LAND USE, LIVESTOCK, AND DISEASE: PATTERNS OF INFECTION IN CATTLE AT THE LIVESTOCK-WILDLIFE INTERFACE

by

MALAVIKA RAJEEV

(Under the Direction of Vanessa Ezenwa)

ABSTRACT

Land use is a major driver of human disease risk. However, few studies have examined this driver in livestock, which are often integral components of human lives and livelihoods. We examined exposure risk of cattle in northern Kenya for three high-impact pathogens: *Brucella spp.* Bovine viral diarrhea virus (BVDV), and *Leptospira spp*.; across two land use types: private ranches (low intensity cattle ranching, high wildlife densities) and communal ranches (high intensity cattle ranching, low wildlife densities). Cattle on communal ranches had higher exposure risk for *Brucella*, while cattle on private ranches had higher *Leptospira* exposure risk. We suggest that variation in contact patterns between cattle and wildlife may be driving the the pathogen specific effects of land use on exposure observed. Ultimately, understanding relationships between land use and disease could help to target specific pathogens, host populations, and sites for disease management and control efforts.

INDEX WORDS: Land use, Livestock infectious diseases, Kenya, Livestock-wildlife interface, *Brucella*, BVDV, *Leptospira*

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MALAVIKA RAJEEV

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MALAVIKA RAJEEV

Major Professor:	Vanessa Ezenwa
Committee:	Sonia Altizer Nicole Gottdenker

Electronic Version Approved:

Maureen Grasso
Dean of the Graduate School
The University of Georgia
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CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

BACKGROUND

Anthropogenic land use is considered to be a major driver of the emergence and spread of infectious diseases in humans, wildlife, and domestic animals (Patz et al. 2004). Recent investigations of trends in human infectious diseases report that a majority of pathogens of human concern have a domestic or wild animal origin (Taylor et al. 2001). Therefore, impacts of land use on infectious disease risk at areas of human-animal interface can have a significant impact on human, domestic animal, and wildlife populations. In this chapter, I review the literature on land use and disease risk. Specifically, I discuss case studies that have explored the mechanisms underlying observed land use-disease patterns, and identify current gaps in research on links between land use and infectious diseases.

Land use as a driver of infectious disease: case studies of known mechanisms

Some of the major land use drivers of infectious diseases include: changes to the physical environment (i.e. deforestation, damming), agriculture (food and livestock production), and urbanization (Patz et al. 2004). These drivers have been associated with increased disease risk in humans, but the mechanisms by which these land use practices affect disease are only understood for a few pathogens (Patz et al. 2004). Human land use can affect abiotic and biotic factors which promote pathogen/vector development and persistence in the environment. A well studied example of this phenomenon is the link between human malaria prevalence and

deforestation. Deforestation alters habitat structure and abiotic conditions which affect the development and reproduction of the malaria vector (Pongsiri et al. 2009). For example, in Kenya, the feeding and reproductive rate of female *Anopheles gambiae*, the common malaria mosquito vector in this region, was 51% higher in cleared areas compared to forested areas, and this coincided with increased daytime and within household temperatures in these deforested areas (Afrane et al. 2005). Additionally, *A. gambiae* larvae in artificial pools in cleared areas are more abundant and develop much faster compared to larvae in natural pools in forested areas (Minakawa et al. 2002, Tuno et al. 2005).

Another mechanism by which land use can affect disease risk is through changes in the abundance, demography, and distributions of intermediate hosts and vectors (Karesh et al. 2012). For example, in the past two decades, overfishing in Lake Malawi has been linked to increased rates of schistosomiasis infection in the surrounding human populations. Specifically, high intensity fishing caused significant population declines of cichlid fish species. This resulted in decreased predation pressure and subsequent booms in the primary prey species, snails, which are the intermediate host for schistosomiasis. This flux of a key host population has been linked to the increased risk of schistosomiasis in humans. Interestingly, recent rebounds of the fish populations in Lake Malawi have been linked to decreased density of snail populations in the lake and decreased prevalence of schistosomiasis in humans (Stauffer et al. 2006).

In addition to changes to intermediate host and vector populations, land use can also alter the community composition of reservoir hosts (i.e. change the relative abundances and distributions of multiple host species) (Patz et al. 2004). Changes to host community composition can impact contact rates between multiple host species, as well as between hosts and vectors. In North America, the effect of land use on host community composition has been

explored for *Borrelia burgdorferi*, the causative agent of Lyme disease, an important tick-borne infection that presents significant human disease risk. In the forests of the NE United States, a wide array of small mammals, from Eastern chipmunks to skunks to white-footed mice, are reservoirs for Lyme disease, and historical patterns of deforestation and habitat fragmentation have shifted the composition of the small mammal communities in these forests resulting in complex relationships between land use and Lyme disease risk (Ostfeld and Keesing 2000). Specifically, long term research on Lyme disease in relationship to host community composition has shown that nymphal tick prevalence is largely dependent on the presence of white-footed mice, which are the most competent reservoirs of Lyme disease. However, the importance of white-footed mice in terms of Lyme disease transmission largely depends on the other host species present and their relative abundances. For example, Eastern chipmunks are also relatively efficient reservoirs for Lyme disease, but they compete with and reduce densities of white-footed mice, potentially mitigating disease transmission risk when they co-occur with mice (Loguidice et al. 2008).

Finally, land use can alter contact patterns between infected and susceptible hosts (Murray and Daszak 2013). This is particularly evident in cases where novel hosts (usually humans) become infected, as in the case of Nipah virus and simian foamy virus. Emergence of simian foamy virus in humans in Central Africa has been linked to primate bushmeat hunting. Hunters, specifically those that reported contact with blood and bodily fluid of wild primates, were found to have antibodies to simian foamy virus (Wolfe et al. 2004). For Nipah virus, agricultural intensification in Malaysia is thought to be the driver of its emergence in humans. Specifically, overlap between pig and fruit production resulted in contact between a wild, frugivorous bat reservoir of the virus and domestic pigs. Ultimately, this overlap in two different

agricultural practices resulted in repeated contacts between wild and domestic hosts, resulting in a sustained outbreak in pigs, which eventually spilled over to humans (Pulliam et al. 2011).

Gaps in current knowledge of links between land use and disease

Other than the case studies detailed above, there are few pathogens for which we have a mechanistic understanding of the effects of land use on transmission and spread. However, there is a significant body of work linking land use to disease risk (Gottdenker 2009). The majority of these studies have focused on emerging diseases rather than endemic infections (Daszak et al. 2001, Patz et al. 2008, Murray and Daszak 2013). Furthermore, the focal pathogens have been mainly vector-borne or trophically transmitted, and of these the majority are protozoa and helminths, potentially because there are multiple ways that land use can affect these often environmentally linked pathogens (i.e. changes in microhabitat/microclimates as in the case of human malaria, Gottdenker 2009). Importantly, most of the work on land use-disease relationships has been done on high-impact diseases of human populations, with fewer studies of animal populations (Brearly et al. 2012, Perry et al. 2011). These trends in current research highlight important areas for future work.

