6. IMPACT OF TUBERCULOSIS AND BRUCELLOSIS ON SURVIVAL

Tuberculosis and brucellosis each have the potential to impact the survival of bison. Tuberculosis can affect many organ systems of bison, with lesions found in the liver, lungs, the pericardium, ribs, and kidneys among others (see Tessaro et al. 1990; Choquette et al. 1961). Tessaro et al. (1990) diagnosed granulomatous encephalitis in a calf infected with tuberculosis. Tessaro et al. (1990) reported that one bison cow died of generalized tuberculosis, and three others would have if wolves had not intervened (4 of 72 or 6%). Each of these latter three bison were emaciated. Meat from tuberculosis-infected bison is considered unfit for human consumption if there is evidence of systemic dissemination of the bacteria. Bison that reach this stage are considered to have poor survival prospects (Fuller 1962). Based on this criterion, Fuller (1962) argued that the condemnation rate of 5% of bison slaughtered in WBNP (1952-56, n = 1508) represented the mortality rate of bison directly attributable to tuberculosis. Brucellosis has the potential to affect bison survival by reducing foraging efficiency and increasing risk of predation by wolves. Arthritis is a common manifestation of brucellosis in WBNP bison (Tessaro et al. 1990). These authors report five of 72 bison (7%) had swollen stifle joints and arthritis associated with brucellosis. Two of these were severely emaciated, which Tessaro et al. (1990) interpreted as a secondary consequence of the brucellar lesions. In total, Tessaro et al. (1990) concluded that 8% of the bison in their

sample of 72 bison died as a consequence of these diseases. Although there are no data on the relative risk of predation on diseased bison, Smith et al. (2000) note that wolves in Yellowstone preyed on injured bison or those in poor condition (unrelated to tuberculosis or brucellosis), suggesting an increased risk of predation related to these pathogens in WBNP.

6.1 Methods

6.1.1 Radio-telemetry

I deployed radio-collars on bison in February-March 1997-1999 (see chapter 2). Each radio-collar was equipped with a motion sensor to detect mortality, and had a battery life of 24 months. In addition, I deployed ear-tag radio transmitters on calves (<12 months old) in 1997-1999. These transmitters had a battery life of 10 months in 1997 and 15 months in 1998 and 1999. I attempted to relocate bison from an airplane once every 10 days in the spring and early summer, and approximately every three weeks the remainder of the year.

When mortality was detected during aerial surveys, I investigated the carcass on the ground. Carcasses were usually investigated with the assistance of a member of the closest community. The cause of death was determined by evaluating the criteria presented in Table 6.1. When sufficient remains were present a standardized protocol was followed during each mortality investigation, based on methods outlined in Wobeser and Spraker (1980) and Roffe et al. (1994). Predation was identified by degree of skeleton disarticulation, evidence of a chase sequence in snow, scattered blood or fur,

Table 6.1 Criteria used to determine whether predation is the cause of mortality for bison found dead during telemetry flights.

	Cause of mortality	
Criteria	Predation	Other
Chase sequence	Yes	No
Body position	Lateral	Sternal
Skeleton disarticulated	Yes	No
Location of stomach	Outside body	Inside body

and location of rumen (in or outside of abdominal cavity). I classified the cause of mortality as "undetermined" if I could not distinguish predation from scavenging or there was no indication as to the cause of death. Causes of mortalities classified as "other" include drowning, hunting, and disease.

6.1.2 Data analysis

Annual survival rate was estimated as described by Heisey and Fuller (1985), except that the analysis was based on seasonal, rather than daily, survival probabilities. Seasons were defined as in section 3 with one exception: winter was divided into two seasons (early 7, November - 1 March; late, 2 March - 4 May) to accommodate addition of new radio collars in 1998 and 1999. I estimated seasonal cause-specific mortality rates as $m_{ij} = y_i / x_i$, where m_i is the probability that a bison dies from cause j during season i, y_{ij} is the number of deaths in season i due to cause j, and x_i is the number of bison at risk during the season (Heisey and Fuller 1985). Annual rates were calculated as the product of the seasonal rates. Ninety-five percent confidence limits were estimated using the Taylor series approximation method in program MICROMORT (Heisey and Fuller 1985).

I censored bison from analysis at time of lost radio-contact except for calves (10-11 months of age at capture) that had a 10-month battery life on their transmitters. I assumed that calves died if I lost contact within 6 months of collar deployment (i.e., start of autumn season). I excluded those bison that were determined to have died from capture-related causes (described in section 2). The following variables were examined

as predictors of seasonal survival probability: year, population (as defined in section 3), brucellosis status (complement fixation titre ≥1:40), tuberculosis status (caudal fold or fluorescent polarization assay positive), age, and sex. In addition, all two-way interactions among biological variables were included. As in section 5, I used backward elimination multiple logistic regression (SPSS 10.05, Chicago, IL) to determine which four terms (main effects or two-way interactions) had the highest value in predicting survival. Independent variables were sequentially removed from the model at $p > 0.10$. I used AICc to rank all models constructed from combinations of the remaining four independent variables (STATISTICA Release 5.5, StatSoft, Inc. Tulsa OK), and model averaging (Burnham and Anderson 1998: 46-48, 123-140; Anderson et al. 2000) to determine confidence limits for each regression coefficient. Results are presented as odds ratios and confidence intervals to facilitate interpretation of data. Model fit was assessed using likelihood ratio tests (Sokal and Rohlf 1995: 686-697).

