Pathogen Transmission in Social Animals

Annotated Bibliography


Wildlife Biologists work with one of the world’s top wolf specialists to explore the potential for using the wolf sanitation effect to manage elk disease. Specifically, this study explores whether or not natural wolf predation restricts the presence of pathogenic microbes in elk populations enough to be given significant weight in elk disease management plans. The study design is a first-ever attempt to compare the presence of pathogenic microbes in Yellowstone elk before and after wolf reintroduction. As such, it paves the way for future research, but it also leaves most questions unanswered. In fact, much of the discussion revolves around what issues still need to be studied before any conclusions about the data collected in this study can be made. Issues brought up include how wolves affect elk grouping in various locations of Yellowstone National Park; how elk grouping affects disease transfer of different pathogenic microbes; and how much researchers misinterpret in their evaluations of the wolf sanitation effect simply because disease symptoms in elk are not visible. The only concrete conclusion that the authors of this article could draw was that the wolf sanitation effect is microbe-specific, rather than universal. The value of this research article is in the long, thoughtful discussion, and in the development of reference values to which future data on potential predator sanitation effects can be compared.


Disease ecologists perform a quantitative risk assessment of Brucella abortus transmission from bison to cattle. While no documented case of B. abortus transmission between bison and cattle in a natural setting exists, the authors still put together a risk model composed of variables thought to increase the likelihood of microbe transmission between these two host species through contact with infected birthing fluids and tissues. Variables include bison population size, winter severity, number of bison leaving Yellowstone National Park (YNP) during the winter, and cattle grazing patterns near YNP boundaries. The model shows B. abortus transmission risk to be spatially and temporally heterogeneous, with local hotspots. Despite the heterogeneity, overall transmission risk has a skewed distribution, where, for the most part, transmission risk is very low. Heterogeneity is attributed to factors such as where/when cattle grazing occurs and how that affects bison-cattle intermingling at various seasons of year. Since B. abortus transmission between bison and cattle was shown to be time and location-specific, the authors recommend that adaptive management be enforced in place of the
current management system (one that is both costly and continuous). While the authors do not develop a risk assessment model that would show the consequences of different management scenarios, their discussion includes insights on several innovative management alternatives.

Wildlife research biologists attempt to predict the risk of B. abortus transmission between elk and livestock. They use GPS data from telemetry-collared elk to develop models of potential host comingling, based on elk resource selection and livestock grazing locations. The best model predicts that B. abortus transmission is strongly related to interactions between hosts and their environment, including predators, vegetation, physical geography, and human presence. These interactions can result in elk resource selection choices that lead to spatial overlap with domestic livestock. Presumably, the models developed in this study can help prevent disease outbreaks by identifying areas where elk and livestock comingling is most likely to occur, so that management plans can target these areas and prevent comingling. However, their true utility is indeterminate since the degree to which hosts’ spatial overlap truly leads to B. abortus transmission is not investigated in this study. To do so would have required the inclusion of additional variables in the models, such as the presence of other microbes in the environment. Further, the predictive accuracy of the models is limited; the best models are site-specific and much more research would be required before the models could be generalized enough to predict spatial overlap between different B. abortus hosts on a larger scale.