One key area where research is needed is on the link between land use and infectious disease in livestock. To date, only a few studies that have examined how land use relates to disease in livestock (Perry et al. 2011). For instance, cropland irrigation in Mali has been associated with increased prevalence of the liver fluke, a zoonotic parasite (Traore 1989); and agricultural intensification has been linked to an increase in the emergence and spread of *Cryptosporidium parvum, Escherichia coli,* and *Listeria monocytogenes*, all food-borne pathogens of humans (Schlundt 2002). These case studies support the need for further work

across a wider range of systems. Given that humans depend on livestock for subsistence and economy in many regions of the world, livestock diseases have the potential to exert strong indirect impacts on human well-being (Randolph et al. 2007). Furthermore, because there is often a high degree of contact between humans and livestock (especially in resource poor regions) livestock can act as a source of zoonotic pathogens (i.e. pathogens that can be passed from animals to humans) (Zinsstag et al. 2007). Thus, livestock disease and human disease are tightly inter-connected in many systems. For these reasons, studying how land use relates to disease risk in livestock is especially important for understanding the role of anthropogenic land use in shaping patterns of infection that can impact human communities.

STUDY SUMMARY AND OBJECTIVES

The objective of this study was to examine how variation in land use is associated with disease risk in livestock populations. To do this, pathogen exposure risk was compared across two different types of livestock production systems that vary in land use practices, specifically private vs. communal ranching in northern Kenya. Private ranches have low intensity land use with lower stocking rates of livestock, and these areas often support a high abundance of wildlife. In contrast, wildlife are largely absent from communal ranches, since these areas have higher intensity land use and high livestock stocking rates, resulting in land degradation and displacement of wildlife (Georgiadis et al. 2007, Sundaresan and Riginos 2010).

Three pathogens were selected for comparison across land use types: *Brucella spp.* which cause brucellosis; bovine viral diarrhea virus (BVDV), which causes bovine viral diarrhea, and *Leptospira spp.*, which cause leptospirosis. These pathogens are all globally distributed in cattle and have significant negative impacts on livestock health and production. In addition, brucellosis

and leptospirosis are common bacterial zoonoses in sub-Saharan Africa (Mcdermott and Arimi 2002, Vijayachari et al. 2008, Lindberg and Houe 2005). The overall goal of the study was to establish whether patterns of disease risk in livestock vary with land use and whether these patterns differ for different pathogens.

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CHAPTER 2

LAND USE, LIVESTOCK, AND DISEASE: PATTERNS OF EXPOSURE IN CATTLE AT THE LIVESTOCK-WILDLIFE INTERFACE[1]

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ABSTRACT

Land use is known to be an important driver of human infectious disease risk. However, few studies have examined land use as a driver of disease risk in livestock, which are integral to human livelihoods and a significant source of human pathogens. We examined exposure risk of cattle in northern Kenya across two land use types: private ranches (low intensity cattle ranching, high wildlife densities) and communal ranches (high intensity cattle ranching, low wildlife densities) for three high-impact pathogens: *Brucella spp.* Bovine viral diarrhea virus (BVDV), and *Leptospira spp* . We found an overall prevalence of 6% for *Brucella spp.*, 74% for BVDV, and 35% for *Leptospira spp*. Cattle on communal ranches had higher exposure risk for *Brucella*, while cattle on private ranches had higher *Leptospira* exposure risk. There was no association between land use and BVDV exposure risk. We suggest that variation in contact patterns between cattle and wildlife across land use types may be driving the variation in exposure risk, and that the effect of land use on exposure is pathogen dependent. Ultimately, understanding relationships between land use and disease risk could help with targeting specific pathogens, host populations, and sites for disease management and control efforts.

INTRODUCTION

Anthropogenic land use is thought to be an important and complex driver of infectious disease dynamics (Patz et al. 2004). For instance, human land use can influence disease transmission patterns by altering environmental conditions that affect the developmental rate of parasites and vectors (e.g. human malaria, Afrane et al. 2005); by changing the abundance and distribution of hosts critical for transmission (e.g. human schistosomiasis, Stauffer et al. 2006); and by affecting contact patterns among host populations (e.g. Nipah virus, Pulliam et al. 2012). Although several studies have examined the relationship between land use and disease risk in humans, less is known about how land use is associated with patterns of infection in animal populations, especially domesticated species (Perry et al. 2011). In many parts of the world, however, human and animal populations are inextricably linked from both a health and economic perspective (Osofsky et al. 2005). This intersection is particularly relevant in resource-poor livestock production systems where human and livestock populations overlap substantially. In such systems, disease in domestic animals can impact humans directly, since these animals often act as sources of infection (zoonoses), and indirectly via economic and subsistence losses (Molyneaux et al. 2011). Thus studying how land use relates to infectious disease risk in livestock can translate directly into human disease risk and overall wellbeing.

In Kenya's rangelands, which encompass approximately 80% of the country, human communities largely depend on livestock for their livelihoods (Otichilo et al. 2000). In addition, these humans and their livestock share resources with abundant wildlife populations, (Grootenhuis and Olubayo 1993). At this interface, changes in land use can be a significant driver of disease in humans, livestock, and wildlife (Murray and Daszak 2013). In northern Kenya, the two dominant forms of livestock production, private and communal ranching, involve

distinct land management and use practices (Sundaresan and Riginos 2010). Private ranches favor low intensity livestock production and the land is often simultaneously used for leisure, tourism, or conservation purposes (Georgiadis et al. 2007, Kinnaird and O'Brien 2012). In contrast, communal ranches are characterized by higher intensity livestock production and resources on the land are typically shared by multiple individuals for pastoralism (Georgiadis et al 2007, Sundaresan and Riginos 2010). These differences in land management result in very distinct herbivore demographic patterns by ranching type that could influence patterns of pathogen transmission.

In the Laikipia district of northern Kenya, for example, a study based on 21 years of district-wide aerial survey data reported a mean cattle density of 2.46 on private ranches versus 2.06-3.93 on communally used properties (Georgiadis et al 2007). By contrast, wild herbivore densities showed the reverse pattern with private ranches hosting over four times higher wild herbivore densities than communal ranches. Estimated cattle to wildlife ratios ranged from 1.6:1 on private ranches to between 6:1-18:1 on communally owned ranches (Georgiadis et. al 2007). Other studies have also quantified the disparity in cattle and wildlife densities by land use type on a smaller scale and found similar patterns. Kinnard & O'Brien (2012) measured total livestock by weight per unit area across eight properties in Laikipia, and reported values of 12-14 on private ranches vs. greater than 25 on communal ranches. The same study also found that communal ranches had significantly lower abundances of wildlife than their private counterparts. Yet another study estimated total livestock per unit area at the household level for three communal properties in Laikipia, and reported estimates ranging from 204–539 cattle per 100 households (Mizutani et al. 2005). In combination, these studies suggest that there are distinct differences in the densities of livestock and wildlife on private vs. communal ranches. One major

way in which these differences could translate into disparate patterns of disease transmission in livestock is by influencing the relative magnitude of within-species (livestock:livestock) vs. between-species (wildlife:livestock) contact rates across ranch types.

