Impact of Infectious Diseases on Wild Bovidae Populations in Thailand: Insights from Population Modelling and Disease Dynamics

Wantida Horpiencharoen[1], Jonathan C. Marshall[1], Renata L. Muylaert [1], Reju Sam John[2], David T S Hayman[1]

[1] Massey University, Private Bag, 11 222, Palmerston North 4442, New Zealand
[2] Department of Physics, The University of Auckland, 38 Princes St, Auckland 1010, New Zealand

*E-mail wantidah@gmail.com

Abstract

The wildlife and livestock interface is vital for wildlife conservation and habitat management. Infectious diseases maintained by domestic species may impact threatened species such as Asian bovids, as they share natural resources and habitats. To predict the population impact of infectious diseases with different traits, we used stochastic mathematical models to simulate the population dynamics 100 times over 100 years for a model Gaur (*Bos gaurus*) population with and without disease. We simulated repeated introductions from a reservoir, such as domestic cattle. We selected six bovine infectious diseases; anthrax, bovine tuberculosis, hemorrhagic septicaemia, lumpy skin disease, foot and mouth disease and brucellosis, all of which have caused outbreaks in wildlife populations. From a starting population of 300, the disease-free population increased by an average of 228%. Brucellosis with frequency-dependent transmission showed the highest average population declines (-97%), with population extinction occurring 16% of the time. Foot and mouth disease with frequency-dependent transmission showed the lowest impact, with an average population increase of 225%. Overall, acute infections with very high or low fatality had the lowest impact, whereas chronic infections produced the greatest population decline. These results may help disease management and surveillance strategies support wildlife conservation.

Keywords : Bovine, disease transmission, prediction, population, wildlife conservation

Introduction

Livestock encroachment into wildlife habitats can drive disease transmission between wildlife and domestic livestock, which is a vital issue for both human public health and wildlife conservation. An effect of agricultural expansion and land-use change is to bring wildlife and livestock close to each other and increase the contact frequency and time between domestic and wildlife populations [1, 2, 3]. This increased contact may increase the risk of disease transmission as they can share the same natural resources (e.g. grassland and water) [4].

Infectious diseases can cause dramatic declines in wildlife populations, as demonstrated by chytridiomycosis, which has been implicated in the likely extinction of over 200 amphibian species [5]. Most infectious bovid pathogens are capable of infecting both domestic and wild species. For example, bighorn sheep populations declined from ovine respiratory disease (*Mycoplasma ovipneumoniae*) acquired when sharing the grazing areas with domestic sheep [6]. Similarly, bovine brucellosis has been transmitted from domesticated yak to wild yak in China [7], and between bison, elk and domestic cattle in the USA [8]. Brucellosis affects these bison and elk populations both indirectly and directly as the seropositive animals may be culled for management and directly as the pathogen affects animal reproductive systems [9]. Critically, the impact of infectious diseases is determined by disease-specific traits, such as infection fatality rates [10].

There are five wild bovids species (Gaur, Banteng, Wild Water Buffalo, Mainland Serow and Chinese Goral) that remain in Thailand. They are experiencing dramatic population declines from habitat destruction, illegal hunting [11], and resource competition with domestic livestock [12]. Infectious diseases transmitted from contact with domestic cattle could cause further declines. Several diseases are circulating in Thai cattle, including endemic diseases like bovine tuberculosis (bTB) from *Mycobacterium bovis* [13], and new infectious diseases, such as the recent lumpy skin disease (LSD) [14].

Infectious disease modelling provides a tool to understand disease dynamics better and predict the potential consequences of infection in a population, helping disease prevention and control programs [15], particularly as collecting field data or conducting experiments on some pathogens and hosts is extremely challenging. Models have, for example, been used to determine the potential impact of disease on endangered species, such as canine distemper in the Amur tiger [16]. Although models contain uncertainty and may not cover all factors, predictions can guide the policies and help decision-making [17].

Here, we use mathematical models to explore the potential consequences of six major bovine infectious diseases on endangered Thai wild bovid populations. Our aim is to estimate the potential population changes after the disease is introduced in the population from a reservoir, such as domestic cattle. The diseases are anthrax, hemorrhagic septicaemia (HS), bTB, LSD, foot and mouth disease (FMD) and bovine brucellosis, which all infect a range of bovid species, are distributed worldwide, including Thailand, and have different characteristics. Our study predominantly focuses on the Gaur (*Bos gaurus*) population as their populations are well described plus, of the Thai wild bovids, they have the greatest opportunity to interact with domestic livestock and humans since they are the most likely to share space and resources (e.g. agricultural areas, watering holes) [18, 19]. We hypothesised that acute infections with very low and very high infection fatality rates would have less impact on populations than those with moderate mortality or chronic diseases, the latter high fatality case because they 'burn out' by removing infectious individuals rapidly [10]. The study aims to help infectious disease surveillance and monitoring prioritisation strategies in wildlife and livestock for wild bovid conservation.

Material and methods

1. Model construction

1.1 Population dynamic models

We selected Gaur populations as a model system because they are widespread across Thailand, overlap with livestock and people, and demographic data are available [18, 20]. Further, their demography is similar to other threatened wild bovids (Figure S1 in supplementary materials).

We built an aged-structured population model without an infectious disease as a baseline model. We used the average of reproductive maturity (first calving is around 3 years) and differing natural death rates to classify the population into three age structures 1) calf (c): aged 0 - 1 year, 2) subadult (sa): aged 1 - 3 years and 3) adult (a): aged >3 years.

We assumed demographic parameters were otherwise constant. Only adult females were assumed to add new calves to the population, which enter the susceptible class at a birth rate $\mu_b N_a$. Animals can leave their compartments at the natural death rate (μ_a, μ_{sa} or μ_c) or ageing from calf to subadult (δ_c) and from subadult to adult (δ_{sa}). The initial population was 300 animals, based on the Gaur population size in the Khao Pheang Ma non-hunting area (8 km^2) in Thailand [21, 22].

If N is the total animal population, N_a is the adult population, N_{sa} is the subadult population and N_c is the calf population, the population dynamic model equations at time t are:

$$
\begin{aligned}
\frac{dN_t}{dt} &= N_c + N_{sa} + N_a \\
\frac{dN_c}{dt} &= \mu_b N_a - \delta_c N_c - \mu_c N_c \\
\frac{dN_{sa}}{dt} &= \delta_c N_c - \delta_{sa} N_{sa} - \mu_{sa} N_{sa} \\
\frac{dN_a}{dt} &= \delta_{sa} N_{sa} - \mu_a N_a
\end{aligned}
\tag{1}
$$

1.2 Infectious disease models

We used the same age-structured population as the baseline model (Equation 1) and incorporated compartments with different parameter values for building the disease models.

We modelled the diseases based on Susceptible-Infected-Recovered (SIR) models and modified them based on the disease parameters of domestic animals (e.g. dairy cattle, domesticated buffalo) and wildlife from the previous studies and background knowledge. Table 1 presents the diseases and model structures we used, and a flow diagram is in the supplementary materials. For the compartments used in the models, S denotes the number of susceptible animals, E denotes the number of exposed animals, I denotes the number of infected animals, R denotes the number of recovered animals, and M denotes the number of calves with maternally derived immunity. We selected six infectious diseases which have been reported to cause outbreaks in wild ungulates and livestock populations in several places, including Thailand, which are: anthrax (*Bacillus anthracis*) with an SI structure, bovine tuberculosis (bTB-*Mycobacterium bovis*) with an SEI structure, hemorrhagic septicaemia (HS -*Pasteurella multocida*) with an SIRS structure, lumpy skin disease (LSD) with an SEIRS structure, and both foot and mouth disease (FMD) and brucellosis (*Brucella abortus*) with an SIERMS/E structure. Table 2 displays the disease parameters used in the models. These infections have a range of key parameters of interest. They include infectious diseases with very short (effectively no) incubation periods (e.g. HS) to long incubation periods (e.g. bTB), and very high mortality (e.g. anthrax) to low mortality (e.g. LSD, FMD). We selected 4 disease parameters (transmission rate, incubation rate, infectious rate and fatality rate) included in all models and identified which most contributed to the total population change using a principal component analysis (PCA).

Table 1. Diseases, pathogens, and the structures adopted in the modelling procedures.