6.2 Results

6.2.1 Radiotelemetry

I deployed 80, 75, and 72 radio-collars on bison in 1997, 1998, and 1999, respectively. Fifty-four radio-telemetry surveys were conducted between April, 1997 and February, 2000 (mean interval, 20 ± 8 [SD] days).

6.2.2 Annual survival and cause-specific mortality

Annual survival of bison varied from a low of 0.77 in the Delta population (1998/1999) to a high of 1 in the Nyarling River population (1997/98 and 1999/00; Table 6.2). Mortality from all causes was highest in the Delta population, particularly due to wolf predation. Wolf predation was a minor source of mortality in the Hay Camp and Nyarling River populations (mean annual probability, <0.01). Two bison from the Hay Camp population were shot outside the WBNP boundary near Mission Farms and the Salt River area. Drowning was a minor source of mortality among radio-collared bison (Table 6.2). One bison died of generalized tuberculosis in the Delta area.

6.2.3 Seasonal survival probabilities.

In spring (5 May - 30 June) and summer (1 July and 31 Aug.) survival probabilities for bison >1 year of age were high, exceeding 0.97 in all populations (Figures 6.1 and 6.2). This high survival probability prevented analysis of factors associated with mortality. Further, I could not assess factors associated with survival in the Nyarling River population, as only two radio-collared bison (>2 years of age) died over the course of the study (Table 6.2). Therefore, I focus on autumn, early and late winter survival in the Hay Camp and Delta populations.

Autumn (1 Sept. - 6 Nov.) survival for bison ≥1 year was high in the Delta, Hay Camp and Nyarling River populations. I excluded all but the following factors associated with autumn survival probability with the backward elimination multiple regression: tuberculosis status, sex, an interaction between sex and age, and an interaction between tuberculosis status and age (remaining variables and interaction

Table 6.2 Annual survival and cause-specific mortality rates for bison (≥ 1 year old) in WBNP, 1997-2000. Ranges in parentheses are 95% confidence intervals.

			Cause-specific mortality rates		
Year	Population	Annual Survival	Undetermined[a]	Wolf predation	Other
1997/98[b]	DT[c]	0.81 (0.68 - 0.96)	0.07 (0 - 0.15)	0.10 (0 - 0.21)	0.02[d] (0 - 0.07)
	HC	0.96 (0.89 - 1)	0 (-)[d]	0 (-)	0.04[e] (0 - 0.11)
	NY	1 (-)	0 (-)	0 (-)	0 (-)
1998/99	DT	0.77 (0.64 - 0.91)	0.06 (0 - 0.14)	0.12 (0.02 - 0.21)	0.05[f] (0 - 0.12)
	HC	0.98 (0.93 - 1)	0.02 (0 - 0.07)	0 (-)	0 (-)
	NY	0.99 (0.77 - 1)	0.01 (0 - 0.23)	0 (-)	0 (-)
1999/00	DT	0.89 (0.79 - 1)	0 (-)	0.09 (0 - 0.18)	0.03[d] (0 - 0.08)
	HC	0.92 (0.86 - 1)	0.04 (0 - 0.09)	0.01 (0 - 0.04)	0.02[e] (0 - 0.06)
	NY	1 (-)	0 (-)	0 (-)	0 (-)

[a] includes mortalities where wolf predation could not be excluded.
[b] year assumed to begin on March 1.
[c] Delta, DT; Hay Camp, HC; Nyarling River, NY.
[d] drowning (n = 1 in 1997/98 and 1997/98).
[e] hunting (n = 1 in each year).
[f] drowning (n = 1), generalized tuberculosis (n = 1).

