Response to the Critique of Brucellosis in Captive Bison

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RESPONSE TO LETTER TO THE EDITOR . . .

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In their extensive review, Drs. Meagher and Meyer had several criticisms of our 1990 paper titled "*Brucella abortus* in Captive Bison. I. Serology, Bacteriology, Pathogenesis, and Transmission to Cattle," Journal of Wildlife Diseases 26: 360–371. Some of the minor criticisms which they enumerate are valid but were beyond our control, such as the small numbers of animals in the experimental groups. Because of budgetary restraints, we rarely had the number of animals preferred to include in experiments, but we did our best to answer as many questions as possible with the available resources. Other valid criticisms such as the error in the literature cited (Davies et al., 1980) were under our control but were just human error that escaped us and the editorial process. Rather than listing and addressing each criticism, for the sake of brevity, we will simply cover the main issues elaborated in their review.

The main objection raised by Drs. Meyer and Meagher seems to be the dose of *Brucella abortus* strain 2308 used to challenge the bison (*Bison bison*); in their opinion, this "was a severe overdose." We find this criticism particularly confusing on several counts. First of all the challenge dose of 1×10^7 colony forming units (CFU) and the organism of *B. abortus* strain 2308 were selected at a meeting of acknowledged brucellosis and wildlife experts including Dr. Tom Thorne in 1984 in Helena, Montana (USA). Also in attendance and participating in the discussion at the same meeting was Dr. Mary Meagher. She voiced no objections to the proposed challenge dose or design at that time but seems very concerned 10 yr later. If she will review her notes or the official minutes of that meeting Dr. Meagher also will recall that the Texas A&M University researchers at that meeting suggested and requested that the dose infecting 50% of the test animals (ID_{50}) for bison should be established prior to the experiment in question, but the majority of the committee decided that "it would be a waste of time and money" and that the challenge dose previously used for cattle and elk (*Cervus elaphus*) would suffice. But returning to the issue of the "massive" challenge overdose, the outcome of the pregnancies of the 12 challenged bison are listed in Table 2 of our publication. As one can readily see, six of 12 of the challenged bison did not abort and two of 12 did not even become infected. These results are not what one would expect in the face of a "severe overdose." Also the decision to use a dose of 1×10^7 CFU to challenge the bison was based partially on the desire to be able to determine the susceptibility of the bison to a dose commonly used in cattle. Dr. Meyer offered examples of challenges utilizing lower doses in cattle in the critique, but Nicoletti (1990) provides a dozen examples of challenges of 1×10^7 CFU in cattle by several authors. Dr. Meyer should be aware of these data because she is an author of another chapter in the same book. No one to our knowledge has established an ID_{50} for bison. After the experiment under discussion, however, we continued to use 1×10^7 CFU to challenge bison in four more brucellosis experiments at Texas A&M University. During those four additional experiments, a total of 75 non-vaccinated control bison were challenged; 18 of the 75 bison did not abort and six of the 75 totally resisted infection. These data again would not support the case for "a severe overdose" as Drs. Meyer and Meagher claim. If, however, someone can provide us with data which establish the "proper" or "natural" challenge dose for bison, we certainly will consider using it in the future.

The other major objection by Drs. Mey-

er and Meagher seems to be centered around the use of "captive" animals and why the experimental results "are at such odds with the manifestations of the disease as it occurs in free-ranging bison." If their criticism of the use of "captive animals" is based on the assumption that "captive" bison differ genetically and are under conditions of stress, and therefore differ immunologically from their "free-ranging" cohorts, then in some cases the criticism might be valid. Wild bison in Yellowstone National Park (YNP), Wyoming (USA), for example, does experience winter stress, nutritional stress, predation, and concomitant disease that could affect their individual response to brucellosis in a manner not parallel to that in captive bison. These factors were held at a minimum or controlled in the captive bison in our experiments. Thus, the results in captive bison should be viewed as a best case scenario, because the effects of brucellosis in stressed "free-ranging" animals would be expected to be more severe. As to the issue of any possible genetic and therefore immunologic differences between "free ranging" bison and the bison in our studies, all of the bison used in the first three experiments of our brucellosis research were from a commercial bison herd from northeastern Wyoming. This privately owned bison herd of approximately 2,000 animals has been on that location since 1964 when over 570 bison trapped in YNP were transported to the ranch as the foundation of the present commercial herd. The bison cows used in our brucellosis research were from a substantial subset of the YNP herd that had been out of the park less than 10 to 15 yr. In retrospect, it is no surprise that the two groups of bison (YNP and our research animals) have not differed significantly on any genetic testing technique (Stormont, 1987; Stormont and Morris, 1992) including those at the mitochondrial DNA sequence as determined by the technique of Strobeck et al. (1993). The vast majority of bison presently in YNP descended from a captive breeding herd that originated from three bison bulls from the Goodnight herd in Texas (USA), 18 bison cows from another privately owned herd in Montana (USA). Hundreds of offspring from the captive breeding herd were released over the years to mix with the 25 "free-ranging" bison in YNP. Therefore, the last "free-ranging" bison herd has over 98% of its genetic roots in captive or ranched bison. These data would support the use of "captive" bison as excellent experimental models for those "free-ranging" bison.