In addition to influencing contact rates, differences in land use could also create variation in the susceptibility of livestock to infection. Specifically, due to land degradation on shared communal ranches, forage is typically of lower quality and quantity than on private properties (Sundaresan and Riginos 2010). Thus, livestock on communal ranches may experience resource limitation, which can increase susceptibility to pathogens if decreased availability of resources translates into reduced immune function (Smith et al. 2005). Differences in socioeconomics associated with land use might also affect disease susceptibility. For example, communal ranches typically have limited access to veterinary care, while private ranches invest more heavily in regular pharmaceutical interventions (e.g. antihelminthics, acaricides). Thus, frequency of veterinary intervention could also influence transmission patterns.

While rates of disease transmission may depend on differences in exposure and susceptibility related to land use, characteristics of specific pathogens may also be important (Karesh et al. 2012). For instance, on communal ranches, higher stocking rates of livestock may increase cattle-to-cattle contact rates exacerbating the transmission of pathogens that are spread by close contact. Alternatively, on private ranches, the high abundance of wildlife could enhance transmission of pathogens with significant wildlife reservoir hosts. To better understand the relationships between land use and livestock disease, we examined the seroprevalence of three pathogens in cattle across private and communal ranches in northern Kenya: *Brucella spp.,* Bovine Viral Diarrhea virus (BVDV), and *Leptospira spp* serovar Hardjo. All three pathogens are globally distributed and have important negative effects on livestock health and production ,

but they vary in key transmission characteristics (see Table 2.1). *Brucella* in cattle is primarily transmitted via direct or close contact with reproductive tissues of infected animals. Environmental persistence of *Brucella* is negligible; therefore close cattle to cattle contact is the most common mode of transmission (Olsen and Tatum 2010). Transmission of BVDV is primarily through direct, nose-to-nose contact with persistently infected individuals. Cattle that are persistently infected with BVDV typically acquire the pathogen as fetuses, and then shed virus at high concentrations throughout their lives (Lindberg and Houe 2005). Finally, *Leptospira* transmission occurs through direct contact with urine of infected individuals and frequently via indirect contact with contaminated water and pasture (Vijayachari et al. 2008).

All three target pathogens have been shown to infect multiple species of wild ungulates across the world, suggesting that cross-species transmission may be feasible particularly in cases where wild hosts are sympatric with cattle (see Table 2.1). *Brucella* infection in wild ungulates has been documented worldwide (Godfroid et al. 2002), however, few wild species are known to be maintenance hosts (i.e. able to maintain and transmit the pathogen without reintroduction from domestic species) (Munoz et al. 2010). One notable exception is the maintenance of *Brucella* in wild elk (*Cervus canadensis*) and bison (*Bison bison)* in the Greater Yellowstone Area in North America (Olsen and Tatum 2010), where transmission to cattle can occur during the winter calving season, when placental and fetal tissues are more common in the environment and limited resources result in increased spatial overlap between wild wildlife and cattle (Proffitt et al. 2011). BVDV exposure has been documented in a wide array of European, North America, and African ungulates (Vilcek and Nettleton 2006), but evidence of persistent infection in wildlife is limited to eland (*Taurotragus oryx)* in sub-Saharan Africa and white-tailed deer (*Odocoileus virginianus*) in North America (Walz et al. 2010). Finally, *Leptospira spp.* have

been documented in a vast array of warm-blooded species spanning multiple taxa (Vijayachari et al. 2008). However, little is known about specific wild species which may act as reservoirs for *Leptospira* serovar Hardjo, but exposure to this organism has been documented in red deer (*Cervus elaphus*) in North America and in buffalo (*Syncerus caffer)* and eland in southern Africa (Aguirre et al. 1995, Anderson and Rowe 1998).

To determine links between land use and exposure risk to each pathogen, we conducted a serosurvey of cattle across six ranches (3 private and 3 communal) in northern Kenya. We predicted that if disease transmission is largely determined by contact rates between cattle, individual risk of pathogen exposure would be higher on communal ranches where cattle densities are higher. However, if contact with a wildlife reservoir contributes significantly to infection, we expected to see greater exposure risk on private ranches. In particular, we predicted that for *Brucella* and BVDV, where direct contact between cattle is central to transmission, exposure risk would be higher on communal ranches where stocking densities are higher (Table 2.1). By contrast, for *Leptospira,* where in addition to direct transmission, there is significant indirect transmission via environmental contamination, we expected that high levels of indirect spatial overlap between cattle and potential wildlife reservoirs on private ranches would elevate exposure risk on these properties (Table 2.1). To account for potential variation in pathogen susceptibility across ranch types, we also measured animal condition, since poor body condition can enhance host susceptibility to pathogens (Beldomenico and Begon 2010). Ultimately, our goal was to establish how patterns of disease risk vary across land use types, and to better understand the ecological and epidemiological links between anthropogenic land use and infectious diseases.

METHODS AND MATERIALS

Study Sites

We sampled cattle originating from six sites located in the Laikipia and Isiolo districts of northern Kenya. Cattle originating from the one site located in Isiolo were sampled in Laikipia having been recently transferred from a communal ranch to a private property. Oral consent was obtained from livestock owners prior to sampling. The Laikipia district, covering an area greater than 9,000 km^2, is made up of a mosaic of land use ranging from government owned, privately owned, to communally owned properties (Figure 2.1). The majority of the district is comprised of ranches; and these properties are important for both livestock production and wildlife conservation (Sundaresan and Riginos 2010). Ranches were classified into two categories - private or communal - based on information on land ownership and use, livestock management, and attitudes towards wildlife (Georgiadis et al. 2007, Kinnaird & O'Brien 2012, Sundaresan and Riginos 2010). Private ranches were designated as properties owned by a single individual or trust with centralized livestock management, and evidence of active investment and/or conservation of wildlife since the early 1990s. Communal ranches were designated as areas with a community structure (e.g. a chief and multiple homesteads) where multiple individuals own livestock, but resources are shared (e.g. communal grazing). These sites are also characterized by little to no active wildlife conservation. Prior to sampling, we verified the vaccination status of herds on all private ranches. Outside of national vaccination programs, livestock vaccination on communal ranches is rare, therefore we assumed that animals in the communal areas had not been vaccinated for the target pathogens.