Disease	Pathogens	Model structure
Anthrax	*Bacillus anthracis*	$S \to I$
Bovine TB	*Mycobacterium tuberculosis*	$S \to E \to I$
Haemorrhagic septicaemia	*Pasteurella multocida*	$S \to I \to R \to S$
Lumpy Skin Disease	*Capripoxvirus*	$S \to E \to I \to R \to S$
Foot and Mouth Disease	*Aphthovirus*	$S \to E \to I \to R \to M \to S/E$
Bovine brucellosis	*Brucella abortus*	$S \to E \to I \to R \to M \to S/E$

Different transmission types can provide different model results [23]. Here, we considered two disease transmission modes; 1) Density-dependent (DD) and 2) Frequency-dependent (FD). DD transmission is assumed when the contact rate is proportionate to the population density, while FD transmission is assumed when the contact rate is independent of the population density [24, 23]. We assumed the transmission modes for each pathogen and then compared them by introducing both transmission modes because, for some infections, there is no clear evidence of which type suits the pathogen's transmissions and these represent both extremes [10]. Transmission is often likely a mix of both DD and FD in many cases such as FMD and bTB [25, 26]. For the transmission rate (β), we used parameter values based on the reference studies with the reported FD or DD

transmission (Table 2), which differs among infectious diseases. However, to test the sensitivity of the results to these assumptions, we also rescaled the β rate to examine the consistency of the results between FD and DD using Equations 2:

$$\begin{aligned} \beta_{DD} &= \frac{\beta_{FD}}{N} \\ \beta_{FD} &= \beta_{DD} \times N \end{aligned} \tag{2}$$

To model the repeated introduction of an infection from a reservoir such as domestic cattle (e.g. for FMD) or the environment (e.g. anthrax), we repeatedly reintroduced infection into our population at rate ϵ independently of any infection in the population. This reintroduction means the impact of infections are not simply estimated by the basic reproductive number $(R0)$, the average number of secondary cases caused by a primary case in a completely susceptible population.

1.2.1 Anthrax (*Bacillus anthracis)*

To model anthrax, we initially assumed the transmission is FD. We used an SI model [27, 28]. We assumed that S animals are exposed to infected animals and then become infectious (I) at β. All infected animals (I) die (100% mortality) [29] at disease-induced death rate (ρ) and the infectious rate (γ), $\gamma\rho I$.

1.2.2 Bovine tuberculosis (bTB - *Mycobacterium tuberculosis*)

Bovine TB is a chronic and zoonotic infection in livestock and wildlife worldwide [30]. We first assumed DD transmission and used an SEI structure for modelling. The flow of the model starts from S, which are exposed to I animals and become exposed (E) at transmission rate (β); then E animals enter the I compartment at the incubation rate (σ). As we assume lifelong infection without recovery [30]; I animals either die with an age-specific disease-induced fatality rate (ρ) or natural death rate (μ). S and E adults give birth with the normal birth rate $\mu_b(S_a + E_a)$ but I adults are assumed to have a lower fecundity rate (reduced by 27%, [31]) at $\mu_{bI} I_a$. Bovine TB has a long incubation period from several months up to 7 years [32], so here we used 5 months based on the mean incubation period in the African buffalo [30]. We also assumed there's no vertical or pseudo-vertical (e.g. in utero or calf rearing) transmission as it is uncommon in bTb [33, 34].

1.2.3 Hemorrhagic septicaemia (HS - *Pasteurella multocida*)

HS is a fatal septicemic disease in cattle and buffalo. We assume DD transmission based on a previous HS modelling study [35]. We used an $SIRS$ model and excluded an E class as the disease can show acute clinical signs with a short incubation period of $\sim$ 18-20 hours [36]. S animals become I at the transmission rate (β). I animals may die from HS at the disease-induced fatality rate (ρ) or survive and become recovered (R) at infectious rate $(1/\gamma)$. We calculated fatality ranged from 0.53 - 5.84% in the cattle population, using the number of deaths from HS (0.21%; assumed from the percentage of deaths from bovine respiratory disease, [37]) divided by the prevalence of HS (3.59 - 40%, [38, 39]). R animals reenter S when they lose immunity at the immunity loss rate (ω). We used the proportion of susceptible animals (0.6) to calculate $R0$ and therefore the β rate using the equation: $R0 = (1/(1 - I)) = 1/S$.

1.2.4 Lumpy skin disease (LSD - Lumpy skin disease virus, *Capripoxvirus*)

We used an $SEIRS$ structure for LSD. We inserted E and R compartments as the disease has an incubation period of between 7 and 14 days and a recovery period of around 4 - 6 months. We initially assumed the transmission is DD as the cattle density could be one of the risk factors to increase the transmission rate within-herd. However, we used both FD and DD β values because the published work has reported differences in incidence rates associated with different transmission modes [40]. We assumed different birth rates for I females (μ_{bI}) from the

natural birth rate, because LSD can reduce the fertility rate by 10% [41]. Also, we applied the highest fatality rate in calves (5%) and lower mortality rates to subadults (3%) and adults (1%) [42].

1.2.5 Foot and mouth disease (FMD - Foot-and-mouth disease virus, *Aphthovirus*) & Bovine Brucellosis (*Brucella abortus*)

We initially assumed the transmission was FD for both FMD and brucellosis. An $SEIRMS/E$ model was applied for FMD and brucellosis. We considered the $SEIR$ model appropriate for both diseases. Recovered FMD and brucellosis cows can pass immunity to their offspring. Therefore, we added a maternally-derived immunity (M) compartment, which refers to the calves born with maternally-derived immunity from recovered mothers (R_a). We assumed that if recovered adults (R_a) calve at the birth rate (μ_b), a calf will receive maternal immunity and stay in M compartment for 6 months [43] before immunity wanes and they become susceptible again (S_m) at a loss of immunity rate (ω_m). S_m calves can either become an exposed calf (E_c) if contact with I or enter a susceptible subadult (S_{sa}) compartment at a loss of immunity rate plus calf ageing rate: $(\delta_m S_m = \delta_c S_c + \omega_m M)$ if they have no contact with I to ensure that animals do not spend too long in the calf age class $(_c)$.

Vertical transmission from mothers to calves can be a consequence of infection among infected mothers with different probabilities for FMD (~ 0.5) and brucellosis (~ 0.9). Infectious adults are assumed to produce an infectious calf (I_c) entering I at the birth rate $\mu_b I$. The proportion of infected females producing infected calf denotes α. So, an infected female can produce an infected calf at a rate $\alpha\mu_{bI}I_a$ and produce a susceptible calf at a rate $(1-\alpha)\mu_{bI}I_a$.

The mathematical ordinary differential equations for the calf population (X_c) are:

$$\begin{aligned}
\frac{dS_c}{dt} &= \mu_b(S_a + E_a) + (1-\alpha)\mu_b I_a - \beta_c S_c(I_c + I_{sa} + I_a) - \delta_c S_c - \mu_c S_c + \omega_c R_c - \epsilon S_c \\
\frac{dE_c}{dt} &= \beta_c S_c(I_c + I_{sa} + I_a) + \beta_c S_m(I_c + I_{sa} + I_a) - \sigma_c E_c - \delta_c E_c - \mu_c E_c + \epsilon S_c \\
\frac{dI_c}{dt} &= \sigma_c E_c - (1-\rho_c)\gamma_c I_c - \rho_c\gamma_c I_c - \delta_c I_c - \mu_c I_c + \alpha\mu_{bI} I_a \\
\frac{dR_c}{dt} &= (1-\rho_c)\gamma_c I_c - \omega_c R_c - \delta_c R_c - \mu_c R_c \\
\frac{dM}{dt} &= \mu_b R_a - \omega_M M - \mu_c M \\
\frac{dS_m}{dt} &= \omega_M M - \delta_m S_m - \beta_m S_m(I_c + I_{sa} + I_a)
\end{aligned} \tag{3}$$

The full sets of equations and all other disease model equations and diagrams can be found in the supplementary materials. Note that all equations are subsets or variants of Equation 3.

