "secondary ranges" focused on areas suspected to contain bison. Only primary ranges were surveyed in 1971-1974 and secondary ranges were added for the remaining years. Observations were recorded on maps (1:250,000, NAD 27) as well as on data sheets (Jane Chisholm, WBNP, personal communication; Carbyn et al. 1993: 94).

All total count data presented herein come from three sources (Table 7.1). Tessaro (1987) summarized data from surveys 1971-1974 for bison counted on "primary ranges" identified as "those areas where large proportions of the bison population could be reliably found" (p. 63); although the actual locations of bison observed are not available. For 1975 - 1980 bison numbers were obtained from unpublished Warden Service Reports (available from WBNP, Box 750, Fort Smith NT X0E 0P0). These reports contained descriptions of the location of flightlines and the number of bison counted on each. Based on these descriptions I have classified the counts into the populations described in chapter 3 where possible. However, I could not objectively evaluate the degree to which these classifications are correct for the Garden River,

Table 7.1. Summary of bison total count surveys in Wood Buffalo National Park (1971-1999). Data sources: Tessaro (1987), 1971-1974; WBNP unpublished warden reports (1975-1980); this study (1981-1999). Population growth rate, *r*, was calculated as the slope of the regression between ln-transformed population size and year. Numbers in italics were not used to calculate population growth rates.

Year	DT	HC	OV	GR	LB	NY
71		*9561*	[a]			
72		*9236*				
73		*8062*				
74		*7415*				
75	3351	1428		*458*[b]	*290*[c]	
76	*4967*	*627*		*240*	*227*	
77	3440	1260		*105*	*365*	
78	3602	1083		*374*	*314*	
79	3631	989		*376*[d]	*543*	
80	3450	704		*282*	*76*	
81	3759	1021	33	790	9	22
82	3084	818	21	*73*		
83	3641	787	81	398	33	48
84	3125	639	449	270	144	35
85	3820	321	70	312	58	12
87	2742	495	150	351	6	145
88	3279	423	81	218	71	5
89	2270	520	83	318	118	36
90	1805	652	68	323	56	112
91	1775	505	94	458	3	144

Year	DT	HC	OV	GR	LB	NY
92	1584	245	22	439	4	236
94	1004	382	103	292	43	196
95	1032	477	38	445	23	174
96	1064	621	52	616		173
97	779	686	55	500	35	49
98	421	577	166	674	105	229
99	429	635	117	707	79	184
r	-0.09 (-0.11- -0.07)	-0.03 (-0.06 - 0.02)		0.02 (-0.01 - 0.06)	0.03 (-0.10 - 0.16)	0.13 (0.04 - 0.22)

[a] Bison counted in OV split between HC and DT at the Peace River (1971 - 1980)

[b] GR includes bison counted at Lake One for the years 1975 - 1980, after which they are included in DT

[c] Two new flight lines added

[d] South of Peace River only, and includes 306 bison at Lake One and west of Lake Claire.

Nyarling River and Little Buffalo populations so I have not included data from these years in calculating population growth rates. Further, numbers of bison assigned to the Delta population during this period include only bison counted in the Peace-Athabasca Delta, and exclude bison counted in the Lake One area and the area just north of the Peace River east of Point Providence (Figure 7.2). As these areas are within the range of the Delta population, I have excluded these years from calculation of Delta population growth rate. Descriptions of surveys in the Hay Camp area are clearly consistent with the range of the Hay Camp bison population (i.e., the meadow-complexes extending north from Murdoch Creek to the Hornaday River and including the Salt Plains area), and so I have elected to include these data from this period for the Hay Camp population.

J. Chisholm (WBNP, Fort Smith, NT) conducted an extensive review of bison observations during total counts for the period from 1981-1999 and created a standardized, spatially referenced digital file for analysis. During the 1980s, the park staff that conducted the surveys tended to rely on data recorded on the datasheets to compile reports for each survey, while data recorded on the maps was used to create an electronic file; the two recording methods were not reconciled (Jane Chisholm, WBNP, personal communication). Locations recorded on maps were used as the basis of the standardized digital file whenever possible.

Each bison location was classified into one of the five WBNP bison populations described in chapter 3, as depicted in Figure 7.2. Note that only locations of bison within WBNP park boundaries were included. As the Little Buffalo population range is contiguous with the Slave River Lowland bison population (see Figure 3.5), estimates of the former population represent underestimates. Bison located within the area marked

OV (overlap) cannot be reliably identified as being from the Delta or Hay Camp populations (chapter 3), and so are listed separately and were excluded from analysis. All classification was done in ArcView (Version 3.1, ESRI, Redlands, CA). Instantaneous population growth rates (*r*) for each population were calculated as the slope of a linear regression between population number (ln-transformed) and year, over the period 1975-1999 (Hay Camp population) and 1981-1999 (Delta, Garden River, Nyarling River, and Little Buffalo populations). I considered a population to be decreasing or increasing if the 95% confidence interval for the slope did not include zero. I excluded the 1976 surveys for the Hay Camp population as there appeared to have been a temporary range shift during that year of bison south into the Peace-Athabasca Delta, followed by a return north the following year (Carbyn et al. 1998). Further, in 1982 the range of the Garden River population was only partially surveyed and so that survey was excluded from analysis. The ranges of the Nyarling River and Little Buffalo populations were not surveyed in 1982, and no surveys were conducted in 1993.

7.1.1 Simulating population growth and maximum predation rate

I constructed an age-specific, discrete population model to simulate population growth in the presence and absence of exotic pathogens. I used 20 age and two sex classes, with each age centered at just after the birth season (June 1), assuming that most calves are born in May. All parameter values used in the simulation are listed in Table 7.2.

Table 7.2. Demographic parameters used in the stochastic population projection.