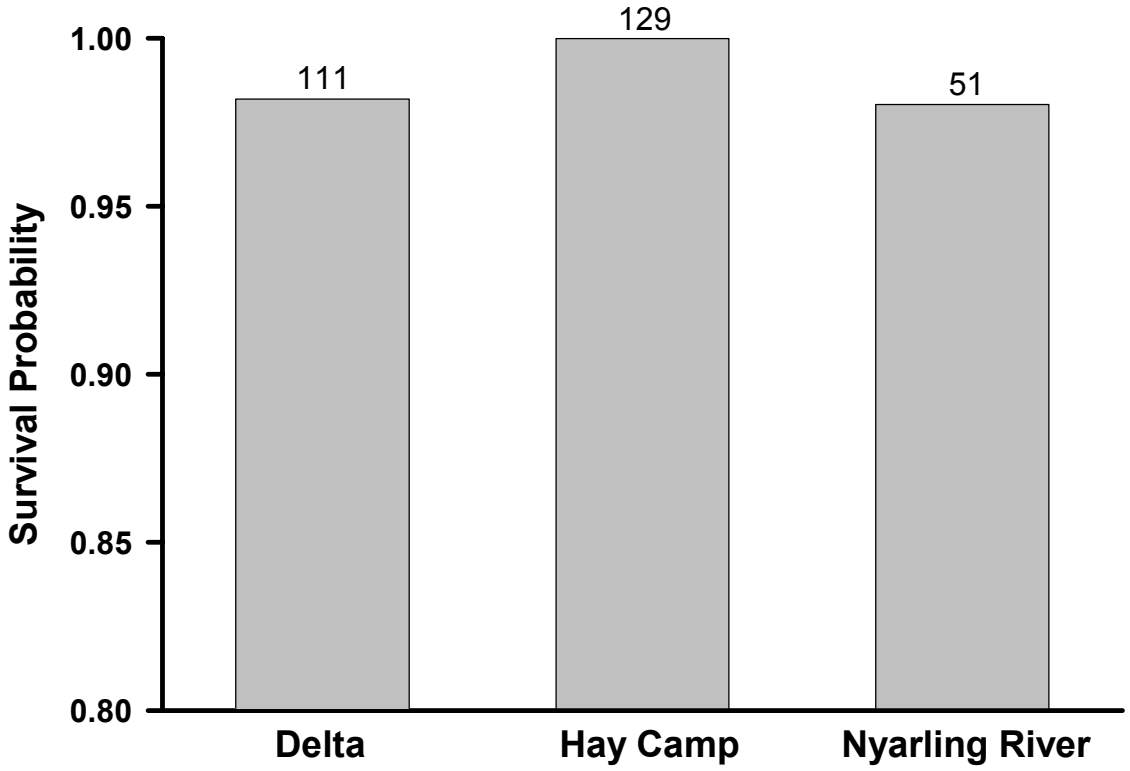

Figure 6.1. Survival probability in spring (5 May - 30 June) of bison from the Delta, Hay Camp and Nyarling River populations. Sample size is indicated at the top of each bar.

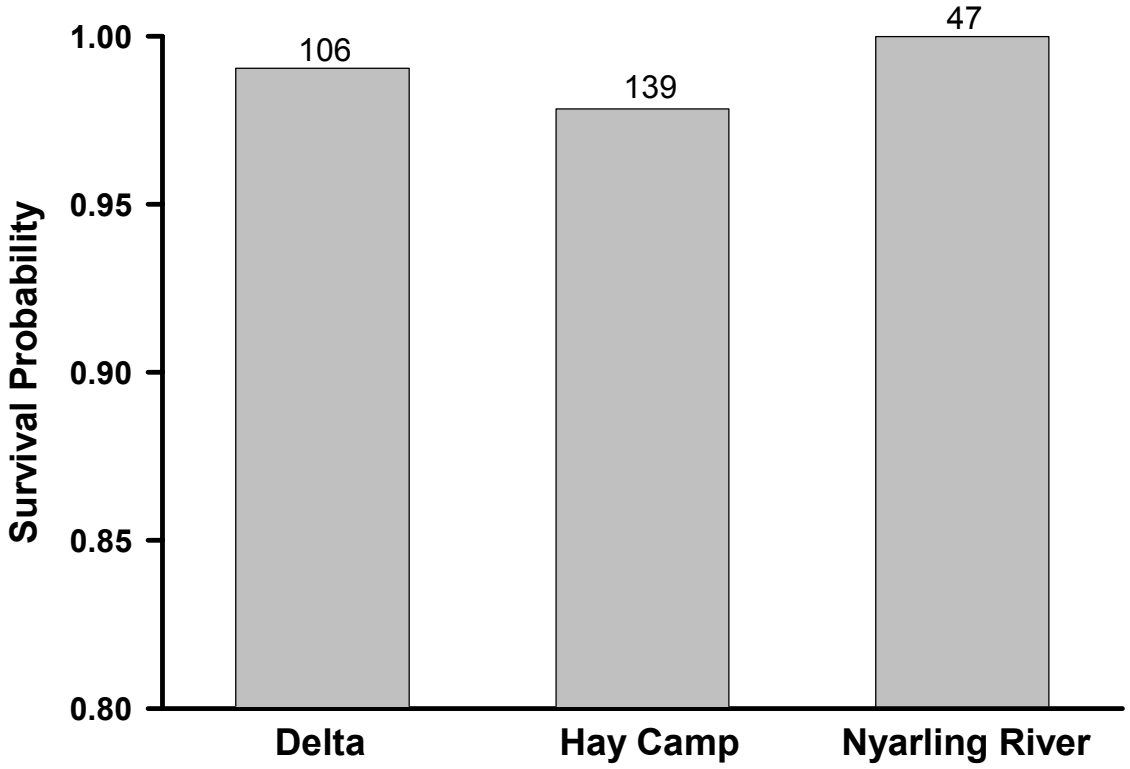

Figure 6.2. Survival probability in summer (1 July - 31 Aug.) of bison from the Delta, Hay Camp and Nyarling River populations. Sample size is indicated at the the of each bar.