2. Model simulations

Because the Gaur and other endangered bovid populations are small and we are interested in how infections might lead to their decline and because we also wanted to allow infection to go extinct in populations if they could not be maintained, we used stochastic models. First, we built the population dynamics model without infectious disease classes and parameters as a baseline model [10]. Then, we introduced an infectious adult $(I_a = 1)$ to the susceptible (S) population. We assumed that I would infect S as a transmission rate, β, and enter the next compartment based on the model structure. Demography (birth rate, natural death rate and ageing rate), the external force of infection (ϵ), and disease-induced fatality (ρ) were included in all disease models.

All models were simulated for 100 years, and the stochastic models were simulated 100 times

to generate the mean and to understand the uncertainty. The parameter values used for modelling were collected from the literature review and observational data (Table 2). We compared the difference in total population (N) between no infection and disease models by calculating the average percentage of the population change using the total population at the start (N_{t0}) minus the total population at the end, (N_{t100}) of the simulation time, divided by $N_{t=0}$, and converted this to a percentage using the following equation:

$$\bar{x} = \sum_{i=1}^{100} \frac{N_t - N_{t0}}{N_{t0}} \times 100 \tag{4}$$

We used R 4.1 [44] to simulate all the models and for further analysis. The R code for reproducing the analyses is available at a GitHub repository https://github.com/Wantidah/InfectiousModel.

Results

We developed stochastic models for a Gaur population, including a baseline model without infection and six infectious disease models. The baseline model of the Gaur population demonstrated significant population growth, increasing from 300 to an average of 685 (range 113 - 1469) animals, which is approximately a 228% (38 - 489%) increase. The average adult and subadult populations consistently increased, while the calf population slightly decreased from 95 to 82 animals (19 - 279) on average (Figures 1 and 3). This gave us a disease-free population to model the impact of disease introduction into.

Brucellosis had the greatest population impacts and FMD the lowest impact. Brucellosis with FD transmission led to a 97% decrease in the average population change (Figure 1) and was most likely to drive the population to local extinction with 16% of the total simulations leading to extinction, mostly occurring from year 80-100 (Figure 2, and supplementary materials). The least impact was seen in the FMD model with FD transmission, which predicted the total population growing by 225%, just 3% less than the disease-free population.

Using different parameter values, fatality rates and modes of transmission yielded different effects on the modelled populations for HS, FMD, LSD and brucellosis. FD brucellosis had the largest population impact, yet DD brucellosis suppressed population growth but led to a stable population. In contrast, HS, LSD and FMD FD models showed continued population increase. Anthrax and bTB showed only a slight difference in the average population change between the two transmission modes. Simply rescaling the β with modelling FD or DD transmission had limited changes. Rescaling the β also reduced the probability of local extinction for FD brucellosis. Figures 2 and 3 present the results of rescaling for LSD, FMD and brucellosis, and more information can be found in the supplementary materials.

For HS, we found that the impact of infection was less dependent on the mode of transmission than case fatality. A ten-fold increase in fatality rate led to a decline in the population, whereas a switch from frequency to density-dependent transmission led to qualitatively similar dynamics. The rescaling results from DD to FD provided similar dynamics trends for HS. HS DD and HS FD models with a rescaling of the reported $\beta = 0.5$ showed the same dynamics, with a population decline of 85% of the average population.

There was uncertainty regarding the mode of transmission in bTB models, however, Cross and Getz [30] showed limited qualitative differences in model outcomes when they used FD or DD transmission. Here, we saw similar results in that overall, the populations tended to decline gradually through the simulation period. Rescaling the β transmission parameter also led to limited qualitative differences in population trends, but we did see differences in predicted classes;

for example, this increased or decreased the number of infected individuals over time (i.e., higher or lower prevalence) (see supplementary materials).

For LSD, we found that the two published transmission parameter values led to differing outcomes that also depended on the mode of transmission. Whist rescaling the parameters did not lead to qualitative differences, the use of the parameter values estimated from direct density-dependent transmission within herds from Magori-Cohen et al. [40] led to a population decline. However, the estimate from the indirect transmission (presumably via mechanical transmission from flies) did not and the modelled population still grew by 155% with FD LSD.

FD FMD transmission with a β transmission rate of 0.115 and the rescaled DD parameter 3e-4 similarly had limited impact on the population growth with an increasing population over time (Figure 2). Increasing β in the DD model, however, decreased the total population and increased I animals. FMD also showed a periodic pattern with outbreaks around every 3 - 5 years (Figure 1). Increasing the β rate from 0.11 to 21 in FD FMD models led to similar dynamics close to DD transmission (Figure S13 - S14 in the supplementary materials).

Our PCA quantitatively shows that pathogens with longer incubation periods, chronic infection and medium to low fatality lead to greater population growth in smaller populations of endangered bovids than a high fatality or high transmission rate alone (Figure S17-S18).

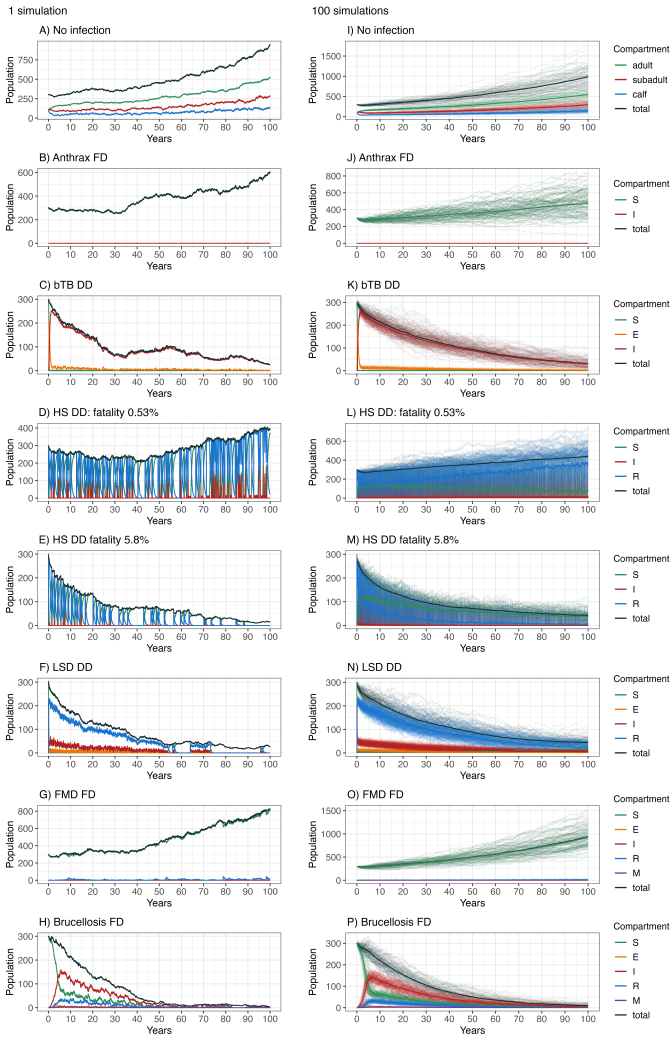

Figure 1. Modelled Gaur population dynamics with and without disease: A-H are single example stochastic simulations for 100 years; I-P are 100 stochastic simulations for 100 years. Mean values are in solid lines. A and I are no infection models and the others are the infectious disease models where bTB is bovine tuberculosis; HS hemorrhagic septicaemia; LSD lumpy skin disease; and FMD foot and mouth disease. Entire model results see the supplementary materials