Parameter	Value (SE)		
	Disease[1]	Healthy	Reference
Pregnancy rate, b_i			
Poor body condition	0.62 (0.05)	0.68 (0.04)	Table 5.1
Good body condition	0.73 (0.06)	0.78 (0.05)	Table 5.1
Adult survival, *d*			
Males	0.83 (0.08)	0.93 (0.09)	Tables 6.3 - 6.5
Females	0.85 (0.08)	0.95 (0.10)	Tables 6.3 - 6.5
Drowning mortality	0-0.25	0-0.25	Assumed
Anthrax mortality			
Juveniles (<2 years)	0	0	Gates et al. 1995
Females (>2 years)	0.02	0.02	Gates et al. 1995
Males: 2-3 years	0.02	0.02	Gates et al. 1995
4-6 years	0.09	0.09	Gates et al. 1995
7-11 years	0.25	0.25	Gates et al. 1995
12+ years	0.21	0.21	Gates et al. 1995

[1] bison tuberculosis test-positive and high Brucella titre, see chapter 5.

The population density at the end of each iteration (N_{t+1}) was projected by:

$$M_{t+1} = 0.5 \bullet (\sum_{i=2}^{19} s_j \bullet b_i \bullet F_{i,t+1}) + [M_{0,t} - k_0(M_{0,t})] + \sum_{i=1}^{19} [d_M \bullet M_{i,t} - k_i(M_{i,t})]$$

$$F_{t+1} = 0.5 \bullet (\sum_{i=2}^{19} s_j \bullet b_i \bullet F_{i,t+1}) + [F_{0,t} - k_0(F_{0,t})] + \sum_{i=1}^{19} [d_M \bullet F_{i,t} - k_i(F_{i,t})]$$

$$N_{t+1} = M_{t+1} + F_{t+1}$$

[7.1]

where b is pregnancy rate on March 1 (time of data collection; see chapter 5), s_j is survival of the foetus/calf from March 1 to June 1 (i.e., the complement of late gestation and perinatal mortality, including abortions and nonviable calves due to brucellosis; Cheville et al. 1998; Rhyan et al. 2000), d is the proportion of females, F_i, or males, M_i, (>1 year) surviving from t to t+1, and k_i is the number killed by wolves in each age and sex class. Pregnancy rate was estimated using the best model in Table 5.1. I assumed that 75% of females were in "good" body condition (D.O. Joly and F. Messier unpublished data). I also assumed a 50:50 sex ratio at birth. I simulated pregnancy rate by randomly picking each coefficient in the logistic regression model from a distribution based on observed mean and standard errors. The average pregnancy rate presented in Table 7.2 is the predicted deterministic pregnancy rate, with the standard error was estimated by simulating pregnancy 1000 times and calculating the standard error of that distribution. Female adult survival, d, was assumed to be 0.95 for bison without pathogens (SE 10% of the mean for all survival rates; Gaillard et al. 2000) as I was concerned that simply censoring wolf kills from my data would result in a negatively biased estimate of survival in the absence of predation (i.e, some bison died from undetermined causes and predation could not be excluded). Female adult survival for

bison with a positive tuberculosis test and high Brucella titre were assumed to have an annual survival rate in the absence of predation of 0.85, based on the difference between the predicted annual survival for infected and infection-free individuals (Tables 6.3-6.5). Male survival was assumed to be 0.02 lower than female survival. I assumed that 10% of the population had both a high titre for brucellosis and was positive for tuberculosis. This implies that pathogen transmission is insensitive to density (see chapter 4), and predation is nonselective with respect to pathogen status. Population growth in the absence of exotic pathogens was simulated by assuming all bison had survival and reproduction rates of "healthy" bison (i.e., low brucella titre and/or tuberculosis test-negative).

The population growth rate of large ungulates responds to increases in abundance by reductions in demographic rates in the following order: juvenile survival, age at first reproduction, fecundity of prime-aged females, and finally adult survival, which is relatively insensitive to increasing abundance (Eberhardt 1977; Fowler 1987; Gaillard et al. 1998, 2000). Calf: cow ratios of bison have been shown to decline in response to density (Fowler 1981; Blyth 1995); however, the density at which this occurs is unknown for northern bison populations. For example, summer calf: cow ratios in the Mackenzie Bison Sanctuary were relatively stable even at high abundance (e.g., 1500 – 2000 bison; Larter et al. 2000). I incorporated density-dependence by reducing the parameter s_j in response to density following Fowler (1981):

$$s_j = s_0 - x \bullet N_t^{\,y} \qquad [7.2]$$

where s_0 is juvenile survival at low bison abundance and x and y are parameters that describe the shape of the reduction in juvenile survival with density. There are no data

available on late gestation and perinatal mortality of bison. I assumed that $s_0 = 0.7$ and 0.9 in the presence and absence of exotic pathogens, respectively. The difference is based on the difference between June calf: cow ratios in WBNP and the Mink Lake bison population (Mink Lake is also at low density and exotic pathogens are not present; Larter et al. 2000, Gates et al. 1995). Incorporating density-dependence in this manner assumes that infected and infection-free bison are affected equally by increases in density. The standard error for s_0 was assumed to be 30% of the mean (Gaillard et al. 2000). The parameter y was set at 5 following Eberhardt (1997, 1998) and x was calculated by solving for $s_j = 0$ at $N_t = 2.5$ bison / km^2 ($x = 0.009$). This latter value is from Campbell and Hinkes (1983) and corresponds to 12,500 bison in WBNP assuming 5,000 km^2 of core bison habitat. The true ecological carrying capacity of bison in WBNP is unknown.

