8.0 DISCUSSION – DISEASE MANAGEMENT IN WBNP

The main focus of this thesis was to evaluate the potential of tuberculosis and brucellosis to have caused the observed decline in bison abundance in WBNP since the 1950s. I have used a building block approach where I evaluated bison metapopulation structure within WBNP, and then used these population units to evaluate epidemiological and demographic aspects of the bison, pathogen, and predation relationship. I have demonstrated several key aspects of the ecology of brucellosis and tuberculosis in the WBNP bison population. First, in chapter 4 I found that the prevalence of each pathogen has not been affected by the 5-fold decline in bison numbers in WBNP. Dobson and Meagher (1996) proposed that brucellosis is not sustained in populations less than 200 bison. I found that brucellosis prevalence in the Nyarling River population was similar to that in the Delta population, although the maximum number of bison counted in that population in the last 20 years was 236 animals. Bison population density must be very low for contact rates among bison to be reduced to the point necessary for brucellosis disappearance. Although I could not directly compare my tuberculosis prevalence estimates to previous surveys, I note that tuberculosis prevalence in1950-1970 was 10% higher in the Hay Camp population than the Delta population (Carbyn et al.1993), consistent with my estimated difference of 8%. This suggests that tuberculosis prevalence is relatively insensitive to changes in bison density. This is likely a consequence of the gregarious nature of bison. In general, I conclude that the relative insensitivity of tuberculosis and brucellosis prevalence to bison density will result in persistence of these pathogens at all but the lowest bison densities.

Second, I found that tuberculosis and brucellosis interact to affect the demography of individual bison (chapters 5 and 6). Bison in the Hay Camp and Delta populations with both tuberculosis and brucellosis had lower winter survival probabilities and reduced pregnancy rates relative to those with one or neither pathogen. The physiological mechanism behind this interaction is not clear, but I suggest that maintaining an immune response to both pathogens is energetically costly. I also found that tuberculosis-positive bison in the Nyarling River population had a reduced pregnancy rate relative to negative bison. I maintain that due to errors in disease testing, my estimate of the effect of these pathogens is constrained to be an underestimation of the true effect.

Third, I have evaluated predictions of the disease-predation hypothesis, and found that this hypothesis is a likely explanation for the decline of bison abundance in WBNP. The trend of population decline is not unique to the Delta population, and so I suggest that the cause of the decline cannot be ascribed to environmental conditions unique to the Peace-Athabasca Delta. I found that predation by wolves was greater in the Delta population than elsewhere in the park (chapter 6); however, I believe this is simply because other populations reached low densities where predation relaxed in advance of the Delta population. I expect predation will decline in the Delta population as well now that numbers are low. Simulation of population dynamics with and without exotic pathogens indicates a high probability that a population harbouring these pathogens will decline to low densities. In the absence of exotic pathogens or predation, a population will likely grow to some unknown high density even if the effects of anthrax and drowning are considered.

The potential role of tuberculosis and brucellosis in the ecology of bison in WBNP has generated much controversy since they were introduced (reviewed by Gates et al. 1997). W.A. Fuller (1962) asked the question, "Should further steps be taken to control tuberculosis, and if so, should the objective be merely control, or elimination?" (p. 41). He concluded that the effect of tuberculosis on the bison population was not sufficient to justify elimination of the pathogen, as it would require "virtual elimination of the bison." As there are no technologies presently available to eradicate brucellosis and tuberculosis without whole herd depopulation and repopulation (e.g., Wobeser 1994b; Cheville et al. 1998), forty years later, we are faced with the same dilemna. A depopulation program would cost many millions of dollars, would necessitate absence of bison from WBNP for several years, (FEARO 1990) and would be a major intrusion into a national park that may affect the perceived aesthetic value of a wilderness park. In other respects, there are significant costs to the *status quo.* Since 1962, the WBNP bison population has declined to a fraction of its former size; while other northern populations appear to be thriving. For example, there are five free-ranging, exotic pathogen-free wood bison populations in northern British Columbia, Alberta, and the Yukon and Northwest Territories, totalling almost 3000 bison (RENEW 2001). All of these populations are increasing in size; however, the National Recovery Plan for Wood Bison (RENEW 2001) lists the presence of exotic pathogens in and around WBNP as the single most important factor affecting further recovery of wood bison in northern