terms, $p > 0.10$). However, none of the models constructed from these four terms adequately fit the data (all models, $p > 0.5$) nor did the odds ratio for any term differ from one (Table 6.3). The most parsimonious model of survival for the early winter season (7 Nov - 1 Mar) indicated that year and an interaction term between high brucellosis titre and tuberculosis status were important factors; however, a model including an interaction term between high brucellosis titre and tuberculosis only provided an almost identical compromise between bias and explained variance (Table 6.4). Model-averaged odds ratios indicated that survival in this season was lower in 1997 than 1999 (odds ratio 2.0, 95% confidence interval, 1.0 - 3.8; Figure 6.3), but there was no difference between 1998 and 1999 (odds ratio 1.0, 95% confidence interval, 0.6 - 2.03; Figure 6.3). Bison that tested positive for tuberculosis and had a high titre for brucellosis were 2.5 times more likely to die during this season than bison that tested positive for one or neither pathogen (95% confidence interval, 1.0 - 6.1; Figure 6.4).

One adult female bison was killed by wolves in late winter (2 Mar. - 4 May) of 1999, two days after capture (see chapter 2, section 2.3.2). Although this mortality was unrelated to capture, no other mortalities occurred during this season. Therefore, I restricted the analysis to late winter of 1997 and 1998. Five terms were not excluded by the backward elimination as predictors of late winter survival: sex, population, year, an interaction between tuberculosis status and age, and an interaction between tuberculosis and a high brucellosis titre (Table 6.5). An interaction term between sex and tuberculosis was also significant in the multiple regression, although I did not consider this term as it unrealistically indicated that tuberculosis increased survival. The first to sixth ranked models provided good fits to the data and only differed by 2.8 AICc values

Table 6.3 Comparison of models of autumn (1 Sept. – 7 Nov.) survival probability for bison in the Delta and Hay Camp populations. The χ^2 and p-value refer to the likelihood ratio goodness of fit test. Relative AIC_c is presented as Δ_i, and the Akaike weight (ω_i) refers to the probability that the model is the Kullback-Liebler best model, given the data (see Anderson et al. 2000).

Model[a]	df	χ^2	p	Δ_i	ω_i
Tb	1	0.32	0.57	0.00	0.15
Sex*age	1	0.06	0.80	0.26	0.13
Sex	1	0.05	0.83	0.28	0.13
Tb*age	1	0.01	0.94	0.32	0.13
Tb, tb*age	2	1.17	0.56	1.21	0.08
Sex, tb	2	0.48	0.79	1.90	0.06
Tb, sex*age	2	0.46	0.79	1.92	0.06
Sex*age, tb*age	2	0.08	0.96	2.30	0.05
Sex, tb*age	2	0.07	0.97	2.31	0.05
Sex, sex*age	2	0.06	0.97	2.32	0.05
Sex, tb, tb*age	3	1.28	0.73	3.18	0.03
Tb, sex*age, tb*age	3	1.23	0.75	3.23	0.03
Sex, tb, sex*age	3	0.48	0.92	3.97	0.02
Sex, sex*age, tb*age	3	0.08	0.99	4.37	0.02
Sex, tb, sex*age, tb*age	4	1.29	0.86	5.26	0.01

[a] model-averaged odds ratios (95% CI): sex, 1.0 (0.5 – 1.7); tb, 1.3 (0.7 – 2.6); sex*age, 1.0 (0.9 - 1.1); tb*age, 1.0 (0.9 – 1.1).
[b] age of bison determined by tooth eruption (< 3 years) or cementum annuli.

Table 6.4. Comparison of models of early winter (7 Nov. – 1 Mar.) survival probability for bison in the Delta and Hay Camp populations. The χ^2 and p-value refer to the likelihood ratio goodness of fit test. Relative AIC_c is presented as Δ_i, and the Akaike weight (ω_i) refers to the probability that the model is the Kullback-Liebler best model, given the data (see Anderson et al. 2000).