Predation by wolves was simulated by dividing the product of the functional and numerical responses of wolves by bison density (e.g., Messier 1994, 1996). I assumed a hyperbolic functional response, as formulated by Real (1977, 1979). Means and standard errors for the asymptotic killing rate (a) and half-saturation constant (b) were estimated by fitting the hyperbolic functional response to data on killing rates of bison by wolves from Carbyn et al. (1993) and Van Camp (1987; number of bison killed per 100 days in winter; Figure 7.3) using a Marquardt-Levenberg algorithm (SPSS 10.05, Chicago, IL). I assumed that this functional response predicts an integrated, rather than instantaneous, killing rate. I scaled the winter killing rate to an annual rate by the product of 3.65 and 0.71 (Messier 1994). I chose to use the hyperbolic over the

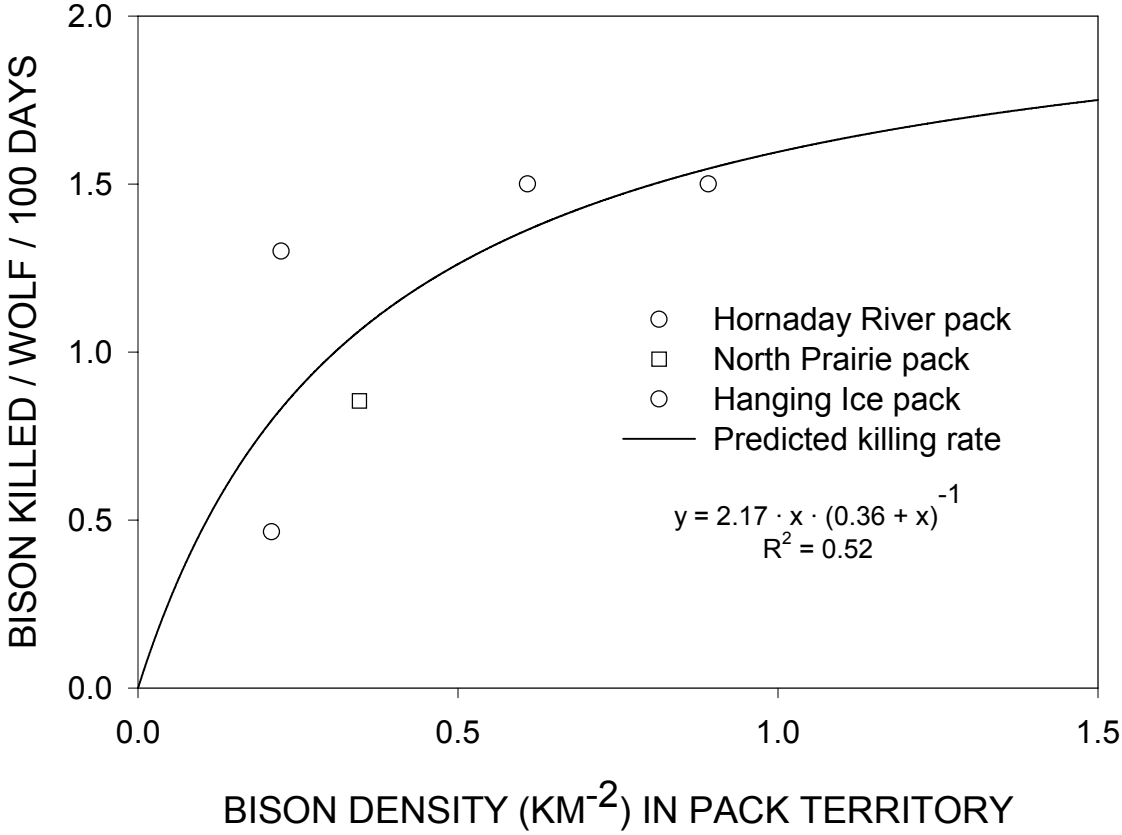

Figure 7.3 The functional response of wolves to changing bison density in winter. Data on killing rate and bison abundance for the Hornaday River wolf pack are from Carbyn et al. (1993: 180, 220). Killing rate and bison abundance data for for the North Prairie and Hanging Ice packs are from Van Camp (1987) and Calef and Van Camp (1987) respectively. I assumed that winter territory size for the Hornaday River pack was 800 km^2 (Carbyn et al. 1993: 170) and estimated winter territory sizes for the North Prairie (520 km^2) and Hanging Ice (230 km^2) packs from Figure 3 in Van Camp (1987). I estimated bison abundance in the North Prairie territory as the number of bison north of transect 14 in 1976 and for the Hanging Ice territory as the number of bison in Area 1 (1976; see Calef and Van Camp 1987).

sigmoidal functional response as the data were insufficient to justify inclusion of a third parameter. I discuss the implications of this decision in the discussion section of this chapter. The mean (SE) for each parameter are: asymptotic annual killing rate, a = 5.54 (2.61) and bison density at half the maximum killing rate, b = 0.36 (0.39) bison / km^2.

Wolves respond numerically to changes in bison abundance (Joly and Messier 2000); however, the form of this relationship is unknown. I assumed a hyperbolic numerical response similar to that presented by Messier (1994). If wolves were not territorial, wolf abundance per unit area could be predicted by:

$$W / A = w_p \bullet N \qquad [7.3]$$

or

$$W = w_p \bullet N \bullet A \qquad [7.4]$$

where W is wolf abundance, A is the unit of area, w_p is a proportionality constant (wolves • prey^{-1}), and N is the number of prey. However, spacing behaviour of wolves will cause the numerical response to deviate from proportionality (e.g., Messier 1994; Messier and Joly 2000) and so assuming an arbitrary area of 1 and minimum territory size of A_{min}, substituting,

$$A = 1 - A_{min} \bullet W \qquad [7.5]$$

into eq. 7.4 and simplifying, results in a hyperbolic numerical response:

$$W = w_p \bullet N \bullet (1 + w_p \bullet A_{min} \bullet N)^{-1} . \qquad [7.6]$$