Sampling

Between 40-75 cattle were sampled at each site (Table 2.2). Across sites, the number of individual herds sampled varied from 1-4 on private ranches, and from 1-20 on communal ranches (Table 2.2). This variation in number of herds sampled was the result of differences in herd structure between land use types since private ranches typically have fewer, larger herds, while communal ranches have multiple small herds. Herds on communal ranches often mix, sharing the same grazing areas, while herds on private ranches are spatially separated and do not mix. Sampling was restricted to cattle over 1 year of age, and age was recorded in months where available. Both sexes were sampled, but at all sites, females made up the majority of herds. The proportion of animals sampled that were female at each site ranged from 67-100%; however, there was no significant difference in the proportion of females sampled across site types (Mean +/- SE: Communal, 0.93 (0.07), Private, 0.81 (0.08); Mann-Whitney U test: $X^2 = 1.23$, $p = 0.27$). For pathogen testing, we collected approximately 7 ml of blood from the jugular vein into red top vacutainer tubes using 18 gauge needles. Immediately after filling each blood tube, a heparinized 100 µL capillary tube was also filled to measure packed cell volume (PCV). Blood samples were transported back to the lab and centrifuged at 3300 rpm for 20 minutes to harvest serum. Serum samples were stored at -20 ºC until processing. Capillary tubes were centrifuged for 10 minutes at 11,000 rpm and PCV was measured using a hematocrit reader card. For each animal sampled, a single observer also collected a series of morphometric measurements, including heart girth (circumference of body at the shoulders), body length (length from point-of-rump to point-of-muzzle with neck extended) and height (from pin bone to hoof).

Serological testing

We tested for antibodies to three different pathogens using commercially available diagnostic assays for *Brucella*. spp., Bovine Viral Diarrheal Disease (BVDV), and *Leptospira spp*. To detect exposure to *Brucella spp.*, we used the IDEXX brucellosis Serum Ab Test an indirect enzyme-linked immuno sorbent assay (ELISA) which measures antibodies against both *Brucella abortus* and *Brucella melitnesis.* For BVDV we used the IDEXX BVDV Total Ab Test for cattle, and for *Leptospira spp.*, we used the Linnodee Lepto Kit (Linnodee Animal Care, Ballyclare, UK), which tests for antibodies to *Leptospira* serovar Hardjo one of the most prevalent serovars found in cattle in sub Saharan Africa (Schoonman and Swai 2010, Niang et al. 1994, Myburgh et al. 1989). All assays were performed according to the manufacturers' specifications.

Statistical Analyses

Test results were calculated using cut-off values provided by each manufacturer (Table 2.3). For all assays, samples that fell outside of the manufacturers recommended range of values designating a positive or negative value (i.e. suspect samples, Table 2.3) were excluded from subsequent analyses. To calculate seroprevalence at the site-level, we accounted for test sensitivity and specificity as described in Rogan and Gladen (1978): $TP = (AP + Sp-1) / (Se +Sp -1)$, where *TP* is the true prevalence, *AP* is the apparent prevalence (# of positives/# tested), *Se* is the sensitivity and *Sp* is the specificity of the assay (see Table 2.3). If true prevalence values were negative, we assumed a seroprevalence of 0%. .

We evaluated whether land use might be associated with variation in susceptibility to pathogen infection, by testing for differences in animal condition across site types. We used PCV

and size-corrected mass as two estimates of cattle condition. Variation in PCV values are indicative of nutritional stress and active parasite infection in livestock (Stockham and Scott 2002, Marufu et al. 2010, Grace et al. 2007); thus an animal with lower PCV may be in poorer condition or suffering from parasite infections. Size corrected for mass can reflect differences in weight that are not simply a function of skeletal size. Individuals that have higher (positive) size-mass residuals weigh more than expected for their body size, and therefore may be in better condition (Schulte-Hostedde et al. 2005). We calculated size-corrected mass from the regression of heart girth, a common proxy for mass in livestock when true weight cannot be measured (Goe et al. 2001, Kashoma et al. 2011), on body length, a size metric. Differences in condition across site types were evaluated using Mann-Whitney U tests.

To investigate the effect of land use on individual exposure risk, we tested for an effect of site type on serostatus for each pathogen using generalized linear mixed effects models with binomial distributions (0= seronegative, 1= seropositive). For each model, land use (private vs. communal), sex, and condition (PCV and size-mass residuals) were included as fixed effects, while site identity was included as a random effect. We also tested for effects of these same predictor variables (land use, condition, sex) on the risk of exposure to multiple pathogens. For this analysis, individuals were divided into two classes, those seropositive for 0-1 pathogens (i.e. exposed one or fewer pathogens), and those seropositive for 2 or more pathogens (i.e. exposed to two or more pathogens). The multiple exposure model was run using a generalized linear mixed effects models with binomial distribution. All mixed effects models were implemented using the lme4 package in R (R core development team). For all significant factors in the models, odds ratios and confidence intervals (CIs) were calculated by taking the exponent of the coefficient of

the fixed effects and the associated upper and lower CIs of these coefficients (i.e. e^x where x is the either the coefficient, the upper CI, or the lower CI).

Finally, since quantitative age data were not available for most cattle on communal properties, we used a subset of individuals from the private properties to examine the effect of age (in months) on exposure risk. In these models, age, sex, and condition were included as fixed effects, and site was included as a random effect. Since there were too few *Brucella* seropositives on privates properties (n = 4/166), we restricted the age analysis to BVDV and *Leptospira* only.

RESULTS

Seroprevalence patterns

The average seroprevalence across sites for the focal pathogens were 6% for *Brucella*, 35% for *Leptospira*, and 74% for BVDV (Table 2.4). Seroprevalence varied greatly by site, ranging between 0-31% for *Brucella*, 0-67% for *Leptospira*, and 34-99% for BVDV. The average seroprevalence also varied by land use type. Communal ranches had a higher seroprevalence of *Brucella:* 11% compared to <1% on private ranches. By contrast, private ranches had a higher seroprevalence of both BVDV (85% vs. 63% on communal ranches) and *Leptospira* (54% vs. 16% on communal ranches) (Table 2.4).

Predictors of individual exposure risk

Both metrics of animal condition were significantly lower on the communal ranches (PCV and size mass residuals: $X^2 = 3.86$, p-value=0.05; Figure 2.2a and 2.2b), so we included both of these indices of condition in our models of individual exposure risk to account for potential differences in pathogen susceptibility across land use types. For *Brucella*, we found that

individual exposure risk was significantly higher on communal ranches compared to private ranches (Table 2.5). Overall, the odds of being exposed to *Brucella* were 22.8 times higher on communal properties (CI:1.08-483.09). In direct contrast, for *Leptospira*, we found that exposure risk was significantly higher on private ranches compared to communal ranches (Table 2.5). The odds of *Leptospira* exposure were 9.6 times higher on private ranches (CI: 2.86-32.36). For BVDV, land use was not a significant risk factor for exposure (Table 2.5). When we evaluated if land use was associated with risk of multiple exposures, we found that animals on private ranches were more likely to be exposed to two or more pathogens (OR=6.7, CI: 1.03-43.16; Table 2.5).