Canada. Not only are further reintroductions of bison prevented within a major portion of their range (Gates et al. 1994), but the Mackenzie Bison Sanctuary and Hay Zama populations are at risk of becoming infected (APFRAN 1998).

What is the answer to the dilemna posted by W.A. Fuller? I believe the answer lies in building consensus among members of the communities who are most affected by the disease issue. In this thesis I have built an argument that introduction of tuberculosis and brucellosis has resulted in the sustained decline in the WBNP bison population. The people who are closest to the bison must now weigh the costs of disease management against the costs of persistence of exotic pathogens in this system. It is clear that any decision to eradicate tuberculosis and brucellosis must be supported by local communities. It is only with the strength of a local consensus behind the decision that governments will be able to conduct a pathogen eradication program.

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APPENDIX A. DRUG DOSAGES USED TO CAPTURE AND HANDLE BISON

Table A1. Drug dosages used during capture and captivity while handling bison in Wood Buffalo National Park, 1997 and 1998. Mean dose from a single injection is indicated by age group; standard error and sample size follows in parentheses.

	Drug	Calves	Sub-adults	Adult females	Adult males
1997					
Day 1	Azaperone (μg/kg)	49.9 (2.37, 21)	49.9 (3.86, 42)	43.5 (0.70, 43)	52.2 (--, 1)
Day 1	Carfentanil (μg/kg)	--	--	5.0 (0, 3)	5.0 (0, 3)
Day 1	Xylazine/Rompun (μg/kg)	--	--	22.5 (0, 3)	63.1 (0, 3)
Day 1	Naltrexone (mg/kg)	0.8 (0.19, 2)	0.9 (0.06, 4)	0.7 (0.04, 2)	0.7 (0.04, 3)
Day 1	LAN (mg/kg)	0.6 (0.03, 22)	0.8 (0.04, 38)	0.8 (0.03, 39)	1.0 (--, 1)
Day 3	Carfentanil (μg/kg)	5.9 (0.46, 22)	5.6 (0.53, 42)	4.3 (0.08, 43)	5.0 (0.33, 3)
Day 3	Xylazine/Rompun (μg/kg)	99.7	63.1	47.8	63.1

	Drug	Calves	Sub-adults	Adult females	Adult males
		(10.57, 6)	(9.95, 5)	(5.04, 7)	(4.59, 3)
Day 3	Naltrexone (mg/kg)	0.7 (0.04, 15)	0.9 (0.08, 38)	0.6 (0.01, 37)	0.6 (0.05, 3)
1998					
Day 1	Azaperone (µg/kg)	64.1 (2.86, 21)	61.5 (1.47, 35)	56.5 (0.16, 51)	--
Day 1	Carfentanil (µg/kg)	--	--	--	7.2 (0, 7)
Day 1	Xylazine/Rompun (µg/kg)	--	--	--	82.3 (0, 4)
Day 1	Naltrexone (mg/kg)	--	--	--	0.9 (0.04, 7)
Day 1	LAN (mg/kg)	0.4 (0.02, 21)	0.5 (0.01, 35)	0.5 (0.01, 51)	--
Day 3	Carfentanil (µg/kg)	3.4 (0.13, 21)	3.7 (0.10, 34)	3.6 (0.07, 48)	6.9 (0.26, 7)
Day 3	Xylazine/Rompun (µg/kg)	59.2 (2.48, 17)	56.2 (2.55, 24)	44.9 (1.31, 40)	82.5 (3.04, 7)