In this formulation, the wolf: bison ratio w_p is the slope of the numerical response at zero prey density. I estimated w_p by fitting eq. 7.6 to data compiled by Messier (1994) on wolf densities at various moose densities using a Marquardt-Levenberg algorithm (SPSS 10.05, Chicago, IL), and converted this ratio from wolf: moose to wolf: bison by

the factor 1 bison = 1.33 moose (Fuller 1989). The resulting $w_p = 0.099$ (SE = 0.051). I assumed that in the presence of exotic pathogens, the wolf: bison ratio increased by 5% to 0.104 (SE = 0.053) as result of scavenging of bison that died of generalized tuberculosis (approximately 5% of the bison population; Fuller 1962; Tessaro et al. 1990). I assumed the asymptotic wolf density would be 1 wolf per 30 km^2 (thus $A_{min} = 30$) based Carbyn et al. (1993), who report observed high wolf densities in WBNP at 1 wolf per 38 km^2. As the numerical response was simulated stochastically for each year independently, there was potential for very wide variation in wolf numbers in the model. I set a maximum rate of increase in wolf numbers at $\lambda = 1.48$ based on Eberhardt (1998) and a maximum decrease at $\lambda = 0.67$ (the inverse of $\lambda = 1.48$). The integrated predation rate was predicted by (Messier 1994, 1996):

$$p = f(N_t) \bullet W(N_t) \bullet N_t^{-1}, \qquad [7.7]$$

which, when substituting each function is:

$$p = (a \bullet c \bullet N_t) \bullet (b + N_t)^{-1} \bullet (1 + c \bullet d \bullet N_t)^{-1}. \qquad [7.8]$$

The functional response was set by the bison density at the beginning of the year, and the number killed in each age and sex class (k_i) were apportioned based on eq. 1 in Chesson (1983). This function describes the probability that the next kill by a predator will be a certain prey type, based on relative availability of each prey type in the prey assemblage. Although the original function is intended to predict the probability of the next kill being of a certain prey type, I have assumed that integrated over the iteration, it estimates the proportion of the diet of the predator made up by that prey type. Preference indices (α) for each bison age and sex class were calculated using equation 2 in Chesson

(1983), based on prey selection of the Hornaday River wolf pack (data in Carbyn et al. 1993:44).

Drowning and anthrax each have the potential to cause mass mortality of bison (Carbyn et al. 1993; Gates et al. 1995; Dragon and Elkin 2001). I repeated the population projection, incorporating each factor as stochastic mortality sources. I assumed there was an annual probability of an anthrax outbreak of 0.2 based on the frequency of outbreaks in WBNP since 1962 (Dragon and Elkin 2001). When an outbreak occurred, adult survival rates (*d*) were adjusted based on data in Table II of Gates et al. (1995), and primarily affected males > 4 years of age. I assumed an annual probability of a major flood of 0.1, based on the flood frequency in the Peace-Athabasca Delta over the period 1803-1996 (PAD Technical Studies 1996). Mortality due to floods when they occurred was simulated by a uniformly distributed random variable ranging from (0 – 0.25). I applied this rate equally to all age classes.

Each simulation was run for 75 years, 1000 times to generate a probability distribution for equilibrium bison population density in the presence and absence of exotic pathogens. The initial population density for each simulation was set at 2.5 bison / km^2. Initial age structure was determined by using the age-structure of bison slaughters in WBNP during the 1950s (Fuller 1962), conducting one projection, then using the final age structure in year 20 as the initial age for each of the 1000 iterations. Equilibrium density was estimated as average density from year 60 – 75 of each population projection. To compare among simulations, I calculated the portion of simulations that resulted in an average density in the low (<0.83 bison / km^2), intermediate (0.83 - 1.67

bison / km^2), and high (> 1.67 bison / km^2) 33% of ecological carrying capacity (2.5 bison / km^2 or 12,500 bison in WBNP).

7.2 Results

7.2.1 Population trends in WBNP

Among the five bison populations in WBNP, two experienced a decline in numbers over the period (1971-1999; Table 7.1). Abundance in the Delta population declined at r = -0.12 from almost 4000 bison in 1976 to ca. 400 bison in 1999. The Hay Camp population declined at r = -0.04; although examination of the Hay Camp numbers reveals that the population trajectory likely had two phases. Results of a change-point test (Siegel and Castellan 1988: 64-70) on Hay Camp population numbers indicated that the population trajectory changed at the 1984 survey (change point test, approximate z = 3.74, $p < 0.001$, n = 22). Over the period 1975-1984 the population declined at r = -0.08 (95% CI, -0.12 - -0.04) while during 1985-1999 the population was stable or increased (r = 0.03, 95% CI, -0.01 - 0.07). The growth rate of the Garden River population did not differ from zero for the period 1981-1999 (Table 7.1). However, the 1981 count for this population is likely an outlier as it is almost double that of the next complete survey (1983; Table 7.1); reanalysis for 1983 -1999 indicated that the population grew during this period (r = 0.05, 95% CI, 0.02 - 0.08). The number of bison classified as in the Little Buffalo population was stable for the period, although these numbers were highly variable. In addition, the number of bison classified in the Nyarling River population

increased over the period; however, the numbers were generally low and variable pre-1990 (Table 7.1).

7.2.2 Simulating population growth

Deterministic population growth in the presence and absence of exotic pathogens, as well as predicted predation rate is depicted in Figure 7.4. Maximum predation rate was 13.3% / year, and peaked at a bison density of 0.35 bison/ km^2. The growth rate of bison populations in the absence of exotic pathogens (maximum 16% / year) exceeded predation until density-dependent reduction in calf survival resulted in an equilibrium density of 2.2 bison / km^2. At this density, predation rate was 6% / year, and was inversely-density dependent. In contrast, the growth potential in the presence of tuberculosis and brucellosis reduced population growth rate sufficiently to create the potential for a multiple-state system (Figure 7.4). Maximum population growth rate was 11.1% and the increase in the wolf: bison ratio attributed to scavenging of diseased carcasses increased maximum predation rate slightly to 13.6%. Thus the equilibrium density of bison in the presence of exotic pathogens was not independent of population trajectory: a population starting at moderate or high density (> 0.8 bison / km^2) would stabilize at 1.75 bison / km^2. At this density, predation rate is 7.4% / year and inversely density-dependent. In contrast an infected population starting at low density (< 0.8 bison / km^2) would stabilize at 0.13 bison / km^2. At this density, predation rate was 11.0% / year and was strongly density-dependent (Figure 7.4). The presence of a predator-pit indicates that variation in survival, reproduction, and predation is expected to have an impact on equilibrium bison densities.