Wildlife health researchers use blood samples, elk count data, and statistical and population modeling methods to link increased occurrence of B. abortus in elk to increases in elk population size and corresponding increases in elk aggregations. They also suggest that increases in B. abortus occurrence may be due to cross reactions with other pathogenic microbes, such as Yersinia enterocolitica, as well as to increased microbe transmission between bison and elk. However, other research appears to refute the bison-elk transmission scenario (see Proffitt et. al 2010), and more research is needed to validate the idea that microbial interactions promote B. abortus incidence in elk. The authors propose that transmission between hosts may be tracked by genetic analysis of brucellosis strains in the various hosts. They blatantly urge for more cooperation with landowners, to allow more hunting and more natural predation on elk herds, so as to limit elk herd size and densities and thereby control the spread of B. abortus. While much of this article is speculative, I found it to be quite interesting because the authors provide a good discussion about the history of the problem they are investigating, describe their own research, and then devote time to brainstorming what more needs to be done to better understand B. abortus dynamics and why it is increasing in host populations. More research is needed on how predation and habitat type interact to affect elk group size and density.
A collaborating team of USGS and WGFD biologists, academics, and researchers use hierarchical Bayesian models to investigate relationships between host density and intraspecific *B. abortus* transmission. While they find a positive relationship between microbe prevalence and host density at a localized scale, whether or not this relationship may be characterized as linear at any spatial scale is not determinable. With many small elk herds and a few large herds inhabiting the study area, previous localized disease management practices targeting elk density may have had little effect on *B. abortus* prevalence in the larger herds, thought to act as key *B. abortus* banks in the overall elk population. Further investigation of seasonal and annual changes in host group size distribution and density would aid the development of *B. abortus* management practices applicable at a large scale. While this particular study leaves questions unanswered, it is valuable as a rare empirical study of microbe-host density relationships in large mammals and offers direction for further research necessary for successful wildlife management.
seem to be qualified scientists and researchers. In this study, they find that culture-independent methods (based on 16s rDNA PCR amplification and phylogenetic analysis) allow them to document higher microbial diversity in bighorn sheep than does biological culturing. While the focus of this study was to identify the best methodology for identifying pathogenic microbes in bighorn sheep, the authors’ findings herald better detection of virulence genes and better ability to track where pathogenic microbes in bighorn sheep originate from, i.e. whether they arise naturally, or are transmitted from domestic animals.


Biomedical experts make use of a rare opportunity to study the etiology of a pneumonic epizootic in bighorn sheep from its onset and as it progresses, rather than post-factum. Oropharyngeal and nasal swabs, as well as tissue and blood samples from bighorn sheep, all indicate that the epizootic was brought about by a large variety of pathogenic microbes; no single pathogenic microbe could be identified as the primary pathogen of the epizootic. Complex dynamic interactions between these pathogens potentially explain why antibiotic treatments were unsuccessful in curing bighorn sheep, and the authors highlight the importance of further research on the interactions between multiple pathogenic microbes in bighorn sheep hosts. The authors suggest that stress factors also contribute to pneumonic epizootics by weakening hosts’ immunity to pathogenic microbes; however, this issue was not directly investigated. Further, the source of the pathogenic microbes remained undetermined. This article is quite good at explaining the challenges of studying pneumonic epizootics in bighorn sheep and what further questions need to be addressed if bighorn sheep conservation efforts are to be successful. As such, it is a good introduction to the topic.


Disease biologists studying bronchopneumoniae in bighorn sheep circumvent difficulties associated with conventional bacteriology by using culture-independent techniques. They find a strong association between pneumonia and Mycoplasma ovipneumoniae presence in bighorn sheep. While, for the most part, M. ovipneumoniae seemed to increase the susceptibility of bighorn sheep to pneumonia, the data collected by Besser et al. 2008 also suggests that on rare occasions, it may actually cause pneumonia. Unlike Dassanayake et al. 2010, Besser et al. 2008 find no evidence to support the idea that, in cases where M. ovipneumoniae serves to increase host susceptibility to pneumonia, the actual causative agent of pneumonia is Mannheimia haemolytica. With such contradictions, more research is necessary to understand the role of both M. ovipneumoniae and M. haemolytica in pneumonia development. This study is interesting because the authors use a series of alternative methods to study bronchopneumoniae. The problem of a small sample size, however, should not be ignored.
In this article, rather than examine interspecific and intraspecific pathogenic microbe transmission, animal health specialists examine microbe-microbe interactions within host individuals. Bighorn sheep inoculated with *M. ovipneumoniae* developed pneumonia only if they also hosted the pathogenic microbe *M. haemolytica*. This occurred both when *M. haemolytica* was present in a host prior to the host’s inoculation with *M. ovipneumoniae* and when *M. haemolytica* was inoculated into a host after *M. ovipneumoniae* inoculation. By itself, *M. ovipneumoniae* did not cause disease in bighorn sheep hosts; rather, it reduced the resistance of bighorn sheep to *M. haemolytica*, which, in turn, led to the development of pneumonia. While the results seem straightforward, the choice of sample size (only four bighorn sheep were tested) is questionable. A repeat of this study using a larger sample size, as well as an investigation of the effect of host characteristics (e.g. age, health history, etc.) and microbe characteristics (e.g. *M. haemolytica* strains differ in virulence levels), would enrich the general findings presented in this study.