Despite the significant difference in body condition across land use types, neither measure of condition (PCV and size-mass residuals) emerged as a significant risk factor for exposure to any of the three pathogens, or for exposure to multiple pathogens (Table 2.5). Sex was a significant predictor of exposure risk for two of the three pathogens. *Brucella* exposure were 7.9 times higher for males (CI:1.94-31.66) than females, while for *Leptospira* the odds of exposure for females were 2.4 times higher (CI: 1.01-5.62) than for males (Table 2.5). Sex was not a predictor of either BVDV or multiple exposure (Table 2.5). For the subset of animals on private ranches for which we tested for an age effect, we found that older animals had higher exposure to BVDV (estimate = 0.112, p=0.0024; OR(CI)=1.2 (1.04-1.20)). However, there was no effect of age on *Leptospira* exposure (estimate = -0.001, p=0.79). Neither condition nor sex was associated with BVDV or *Leptospira* risk in the age models.

DISCUSSION

To investigate how land use is associated with patterns of infection in livestock, we examined patterns of exposure risk to *Brucella spp.*, BVDV, and *Leptospira spp.* in cattle on private and communal ranches in northern Kenya. Across sites we found an average seroprevalence of 6% for *Brucella*, 74% for BVDV, and 35% for *Leptospira*. *Brucella* seroprevalence tended to be higher on communal ranches, while BVDV and *Leptospira* tended to be higher higher on private ranches. Individual risk of exposure varied by land use type for two out of the three pathogens. *Brucella* risk was significantly higher on communal ranches, while *Leptospira* risk was significantly higher on private ranches. There was no association between land use and BVDV exposure risk. Overall, our results suggest that land use is associated with variation in livestock infection risk. However, the directionality of the effect was dependent on pathogen identity, suggesting that the specific effects of land use infection risk are pathogen dependent.

Our estimate of *Brucella* seroprevalence by site showed a 24-fold difference by land use type (12% on communal vs. 0.5% on private). Importantly, individual risk of exposure was significantly higher for cattle on the communal ranches, with individuals on communal properties having 22.8 greater odds of exposure. Since *Brucella* transmission is largely dependent on direct contact between infected and susceptible animals (Olsen and Tatum 2010), it is likely that within-herd cattle contact drives the majority of transmission in both communal and private ranching systems. In our study area, higher cattle densities and shared grazing are characteristic of communal ranches, while private ranches are characterized by spatial segregation of herds across the landscape. This difference in livestock management may explain the variation in *Brucella* exposure risk we observed between land use types. Indeed, previous

studies have found that similar risk factors (e.g. higher cattle densities, communal grazing) are associated with *Brucella* exposure in cattle in sub-Saharan Africa (McDermott and Arimi 2002, Muma et al. 2007). There are also reports of *Brucella* infection in wild herbivore species in sub-Saharan Africa (McDermott and Arimi 2002), and some studies have reported proximity to wildlife as a risk factor for cattle exposure (Mazeri et al. 2013, Muma et al. 2007). In particular, African buffalo (*Syncerus caffer*) are considered to be the major reservoir for *Brucella* in sub-saharan Africa (McDermott and Arimi 2002), however the densities of buffalo are very low on communal ranches in Laikipia (Georgiadis et al. 2007), which suggests they likely play a limited role in *Brucella* transmission to cattle in this system.

In our study, private ranches had a higher prevalence of BVDV (84%) than communal ranches (65%), however individual exposure risk did not vary significantly by land use type. This may be due to the epidemiology of BVDV. For this pathogen, persistently infected (PI) individuals, although comprising less than 1% of the herd, typically account for a large proportion of the infections that occur within and between herds (Lindberg and Houe 2005). Thus, it is likely that the presence of PI individuals may account for the very high (close to 100%) seroprevalence we observed at some of our sites. Although, BVDV has been documented in a wide array of African wildlife, and persistent infection has been shown to occur in eland (Vilcek and Nettleton 2006, Anderson and Rowe, 1998), it is unlikely that contact with wildlife is the main driver of transmission because BVDV persists poorly in the environment and indirect contacts contribute a negligible amount to transmission (Courcoul and Ezanno 2010, Lindberg and Houe 2005). PI individuals cannot be detected using antibody tests so we could not account for the presence of these individuals in the cattle herds in our study; however, variation in the

frequency of PIs may potentially account for the differences in BVDV seroprevalence across study sites.

At the site level, *Leptospira* prevalence was over 3 times higher on private ranches than on communal ranches (53.5% vs.15.6%, respectively), and cattle on private ranches had significantly higher exposure risk. Previous studies on *Leptospira* suggest that risk factors for exposure in cattle include large herd size, access to contaminated pasture and water sources, and co-grazing with other infected livestock species (e.g. sheep and goats) (Mazeri et al. 2013). Since we observed elevated risk on private ranches where sharing of pasture and water sources between cattle and wild herbivores is common (Kinnaird and O'Brien 2012), our results suggest that overlap between cattle and wild species may be an important risk factor for *Leptospira* in areas where wildlife are abundant. Although there is limited information on *Leptospira* serovar Hardjo in wildlife, a study in Zimbabwe found that both eland and buffalo tested positive to Hardjo antibodies (Anderson and Rowe 1998). For other serovars, wildlife are known to be significant reservoirs and contamination of water sources by these species is considered to be a major risk factor for cattle and human exposure (Andre-Fontaine and Ganiere 1990, Vijayachari et al. 2008). Altogether, these studies suggest that *Leptospira* may circulate among at least some of the wildlife species present on the private ranches in our study region, and could contribute to cattle transmission via contamination of shared habitat.

In our model of multiple exposure, cattle on private ranches had higher risk of exposure to multiple pathogens. This effect could be due to differences in livestock mortality and morbidity patterns between communal and private ranches. Because cattle on communal ranches are generally in poorer condition, have lower quality forage, and lack veterinary care, a single infection in these animals may result in a higher probability of mortality. Thus, fewer animals

may survive multiple infections on the communal ranches. Furthermore, since owners may tend to sell cattle that show overt symptoms of disease (Boukary et al. 2011), if multiple infections result in more severe signs of morbidity in individuals with lower body condition, animals that are exposed to multiple pathogens on communal ranches may be more likely to be sold. This potential bias in selling of animals could further reduce the occurrence of multiply-infected animals on communal ranches.

Although we found that land use type was a significant predictor of exposure risk for two out of three pathogens, we found no evidence that body condition was associated with individual risk of exposure. This was despite the fact that cattle on communal ranches had significantly lower condition scores (i.e. PCV and size-mass residuals), than animals on private ranches. These results suggest that differences in condition cannot account for the differences in exposure risk we observed between land use types. Other factors included in our analyses were sex and age, both frequently assessed as risk factors for pathogen exposure (Mazeri et al. 2013, Muma et al. 2007, Schoonman and Swai 2010, Handel et al. 2011). In our study, males had higher exposure risk to *Brucella*, but lower exposure risk to *Leptospira*, while there was no association between sex and BVDV exposure. Previous studies have also found conflicting results with respect to sex, including significant male bias in exposure (BVDV, Nigussie et al. 2010), significant female bias in exposure (*Brucella*: Muma et al. 2007), and no sex effect (*Brucella*, Matope et al. 2011; *Leptospira*, Schoonman and Swai 2010; BVDV, Handel et al. 2011). Our results along with those of other studies suggest that sex can influence exposure risk, but its specific role may depend on the pathogen and the population. With respect to age, on private ranches BVDV exposure increased with age, but there was no effect of age on *Leptopsira* exposure risk. We were unable to examine age effects for *Brucella*. Previous studies have

reported age class as a consistent risk factor for all three pathogens, with older age classes having higher exposure risk (Muma et al. 2006, Handel et al. 2011, Mazeri et al. 2013). Since we could not obtain fine-scale age data for animals on communal ranches, age could account for some of the variation in exposure we observed between land use types. However, we have no evidence to suggest that age structure varied between land use types, so age variation is unlikely to fully account for the land use differences we found.