Model[a]	df	χ^2	p	Δ_i	ω_i
Year, tb*bruc	3	4.17	0.04	0.00	0.24
Tb*bruc	1	7.25	0.06	0.06	0.17
Year, tb*age[b], tb*bruc	4	4.86	0.09	1.53	0.11
Year, age, tb*bruc	4	4.20	0.12	1.71	0.10
Tb*age, tb*bruc	2	7.85	0.10	2.08	0.08
Age, tb*bruc	2	3.31	0.19	2.32	0.07
Year, age, tb*age, tb*bruc	5	7.28	0.12	3.24	0.05
Year	2	4.90	0.18	3.14	0.05
Age, tb*age, tb*bruc	3	0.31	0.58	3.65	0.04
Year, age	3	0.09	0.76	4.32	0.03
Age	2	7.88	0.16	5.04	0.02
Year, tb*age	3	3.53	0.32	5.10	0.02
Tb*age	1	3.35	0.34	6.04	0.01
Year, age, tb*age	4	0.34	0.85	6.38	0.01
Age, tb*age	2	3.54	0.47	7.07	0.01

[a] model-averaged odds ratios (95% CI): 1997 vs. 1999, 2.0 (1.0 - 3.8); 1998 vs. 1999, 0.96 (0.5 – 2.0); age, 0.98 (0.92 - 1.04); tb*age, 1.0 (0.96 – 1.03); tb*bruc, 2.5 (1.0 – 6.6).

[b] age of bison determined by tooth eruption (< 3 years) or cementum annuli.

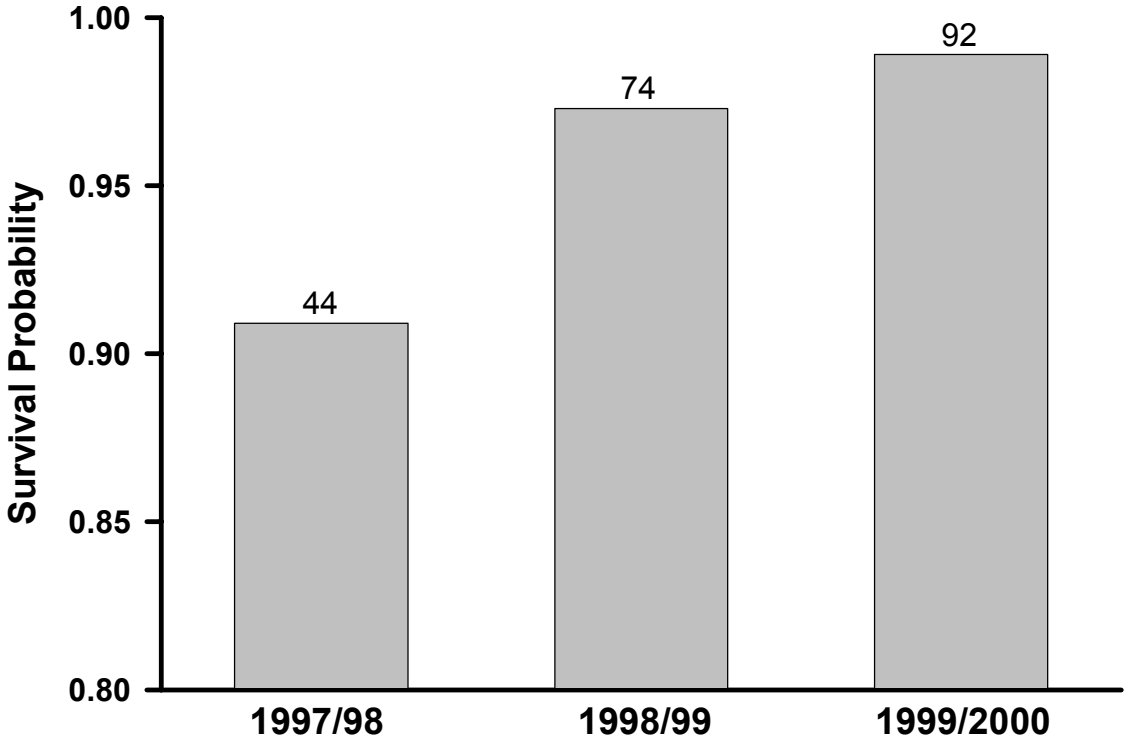

Figure 6.3. Survival probability in early winter (7 Nov. - 1 Mar.) of bison from the Delta and Hay Camp populations from 1997-1999. Sample size is indicated at the top of each bar.