Statisticians, biologists, and animal health specialists join forces to evaluate results from a badger culling field trial done in an attempt to prevent the transmission of a pathogenic microbe (*Mycobacterium bovis*) from badgers to cattle. They compare *M. bovis* presence in cattle subjected to different badger culling treatments using log-linear Poisson regression and find that localized badger culling actually corresponds to an increase of *M. bovis* in cattle. This seemingly counterintuitive result suggests that *M. bovis* transmission dynamics are complex. One probable explanation for the surprising relationship is that culling disrupts badger social organization, triggering dispersal and *M. bovis* transmission among badgers, which, in turn, increases the risk of *M. bovis* transmission to cattle. While localized culling (done solely in response to *M. bovis* breakouts in cattle) is shown to be ineffective, this study does not delve into the effects of widespread culling and no culling treatments. Nonetheless, it has value for eliminating one of the possible *M. bovis* management choices, and pointing out what management options should still be explored.


Scientists use radio-telemetry, direct observation, and culture-test data to draw connections between badger ranging behavior and *M. bovis* seroprevalence. They discover that ranging behavior in badgers that host *M. bovis* differs strongly from that of *M. bovis*-free badgers. Badgers that host *M. bovis* have larger home ranges that overlap more frequently with adjacent territories, and are observed to move about their home ranges more frequently and forage farther away from their main sett than healthy
badgers. Whether or not *M. bovis* causes differences in host ranging behavior or is a result of differences in ranging behavior still needs to be investigated: either the pathogenic virus indirectly leads to behavioral changes in its hosts (e.g. inability to compete with other badgers for food causes sick badgers to search wider for food), or badgers with larger ranges are simply more likely to come across sources of *M. bovis* in the environment.


Many of the same statisticians and biologists as in Donnelly et al. 2003 use badger necropsies to develop a base model (using linear regression) of *M. bovis* transmission between hosts of different species. They confirm that culling can actually increase *M. bovis* infection in badgers, but go further to explain that this phenomenon is observed specifically in areas located near where culling has occurred, and in areas that are not geographically isolated. In other words, badger culling will actually promote *M. bovis* transmission in areas that immigrant badgers can easily reach. The authors also observe that *M. bovis* presence in cattle rises in these same areas, most likely because increased badger density increases pathogen transmission to cattle. In addition, delayed removal of sick cattle is associated with an increase in badger illness. Unlike many other studies on *M. bovis* prevalence in badgers and cattle, this study actually highlights that *M. bovis* transmission from cattle to badgers is just as much of a problem – if not more – as *M. bovis* transmission within each host species and *M. bovis* transmission from badgers to cattle. A potential new management approach stemming from these findings is to focus more on controlling *M. bovis* seroprevalence in cattle. This could reduce cattle-to-cattle transmission, as well as cattle-to-badger transmission, which, in turn, could reduce the possibility of cattle reinfection by badgers. In general, the results of this study connote the idea that microbe transmission is more complex than previously thought; transmission both within and between alternative hosts should be considered in management plans.