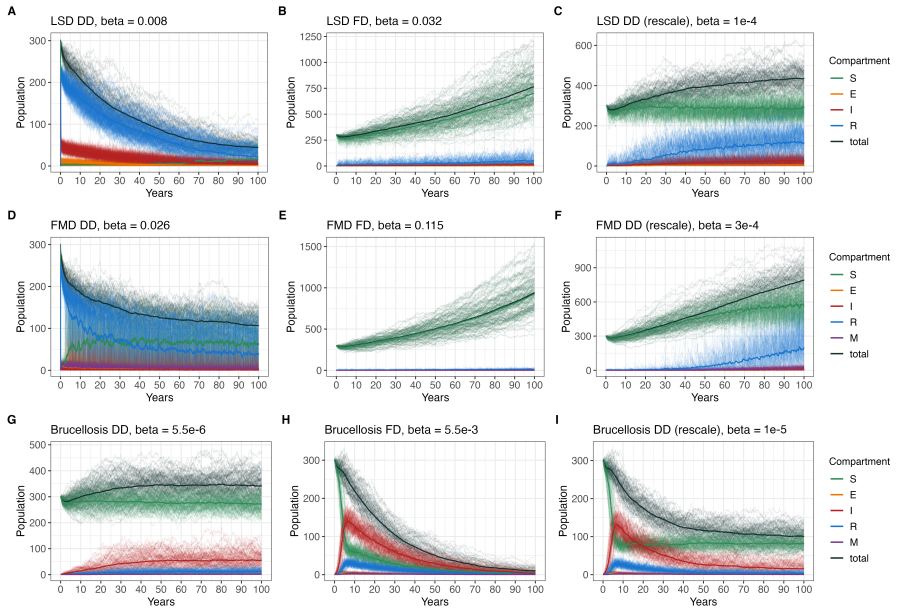

Figure 2. Population dynamics for LSD (A-C), FMD (D-F) and brucellosis (G-I) with different transmission modes and rescaled β transmission coefficient values to isolate the effect of the mode of transmission. A, D, G (left) are DD models; B, E, H (centre) are FD models, and C, F, I (right) are DD models with rescaled β transmission of FD parameters. Rescaling LSD (A-B) and FMD (D-E) parameters have limited impact over the period modelled, but rescaling the brucellosis β shows a reduction in FD transmission.

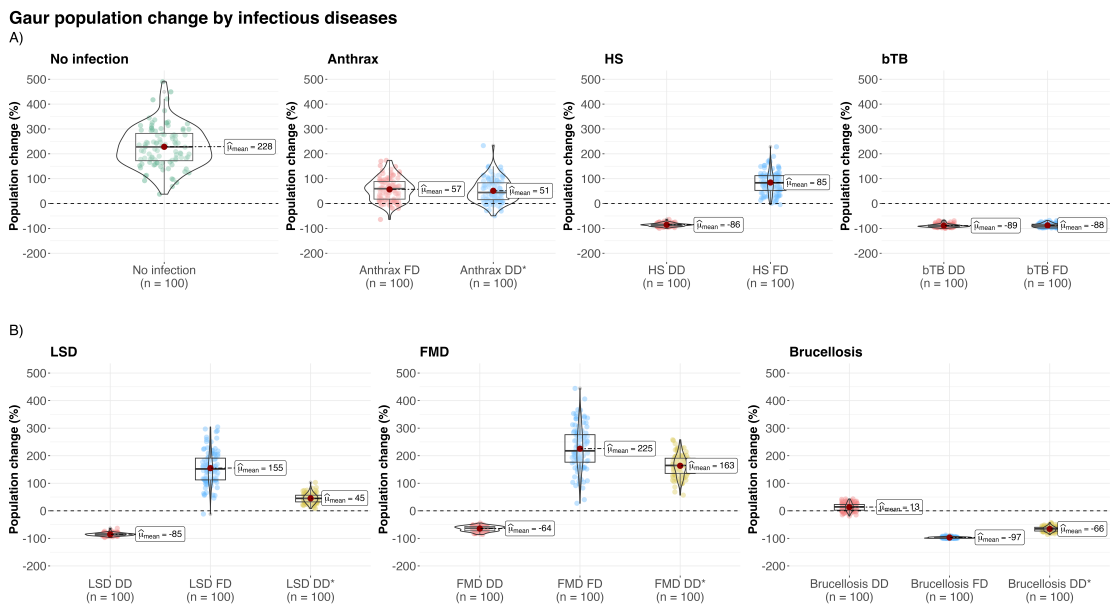

Figure 3. Overall modelled Gaur population changes for each infection. Shown are the 100 results after 100 years of 100 stochastic simulations. The x-axis is the type of disease transmission and y axis is the population change in percentage. A (top) compares no infection, FD and DD for anthrax, HS and bTB; B (bottom) compares FD and DD transmission and DD* that uses the rescaled β transmission from the FD model with DD transmission for LSD, FMD, and brucellosis.

Table 2. Parameters and variables.

Symbol	Description	Species						Units	Reference
		Gaur	Banteng	Buffalo	Serow	Goral			
N_0	Starting total population	300	470	69	120 (assume)	292 (assume)		animal	[21, 45, 46]
μ_b	Birth rate	0.34	0.35	0.40	0.70	0.50		year^{-1}	[47, 48, 49]
μ_c	Calf death rate	0.27	0.26	0.27	0.50	0.45		year^{-1}	[47, 50, 51, 52]
μ_{sa}	Subadult death rate	0.15	0.26	0.15	0.15	0.28		year^{-1}	[47, 50, 49, 52]
μ_a	Adult death rate	0.17	0.15	0.20	0.28	0.18		year^{-1}	[47, 50, 49, 52]
δ_c	Calf ageing	2.7x10^{-3}	2.7x10^{-3}	2.7x10^{-3}	2.7x10^{-3}	2.7x10^{-3}		day^{-1}	[53]
δ_{sa}	Subadult ageing	9x10^{-4}	9x10^{-4}	9x10^{-4}	9x10^{-4}	9x10^{-4}		day^{-1}	[53]
		Disease							
		Anthrax	bTB	HS	LSD	FMD	Brucellosis		
β	Disease transmission rate	0.01- 3x10^{-5}	1.4x10^{-3}	0.33	0.008-0.032	0.15 - 0.026	5.5x10^{-3} - 5.5x10^{-6}	day^{-1}	[28, 30, 40, 54, 55, 56]
σ	1/Incubation period	0.14	6.7x10^{-3}	0.3	0.14	0.13	0.07	day^{-1}	[57, 30, 58, 40, 59, 60]
γ	1/Infectious period	1	-	0.33	0.03	0.2	1.4x10^{-3}	day^{-1}	[29, 58, 61, 62, 63]
ρ_c	Disease-induced fatality in calf	1	0	0.53-5.84	0.05	0.1	0.1	%	[29, 31, 64, 42, 65, 66]
ρ_{sa}	Disease-induced fatality in subadult	1	0	0.53-5.84	0.03	0.05	0.05	%	[29, 31, 64, 42, 65, 66]
ρ_a	Disease-induced fatality in adult	1	0.11	0.53-5.84	0.01	0.03	0.03	%	[29, 31, 64, 42, 65, 66]
α	Infected female will produce infected calf	-	-	-	-	0.5	0.9	%	[56]
μ_{bI}	Birth rate for infectious individuals	-	0.23	-	0.31	0.31	0.17	day^{-1}	[31, 41, 56]
ω_c	Loss of immunity for calves	-	-	5.6x10^{-3}	5.6x10^{-3}	8.3x10^{-3}	5.6x10^{-3}	day^{-1}	[67, 68, 69, 63]
ω_{sa}	Loss of immunity for subadults	-	-	5.6x10^{-3}	5.6x10^{-3}	8.3x10^{-3}	5.6x10^{-3}	day^{-1}	[67, 68, 69, 63]
ω_a	Loss of immunity for adults	-	-	5.6x10^{-3}	5.6x10^{-3}	1.7x10^{-3}	5.6x10^{-3}	day^{-1}	[67, 68, 69, 63]
ω_m	Waning of maternal immunity	-	-	-	-	6.9x10^{-3}	5.6x10^{-3}	day^{-1}	[69, 63]
ϵ	External force of infection rate	2x10^{-5}	2x10^{-5}	2x10^{-5}	2x10^{-5}	2x10^{-5}	2x10^{-5}	day^{-1}	[10]

[To interpret the parameters, any rate r can be converted to probability $\mathbb{P}(t)$ using 1-exp^{rt}, where t is the time period, e.g. for ϵ, $\mathbb{P}(t)$ = 1-exp$^{\epsilon t}$ = 1-exp$^{2\times10^{-5}*365}$ = 0.007, or if the total S in the population is 300, $\approx$ 2 events per year. The dashed line (-) means no parameters were used in the models.]