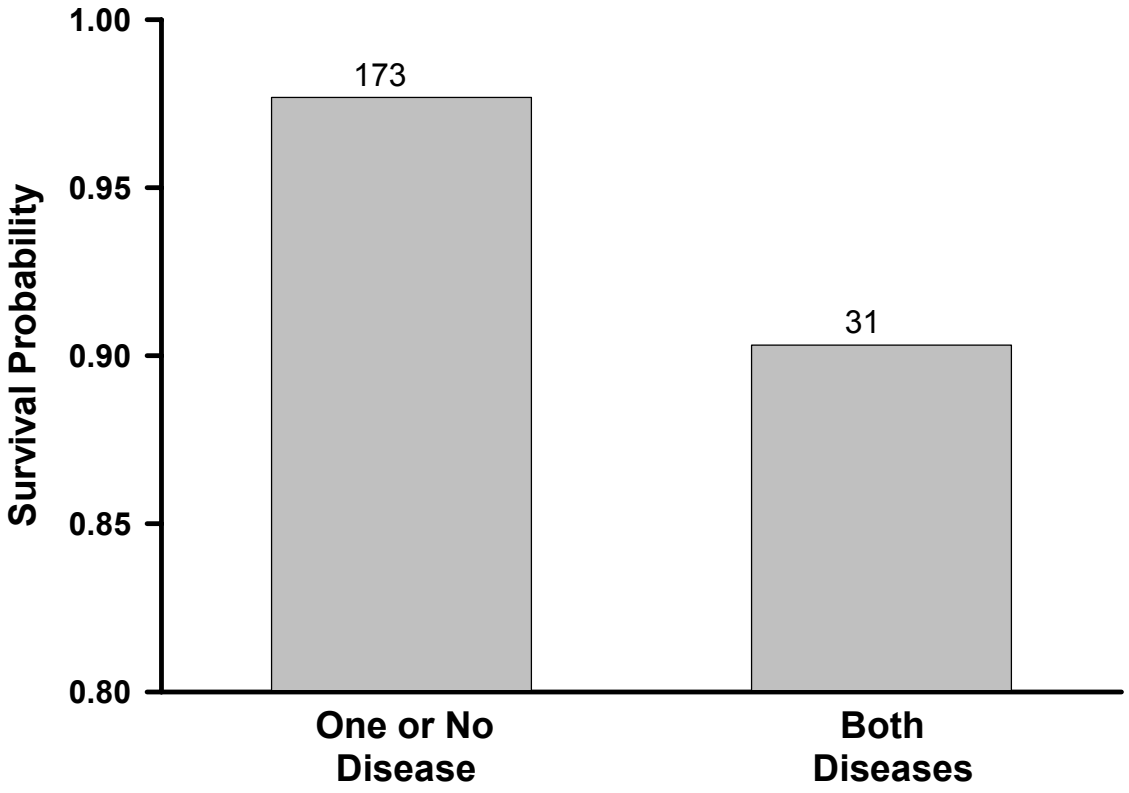

Figure 6.4. Survival probability in early winter (7 Nov. - 1 Mar.) of bison from the Delta and Hay Camp populations based on presence of bovine diseases. Sample size is indicated at the top of each bar.

Table 6.5 Comparison of models of late winter (2 Mar. – 4 May) survival probability for bison in the Delta and Hay Camp populations. The χ^2 and p-value refer to the likelihood ratio goodness of fit test. Relative AIC_c is presented as Δ_i, and the Akaike weight (ω_i) refers to the probability that the model is the Kullback-Liebler best model, given the data (see Anderson et al. 2000).

Model[a]	df	χ^2	p	Δ_i	ω_i
Sex, pop, year, tb*bruc	4	23.81	<0.001	0.00	0.29
Pop, year, tb*bruc	3	20.98	<0.001	0.70	0.20
Sex, pop, year, tb*age, tb*bruc	5	23.84	<0.001	2.12	0.10
Sex, pop, tb*bruc	3	19.77	<0.001	1.19	0.11
Pop, year, tb*age, tb*bruc	4	21.19	<0.001	2.61	0.08
Pop, tb*bruc	2	16.77	<0.001	2.80	0.07
Sex, pop, tb*age, tb*bruc	4	19.87	0.001	3.94	0.04
Pop, tb*age, tb*bruc	3	16.94	0.001	4.74	0.03
Sex, tb*bruc	2	13.42	0.001	6.15	0.01
tb*bruc	1	11.35	0.001	6.15	0.01
Year, tb*bruc	2	13.34	0.001	6.23	0.01
Sex, year, tb*bruc	3	15.04	0.002	6.64	0.01
tb*age, tb*bruc	2	11.91	0.003	7.67	0.01
Year, tb*age, tb*bruc	3	13.91	0.003	7.77	0.01
Sex, pop, year, tb*age	4	15.81	0.003	8.00	0.01
Sex, tb*age, tb*bruc	3	13.47	0.004	8.21	0.00
Sex, year, tb*age, tb*bruc	4	15.13	0.004	8.68	0.00
Sex, pop, tb*age	3	12.72	0.005	8.95	0.00
Sex, pop, year	3	12.23	0.007	9.44	0.00

Model[a]	df	χ^2	p	Δ_i	ω_i
Pop, year, tb*age	3	12.19	0.007	9.49	0.00
Pop, year	2	9.84	0.007	9.74	0.00
Sex, pop	2	9.65	0.008	9.93	0.00
Pop	1	7.11	0.008	10.39	0.00
Pop, tb*age	2	8.98	0.011	10.60	0.00
Sex	1	2.44	0.118	15.06	0.00
Sex, tb*age	2	4.07	0.131	15.51	0.00
Sex, year	2	3.59	0.166	15.99	0.00
Sex, year, tb*age	3	5.42	0.144	16.26	0.00
Year	1	1.31	0.252	16.19	0.00
Tb*age	1	0.84	0.360	16.66	0.00
Year, tb*age	2	2.34	0.311	17.24	0.00

[a] model-averaged odds ratios (95% CI): sex, 0.7 (0.5 – 1.1); pop, 3.0 (1.0 – 9.0); year, 0.6 (0.4 - 1.0); tb*age, 1.00 (0.97 – 1.03); tb*bruc, 3.7 (1.3 – 11.1).

