BRUCELLOSIS AND TUBERCULOSIS AS FACTORS LIMITING POPULATION GROWTH OF NORTHERN BISON

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by

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ABSTRACT

Tuberculosis (*Mycobacterium bovis*) and brucellosis (*Brucella abortus*) were introduced to Wood Buffalo National Park (WBNP), Canada in the late 1920s. In the last thirty years, the bison population has declined to less than 25% of its former size. My main objectives were to assess:

1) prevalence of tuberculosis and brucellosis in bison;

2) the impacts of tuberculosis and brucellosis on bison reproduction and survival

3) the disease-predation hypothesis as the cause of population decline.

Serological testing indicated disease prevalences of 49% and 31% for tuberculosis and brucellosis, respectively, in 1997 - 1999. Prevalence for both diseases increased with age, and males were more likely to test positive for tuberculosis. Historical data from WBNP indicate that prevalence of neither disease is a direct function of bison density. These diseases are endemic and unlikely to disappear as the population of bison in WBNP declines.

Brucellosis and tuberculosis interacted to affect survival and reproduction of bison. Among female bison captured in the Delta and Hay Camp populations, bison that tested positive for tuberculosis and had a high titre for brucellosis were less likely to be pregnant relative to bison with one or neither disease. In the Nyarling River population, bison tuberculosis was associated with a significant reduction in pregnancy rate. Annual survival rate varied within the park, with annual survival being lowest in the Delta population and highest in the Nyarling River population. Wolf predation rate on bison > 1 year of age was highest in the Delta population, but was a minor source of mortality

elsewhere in the park. Tuberculosis-infected bison with a high brucellosis titre were 2.5 and 3.7 times more likely to die during early and late winter, respectively, than other bison.

Stochastic population simulation indicated that in the absence of tuberculosis and brucellosis, there is a high likelihood of bison persisting at high densities. In contrast, tuberculosis and brucellosis resulted in a high probability of bison persisting at low densities. I conclude that the decline in bison abundance in WBNP can be attributed to the presence of tuberculosis and brucellosis, and that the population will likely persist at low densities with the continued presence of these introduced diseases and natural levels of wolf predation.

ACKNOWLEDGEMENTS

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Special thanks are due to Amanda Plante and Bonita Wong as well as Tim Evans, Trevor Evans, and Michelle MacDonald for field assistance. Vern Neal, Morris Boucher, Willie Courtoreille, and Danny MacDonald were the community representatives for the project. Todd Shury was a critical member of the capture team. I owe thanks to the staff of WBNP as well as the people of Fort Smith and Fort Chipewyan. Big River Air (Fort Smith), Helicopter Capture Services, and Helicopter Wildlife Management (both of Salt Lake City) were incredible in their dedication to this project.

I was told before I came to work with Dr. Messier that he did not tolerate "tomfoolery." Accordingly, I must convey gratitude to my supervisor for years of financial support and academic guidance, and the occasional "tomfoolery"-correction. I also thank Drs. Ramsay (d.), Leighton, Neal, and Bortolotti for their advice and support. My student colleagues created a stimulating environment: Terry Armstrong, Kim Brown, Usne Butt, Candace Elchuk,. Steve Ferguson, David Gummer, Yeen Ten Hwang, Serge Larivière, Phil McLoughlin, Brent Patterson, Jim Rettie, and Lyle Walton.

Most importantly, my family has all sacrificed so that I could be a student: Celina, Hugh, Aidan, Guy, Trish, Nichole, Hugues, Suzanne and Jamie. I can never repay that debt.