Overall we found that land use type was associated with pathogen exposure risk in cattle for two out of three pathogens we studied: *Brucella spp* and *Leptospira spp.*, but not BVDV. Exposure risk for *Brucella* was higher on communal ranches, while risk for *Leptospira* was higher on private ranches. We suggest that variation in livestock and wildlife densities and resulting differences in contact patterns on different ranch types can at least partially explain these results. We also found that cattle on private ranches were at higher risk for exposure to two or more infections, but this result could be a by-product of higher morbidity and mortality of multiply infected individuals on communal ranches. In future studies, directly measuring contact rates between livestock-livestock and livestock-wildlife across land use types will be critical for definitively linking land use to differences in transmission probabilities for specific pathogens. Despite these gaps in data, our results strongly suggest that land use can influence cattle infection risk in this system, and that these effects vary with pathogen biology, specifically transmission mode.

CONCLUSION

At the human-livestock-wildlife interface, livestock disease can have a large impact on human communities. For instance, in a survey of households on communal ranches in Laikipia,

people reported that disease was the leading cause of livestock mortality (Mizutani et al. 2005). Importantly, approximately 40% of human pathogens are shared with domestic animals, thus livestock diseases pose an important health risk to humans (Cleaveland et al. 2001). The links between land use and pathogen exposure risk we found in our study have implications for where different pathogens have the most impact. For example, on private ranches, *Leptospira* may be of concern in terms of both livestock production and human health, whereas on communal ranches *Brucella* may be more of a concern. Ultimately, understanding relationships between land use and disease could help with targeting specific pathogens, host populations, and sites for disease management and control efforts, a strategy that may help limit the burden of disease in countries with fewer resources available for disease control.

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Table 2.1. Summary of disease characteristics, transmission, and predictions by land use. Specifically, details on the pathogen, associated hosts, primary transmission mode, and our prediction for where prevalence would be higher (private vs. communal).

Pathogen	**Disease and impacts**	**Hosts**	**Presence in wildlife**	**Transmission mode**	**Predicted variation with land use**
Brucella spp. (B. abortus and *B. melitenensis*	Bovine brucellosis; abortions, infertility, reduced milk production in cattle (Olsen and Tatum 2010); in humans, chronic systemic illness (Godroid et al. 2012)	Cattle, sheep, goats, wild herbivores (primarily cervids/bovids, humans (Godfroid et al. 2012)	North America (elk, bison); Europe (red deer, chamois, ibex, wild boar); Africa (buffalo) (Munoz et al. 2010, Godfroid et al. 2002)	Direct or close contact w/ infected individuals via reproductive and abortive materials (Olsen and Tatum 2010)	Higher on communal land use
Pestivirus spp.	Bovine viral diarrhea virus (BVDV); abortions, hemorrhagic disease, mucosal disease, mortality of PI individuals, immunosuppresion (Walz et al. 2010	Cattle, sheep, goats, wild herbivores (Walz et al. 2010)	Europe (roe deer, red deer, reindeer) and Africa (buffalo, waterbuck, hartebeest, oryx); PI only documented in eland (sub-Saharan Africa) and white-tailed deer (N. America) (Vilcek and Nettleton 2006);	Direct contact (often nose to nose) with infected individuals and PI individuals (Walz et al. 2010)	Higher on communal land use
Leptospira spp. (L. interrogans and *L. borgpeterseni)* serovar Hardjo	Bovine leptospirosis (serovar Hardjo); abortions, mastitis, reproductive failure in cattle; in humans, acute febrile illness and Weil's disease (associated with renal failure) (Vijayachari et al. 2008)	Cattle, wild herbivores, humans (Vijayachari et al. 2008)	North America (red deer) and Africa (eland and buffalo) (Aguirre et al. 1995, Anderson and Rowe 1998);	Direct and indirect contact with urine of of infected individuals (often via contaminated water/pasture) (Vijayachari et al. 2008)	Higher on private land use

Table 2.2. Description, sample sizes, number of herds sampled, and average herd size for each site. “unk” =unknown.

Type	Description	Sample size(n)	Herds sampled	Average herd size
Private 1	Conservancy; managed primarily for wildlife research with a ranch component; multiple cattle herds;	75	3	93
Private 2	Single-owner ranch; managed for leisure; both wildlife and cattle;	40	1	100
Private 3	Single-owner ranch; managed commercially for livestock production; no active wildlife management; stocked with multiple herds of cattle;	75	4	120
Communal 1	Small group ranch; community structure with an appointed chief and shared grazing; distinct homesteads but with a single communal cattle herd;	75	1	100
Communal 2	Peri-urban community area; community structure with an appointed chief and shared grazing; distinct homesteads and herds with lower numbers of cattle;	75	20	5
Communal 3	Community conservancy; managed for livestock and wildlife through a pro-wildlife trust; community structure with shared grazing; herd composition and structure unknown;	75	Unk	Unk

Table 2.3. Summary of ELISA cut-off values and sensitivity/specificity for each test. Values are taken from the most recent literature or when no literature on the validation was available, from the manufacturer directly. S/P = (optical density (OD) of the sample – OD of the negative control) / (OD of positive control – OD of negative control).

Test	Cut-off values	Sensitivity/ Specificity (%)	Source
Brucella	S/P ≤ 1.10, negative 1.20 < S/P < 1.10, suspect S/P ≥ 1.20, positive	96/97	Manufacturer (IDEXX)
BVDV	S/P ≤ 0.20, negative 0.20 < S/P < 0.30 suspect S/P ≥ .30 is positive	96.1/97.1	Lanyon et al. 2012
Leptospira	S/P ≤ 0.05, negative 0.05 < S/P ≤ 0.12, suspect S/P > .30, positive	94.8/94.1	Manufacturer (Linnodee)

Table 2.4. The number positive/number tested (excluding suspects) and seroprevalence of pathogens by site. Differences in seroprevalence between private and communal sites were not statistically significant (Mann-whitney U: *Brucella*: X^2=1.34, p-=0.25; BVDV: X^2 = 1.19, p =0.28; *Leptospira*: X^2=2.33, p =0.13).