There are features in the dynamics of brucellosis in wild populations of bison that are not observed in most populations of infected cattle. In discussing or studying the differences in the manifestations of the disease, one also should consider the differences in the population structure of each. The YNP bison herd is an excellent example. Males comprise nearly one-half of the bison population in all age categories, the age structure of the bison population includes a large proportion of animals over 5-yr old, and calf survival of bison is extremely low when compared to a commercial cattle herd. Adult intact males in cattle herds generally are less than 5 to 10%, very few cattle are kept in the herd for more than 5 to 6 yr, and calf survival commonly can exceed 90%. With such major differences in population structure and composition between "free ranging" bison and commercial cattle, it is to be expected that results of serologic studies for *Brucella* specific antibodies found in nonrandom subsets of unmanaged bison herds will differ from those observed in cattle herds where most animals are sampled.

We did not suggest that our research on brucellosis in bison is designed to definitively answer all the questions on this issue. Our research was limited in scope by budget and design to document some aspects of the disease in bison and to offer scientists some data on which to base decisions rather than "I think and I believe" and not be limited to anecdotal or observational science. Nothing that has been subsequently

observed in free-ranging bison has altered the results or conclusions of our research. Bison, like cattle and elk, are susceptible to brucellosis. In the opinion of Drs. Meyer and Meagher, "under natural (field) conditions, brucellosis of bison is not mimetic of bovine brucellosis." The generalized term "bovine brucellosis" used in this context has little relevance; while there are subtle differences to the disease within and between each host species, the range of the differences between bison and cattle in response to *B. abortus* appears to be no greater than those documented among breeds of cattle. There are differences to be sure. For example, most bison are not as capable of resisting Strain 19 vaccine as most cattle are capable of resisting. However, the outcomes of the disease in both bison and cattle are most similar in the field or under experimental conditions. Once infected with *B. abortus*, bison generally react to the organism both in humoral and cellular responses in a fashion similar to that seen in most cattle. Most pregnant bison exposed to an appropriate dose at the proper time will, like most pregnant cattle, abort. Most infected bison can be properly diagnosed with commonly used serologic techniques as can most but not all cattle. *Brucella abortus* can be eradicated from infected bison and cattle herds with current methodology without eradication or depopulation of the infected herds. It is a matter of public record that this has been accomplished in private bison herds in the United States such as the Durham Ranch, Gillete, Wyoming (USA), and "free-ranging" public bison herds in the United States such as the National Bison Range, Moiese, Montana, Custer State Park, South Dakota, Wind Cave National Park, South Dakota, and Antelope Island State Park, Utah.

Studying diseases in "captive animals" under experimental conditions has both limitations and benefits. Controlled situations can eliminate or equalize most confounding variables; but other aspects, particularly those associated with behavior resulting from overcrowding, may be difficult to control. The exposure at challenge under controlled conditions can be relatively precise in dose and time. On the other hand, bison and cattle in *B. abortus*-infected herds may be exposed to the etiologic agent at doses varying from one bacterium to more than 1×10^{13} CFU on many occasions over years. Accepting these inherent differences, is it any wonder that serologic and bacteriologic data from and under field conditions may differ somewhat from that collected under more controlled circumstances? Do these differences negate the results found under experimental conditions? We would hope not because if this is true then much of the experimental biomedical research conducted over the last hundred years may need to be repeated.

When all the subtleties are ignored, certain facts concerning bison and brucellosis are not disputable. Bison can and do become infected with *B. abortus* (probably originally from cattle). Infected bison can and do abort. The aborted fetus and birth products are highly infective; 1 g of infected placenta may contain up to 1×10^{13} CFU. Therefore, bison are capable of, and do transmit the organism to, any susceptible individual of a number of susceptible species (including humans) that may come into contact with the infective material. Infected bison can be diagnosed, and the disease has been eradicated from captive and large "free ranging" bison herds without the elimination of the herd. So what is the argument?

The issue of bison brucellosis has become intensely emotional, politically charged, and highly visible with the media. As professionals aware of the sensitivity of the situation, we have attempted in all occasions to restrict our official comments to the data generated by our research or documented by others. It is part of the scientific method to question everything, and as scientists, we appreciate and encourage critical review done in a professional and respectful manner. In this instance after review and reflection, how-

ever, we stand by our data, our analyses, and our interpretations of the same without reservation, and will maintain that position until someone else can provide sufficient conflicting data on brucellosis in bison of equal or greater strength.

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