	Drug	Calves	Sub-adults	Adult females	Adult males
Day 3	Naltrexone (mg/kg)	0.5 (0.02, 21)	0.6 (0.02, 33)	0.6 (0.02, 47)	1.0 (0.03, 6)
Day 3	Tolazoline (mg/kg)	1.3 (0.07, 21)	1.9 (0.07, 31)	1.8 (0.04, 42)	--

Table A2. Drug dosages used for the capture and captivity techniques to handle bison in Wood Buffalo National Park, 1997 and 1998 (bison with multiple injections). Mean dose from each injection is indicated by age group; standard error and sample size follows in parentheses.

		Subadults		Adult Females			Adult Males	
	Drug	1st Injection	2nd Injection	1st Injection	2nd Injection	3rd Injection	1st Injection	2nd Injection
1997								
Day 1	Azaperone (µg/kg)	53.8 (--, 1)	--	48.1 (2.10, 2)	--	--	--	--
Day 1	Carfentanil (µg/kg)	--	--	7.5 (--, 5)	--	--	5.1 (0.65, 5)	3.9 (2.99, 2)
Day 1	Xylazine/Rompun (µg/kg)	--	--	75.4 (--, 1)	--	--	55.3 (5.44, 4)	60.5 (11.47, 2)
Day 1	Naltrexone (mg/kg)	--	--	1.0 (--, 1)	--	--	0.7 (0.13, 5)	--
Day 1	LAN (mg/kg)	--	--	1.0 (0.05, 2)	--	--	--	--
Day 3	Carfentanil (µg/kg)	4.6 (--, 1)	1.7 (--, 1)	5.6 (1.17, 2)	5.6 (1.17, 2)	--	4.6 (0.37, 4)	4.6 (0.52, 3)
Day 3	Xylazine/Rompun (µg/kg)	--	--	75.4 (--, 1)	--	--	56.5 (4.68, 4)	58.2 (6.17, 3)

		Subadults		Adult Females			Adult Males	
	Drug	1st Injection	2nd Injection	1st Injection	2nd Injection	3rd Injection	1st Injection	2nd Injection
Day 3	Naltrexone (mg/kg)	1.1 (--, 1)	--	1.2 (0.34, 2)	--	--	0.9 (0.04, 4)	--
1998								
Day 1	Azaperone (µg/kg)	62.7 (3.96, 3)	--	57.7 (1.34, 5)	--	--	--	--
Day 1	Carfentanil (µg/kg)	--	--	--	--	--	6.1 (0.36, 6)	6.5 (1.63, 2)
Day 1	Xylazine/Rompun (µg/kg)	--	--	--	--	--	68.4 (6.08, 4)	--
Day 1	Naltrexone (mg/kg)	--	--	--	--	--	1.0 (0.11, 6)	--
Day 1	LAN (mg/kg)	0.6 (0.05, 3)	--	0.5 (0.01, 5)	--	--	--	--

		Subadults		Adult Females			Adult Males	
	Drug	1st Injection	2nd Injection	1st Injection	2nd Injection	3rd Injection	1st Injection	2nd Injection
Day 3	Carfentanil (μg/kg)	3.7 (0.33, 3)	2.5 (0.68, 3)	3.7 (0.15, 5)	1.8 (0.74, 3)	--	6.0 (0.35, 6)	5.8 (0.54, 4)
Day 3	Xylazine/Rompun (μg/kg)	40.9 (--, 1)	40.9 (--, 1)	42.7 (4.98, 4)	61.4 (6.07, 4)	62.2 (--, 1)	70.9 (4.21, 6)	69.1 (6.40, 4)
Day 3	Naltrexone (mg/kg)	0.7 (0.12, 3)	--	0.6 (0.06, 4)	--	--	1.1 (0.17, 6)	--
Day 3	Tolazoline (mg/kg)	2.0 (0.18, 3)	--	1.9 (0.10, 5)	--	--	--	--

Table A3. Mean naltrexone:carfentanil ratio (standard error, sample size) used in handling bison (single and multiple injections on days 1 and 3 of capture) in Wood Buffalo National Park, 1997 and 1998.