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1. INTRODUCTION

Between 1925 and 1929, plains bison (*Bison bison bison*) were relocated from Wainwright Buffalo Park to the newly created Wood Buffalo National Park (WBNP; Soper 1941; Figure 1.1). This measure was undertaken to relieve overcrowding at Wainwright, and "preserve the surplus for future economic purposes in northern regions" (Soper 1941:375). Over 6,000 bison were shipped and "released at several points along the eastern park boundary below the junction of the Peace (River)" (Soper 1941), although many are reported to have perished on route or euthanized upon arrival (Carbyn et al. 1993:27). The plains bison mixed with the indigenous wood bison (*Bison bison athabascae*), contrary to prior expectations (Graham 1924). By 1934 the population numbered approximately 12,000 bison (Soper 1941). Bovine brucellosis (*Brucella abortus*) and tuberculosis (*Mycobacterium bovis*) were discovered in WBNP in the 1950s (Corner and Connel 1958, Fuller 1959a). The two diseases were likely introduced with the bison from Wainwright National Park (Tessaro 1986). Seventy-years later, the diseases maintain enzootic proportions in the population (Joly et al. 1998).

Bison in Wood Buffalo National Park have experienced a sustained decline in numbers, dropping from approximately 11,000 bison in 1970 to 2,300 bison in 1997 (Figure 1.2; Carbyn et al. 1993, 1998; WBNP unpublished data). The most extensive study on this ecosystem concluded "wolf predation was the single most important direct

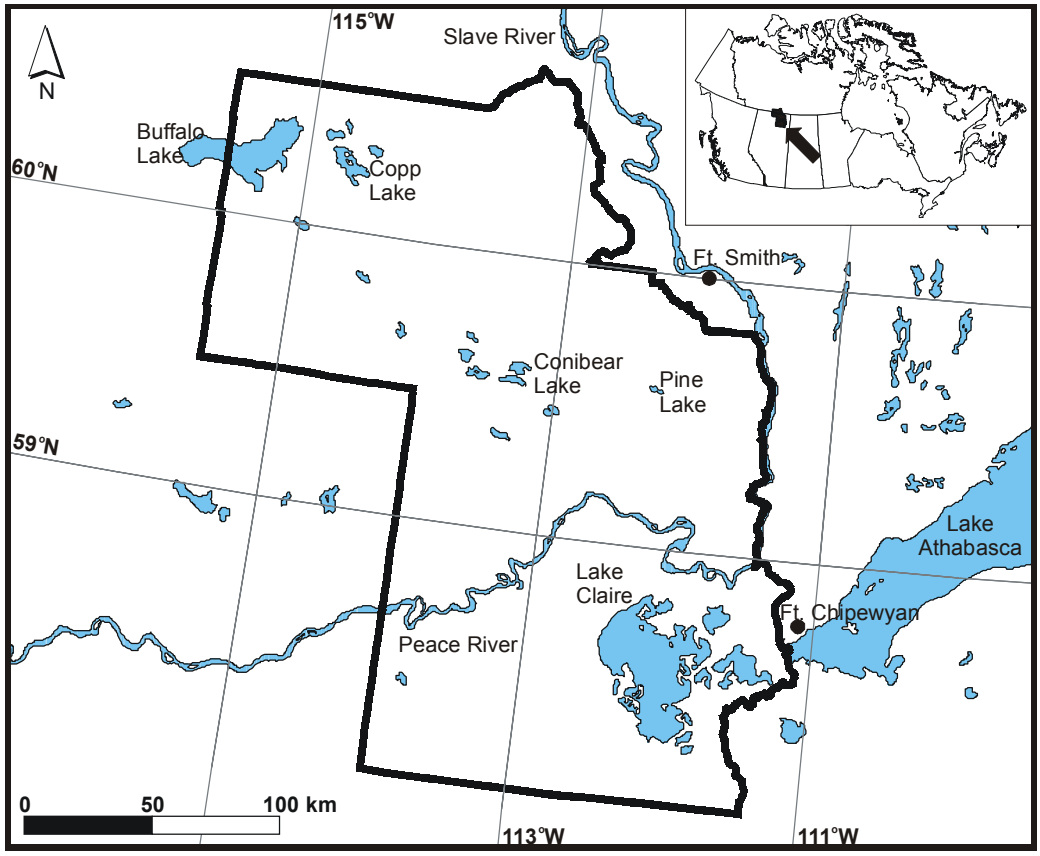

Figure 1.1 Wood Buffalo National Park, Canada.