[b] age of bison determined by tooth eruption (< 3 years) or cementum annuli.

(Table 6.5). Bison in the Delta population were 3 times more likely to die during this season than bison in the Hay Camp population (95% confidence limit, 1.0 - 9.0). Tuberculosis-positive bison with high brucellosis titre were 3.7 times more likely to die than bison with one or neither pathogen (95% confidence limit 1.3 - 11.1; Figure 6.5). There was some indication that survival in this season was lower in 1998 than 1997, although the confidence limits did not differ from one (Table 6.5).

6.3 Discussion

Annual survival rates of bison (≥1 year) were consistent with those of bison in the Mackenzie Bison Sanctuary (0.75 - 0.96; Larter et al. 2000) and previous radio-collaring studies in WBNP (0.87; recalculated from WBNP 1995). Adult survival in large ungulates is often high, and relatively insensitive to environmental variability (Fowler 1987; Gaillard et al. 1998). However, I did observe temporal and geographical variation in bison survival in WBNP. Early winter survival improved in the park from 1997 to 1999, particularly in the Delta population, and survival was higher in late winter in the Nyarling River and Hay Camp populations than in the Delta.

It is important to note that I was conservative in determining whether a particular bison had been depredated, electing to attribute a mortality to wolf predation only when the evidence was strong (i.e., obvious chase sequence, signs of struggle, highly disarticulated skeleton, etc.). However, even if I assume that half of the mortalities classified as "undetermined" were wolf kills, there would still remain a disparity in wolf predation rates among the regions of the park. It is unlikely that kills by black bears were incorrectly attributed to wolves (Carbyn et al. 1993: 232, 239).

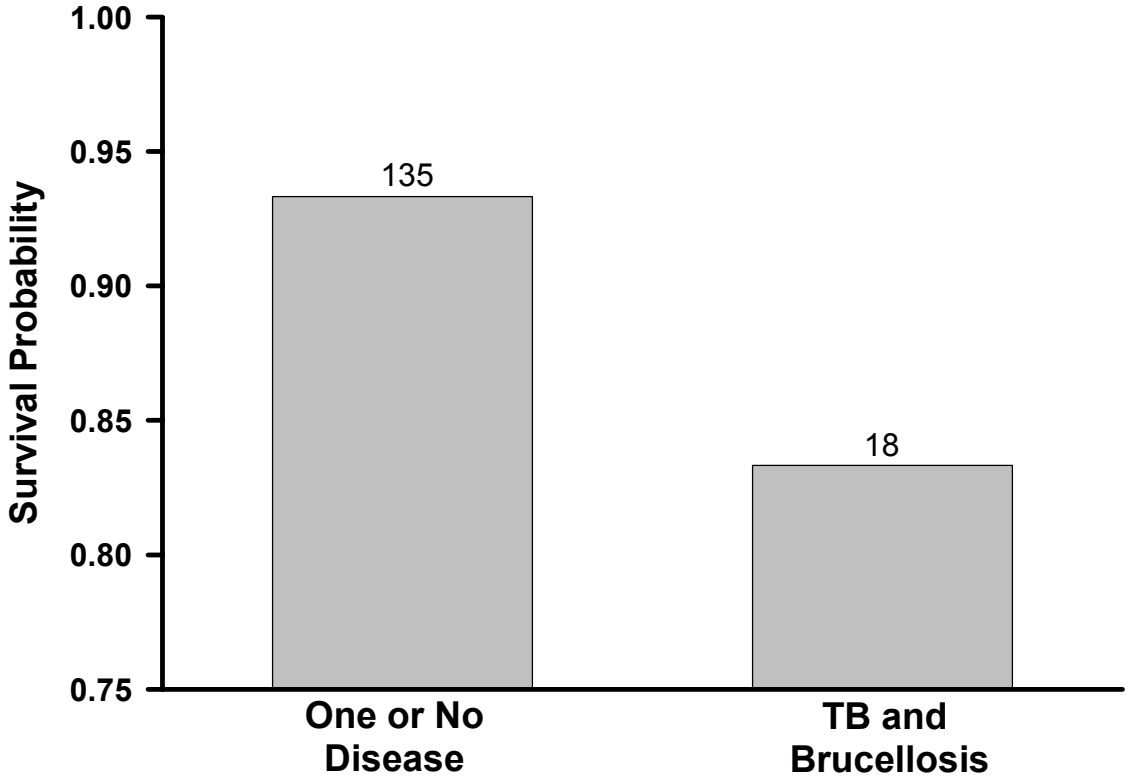

Figure 6.5. Survival probability of bison from Delta and Hay Camp subpopulations in late winter (2 Mar. - 4 May) based on prevalence of disease. Sample size is indicated at the top of each bar.