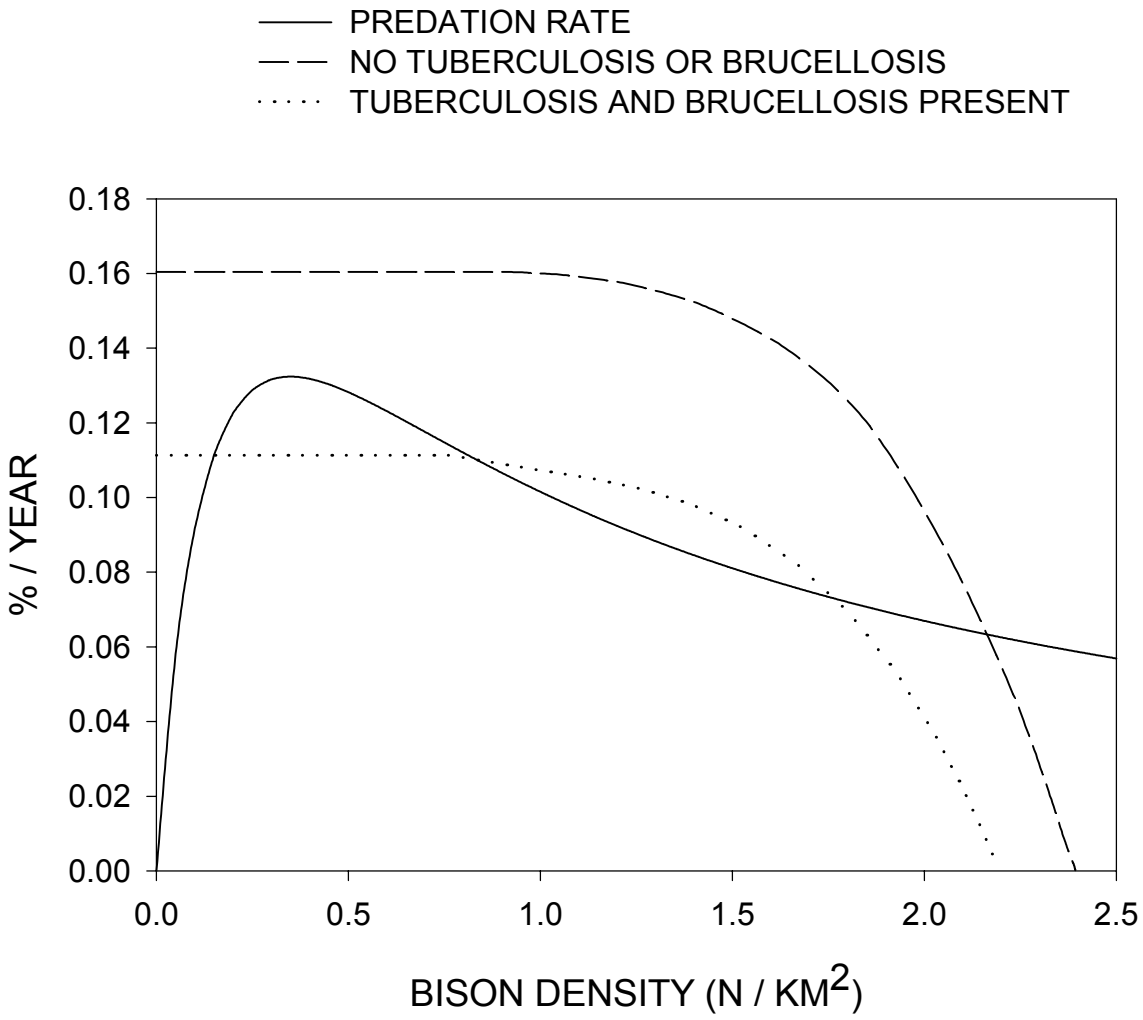

Figure 7.4 Simulated population growth (dashed lines) and predation rate (solid line) in the presence and absence of tuberculosis and brucellosis.

When stochastic variation was added to the simulation, the presence of tuberculosis and brucellosis caused the majority of population simulations to persist at low (68.5%) or intermediate (28.3%) densities (Figure 7.5). There was a low likelihood (3.2%) of a high-density equilibrium. The multiple-equilibrium system became unlikely when anthrax and mass drowning were added as mortality sources: 93.6% of the simulations resulted in low bison density, while only 6.2% and 0.2% were of intermediate or high density (Figure 7.5). In contrast, in the absence of exotic pathogens, there was almost no potential for a multiple-equilibrium system, as the majority (83.8%) of population simulations resulted in a high-density bison population, whereas only 13.4% and 2.8% of the simulations resulted in intermediate or low densities respectively (Figure 7.5). This result was robust in the presence of anthrax and mass drowning (8% low, 29.5% intermediate and 62.5% high density populations; Figure 7.5). It is important to note that none of the simulations resulted in extinction of the bison population.

7.3 Discussion

7.3.1 Population trends in WBNP

Two aspects of the validity of the data used in this analysis must be examined. Tessaro (1987) described several reasons why estimation of bison abundance in WBNP is difficult, including the size of the park, spatial heterogeneity of habitat, spatial and temporal heterogeneity in bison distribution, and low visibility of bison in treed or shrubby areas. The use of standardized methodologies over a long period of time compensates for these difficulties to some extent as individual estimates of bison