Discussion

Interactions between wildlife and livestock can facilitate the transmission of emerging infectious diseases [70], making this interface an essential zone of concern to public health, animal production and wildlife conservation. We identified the potential consequences and severity of six bovine infectious diseases present in Thailand (anthrax, HS, bTB, LSD, FMD and brucellosis) in a model wild bovid population, using different infectious disease model compartments based on the current literature (Table 2). Brucellosis had the greatest population impacts and FMD the lowest, despite the same model structures being used for these two pathogens. Overall, our base model predicted population growth with varying impacts of diseases, and our analyses matched our expectation that those acute infections with very high fatality rates (anthrax and HS) have less impact than chronic infections with lower infectious rates (bTB, brucellosis), as infected individuals are rapidly removed from populations [10]. Therefore, our analyses suggest that pathogens with longer incubation periods, chronic infection and low to medium fatality rates have a greater negative impact on population growth in small populations of endangered bovids (Figure S5, S6, S15). This is likely mostly because these traits allow infections to persist, allowing long-term infection effects on demographic structures (e.g. reduced birth rate, increased death rate).

We used 100% fatality rates in all infected animals as the worst-case scenario for the anthrax model, which led to limited impact over the 100 years, likely because of this rapid removal of infected individuals (I), despite repeated reintroduction of infection [10]. We first considered anthrax transmission between infected and susceptible animals as FD transmission, assuming contact rate is more influential than host density [28]. Transmission could also be DD, based on the density of spores in the contaminated environment (e.g. infected carcass, soil) [27]. Cattle density is also one of the risks for anthrax transmission to wildlife [71], contributing to between-species transmission and we modelled repeated introductions through ϵ to cover the external force of infections other than within-herd transmission.

Bovine tuberculosis causes chronic, fatal infection and reduces pregnancy rates and, therefore, the population growth of wild bovids [31, 72]. There is no current evidence of bTB infection driven population declines in Asian wild bovids, however our study found that, regardless of both transmission modes, the long-term effect of bTB would be to reduce the expected total population by around 88-89%. This is similar to findings by Jolles and colleagues [31], who showed that bTB persisted in African buffalo populations and reduced adult buffalo numbers primarily through mortality of animals more than 4.5 years old. The transmission coefficient (β) was noted as one of

the most important parameters for bTB in African buffalo [30]. In our work, we found consistent population dynamics between FD and DD transmission when changing β between the original value (from several studies) and the rescaled values (Figure 3 and Figure S5,S6 in supplementary materials). This is likely due to the duration of the infection, which might increase the probability of contact with infectious animals in the population and the number of transmissions.

For HS, we used two infection fatality rates since case fatality significantly underestimates infection fatality, because a large proportion of animals are infected but do not develop clinical disease or signs of diseases. We calculated the fatality rate using the disease prevalence and then including seropositive animals from reported study populations of cattle, which decreased the case fatality from 90% to around 6% of animals [73, 38]. Our model shows that changing the mortality from 0.53% to 5.8% affects the total population numbers more than changing the transmission modes, by more strongly reducing the population sizes. HS antibodies were found in free-ranging buffalo in Asia so this population might be a reservoir, but this needs further investigation [74]. HS is endemic in Thailand [73], and mortality in wild ungulates has been reported historically [75], so the mortality and infection status of HS should be considered in the mitigation plans for endangered species (e.g. Wild Water Buffalo and Banteng). Both FMD and LSD with FD transmission had the least impact on populations, with acute, short infections with lower overall mortality [68, 76].

Both FMD and LSD with FD transmission showed the least impact on populations, with both having acute, short infections with lower overall mortality [68, 76]. FMDV in particular, is highly contagious among cattle with a very high β coefficient compared to the other diseases [25]. Yet, although Beck-Johnson et al. [25] found little effect of FMD with either transmission modes within-herds, our results showed that DD transmission led to greater population declines as did rescaling the parameter used for FD models. The reason for the latter observation is not clear but might be because the dynamics with reintroduction allow more infection to persist and so suppress the population (Figure 2). Note that with reported wild bovid herd sizes, acute transmission is unlikely to allow FMDV to persist, but reintroductions from cattle reservoirs (modelled through ϵ) are likely [69]. Our result for FMD DD transmission also showed cyclic patterns in outbreaks consistent with seasonal patterns of outbreaks observed in Thailand [77].

We found that brucellosis with FD transmission and its reported β rate might cause extinction 16% of the time, whereas DD transmission may suppress population growth, but not enough to cause population declines, even with the published FD rate rescaled and used in a DD model. Brucellosis has caused population declines among African buffalo, especially when there is co-infection with tuberculosis [78]. However, brucellosis only caused limited population growth impacts in American bison, even though the disease persisted in the population over time [79, 56]. In Dobson and Meagher's study [56], their FD brucellosis models showed bison populations would increase in numbers, whereas our models predicted a decrease, perhaps because our model species' population size and structure differed from their study. Notably, *Brucella* can infect multiple species, and the transmission source may not be obvious when multiple species interact. For example, brucellosis outbreaks in Yellowstone National Park, USA, were not from wild bison as first thought, with elk the likely primary host [80]. Understanding the potential transmission among and from other wild Asian ungulates may be necessary to fully understand potential brucellosis impacts.

We assumed a single, closed (no migration) population with constant natural birth and death rates. Therefore, our models explore the intrinsic population dynamics without considering the influence of other positive (e.g. conservation) or negative factors (e.g. habitat destruction, competition). Furthermore, it is unclear what population changes occur during migration [81], so a closed population model can only simulate within-herd dynamics and reflect the population impacts in a small population, such as in small protected areas [21, 20].

The population dynamics of four other wild bovid species show similar trends to the Gaur

population (Figure S1), so we assumed there will be similar trends for other two large bovids (Banteng, Wild Water Buffalo) that have similar herd sizes and social behaviours to Gaur [82]. However, population dynamics may differ from the medium-sized bovids (Chinese Goral and Mainland Serow) that live in smaller groups or even pairs and can be isolated from each other [83].

Selecting the appropriate transmission mode for modelling is challenging [84]. The infectious disease parameter values themselves are mostly estimated from livestock outbreak data, which can vary among the regions and rarely is infection 'natural' without intervention through disease control [25]. Although the transmission type for some pathogens have been recorded as FD or DD in previous studies, these were mainly conducted under farm husbandry or experimental conditions in captive or closed systems. This can make animal density have a major effect on contact rates. However, our study focused on wildlife populations that are distributed in areas in which the frequency of contact could have more influence. Moreover, some infectious diseases can display aspects of FD and DD depending on the conditions, such as within- or between herd transmission, herd size, density, contact with other reservoirs and contact mode (indirect, direct). For example, in larger herds, bTB transmission might be based on the frequency of contact, while for smaller herds, it might be based on the density. Also, the transmission mode for anthrax spores from animal to animal is FD, but from the environment to an animal is based on the density of the spores in the areas. We, therefore, took the strategy of assuming the most extreme scenarios, fully FD and DD, and used both for modelling.

Our models also added the external force of infection (ϵ), which represents the re-introduction of pathogens. ϵ is assumed to include transmission from other sources of infection other than just infectious animals, such as transmission due to environmental factors (e.g. soil, carcasses) or vectors (e.g. blood-sucking fly) to susceptible animals [10]. This transmission can theoretically cause population extinctions if agents have high case fatality rates. Here, we chose a relatively high reintroduction rate (~ 2 per year into the initial population), which likely represents a worst-case scenario. However, to improve this study, we encourage adding the specific environmental factors for each disease and incorporating spatial analyses [85, 86].

Further studies might also consider adding the potential reservoir hosts and their dynamics into the models and building two or more host models to examine the transmission among the potential hosts [87, 88, 89, 90, 91] and modelling coinfection, as there are interactions between infections such as FMDV and HS (*Pasteurella multocida*), which seen as a secondary infection in FMD outbreaks [92] and brucellosis and bTB [78]. However, our analyses provide an approach to understanding the *relative* likely impact of common endemic and emerging diseases with different traits and is a tool for understanding gaps in disease surveillance and control systems.

Strengthening disease surveillance and mitigation programs may be further achieved by targeting virulent diseases through passive and active surveillance data, such as collecting the frequency of infections, number and species of wild ungulates, behaviour and time spent together between wild and domestic livestock (particularly in the high-risk areas) [93, 94]. It may be useful for disease mitigation to largely focus on domestic animal disease control and preventing transmission to wildlife as an amenable approach [94]. Moreover, conserving wildlife habitat can reduce the probability of contact and the risk of disease transmission between wildlife and domestic livestock [95, 1]. Limiting the contact between wildlife and livestock could reduce species extinction [96].