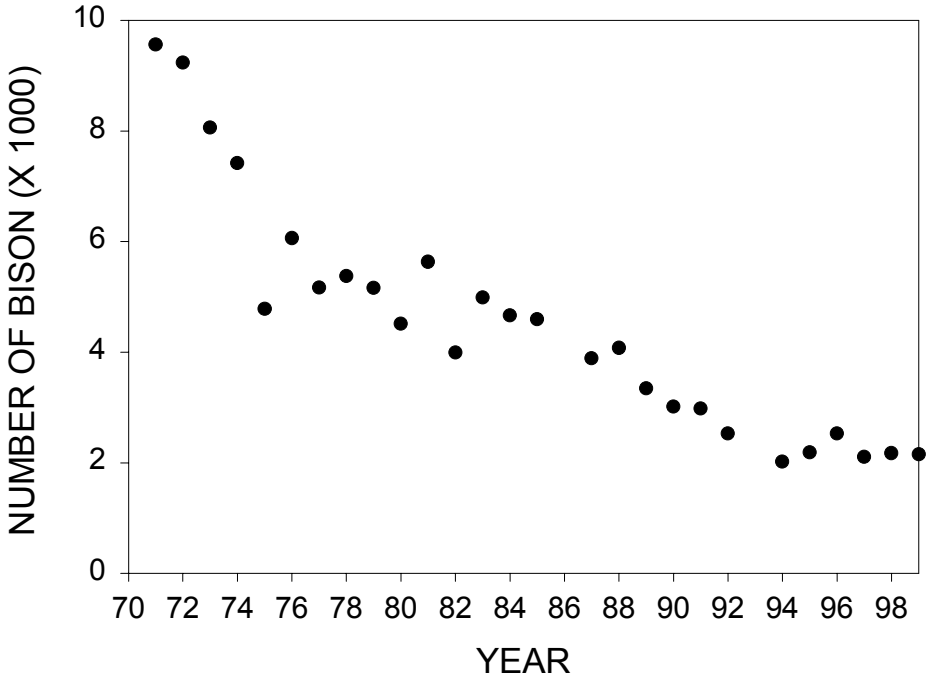

Figure 1.2. Change in bison abundance in Wood Buffalo National Park. 1971-1974 data include only those bison counted in "primary ranges" (see Table 7.1)

cause for the observed and well documented decline of bison in Wood Buffalo National Park" (Carbyn et al. 1993:235). These authors postulate that drying of the Peace-Athabasca Delta, as a result of modification of water flows by the W.A.C. Bennett Dam, is causing bison in the Delta to clump in spatially predictable groups and thus experience abnormal levels of predation by wolves (*Canis lupus*). Disease was ruled out as the underlying cause of the population decline, as although disease is distributed throughout the park, some areas have not experienced the same level of population decline (Carbyn et al. 1993).

In contrast to this view, the available literature suggests that both brucellosis and tuberculosis could have potentially dramatic impacts on bison population demography. Tessaro et al. (1990) examined 72 bison found dead in and around WBNP, and reported that 6 died from causes related to disease. Abortion caused by brucellosis has been reported in bison from field (Williams et al. 1993; Rhyan et al. 1994) and experimental conditions (Davis et al. 1990). Messier and Blyth (1995) demonstrated that the reduction in population growth rate from disease, coupled with the effects of wolf predation, could shift bison from a population density near food carrying capacity to a density less than 10% of the food carrying capacity. Diseases could impair reproduction and, through the effects of debilitation, predispose bison to predation by wolves (Tessaro et al. 1990).