Annual wolf predation rates varied from 0.09 - 0.10 in the Delta population, high relative to predation rates elsewhere in the park (Table 6.2). The low rate of wolf predation observed in the Hay Camp and Nyarling River populations was not expected. There are several possible explanations for this trend. First, wolf density is a function of prey density (Gasaway et al. 1992; Messier 1994; Fuller and Murray 1998) including bison (Joly and Messier 2000). Perhaps densities of bison in the Hay Camp and Nyarling River areas are not sufficient to maintain wolves at a density at which predation would be a significant source of mortality. Only one pack of wolves (>7 animals) was observed in the Hay Camp area on a recent aerial survey (Mitchell et al. 2000), although this information should be viewed cautiously as parts of this area are heavily wooded. A second explanation is that wolves prefer to target moose (*Alces alces*) in the area. Messier (1995) argued that in the presence of prey with different vulnerabilities to predators, wolves would target the more vulnerable prey unless the less vulnerable prey was at high densities. Consistent with this prediction, Larter et al. (1994) found wolves in the Mackenzie Bison Sanctuary preferred moose than bison. Smith et al. (2000) found that wolves in Yellowstone National Park are more successful at killing elk (*Cervus elaphus*) than bison. Further, wolves in Poland preyed on European bison (*Bison bonasus*) less often than would be expected based on bison biomass (Jedrezejewski et al. 2000). Instead, their diet consisted mainly of the more vulnerable red deer (*Cervus elaphus*). Perhaps densities of bison in the Hay Camp area are not sufficient to be a profitable prey choice for wolves. I believe this is a major factor, and return to this point in the next chapter. A third possible explanation for the low wolf predation rate observed in the Hay Camp area is that wolves target mainly juvenile bison. I have no

data on annual survival of bison <1 year of age, and only limited data on bison 1-2 years of age, and so I cannot directly address this question. More research is necessary on the relationship between bison density and relative preference of wolves for moose and bison.

I did not detect a main effect of tuberculosis or brucellosis on survival of bison. There is little dispute that tuberculosis causes an increase in mortality in a range of species including bison (e.g. Tessaro 1986; Tessaro et al. 1990; O'Reilly and Daborn 1995), and as I observed at least one bison that died of generalized tuberculosis, I reject the view that tuberculosis causes *no* disease in bison. Previous estimates of tuberculosis-induced mortality range from 5-6% (Fuller 1962; Tessaro et al. 1990). I was unlikely to detect an effect of this magnitude, given the reduction in statistical power associated with errors in testing (see chapter 4.3.1). However, I did observe an interactive effect between tuberculosis and brucellosis on winter survival of bison. Tuberculosis-positive bison that also had a high titre (complement fixation ≥1:40) for brucellosis were 2.5 times more likely to die during the early winter season (7 Nov. - 1 Mar) than bison with one or neither pathogen. The sum of the Akaike weights for models including the tuberculosis-high brucellosis titre interaction term was 0.87. This indicates strong evidence for an interactive effect of brucellosis and tuberculosis on survival during winter (Anderson et al. 2000). In late winter (2 Mar - 4 May), bison with both pathogens were 3.7 times more likely to die than bison with only one or neither pathogen. The sum of the Akaike weights for models including this term was 0.98, again indicating strong evidence for an interactive effect of both pathogens on survival.

The physiological mechanism for an interaction between tuberculosis and brucellosis to affect survival of bison is not known. Brucellosis-induced arthritis has been reported in bison in WBNP (e.g., Tessaro et al. 1990) and in cattle (Fraser et al. 1991); however, I was unable to find a published account for brucellar arthritis in bison in Yellowstone National Park, where brucellosis is also endemic yet tuberculosis is not present (Cheville et al. 1998). The lack of a published report does not necessarily mean that this aspect of brucellosis pathology does not occur, but it must be rare as there are no reports of these lesions among ~1000 bison slaughtered as they left Yellowstone National Park in the winter of 1996/97. However, these brucellar lesions are common among WBNP bison (e.g., Tessaro et al. 1990), where tuberculosis is present. My data indicate that bison in WBNP that have both pathogens have lower winter survival rates and have reduced pregnancy rates (see section 5). Perhaps the physiological cost of mounting an immune response against both brucellosis and tuberculosis exacts a major toll on bison, resulting in the observed decline in demographic rates.