Conclusion

Our study has provided a prediction of the potential consequence of disease in wild bovid populations considering six important bovine infectious diseases; anthrax, HS, bTB, LSD, FMD and brucellosis. The baseline population model shows a natural population growth of $\sim 228\%$, suggesting maintaining healthy vulnerable populations could allow them to reestablish and overcome

current levels of extinction threats while diseases and other factors may regulate population growth. The inclusion of different consequences on the population dynamics depends on the transmission, incubation, fatality and infectious rates. Brucellosis and bTB models show the greatest, long-term impact among all the models, whereas FMD and LSD showed the least impact, suggesting common but more chronic or 'slow' infections with relatively high mortality may pose the greatest threat to smaller, threatened bovid populations.

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References

[1] David TS Hayman. "Conservation as vaccination: Integrated approaches to public health and environmental protection could prevent future disease outbreaks". In: *EMBO reports* 17.3 (2016), pp. 286–291.

[2] James M. Hassell et al. "Urbanization and Disease Emergence: Dynamics at the Wildlife–Livestock–Human Interface". In: *Trends in Ecology & Evolution* 32.1 (Jan. 1, 2017). Publisher: Elsevier, pp. 55–67. ISSN: 0169-5347. DOI: 10.1016/j.tree.2016.09.012. URL: https://www.cell.com/trends/ecology-evolution/abstract/S0169-5347(16)30184-7 (visited on 07/27/2022).

[3] Andrew W Byrne et al. "Disturbance Ecology Meets Bovine Tuberculosis (bTB) Epidemiology: A Before-and-After Study on the Association between Forest Clearfelling and bTB Herd Risk in Cattle Herds". In: *Pathogens* 11.7 (2022), p. 807.

[4] Ferran Jori et al. "Wildlife–livestock interactions in animal production systems: what are the biosecurity and health implications?" In: *Animal frontiers* 11.5 (2021), pp. 8–19.

[5] Lee Francis Skerratt et al. "Spread of chytridiomycosis has caused the rapid global decline and extinction of frogs". In: *EcoHealth* 4.2 (2007), pp. 125–134.

[6] Tim E. Carpenter et al. "A spatial risk assessment of bighorn sheep extirpation by grazing domestic sheep on public lands". In: *Preventive Veterinary Medicine* 114.1 (2014). Special Issue: GEOVET 2013, pp. 3–10. ISSN: 0167-5877. DOI: https://doi.org/10.1016/j.prevetmed.2014.01.008. URL: https://www.sciencedirect.com/science/article/pii/S0167587714000099.

[7] Qijing Shi et al. "Endangered wild yak (Bos grunniens) in the Tibetan plateau and adjacent regions: Population size, distribution, conservation perspectives and its relation to the domestic subspecies". In: *Journal for Nature Conservation* 32 (2016), pp. 35–43.

[8] Gavin G Cotterill et al. "Parsing the effects of demography, climate and management on recurrent brucellosis outbreaks in elk". In: *Journal of Applied Ecology* 57.2 (2020), pp. 379–389.

[9] Brant A Schumaker, Dannele E Peck, and Mandy E Kauffman. "Brucellosis in the Greater Yellowstone area: disease management at the wildlife–livestock interface". In: *Human-Wildlife Interactions* 6.1 (2012), pp. 48–63.

[10] David T. S. Hayman, Reju Sam John, and Pejman Rohani. "Transmission models indicate Ebola virus persistence in non-human primate populations is unlikely". In: *Journal of The Royal Society Interface* 19.187 (Feb. 2022), p. 20210638. ISSN: 1742-5662. DOI: 10.1098/rsif.2021.0638. URL: https://royalsocietypublishing.org/doi/10.1098/rsif.2021.0638 (visited on 01/09/2023).

[11] Kate E. Jenks, JoGayle Howard, and Peter Leimgruber. "Do Ranger Stations Deter Poaching Activity in National Parks in Thailand?" In: *Biotropica* 44.6 (2012), pp. 826–833. DOI: https://doi.org/10.1111/j.1744-7429.2012.00869.x. eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1744-7429.2012.00869.x. URL: https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1744-7429.2012.00869.x.

[12] Rattanawat Chaiyarat and Sompod Srikosamatara. "Populations of domesticated cattle and buffalo in the Western Forest Complex of Thailand and their possible impacts on the wildlife community". In: *Journal of environmental management* 90.3 (2009), pp. 1448–1453.

[13] Tawatchai Singhla and Sukolrat Boonyayatra. "Prevalence, Risk Factors, and Diagnostic Efficacy of Bovine Tuberculosis in Slaughtered Animals at the Chiang Mai Municipal Abattoir, Thailand". In: *Frontiers in Veterinary Science* 9 (2022).

[14] Veerasak Punyapornwithaya et al. "Spatio-temporal patterns of lumpy skin disease outbreaks in dairy farms in northeastern Thailand". In: *Frontiers in Veterinary Science* (2022), p. 1132.

[15] Carsten Kirkeby et al. "A practical introduction to mechanistic modeling of disease transmission in veterinary science". In: *Frontiers in veterinary science* 7 (2021), p. 546651.

[16] Martin Gilbert et al. "Estimating the potential impact of canine distemper virus on the Amur tiger population (Panthera tigris altaica) in Russia". In: *Plos one* 9.10 (2014), e110811.

[17] Alexander Singer, Mo Salman, and Hans-Hermann Thulke. "Reviewing model application to support animal health decision making". In: *Preventive Veterinary Medicine* 99.1 (2011), pp. 60–67.

[18] Rattanawat Chaiyarat, Suphat Prasopsin, and Naris Bhumpakphan. "Food and nutrition of Gaur (Bos gaurus C.H. Smith, 1827) at the edge of Khao Yai National Park, Thailand". In: *Scientific Reports* 11.1 (Feb. 8, 2021). Number: 1 Publisher: Nature Publishing Group, p. 3281. ISSN: 2045-2322. DOI: 10.1038/s41598-021-82858-1. URL: https://www.nature.com/articles/s41598-021-82858-1 (visited on 07/27/2022).

[19] Ratchanee Chockcharoen et al. "Gaur recovery and management in Khao Phang Ma Non-Hunting Area, northeastern Thailand". In: *BULLetin* 3 (2020), pp. 15–22.

[20] Umphornpimon Prayoon et al. "Past, present and future habitat suitable for gaur (Bos gaurus) in Thailand". In: *Agriculture and Natural Resources* 55.5 (Oct. 31, 2021). Number: 5, pp. 743–756. ISSN: 2452-316X. URL: https://li01.tci-thaijo.org/index.php/anres/article/view/252964 (visited on 07/27/2022).

[21] Prateep Duengkae et al. "Coincidence of low genetic diversity and increasing population size in wild gaur populations in the Khao Phaeng Ma Non-Hunting Area, Thailand: A challenge for conservation management under human-wildlife conflict". In: *Plos one* 17.8 (2022), e0273731.

[22] Phachara Laichanthuek, Ronglarp Sukmasuang, and Prateep Duengkae. "Population and habitat use of gaur (Bos Gaurus) around Khao Phaeng Ma non-hunting area, Nakhon Ratchasima province". In: *Journal of Wildlife in Thailand (Thailand)* (2017).

[23] Hamish McCallum, Nigel Barlow, and Jim Hone. "How should pathogen transmission be modelled?" In: *Trends in ecology & evolution* 16.6 (2001), pp. 295–300.

[24] Matthew J Smith et al. "Host–pathogen time series data in wildlife support a transmission function between density and frequency dependence". In: *Proceedings of the National Academy of Sciences* 106.19 (2009), pp. 7905–7909.

[25] Lindsay M Beck-Johnson et al. "An exploration of within-herd dynamics of a transboundary livestock disease: A foot and mouth disease case study". In: *Epidemics* (2023), p. 100668.

[26] Giovanna Ciaravino et al. "Assessing the variability in transmission of bovine tuberculosis within Spanish cattle herds". In: *Epidemics* 23 (2018), pp. 110–120.