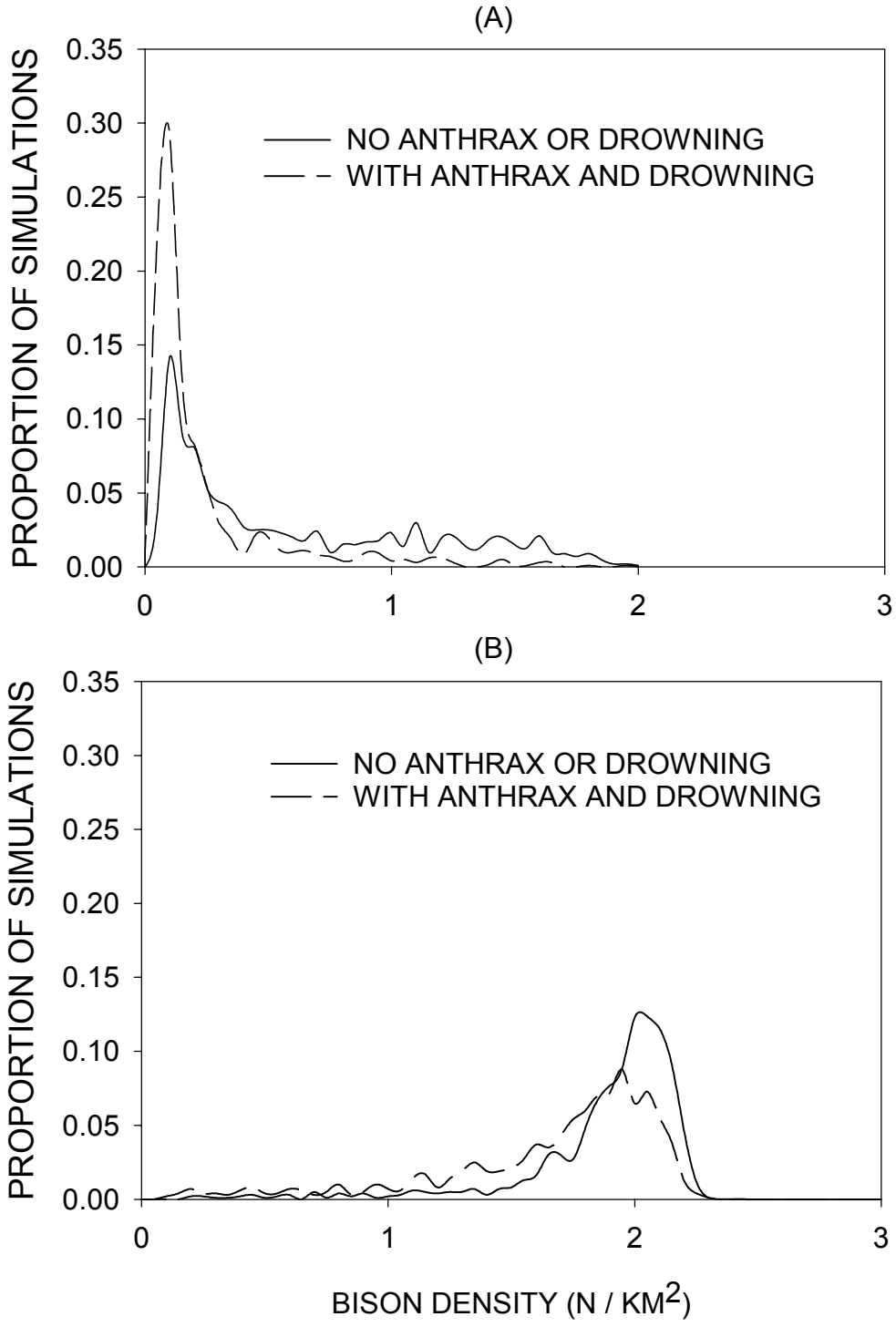

Figure 7.5. Simulated population densities in the presence (A) and absence (B) of tuberculosis and brucellosis. Bison density is the average density from year 60 - 75 of the population projection (n = 1000 for each line). Each simulation was conducted with and without anthrax and mass drowning as sources of mortality.

abundance, although imprecise, would reflect the overall trend in bison numbers. I have particular confidence in estimates of population trend for the Delta and Hay Camp populations as these areas were surveyed with standard flight lines each year and the presence of large meadow complexes (particularly in the Peace-Athabasca Delta) results in relatively good visibility of bison. However, the Garden River, Nyarling River, and Little Buffalo population ranges are surveyed in a more *ad hoc* manner, resulting in large inter-annual variation in abundance estimates for these populations. This variation could result simply from failing to locate a few herds during a survey as the number of bison in these populations is low. I suspect that this problem is particularly evident in the Nyarling River and Little Buffalo populations due to their small numbers and the large areas to be surveyed, as illustrated by comparing bison counts in the 1980s and 1990s for the Nyarling River population (Table 7.1). Counts are low and particularly variable pre-1990. Further, the Little Buffalo population is not contained within WBNP (see Figure 3.6) although only locations of bison within park boundaries were available. I would recommend that WBNP staff consider adopting a stratified random sampling technique (e.g., following Larter et al. 2000) to estimate bison numbers in these areas in order to standardize sampling and allow estimation of confidence limits. At present, it is not possible to determine whether the increase in numbers for the Garden River and Nyarling River populations represent a true increase or simply an artifact of sampling strategy.

The second aspect of the validity of this analysis requiring examination is the implicit assumption that bison metapopulation structure determined during 1990-93 and 1997-2000 represents the metapopulation structure during the period 1975-1990. The

WBNP movement study (1995) was the first study to follow movements of bison using radiotelemetry, and so previous data are limited, particularly for the Little Buffalo, Garden River and Nyarling River populations. Bison movement routes described by warden staff for the period 1971-1981 (see Carbyn et al. 1993: 104-110) in the Hay Camp and Peace-Athabasca Delta are consistent with the metapopulation structure observed here. However, the best evidence for stability in metapopulation structure comes from Wilson and Strobeck (1999). Analysis of microsatellite DNA indicated that there was a metapopulation structure within WBNP, and movement among populations was not sufficient to obscure genetic differences among the populations (referred to as subpopulations in Wilson and Strobeck 1999). Analysis of bison phenotypes reinforces these conclusions (Van Zyll de Jong et al. 1995). Consequently, I conclude that the metapopulation structure determined for bison for 1990-93 and 1997-2000 was sufficiently stable to *a posteriori* classify bison observed during past surveys.