In this study, the authors use phylogenetic comparative methods and parasite species richness data to test opposing hypotheses about how primate ranging patterns influence parasite transmission. In analyzing 825 host–parasite combinations involving 330 parasite species and 119 primate species, they find support for the hypothesis that intensive territory use by hosts increases exposure to parasites that accumulate in soil. The data used in this study does not support the competing hypothesis – one frequently brought up in badger studies – that territorial defense reduces parasitism levels by decreasing home range overlap between social groups. Perhaps one explanation for the latter result is that contact among primate social groups is so high that it overcomes any constraints on parasite transmission. What remains unexplained in this study is why the two competing hypotheses are treated as mutually exclusive.

This study does not prove anything new; however, it provides additional evidence to support the findings of earlier studies that badger culling can actually cause increased presence of *M. bovis* in nearby hosts. Genetic signatures of badger populations confirm that culling prompts badgers dispersal, and more so than previously thought. The effects of culling on *M. bovis* transmission from one host to another seem to drop beyond a certain threshold distance; however, further study is needed to truly understand the effects of long-distance travel on cattle susceptibility to *M. bovis* transmission. Issues that remain unresolved include the reason for why badger populations retain genetic similarity even though their movement (i.e. potential interaction with badgers from other social groups) increases after culling. Another question that has not yet been answered is whether *M. bovis* actually causes badgers to disperse more, or whether widely moving badgers are simply more likely to catch *M. bovis*. The main idea, however, is clear and supports the implications of other studies: that culling is counter-productive.


This study is performed by practically the same group of statisticians, biologists, and animal health specialists as in Woodroffe et al. 2006. The authors reunite to investigate the effects of badger culling on *M. bovis* prevalence in both badgers and cattle. As before, they conclude that proactive culling outperforms reactive culling; however, neither of these two disease management approaches achieves the desired results. Proactive culling significantly reduces badger population size, but produces only small reductions in cattle illness in culled areas and actually leads to increased cattle illness in nearby unculled areas. Reactive culling results in small reductions in badger population size, and overall increased cattle illness. These results suggest a complex, nonlinear relationship between badger population reduction and *M. bovis* transmission to cattle. Most likely, disruption of badger social organization and subsequent migration to new areas and/or immigration of outside badgers into the vacated culled areas results in increased contact between badgers and thus more badger-to-badger microbe transmission. This, together with increased ranging behavior, probably increases contact between badgers and cattle and ultimately results in increased badger-to-cattle and cattle-to-badger microbe transmission. The data collected in this study is difficult to interpret for a variety of reasons. For example, the authors state that culling occurred in all areas where landowners permitted access; however, they do not specify how much access was denied, and how this could have affected data interpretation. Further, the relationship between badger capture rate and badger population density is unclear, so uncertainty remains about the true effects of culling on badger population density and disease transmission. What if badgers simply learned to avoid traps, and their population densities were higher than presumed in this study? Another consideration that the authors themselves point out is that proactively culled areas could very well have served as sink patches influencing badger population dynamics – and, therefore, *M. bovis* transmission – over larger areas. Despite the difficulty of
interpreting the results of this study, the take-home message is that large reductions in badger populations are difficult to achieve and have either minimal or negative results for cattle. Disease management plans should consider alternatives to badger culling.


This study stresses the importance of social behavior on host-pathogen interactions. Woodroffe et al. find that M. bovis is more often found in immigrant badgers, and that it correlates with low badger densities and small badger social group size. The negative relationship between badger abundance (or group size) and M. bovis prevalence does not correspond to predictions made by existing models, and suggests that other factors must be involved in M. bovis transmission. This study is interesting because it includes supplementary analyses to try and explain its own results. However, these analyses are based on indirect measurements and data collected over a relatively short time period. Direct, longer-term studies would yield more reliable explanations. Two issues that beg special attention are (a) whether the negative relationship between badger social group size and M. bovis prevalence is due to badgers in small groups interacting more with neighboring groups, or (b) whether this negative relationship is a result of M.bovis-related mortality, from which host groups become smaller.