[27] Avner Friedman and Abdul-Aziz Yakubu. "Anthrax epizootic and migration: Persistence or extinction". In: *Mathematical Biosciences* 241.1 (Jan. 1, 2013), pp. 137–144. ISSN: 0025-5564. DOI: 10.1016/j.mbs.2012.10.004. URL: https://www.sciencedirect.com/science/article/pii/S0025556412002064 (visited on 11/11/2022).

[28] C. M. Saad-Roy, P. van den Driessche, and Abdul-Aziz Yakubu. "A Mathematical Model of Anthrax Transmission in Animal Populations". In: *Bulletin of Mathematical Biology* 79.2 (Feb. 2017), pp. 303–324. ISSN: 0092-8240, 1522-9602. DOI: 10.1007/s11538-016-0238-1. URL: http://link.springer.com/10.1007/s11538-016-0238-1 (visited on 11/10/2022).

[29] Zerihun Mamo Sinkie. "Modeling And Simulation Study Of Anthrax Attack On Environment". In: 3.4 (2016), p. 5.

[30] Paul C. Cross and Wayne M. Getz. "Assessing vaccination as a control strategy in an ongoing epidemic: Bovine tuberculosis in African buffalo". In: *Ecological Modelling* 196.3 (July 25, 2006), pp. 494–504. ISSN: 0304-3800. DOI: 10.1016/j.ecolmodel.2006.02.009. URL: https://www.sciencedirect.com/science/article/pii/S0304380006000706 (visited on 11/01/2022).

[31] Anna E. Jolles, David V. Cooper, and Simon A. Levin. "Hidden Effects of Chronic Tuberculosis in African Buffalo". In: *Ecology* 86.9 (2005). _eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1890/05-0038, pp. 2358–2364. ISSN: 1939-9170. DOI: 10.1890/05-0038. URL: https://onlinelibrary.wiley.com/doi/abs/10.1890/05-0038 (visited on 11/22/2022).

[32] ND Barlow et al. "A simulation model for the spread of bovine tuberculosis within New Zealand cattle herds". In: *Preventive veterinary medicine* 32.1-2 (1997), pp. 57–75.

[33] CJC Phillips et al. "The transmission of Mycobacterium bovis infection to cattle". In: *Research in veterinary science* 74.1 (2003), pp. 1–15.

[34] FD Menzies and SD Neill. "Cattle-to-cattle transmission of bovine tuberculosis". In: *The Veterinary Journal* 160.2 (2000), pp. 92–106.

[35] Deepak D. "Modeling Vaccination Programs in Outbreaks of Hemorrhagic Septicemia in India". In: *Journal of Animal Research* 10.1 (Feb. 19, 2020). ISSN: 22496629, 2277940X. DOI: 10.30954/2277-940X.01.2020.2. URL: https://ndpublisher.in/admin/issues/JARv10n1b.pdf (visited on 01/11/2023).

[36] MCL De Alwis. "Haemorrhagic septicaemia—a general review". In: *British Veterinary Journal* 148.2 (1992), pp. 99–112.

[37] Anderson Lopes Baptista et al. "Bovine respiratory disease complex associated mortality and morbidity rates in feedlot cattle from southeastern Brazil". In: *The Journal of Infection in Developing Countries* 11.10 (2017), pp. 791–799.

[38] Diego Nobrega et al. "Prevalence, risk factors, and antimicrobial resistance profile of respiratory pathogens isolated from suckling beef calves to reprocessing at the feedlot: A longitudinal study". In: *Frontiers in Veterinary Science* 8 (2021), p. 764701.

[39] Asadullah Khan et al. "Serosurveillance of hemorrhagic septicemia in cattle and buffaloes in district Malakand, NWFP". In: *J Agric Biol Sci* 1 (2006), pp. 11–14.

[40] Reuma Magori-Cohen et al. "Mathematical modelling and evaluation of the different routes of transmission of lumpy skin disease virus". In: *Veterinary research* 43 (2012), pp. 1–13.

[41] HY Abdulqa et al. "Lumpy skin disease". In: *Reproductive Immunology: Open Access* 1.4 (2016), p. 25.

[42] Moumita Das et al. "An updated review on lumpy skin disease: perspective of Southeast Asian countries". In: *J. adv. biotechnol. exp. ther* 4.3 (2021), pp. 322–333.

[43] Liesel Laubscher and Louwrens Hoffman. "An overview of disease-free buffalo breeding projects with reference to the different systems used in South Africa". In: *Sustainability* 4.11 (2012), pp. 3124–3140.

[44] R Core Team. *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing. Vienna, Austria, 2022. URL: https://www.R-project.org/.

[45] Sompoad Srikosamatara et al. *Populations of gaur and banteng and their management in Thailand*. Tech. rep. Thailand Science Research and Innovation, 1998.

[46] Noraset Khiowsree, Ronglarp Sukmasuang, and Utis Kutintara. "POPULATION MONITORING AND DISTRIBUTION RANGE OF WILD WATER BUFFALO (Bubalus bubalisLINNAEUS, 1758) IN HUAI KHA KHAENG WILDLIFE SANCTUARY, UTHAI THANI PROVINCE". In: *Journal of Wildlife in Thailand* 22.1 (2015), pp. 111–118.

[47] Farshid S Ahrestani et al. "Life-history traits of gaur Bos gaurus: a first analysis". In: *Mammal Review* 41.1 (2011), pp. 75–84.

[48] David Choquent. "Growth, body condition and demography of wild banteng (Bos javanicus) on Cobourg Peninsula, northern Australia". In: *Journal of Zoology* 231.4 (1993), pp. 533–542.

[49] Anthony Ronald Entrican Sinclair. *The African buffalo; a study of resource limitation of populations*. Chicago, Ill.(USA) Univ. of Chicago Press, 1977.

[50] PC Gardner et al. *Banteng Bos javanicus d'Alton, 1823., Ecology, Evolution and Behaviour of Wild Cattle: Implications for Conservation, ed. M. Melletti and J. Burton*. Published by Cambridge University Press, 2014.

[51] Joel T Heinen. "Population viability and management recommendations for wild water buffalo Bubalus bubalis in Kosi Tappu Wildlife Reserve, Nepal". In: *Biological Conservation* 65.1 (1993), pp. 29–34.

[52] ShuPei Tang et al. "Viability analysis of Chinese goral population in Saihanwula National Nature Reserve, Inner Mongolia." In: *Scientia Silvae Sinicae* 55.3 (2019), pp. 118–124.

[53] Kate E Jones et al. "PanTHERIA: a species-level database of life history, ecology, and geography of extant and recently extinct mammals: Ecological Archives E090-184". In: *Ecology* 90.9 (2009), pp. 2648–2648.

[54] Kachen Wongsathapornchai et al. "Use of epidemiologic risk modeling to evaluate control of foot-and-mouth disease in southern Thailand". In: *American Journal of Veterinary Research* 69.2 (2008), pp. 240–251.

[55] Aurelio H Cabezas, Michael W Sanderson, and Victoriya V Volkova. "A meta-population model of potential foot-and-mouth disease transmission, clinical manifestation, and detection within us beef feedlots". In: *Frontiers in veterinary science* 7 (2020), p. 527558.

[56] Andrew Dobson and Mary Meagher. "The Population Dynamics of Brucellosis in the Yellowstone National Park". In: *Ecology* 77.4 (1996). Publisher: Ecological Society of America, pp. 1026–1036. ISSN: 0012-9658. DOI: 10.2307/2265573. URL: http://www.jstor.org/stable/2265573 (visited on 11/21/2022).

[57] World Health Organization et al. *Anthrax in humans and animals*. World Health Organization, 2008.

[58] *Haemorrhagic septicaemia*. https://www.woah.org/app/uploads/2021/09/haemorrhagic-septicemia.pdf. Accessed: 2023-03-14.

[59] ME Hugh-Jones and RR Tinline. "Studies on the 1967–68 foot and mouth disease epidemic: incubation period and herd serial interval". In: *Epidemiology & Infection* 77.2 (1976), pp. 141–153.

[60] Gui-Quan Sun et al. "Transmission dynamics of brucellosis: Mathematical modelling and applications in China". In: *Computational and Structural Biotechnology Journal* 18 (2020), pp. 3843–3860.