	Injection	Calves	Subadults	Adult Females	Adult Males
1997					
Day 1	Single	--	--	133.3	139.0
				(0, 2)	(8.3, 3)
Day 3	Single	141.1	155.8	147.9	127.2
		(8.1, 15)	(3.0, 38)	(2.0,36)	(1.0, 3)
Day 1	Multiple	--	--	133.3	122.0
				(--, 1)	(9.4, 5)
Day 3	Multiple	--	166.7	101.9	125.6
			(--, 1)	(9.3, 2)	(18.1, 4)

	Injection	Calves	Subadults	Adult Females	Adult Males
1998					
Day 1	Single	--	--	--	130.6 (2.0, 7)
Day 3	Single	140.5 (4.0, 21)	151.8 (3.5, 33)	163.6 (2.8, 47)	146.8 (2.5, 6)
Day 1	Multiple	--	--	--	132.0 (13.2, 6)
Day 3	Multiple	--	119.8 (2.8, 3)	154.1 (17.1, 4)	115.1 (14.3, 6)

APPENDIX B. DISCUSSION OF CAPTURE-RELATED MORTALITY

B1. Capture and captivity

There were nine acute (i.e., within 3 days of capture) mortalities in the three years of the study. In 1997, an adult female bit off her tongue during capture, severing it approximately 15 cm posterior to the tip. The bison was euthanized as I concluded she would not have been able to forage without a tongue and would starve. The second mortality was an adult female who died in the pen two days after capture. The bison was found in right lateral recumbency and had large amounts of internal fat deposits suggesting the animal was not in nutritional stress. This individual was among the first few bison handled, and among those few that were administered a general sedative (carfentanil and xylazine) at the corral, and after the sedative was reversed, the bison had to be aided in returning to it sternum. Haigh and Gates (1995) recommend a Naltrexone to Carfentanil ratio of at least 125 mg Naltrexone: 1 mg Carfentanil when reversing a sedated animal. This animal received a more conservative ratio of 133 mg Naltrexone: 1 mg Carfentanil, therefore re-narcotization is not likely to have been the cause of death. A slight pale colour in the semitendinosis muscle as well as slight edema found between the semimembranosus and semitendinosis muscles is consistent with reported symptoms of capture myopathy in bison, pronghorn antelope, and elk (Hudson et al. 1976, Chalmers and Barrett 1977, Lewis et al. 1977). The third acute mortality in 1997 was an adult female who appeared normal at time of capture. She arrived dead at the pen after a four minute transport. The bison had excellent fat deposits. Both the lungs and heart appeared normal, and no gross signs of capture myopathy were visible.

In 1998 there four acute mortalities. In the first case an adult female was found in sternal recumbency in the pen. She had melted through the ice in the bottom of the pen, and was found sitting in approximately two feet of water. Although still alive, she was slightly hypothermic (body temperature 36.5ºC). She died the following day. I suspect that she was suffering from capture myopathy and was unable to stand up. Because of this mortality, the placement of pens was changed; subsequently, pens were only located where there was evidence of active bison feeding as an indication that the ground was solid under the snow. In the second case a female yearling was found dead in the pen on the third day of captivity. She was in right lateral recumbency. It appears as if she became "cast" (i.e., on it side) and was unable to right herself. The cause of death was aspiration of rumen contents. The third mortality, that of an adult female, was also a consequence of capture myopathy. Although she was able to walk with difficulty upon release from the pen, in two days she had only moved 15 metres away from the pen. It rear legs were dysfunctional, and she was euthanized to prevent further suffering. All of these mortalities occurred in animals that were captured and handled using the net gun and captivity technique. Overall, 2.5% (6/236) of the bison handled with the capture and corall technique suffered acute mortality (i.e., within three days of capture).