The effect of infectious diseases on the population dynamics of wildlife is poorly understood (Yuill 1987; Gulland 1997). Those diseases that cause major die-offs have long been recognized as important factors in demography (e.g., rinderpest, Sinclair 1979; Prins and Weyerhaeuser 1987). However, due to their chronic nature diseases such as

bovine tuberculosis and brucellosis are particularly poorly understood (e.g., Rodwell et al. 2001). The lack of knowledge regarding these diseases has hampered wood bison recovery efforts. In particular, uncertainty surrounding the role of these diseases in the bison decline has lead to a conservative approach to recovery of bison in northern Canada. Gates et al. (1994) proposed large portions of the historic range of wood bison is unavailable for reintroduction of wood bison for fear of infection through contact with the WBNP population. There is also a concern that contact with the WBNP population will spread tuberculosis and brucellosis to the disease-free wood bison of the Mackenzie Bison Sanctuary (Gates et al. 1994; Tessaro et al. 1993).

In 1990, a Federal Environmental Assessment Review Panel recommended depopulation of bison in Wood Buffalo National Park and reintroduction of disease-free bison (Federal Environmental Assessment and Review Office 1990). However, the Northern Bison Management Review Board (1992) recommended further research to facilitate management decisions. In response to this recommendation, the Minister of Canadian Heritage announced in 1995 a five-year Bison Research and Containment Program (BRCP), with a mandate to "support research efforts that work towards a better understanding of bison ecology within the greater WBNP ecosystem". The first research component of this program forms the core of my thesis.

There are four specific objectives to this research:

1) to assess the prevalence of tuberculosis and brucellosis in bison in Wood Buffalo National Park;
2) to assess the impact of brucellosis and tuberculosis on bison reproduction in Wood Buffalo National Park;

3) to assess the impact of brucellosis and tuberculosis on bison survival in Wood Buffalo National Park; and,

4) to determine how the bovine diseases interact with wolf predation to affect bison population dynamics.

The basic premise of this research project is to use standard disease testing methods to estimate the infection status of individual bison, and then compare demographic rates among bison that test positive or negative on these tests. At the outset I would like to state two major limitations of this approach. First, no test is 100% accurate at determining whether a particular animal has a disease or not. All tests used in this research evaluate an animal's immunological response to challenge with pathogen antigens. This response can vary as a complex function of the time since first exposure to the pathogen, age, nutritional status, and stage of infection among other factors. Further, the tests used in this study were initially designed to detect infection in domestic cattle at the herd level. Specifically, in a program designed to eradicate brucellosis or tuberculosis from a domestic ungulate herd, it is only necessary to demonstrate that the disease is present in the herd. In most of these programs, the entire herd is destroyed once the pathogen is detected, regardless of individual infection status. In the case of brucellosis, the accuracy of these tests has only recently been evaluated in bison, and even then only in a preliminary fashion. I have no controlled studies evaluating the accuracy of the tuberculosis test in bison. Errors in testing in the present study would result in an underestimation of the effect of disease on a demographic parameter (Figure 1.3). Therefore, I view all results here as conservative (i.e., the effects of tuberculosis or brucellosis are likely greater than the effects presented herein).