[61] Burak Mat et al. "Determination of production losses related to lumpy skin disease among cattle in Turkey and analysis using SEIR epidemic model". In: *BMC Veterinary Research* 17 (2021), pp. 1–10.

[62] Fernando Mardones et al. "Parameterization of the duration of infection stages of serotype O foot-and-mouth disease virus: an analytical review and meta-analysis with application to simulation models". In: *Veterinary research* 41.4 (2010).

[63] N Thompson Hobbs et al. "State-space modeling to support management of brucellosis in the Yellowstone bison population". In: *Ecological Monographs* 85.4 (2015), pp. 525–556.

[64] Anderson Lopes Baptista et al. "Bovine respiratory disease complex associated mortality and morbidity rates in feedlot cattle from southeastern Brazil". In: *The Journal of Infection in Developing Countries* 11.10 (2017), pp. 791–799.

[65] G Govindaraj et al. "Foot and Mouth Disease (FMD) incidence in cattle and buffaloes and its associated farm-level economic costs in endemic India". In: *Preventive Veterinary Medicine* 190 (2021), p. 105318.

[66] Simon More et al. "Assessment of listing and categorisation of animal diseases within the framework of the Animal Health Law (Regulation (EU) No 2016/429): Venezuelan equine encephalitis". In: *EFSA Journal* 15.8 (2017).

[67] Rishendra Verma and TN Jaiswal. "Haemorrhagic septicaemia vaccines". In: *Vaccine* 16.11-12 (1998), pp. 1184–1192.

[68] ESM Tuppurainen and CAL Oura. "lumpy skin disease: an emerging threat to Europe, the Middle East and Asia". In: *Transboundary and emerging diseases* 59.1 (2012), pp. 40–48.

[69] Anna Jolles et al. "Endemic persistence of a highly contagious pathogen: Foot-and-mouth disease in its wildlife host". In: *Science* 374.6563 (Oct. 2021). Publisher: American Association for the Advancement of Science, pp. 104–109. DOI: 10.1126/science.abd2475. URL: https://www.science.org/doi/full/10.1126/science.abd2475 (visited on 10/24/2022).

[70] Anke K Wiethoelter et al. "Global trends in infectious diseases at the wildlife–livestock interface". In: *Proceedings of the National Academy of Sciences* 112.31 (2015), pp. 9662–9667.

[71] Fredrick Tom Otieno et al. "Modeling the spatial distribution of anthrax in southern Kenya". In: *PLOS Neglected Tropical Diseases* 15.3 (Mar. 29, 2021). Publisher: Public Library of Science, e0009301. ISSN: 1935-2735. DOI: 10.1371/journal.pntd.0009301. URL: https://journals.plos.org/plosntds/article?id=10.1371/journal.pntd.0009301 (visited on 11/10/2022).

[72] Damien O Joly and Francois Messier. "The effect of bovine tuberculosis and brucellosis on reproduction and survival of wood bison in Wood Buffalo National Park". In: *Journal of Animal Ecology* (2005), pp. 543–551.

[73] Tawatchai Singhla, Pallop Tankaew, and Nattawooti Sthitmatee. "Validation of a novel ELISA for the diagnosis of hemorrhagic septicemia in dairy cattle from Thailand using a Bayesian approach". In: *Veterinary Sciences* 7.4 (2020), p. 163.

[74] Malcolm CL De Alwis. *Haemorrhagic septicaemia*. 1999.

[75] Sarah Robinson et al. "Opportunistic bacteria and mass mortality in ungulates: lessons from an extreme event". In: *Ecosphere* 10.6 (2019), e02671.

[76] Ferran Jori and Eric Etter. "Transmission of foot and mouth disease at the wildlife/livestock interface of the Kruger National Park, South Africa: Can the risk be mitigated?" In: *Preventive veterinary medicine* 126 (2016), pp. 19–29.

[77] Thanicha Chanchaidechachai et al. "Epidemiology of foot-and-mouth disease outbreaks in Thailand from 2011 to 2018". In: *Transboundary and Emerging Diseases* (2022).

[78] Erin E. Gorsich et al. "Opposite outcomes of coinfection at individual and population scales". In: *Proceedings of the National Academy of Sciences* 115.29 (July 17, 2018), pp. 7545–7550. ISSN: 0027-8424, 1091-6490. DOI: 10.1073/pnas.1801095115. URL: https://pnas.org/doi/full/10.1073/pnas.1801095115 (visited on 01/09/2023).

[79] Engineering National Academies of Sciences, Medicine, et al. *Revisiting brucellosis in the greater Yellowstone area*. National Academies Press, 2021.

[80] Jacques Godfroid. "Brucella spp. at the wildlife-livestock interface: an evolutionary trajectory through a livestock-to-wildlife "host jump"?" In: *Veterinary sciences* 5.3 (2018), p. 81.

[81] Farshid S Ahrestani, K Ullas Karanth, et al. *Gaur (Bos gaurus CH Smith, 1827)*. 2014, pp. 174–193.

[82] Sompoad Srikosamatara. "Density and biomass of large herbivores and other mammals in a dry tropical forest, western Thailand". In: *Journal of Tropical Ecology* 9.1 (1993), pp. 33–43.

[83] Yixin Chen et al. "Activity rhythms of coexisting red serow and Chinese serow at Mt. Gaoligong as identified by camera traps". In: *Animals* 9.12 (2019), p. 1071.

[84] Angela D Luis, Amy J Kuenzi, and James N Mills. "Species diversity concurrently dilutes and amplifies transmission in a zoonotic host–pathogen system through competing mechanisms". In: *Proceedings of the National Academy of Sciences* 115.31 (2018), pp. 7979–7984.

[85] Kim M Pepin et al. "Optimising response to an introduction of African swine fever in wild pigs". In: *Transboundary and Emerging Diseases* 69.5 (2022), e3111–e3127.

[86] Pejman Rohani et al. "Environmental transmission of low pathogenicity avian influenza viruses and its implications for pathogen invasion". In: *Proceedings of the National Academy of Sciences* 106.25 (2009), pp. 10365–10369.

[87] Xavier Roche et al. *Introduction and spread of lumpy skin disease in South, East and Southeast Asia: Qualitative risk assessment and management*. Food & Agriculture Org., 2021.

[88] Alicia Aranaz et al. "Bovine tuberculosis (Mycobacterium bovis) in wildlife in Spain". In: *Journal of clinical microbiology* 42.6 (2004), pp. 2602–2608.

[89] Jacques Godfroid et al. "Brucellosis in terrestrial wildlife". In: *Revue Scientifique et Technique. Office International des Epizooties* (2013).

[90] Abdifetah Mohamed. "Bovine tuberculosis at the human–livestock–wildlife interface and its control through one health approach in the Ethiopian Somali pastoralists: A review". In: *One Health* 9 (2020), p. 100113.

[91] Katherine Royce and Feng Fu. "Mathematically modeling spillovers of an emerging infectious zoonosis with an intermediate host". In: *PloS one* 15.8 (2020), e0237780.

[92] Basavegowdanadoddi Marinaik Chandranaik et al. "Serotyping of foot and mouth disease virus and Pasteurella multocida from Indian gaurs (Bos gaurus), concurrently infected with foot and mouth disease and haemorrhagic septicaemia". In: *Tropical Animal Health and Production* 47.5 (June 2015), pp. 933–937. ISSN: 0049-4747, 1573-7438. DOI: 10.1007/s11250-015-0811-x. URL: http://link.springer.com/10.1007/s11250-015-0811-x (visited on 01/11/2023).

[93] Matthew J Silk et al. "Integrating social behaviour, demography and disease dynamics in network models: applications to disease management in declining wildlife populations". In: *Philosophical Transactions of the Royal Society B* 374.1781 (2019), p. 20180211.

[94] RG Bengis, RA Kock, and J Fischer. "Infectious animal diseases: the wildlife/livestock interface." In: *Revue scientifique et technique (International Office of Epizootics)* 21.1 (2002), pp. 53–65.

[95] Alexandre Caron et al. "Relationship between burden of infection in ungulate populations and wildlife/livestock interfaces". In: *Epidemiology & Infection* 141.7 (2013), pp. 1522–1535.

[96] Amy B Pedersen et al. "Infectious diseases and extinction risk in wild mammals". In: *Conservation biology* 21.5 (2007), pp. 1269–1279.