There were 11 bison mortalities within 60 days of capture in 1997. Three bison were killed by wolves within 10 days of release from the pen. Insufficient remains were present upon investigation to determine whether these bison suffered from capture myopathy; however, to be conservative I assume these deaths were related to capture.

Four bison mortalities were discovered first telemetry flight after the capture operation (April 6, 1997), so these mortalities were detected 32-37 days after release from the pen. In the first case a pack of wolves was found present on the carcass upon location 37 days after release. The carcass was not yet fully frozen indicating a recent (< 2 days) mortality, and there was evidence of a chase sequence in the snow as well as other evidence of a struggle. I conclude that wolves were likely the proximate source of mortality, and due to the time elapsed since release from the pen (35-37 days) I do not believe capture was involved in the mortality of the bison. In two of the four cases, evidence of chase and struggle and disarticulation of skeletal material also indicated that wolves were the proximate source of mortality. Insufficient remains were found to determine timing of mortality, therefore I cannot exclude after-effects of capture. The fourth bison was determined to be dead on an aerial survey 33 days after release from the pen; however I was unable to conduct a ground investigation until much later in the summer so I do not know the time or cause of death of this bison. I cannot exclude, and therefore assume, a capture-related source of mortality for this bison. All three of the remaining bison had been located alive in herds of 21-50 bison 49-52 days after release. One of these apparently drowned during a spring flood along the Peace River, and the cause of death could not be determined in the other two bison. I conclude that capture was not implicated in the mortality of these three bison.

There were eight mortalities of female bison within 60 days of release from the pen in 1998. The first was a four year old female discovered five days after release from the pen. The carcass was found intact indicating that predation had not occurred. Logistical reasons prevented a full necropsy of this individual; however, I strongly suspect that this bison died as a consequence of capture-related stress, possibly capture myopathy. The second individual, a 17 year old female, was discovered six days after release from the pen in lateral recumbency. It colon serosa was dark red and the serosal surface exhibited ecchymoses. The colon contents were hard and dry, and copious quantities of fibrin was present in the colon and cecum. In addition, about 400 ml of straw coloured fluid was present in the abdomen. No fecal matter was present in the body, and only raisin sized fecal pellets were located in the vicinity of the carcass. The cause of death was determined to be colonic impaction related to dehydration during captivity. The third bison, a two year old female, was discovered 12 days after release from the pen. The carcass was discovered in lateral recumbency, and there was evidence of thrashing as if she was attempting to stand. Although I could not conduct a full necropsy, I conclude that this bison died of capture-related stress. The fourth bison was discovered within 10 days of release from the pen, and on investigation it was determined that wolf predation was the proximate cause, although I do not exclude capture-related stress due to the short time since release. The fifth bison, a subadult female, was discovered 15 days after release from the pen, clearly having been killed by wolves. Straw-coloured fluid had been extracted from the left carpal joint at capture, and she had a very high serological titre for brucellosis (complement fixation assay titre 1:5120). I cannot exclude capture-related stress due to the short time since release, although the presence of an active brucellosis infection and associated lesions may have predisposed it to wolf predation. Two bison were discovered dead 45 days after release from captivity, both 13 year old females. I could not determine if the first bison was killed by wolves or scavenged post mortem, although wolves had almost completely consumed the carcass. As this bison had not been relocated since release from the pen and so time of death is unknown, I cannot exclude capture related mortality. In the second case the carcass was discovered in 45 cm of water from the spring melt. The carcass was disarticulated, with the bones scattered over a large area. Although the spring melt had washed away any other evidence of a kill, I believe that wolf predation was the proximate cause of mortality in this case. This bison gave no indication of being in distress upon release, and was relocated alive 24 days after release in a large (21-50 animals) herd of bison. Therefore, I strongly believe that this bison died of natural predation unrelated to capture.