Type	***Brucella***		**BVDV**		***Leptospira***	
	+/tested	Prev (%)	+/tested	Prev (%)	+/tested	Prev (%)
Private 1	0/52	0.0	72/75	98.8	44/68	66.9
Private 2	0/39	0.0	23/40	57.5	11/36	28.5
Private 3	4/75	1.4	71/74	98.7	41/65	65.1
Communal 1	5/75	2.9	25/71	33.5	14/73	15.7
Communal 2	2/75	0.0	57/74	78,4	2/75	0.0
Communal 3	24/74	30.5	56/74	77.0	26/74	33.6
Avg. Private	4/166	0.5	166/189	85.0	96/189	53.5
Avg. Communal	31/225	11.1	138/145	63.0	42/222	15.6
Total Avg.	**35/391**	**5.8**	**304/334**	**74.0**	**138/391**	**34.5**

Table 2.5. Predictors of individual pathogen exposure risk in cattle. Bold values are statistically significant at p-value < 0.05.

Risk Factors	*Brucella*		BVDV		*Leptospira*		Multiple	
	estimate	**p-value**	**estimate**	**p-value**	**estimate**	**p-value**	**estimate**	**p-value**
Land use (Private)	-3.13	**0.044**	1.39	0.179	2.26	**<0.001**	1.90	**0.046**
Sex (Male)	2.06	**0.004**	-0.16	0.744	-0.87	**0.047**	-0.19	0.669
PCV	0.02	0.541	0.03	0.065	0.02	0.476	-0.01	0.673
Size-mass Res.	0.09	0.089	-0.002	0.953	0.03	0.377	0.02	0.157

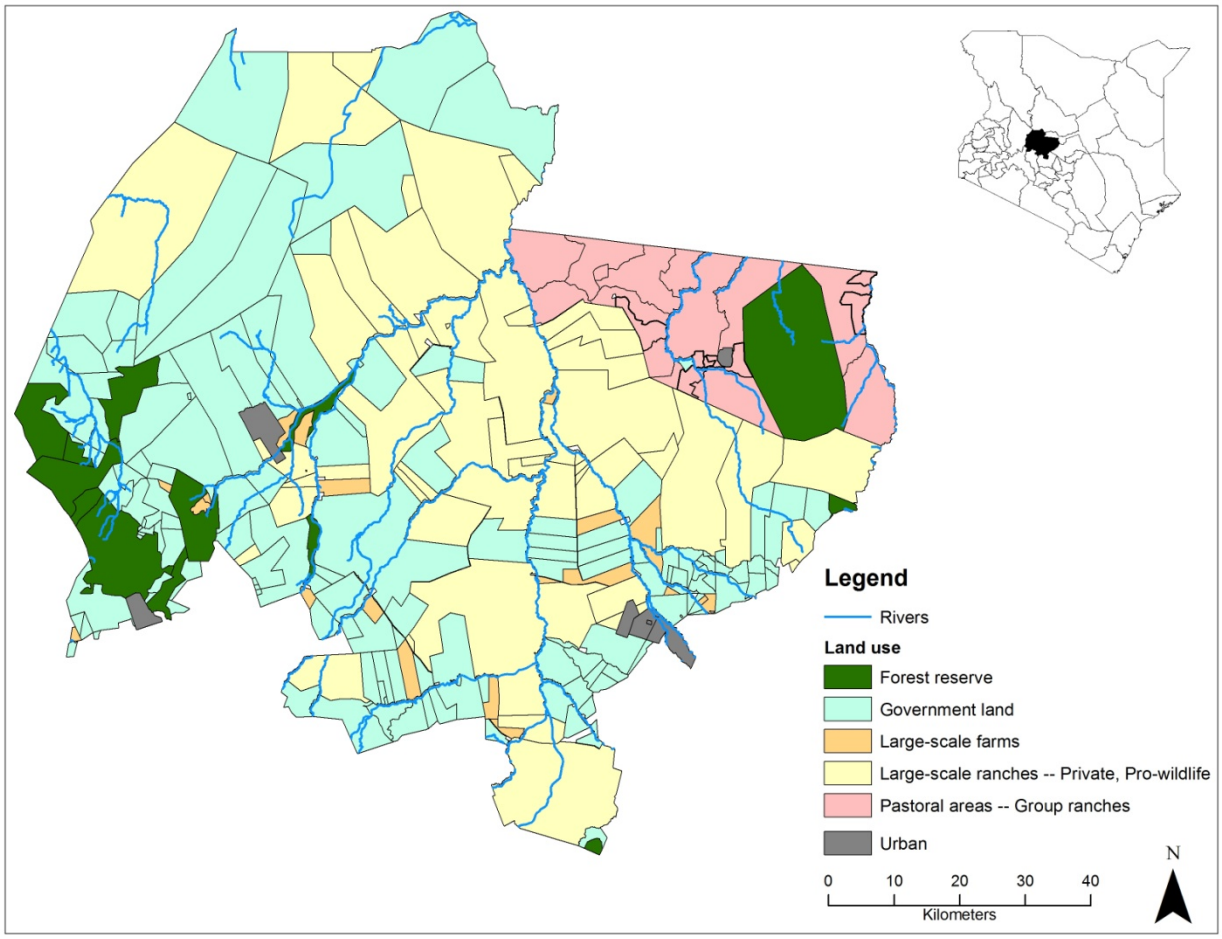

Figure 2.1. Major types of land use in the Laikipia district in Kenya. Most private (non-government owned) properties fall into two land use categories: pastoral areas (group ranches), which include properties we classified as communal and large-scale ranches (private, pro-wildlife), which include properties we classified as private. From Sundaresan and Riginos 2010.