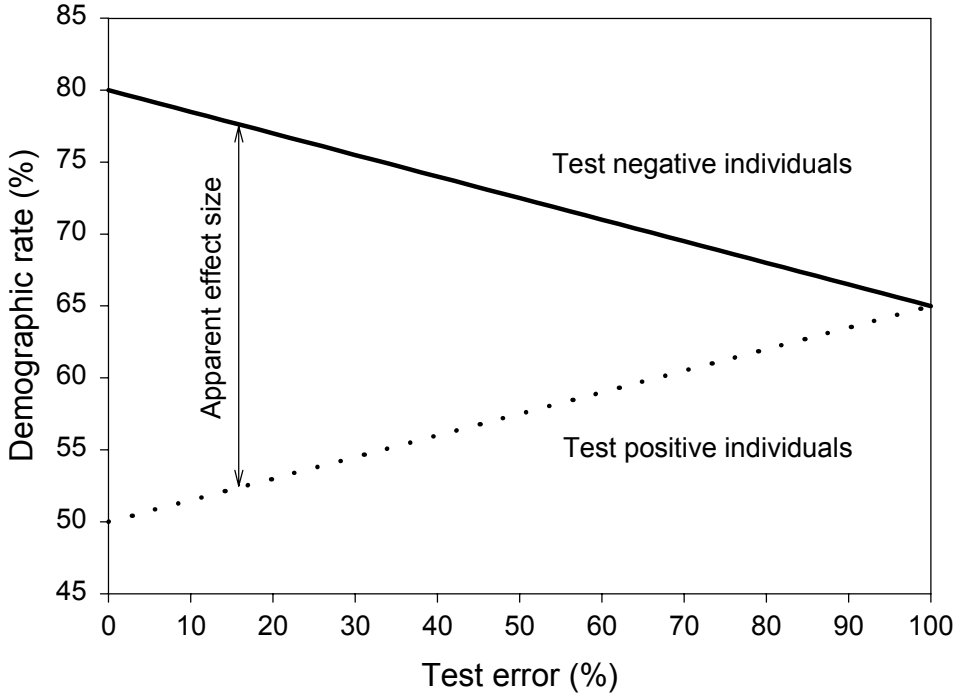

Figure 1.3. Relationship between errors in disease testing and estimates of the effect of disease on demographic parameter of the host. The effect size is the vertical distance between the apparent demographic rates for test positive (dotted line) and test negative (solid line) individuals. Percent test error is calculated as the sum of proportions of individuals that the test incorrectly classifies as diseased or healthy. This figure assumes a parametric demographic rate of 50% and 80% for test positive and negative individuals, respectively.

The second limitation involves the definition of a "disease." The concept of disease encompasses "any impairment that interferes with or modifies the performance of normal functions, including responses to environmental factors such as nutrition, toxicants, and climate; infectious agents; inherent or congenital defects, or combinations of these factors" (Wobeser 1981). In this definition, infection by a pathogen does not necessarily equate to "disease" unless the pathogen is impairing a biological function. Often, the relationship between infection and disease is complex, subtle, and consequently poorly understood (e.g., Yuill 1987). This is particularly true for tuberculosis and brucellosis, where the course of the infection is chronic and insidious.

My study was designed to estimate the infection status of an animal based on their immunological response to a challenge by a disease antigen, and then correlate this response to demographic parameters such as survival or reproduction. However, my tests hinge on the immunological response, which may or may not correlate well the actual stage of infection. Similar to HIV in humans, an individual may harbour the bacteria but not actually be experiencing an impairment of function. The difference between HIV in humans and these bacterial diseases in bison is that the human physician is able to evaluate the actual degree of impairment of immune system function, whereas in this study I only know whether the immune system of a bison has been exposed to tuberculosis or brucellosis in their history. There is no measure of actual impairment of function at the individual level. The inability to distinguish between infected bison with and without pathological signs results in a further underestimation of the effect of infection on demographic parameters, similar to that described above. This complication

increases my belief that the results presented here are conservative, at least for bison with pathological signs.

I have structured this thesis so that each chapter builds on previous chapters. First, I evaluate the degree to which capture and handling affects survival of bison. This is very important, as a major focus of this study is to develop unbiased estimates of bison survival. I must be able to quantify the degree to which my methods influence the results of this study. Second, I evaluate bison metapopulation structure within WBNP. All aspects of the study require a good understanding of how bison arrange themselves in a population structure. As I demonstrate below, arbitrary classification of bison into populations can result in erroneous estimates of population demographic parameters such as population growth rates. Third, I evaluate factors affecting the distribution of brucellosis and tuberculosis in the bison population. The disease status for individual animals is then used in subsequent chapters to evaluate the effect of disease on survival and reproduction. Finally, I build a stochastic population model to evaluate the potential of brucellosis and tuberculosis to alter population densities of bison in WBNP.