I excluded male bison less than one year of age in this analysis due to small sample sizes and a lack of a control. However, two of six male calves were killed by wolves in 1998. Upon first relocation 15 days after release, the first calf was located alive in a small herd of bison. On the next flight 10 days later the bison was found to have been killed by wolves (indicated by a chase sequence in the snow). Bison of this age experience high rates of mortality (e.g., compare calf:cow to yearling:cow ratios in Carbyn et al. 1998); and so I believe this to be natural predation. In the second case of mortality among this age class, the male calf was found to have been killed by wolves on a survey 15 days after release. I cannot exclude a capture-related cause of mortality in this case. There were no mortalities among eight male calves handled with this technique in 1997.

B2. Net-gun

There were no mortalities among bison handled with this technique in 1998 (n = 11). In 1999, an adult female was captured using a net gun and released after handling. While processing, wolves were heard howling in the vicinity of the capture site. She was found dead two days after release approximately 20 km from the capture site, clearly having been killed by wolves (e.g., a chase sequence and signs of a struggle were visible). While this mortality occurred within 3 days of capture, I elected to treat this mortality in the analysis as a "chronic" mortality as the distance travelled from the capture site (20 km) suggests she did not suffer from capture myopathy. One other mortality occurred within 60 days of capture using this technique: a subadult female was relocated dead eight days after release. The mortality was in a remote location and so I could not investigate the mortality until late spring, at which point I was unable to determine the cause of mortality. I suspect capture myopathy played a role in this mortality.

B3. Darting

Two adult males died within three days of capture (2/34 or 5.8%). In the first case, an adult male appears to have tread on thin ice a day after release from the first handling event, fallen through and drowned. I cannot exclude re-narcotization as a possible factor in this mortality, although nearby snowmobile tracks suggest that he was startled and consequently ran on to the thin ice. In the second case, an adult male died after chemical immobilization with carfentanil. The bull showed no sign of sedation 11 minutes after the first dart was injected, and so a second was fired. Five minutes after the second dart the bull laid down and after fifteen minutes (while another male was being processed) the bull died. The bull was in good physical condition, although lesions consistent with tuberculosis as well as brucellosis were found internally. The immobilizing drug carfentanil has a wide margin of safety in bison and so I would not expect the cause of death to be a drug overdose. Consequently, I conclude that the cause of death is acute capture myopathy.

There were two mortalities among darted adult males that occurred within 60 days of capture (n = 1 in 1998 and 1999), or 9.5% of bulls handled in this matter (2/21). In 1998, a 14 year old bull was darted and reversed uneventfully (naltrexone: carfental ratio 142:1). He was in poor body condition at capture (i.e., had poor fat deposits, body condition score 1/5). He was relocated alive 12 and again 22 days after release alive; however, he was discovered dead 31 days after release. The carcass was intact (i.e., not a wolf kill), although some scavenging had occurred. I conclude that although capture may have accelerated the process of mortality, this bull had poor survival prospects during the most difficult time of year for bison (late winter). The second mortality was of an adult male 10 days after release in 1999. The first day capture was relatively uneventful; two doses of carfentanil and xylazine (4 mg of carfentanil and 50 mg of xylazine) were required to immobilize the bison although this may have been the result of poor placement of the first dart. I did have to inject another 60 mg of xylazine to maintain sedation during handling. He was reversed with naltrexone and tolazoline uneventfully. The third day capture was more difficult, as two darts of carfentanil and xylazine as well as another 300 mg of xylazine were required to maintain sedation. He was injected with 24 cc of each of tolazoline and naltrexone to reverse the xylazine and carfentanil respectively after handling. He was relocated dead 10 days after release, and the necropsy conducted 5 days later revealed that he had been killed by wolves. As wolves seem to rarely target adult males, I believe that re-narcotization may have occurred and contributed to the death of this male.