I found that the decline in bison abundance in the Peace-Athabasca Delta was not unique within the WBNP metapopulation, and thus reject H_1. Bison abundance in the Hay Camp population declined from the mid-1970s to the mid-1980s at which point it stabilized, and grew slightly (I discuss this phase below). The decline over this interval was indistinguishable from that seen in the Delta population during 1981-1999 (i.e., 95% confidence intervals for *r* overlap), and therefore I fail to falsify the disease-predation hypothesis based on bison abundance data. My findings contradict Carbyn et al. (1993: 240; 1998), who argued that population decline was most precipitous south of the Peace River, while bison abundance north of the Peace was stable. However, the results herein suggest that delineation of bison populations based on the Peace River obscured the local

population dynamics of the Hay Camp population, which in fact did experience a decline indistinguishable from that in the Delta population. The results of chapter 3 clearly demonstrate that the Peace River is an artificial boundary that does not adequately delineate the Delta and Hay Camp populations.

It is important to stress that rejection of H_1 simply removes a major objection to the disease-predation hypothesis of Carbyn et al. (1993: 240; 1998), but does not prompt acceptance of the disease-predation hypothesis. However, it is instructive to compare population trajectories among the three bison populations infected with brucellosis and tuberculosis for which there is good data on population numbers (Delta, Hay Camp, and the bison population of the Slave River Lowlands). Each of these populations experienced a decline in abundance in the presence of exotic pathogens and predation (see chapter 6; Van Camp and Calef 1987; Messier 1989; Carbyn et al. 1993), while other northern, exotic pathogen-free bison populations grew and continue to grow in abundance (e.g., Larter et al. 2000). This commonality suggests that the search for a cause of decline in bison abundance should focus on common elements among the three populations, rather than factors unique to one population. In particular, it appears as if the decline in bison abundance in the Peace-Athabasca Delta is not unique for bison populations infected with tuberculosis and brucellosis in the presence of predation. Admittedly, the Nyarling River and Garden River populations may have increased in size (Table 7.1), despite the presence of introduced pathogens in these populations (chapter 4; Tessaro et al. 1990); however, I believe this growth is related to their low densities and prey-switching by wolves. I return to this point below.

7.3.2 Simulating population growth and maximum predation rate

The results of the stochastic population simulation were consistent with the disease-predation hypothesis. I found that growth of bison populations in the absence of exotic pathogens exceeded the maximum wolf predation rate and thus failed to reject H_2. Stochastic population modeling indicated that exotic disease-free bison populations, with or without anthrax and drowning, are highly likely to persist at high densities close to ecological carrying capacity. In contrast, a bison population in the presence of tuberculosis and brucellosis was likely to decline to low densities, particularly when drowning and anthrax were added to the model. The failure to reject H_3 indicates that bison populations in which these pathogens are present will likely be regulated at low densities by wolf predation. It is important to note that although a predator-pit was predicted based on the deterministic predation and population growth curves, very little evidence for multiple stable states was evident in the Monte Carlo simulations. I recognize that an unknown and probably large proportion of the variability can be attributed to measurement error, and this uncertainty will substantially reduce our ability to detect multiple stable states. At the very least, I can conclude that if a predator-pit exists, it is beyond our ability to detect.

I have assumed a hyperbolic functional response (Holling 1959, 1965; Real 1977, 1979) based on killing rate data collected in declining populations. This functional response assumes that wolves do not show a density-dependent preference for bison (i.e., do not "prey-switch"; Chesson 1983). Messier (1996) argued that in the presence of two prey types with different vulnerabilities to predation (e.g., moose and bison), wolves should ignore the less vulnerable prey type at low densities and only incorporate it into

their diet at high densities. This density-dependent preference for a particular prey type would result in a sigmoidal functional response (e.g., Chesson 1983). Insufficient data exist to determine whether wolf preference for bison is density dependent. However, I hypothesize that the form of the functional response of wolves is not independent of the prey population trajectory. Following Messier (1996), I suggest that wolves will incorporate bison in their diet only when an increasing bison density reaches some unknown threshold. However, I believe that due to the gregarious nature of bison, wolf preference for bison will not decline in response to a declining bison population until it reaches very low densities. Living in groups can reduce predation risk through the predator dilution effect, where increasing group size reduces the individual's likelihood of being preyed upon; however, predation risk can be also increased if groups increase risk of detection by the predator (encounter effect; reviewed by Wrona and Dixon 1991). The number and typical size of groups counted on total count surveys as a function of bison abundance in the Delta population is depicted in Figure 7.6. The number of groups counted appears to have been relatively stable as the population declined from 4000 to 1500 bison, after which the count begins to reduce. In contrast, the typical group size declines proportionately to bison abundance. If, as proposed by Huggard (1993), the functional response of wolves is linked to the number of groups, not the number of individuals, killing rate would be expected to stay high until bison abundance declines to low density. Therefore, although I recognize this is an unproven hypothesis, I believe the hyperbolic functional response that I assumed in this study is appropriate for simulating declining populations. Examination of the relationship between bison abundance, typical group size, and numbers of groups is necessary for both increasing