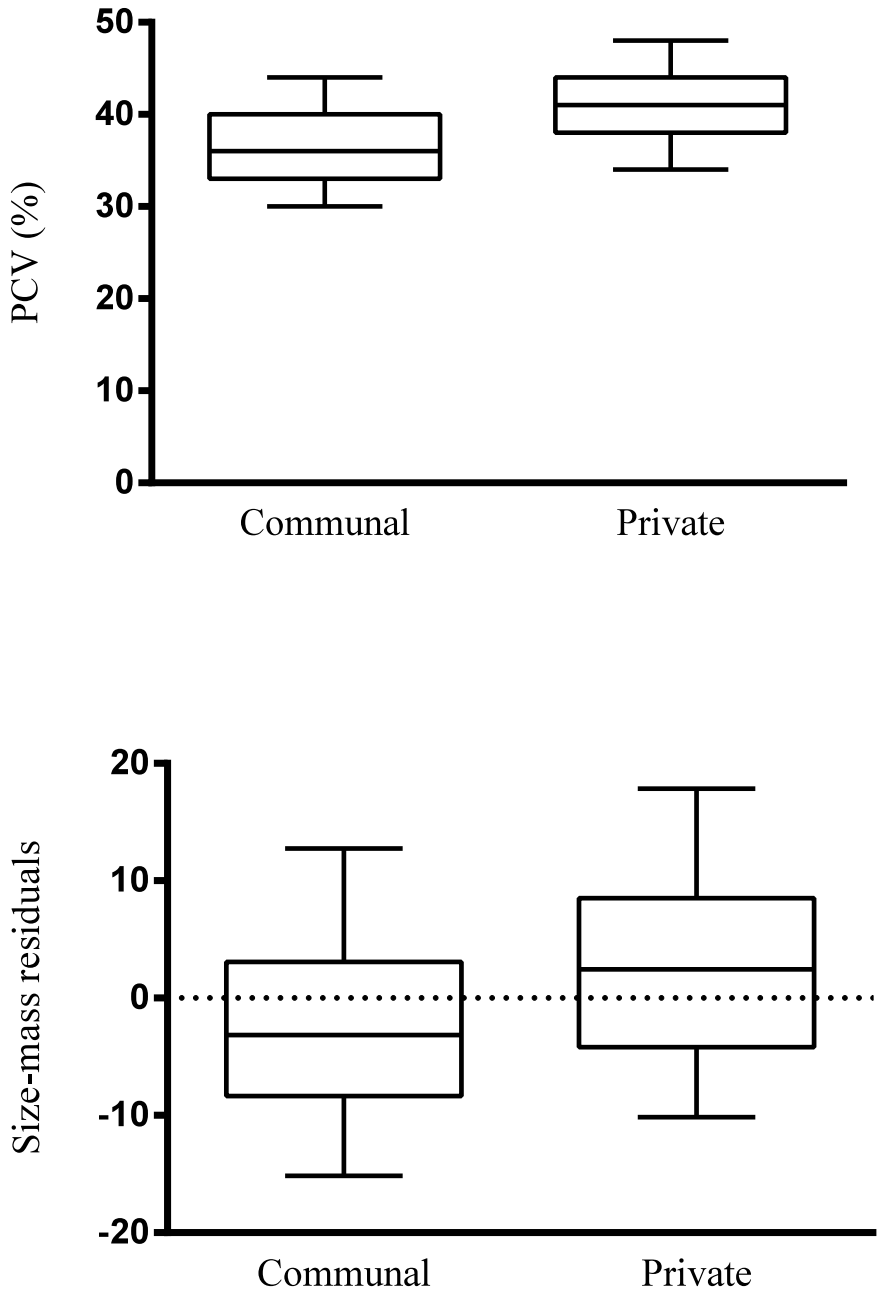

Figure 2.2. Mean (with 95% CI) of condition metrics: **a)** PCV and **b)** size-mass residuals on communal vs. private ranches.

CHAPTER 3

CONCLUSION

Anthropogenic land use change is thought to be an important driver of infectious disease risk in humans (Patz et al. 2004). However, there are few studies which examine the relationship between land use and disease in livestock (Perry et al. 2011), which in many regions of the world are both an integral component of human subsistence and livelihoods and can act as sources of pathogens infecting humans (Molyneaux et al. 2002). This study examined associations between land use and disease in cattle in northern Kenya. Specifically, we compared the seroprevalence of three high-impact pathogens: *Brucella spp.*, BVDV, and *Leptospira spp.* in cattle on private vs. communal ranching systems where differences in land management with respect to livestock shape herbivore densities on the landscape (Georgiadis et al. 2007). Specifically, private ranches are characterized by low intensity livestock ranching and can support high abundances of sympatric wild herbivores. Alternately, communal ranches have higher intensity livestock ranching, resulting in largely degraded habitat with poor capacity to support wild herbivores (Kinnaird and O'Brien 2012, Sundaresan and Riginos 2010).

We predicted that the effects of land use would be pathogen dependent. Specifically, for *Brucella* and BVDV, for which transmission is primarily driven by direct contact between cattle (Olsen and Tatum 2010, Lindberg and Houe 2005), we expected individual exposure risk to be higher on communal ranches where there are high densities of cattle, and cattle belonging to multiple owners commonly mix during grazing. In contrast, for *Leptospira*, which has a significant environmental component to transmission (Vijayachari et al. 2008), we expected that

exposure risk would be higher on private ranches where there is potential for significant indirect contact between cattle and wildlife reservoir hosts.

We found an overall prevalence of 6% for *Brucella*, 74% for BVDV, and 35% for *Leptospira*. Individual exposure risk varied with land use for two out of three pathogens. Cattle on communal ranches had higher exposure risk to *Brucella*, while cattle on private ranches had higher *Leptospira* exposure risk. There was no effect of land use on exposure risk to BVDV. Our results suggest that effects of land use in this system are pathogen dependent. Based on effects of ranching system on herbivore densities in the study system, variation in cattle:cattle and cattle:wildlife contact patterns could be the mechanism driving these pathogen-specific effects on land use. Other factors could also account for some of this variation. Specifically, we found that cattle on communal ranches were in poorer condition than their private counterparts, which may enhance their susceptibility to infection. However, condition did not emerge as a significant predictor of exposure risk for any of the pathogens, suggesting that variation in susceptibility cannot fully explain the patterns we observed.

In future work, it will be important to test potential mechanisms underlying the patterns we observed. Quantitative data on contact patterns within cattle and between cattle and wildlife will be critical for testing whether patterns of relative abundances of these populations drive differences in the transmission of different pathogens. In addition, this type of analysis could be expanded to other livestock species. In the Laikipia district of northern Kenya, trends in sheep and goat densities are on the rise in communal ranches, and to a lesser extent, sheep and goats are also maintained on private ranches (Georgiadis et al. 2007). In a preliminary investigation of sheep and goat pathogen exposure across land use types in the district, we sampled 220 sheep and 147 goats across three private and three communal ranches. We screened animals for

exposure to *Brucella spp*, of small ruminants (e.g. *Brucella abortus and Brucella melitensis*), which parallels bovine *Brucella* in transmission being primarily driven by close intraspecific contact (McDermott and Arimi 2002). Although seroprevalence was very low overall, some interesting qualitative patterns emerged when compared to the cattle analysis. *Brucella spp*. was present in sheep and goats on communal ranches and completely absent from private ranches (Table 3.1). These preliminary results on *Brucella* in sheep and goats support the same trend of increased risk on communal properties that we observed for *Brucella* in cattle.

In conclusion, the patterns we observed could have important implications for which livestock and human populations are at the highest risk for different pathogens. For example, *Leptospira* infection may be of greater concern on private ranches, both in terms of livestock production and human health. Whereas on communal ranches, *Brucella* infection could pose the greater concern to human health, particularly since humans are in very close contact with their livestock on these properties, and depend on them heavily for subsistence. Ultimately, understanding relationships between land use and disease could help to better target future pathogen management and control efforts.

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Table 3.1. The number positive/number tested (excluding suspects) and seroprevalence (uncorrected for test sensitivity and specificity) of small ruminant *Brucella* in sheep and goats at each site.

Type	*Brucella*	
	+/tested	AP (%)
Private 1	0/53	0.0
Private 2	0/75	0.0
Private 3	0/75	0.0
Communal 1	1/70	1.4
Communal 2	1/91	1.1
Communal 3	3/133	2.3
Avg. Private	0/203	0.0
Avg. Communal	5/294	1.6
Total Avg.	**5/497**	**0.8**