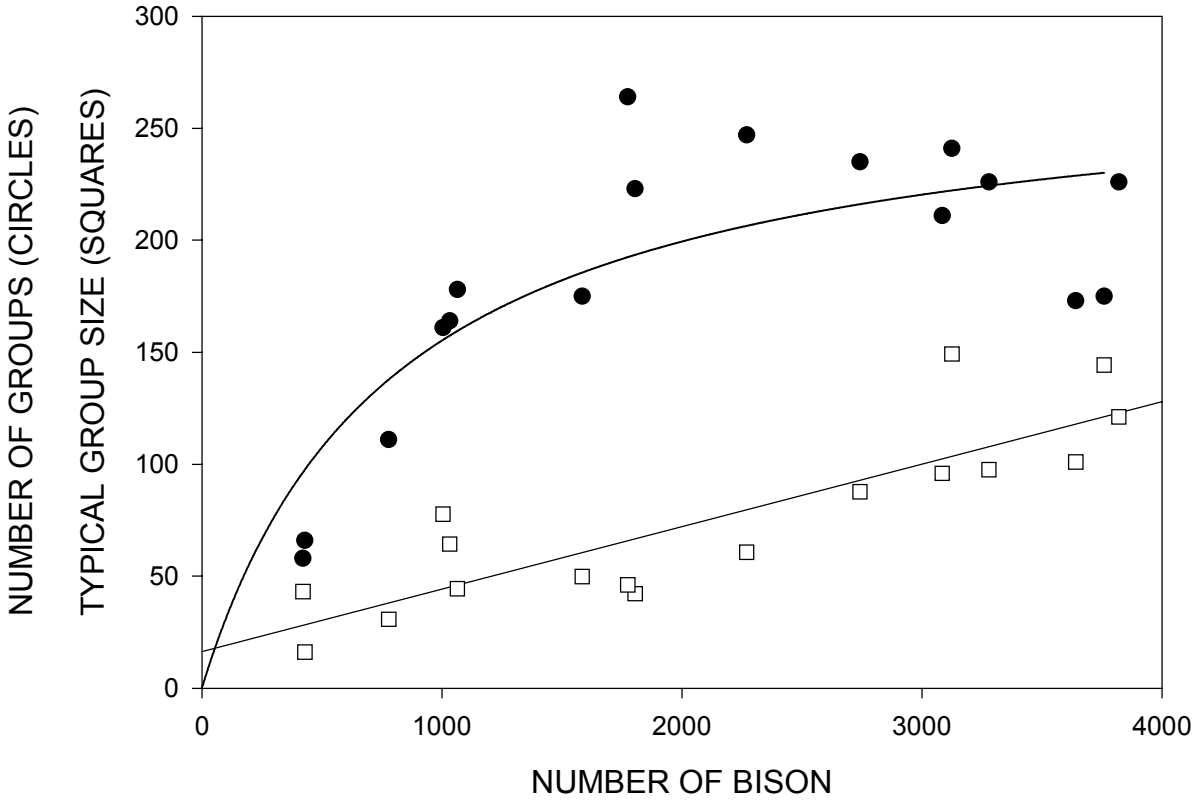

Figure 7.6. The relationship between abundance, typical group size, and number of groups of bison observed in the Delta population during late winter surveys (1981-1999).

and decreasing populations. It is interesting to note that if the typical group size declines substantially faster than the number of groups, then the predator-dilution benefits of group living may decline faster relative to the encounter costs of group living, further exacerbating the impact of predation on a declining population.

If this hypothesized relationship between bison density, population trajectory, and wolf predation is tenable, it is possible to speculate as to the future trends of bison numbers in WBNP. An introduced, low density bison population would experience a "honeymoon" period where predation would be low due to wolf preference for moose, and would increase in abundance. This initial population growth would occur with or without the presence of tuberculosis and brucellosis. Consistent with this hypothesis, wolf predation was not recorded in the Mackenzie Bison Sanctuary bison population as it grew until it exceeded 600 bison (Calef 1984; Gates and Larter 1990). However, as bison abundance increase, wolf preference for bison may increase and thus increased predation levels would cause reduced bison population growth. However, populations free of tuberculosis and brucellosis would continue to increase in size until density-dependent competition for food would initiate bison range expansion (e.g., as in the Mackenzie Bison Sanctuary; Larter et al. 2000) and eventually cause a reduction in juvenile calf survival (e.g., Blyth 1995; Fowler 1981). In contrast, the stochastic simulations presented here demonstrate populations with tuberculosis and brucellosis would not be sufficiently productive to offset predation losses, and population growth would decline. Wolves would continue to prey on bison during this decline, as seen in the Slave River Lowlands (Van Camp 1987) and in the Delta population (chapter 6). At some low bison abundance, wolves may reduce their focus on bison and incorporate

moose to a higher degree in their diet. This threshold abundance is unknown, but it is interesting to note that Carbyn et al. (1993) reported extensive predation on bison in the Hay Camp population when it was in excess of 700 bison (1978-1981). Subsequently, the population declined in size to a low of 300 bison in the mid-1980s and two subsequent studies there reported low levels of predation (1990-1993, WBNP 1995; 1997-2001, chapter 6). Further, predation was not recorded in the low abundance Nyarling River population (<230 bison) during 1997-2001. Once predation pressure relaxes, the population would again grow in the absence of predation (e.g., Hay Camp [mid-1980s to present] and Nyarling River [1981-1999] populations), and the cycle would repeat.

I must stress that the difference between the simulated growth of bison populations with and without tuberculosis and brucellosis is likely an underestimate relative to the true impact of exotic pathogens on bison productivity. First, as discussed in chapter 1, disease-testing error results in an underestimate of the effect of pathogens on a particular demographic parameter. Thus the effect of exotic pathogens on adult survival was likely underestimated in this model. In addition I believe that this reduction in statistical power contributed to an inability to detect a main effect of tuberculosis on bison survival (chapter 6) and pregnancy rate in the Delta and Hay Camp populations (chapter 5). Further, the timing of brucellosis-induced abortions meant that I was unable to adequately test the full effect of brucellosis on reproductive success of bison. I limited the effect of exotic pathogens to those bison that tested positive for tuberculosis and had a high titre for brucellosis; this applied to only 10% of the population. Finally, the only data on wolf killing rate of bison comes from two populations, both of which

are infected with exotic pathogens. Exotic pathogens likely increase killing success of wolves to some degree through debilitation of bison (see chapter 6); I was unable to evaluate the degree to which this occurs and therefore used a common functional response for infected and infection-free populations. I modified the numerical response of wolves to account for extra biomass provided by scavenging diseased-carcasses; however, this effect was very slight (a 5% increase in wolf: bison ratio) and consequently did not impact the results (data not shown). The fact that bison population dynamics can be altered so dramatically in the presence of simulated exotic pathogens is revealing, given these constraints.