1   **Impact of Infectious Diseases on Wild Bovidae Populations in Thailand: Insights**

2   **from Population Modelling and Disease Dynamics**

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8   **Abstract**

9   The wildlife and livestock interface is vital for wildlife conservation and habitat

10   management. Infectious diseases maintained by domestic species may impact threatened

11   species such as Asian bovids, as they share natural resources and habitats. To predict the

12   population impact of infectious diseases with different traits, we used stochastic mathematical

13   models to simulate the population dynamics 100 times over 100 years for a model gaur (*Bos

14   gaurus*) population with and without disease. We simulated repeated introductions from a

15   reservoir, such as domestic cattle. We selected six bovine infectious diseases; anthrax, bovine

16   tuberculosis, hemorrhagic septicaemia, lumpy skin disease, foot and mouth disease and

17   brucellosis, all of which have caused outbreaks in wildlife populations. From a starting population

18   of 300, the disease-free population increased by an average of 228% over 100 years. Brucellosis

19   with frequency-dependent transmission showed the highest average population declines (-97%),

20   with population extinction occurring 16% of the time. Foot and mouth disease with frequency-

21   dependent transmission showed the lowest impact, with an average population increase of

22   200%. Overall, acute infections with very high or low fatality had the lowest impact, whereas

23   chronic infections produced the greatest population decline. These results may help disease

24   management and surveillance strategies support wildlife conservation.

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

26   **Introduction**

27   Livestock encroachment into wildlife habitats can drive disease transmission between

28   wildlife and domestic livestock, which is a vital issue for both human public health and wildlife

29   conservation. An effect of agricultural expansion and land-use change is to bring wildlife and

30   livestock close to each other and increase the contact frequency and time between domestic and

31   wildlife populations (1-3). This increased contact may increase the risk of disease transmission as

32   they can share the same natural resources (e.g. grassland and water) (4).

33   Infectious diseases can cause dramatic declines in wildlife populations, as demonstrated

34   by chytridiomycosis, which has been implicated in the likely extinction of over 200 amphibian

35 species (5). Most infectious bovid pathogens are capable of infecting both domestic and wild
36 species. For example, bighorn sheep populations declined from ovine respiratory disease
37 (*Mycoplasma ovipneumoniae*) acquired when sharing the grazing areas with domestic sheep (6).
38 Similarly, bovine brucellosis has been transmitted from domesticated yak to wild yak in China (7),
39 and between bison, elk and domestic cattle in the USA (8). Brucellosis affects these bison and elk
40 populations both indirectly and directly as the seropositive animals may be culled for
41 management and directly as the pathogen affects animal reproductive systems (9). Critically, the
42 impact of infectious diseases is determined by disease-specific traits, such as infection fatality
43 rates (10).

44 There are five wild bovids species (gaur, banteng, wild water buffalo, mainland serow
45 and Chinese goral) that remain in Thailand. They are experiencing dramatic population declines
46 from habitat destruction, illegal hunting (11), and resource competition with domestic livestock
47 (12). Infectious diseases transmitted from contact with domestic cattle could cause further
48 declines. Several diseases circulate in Thai cattle, including endemic diseases like bovine
49 tuberculosis (bTB) from *Mycobacterium bovis* (13), and new infectious diseases, such as the
50 recent lumpy skin disease (LSD) (14).

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

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

72 **Material and methods**

73   **1. Model construction**

74   **1.1. Population dynamic models**

75   We selected gaur populations as a model system because they are widespread across
76 Thailand, overlap with livestock and people, and demographic data are available (18, 20).
77 Further, their demography is similar to other threatened wild bovids (Figure S1 in supplementary
78 material). We used the same model structure for all species. The demographic parameters for
79 the remaining four bovid species used in simulations are provided in Table 2 because they exhibit
80 variations in population sizes, social behaviours, and distribution, making them interesting for
81 further infectious disease modelling of population impact.

82   We assumed demographic parameters were otherwise constant. If N is the total animal
83 population, N_a is the adult population, N_{sa} is the subadult population, N_c is the calf population
84 and μ is the annual birth rate. Only adult females were assumed to add new calves to the
85 population, which enter the susceptible class at a birth rate $\mu_b N_a$. Animals can leave their
86 compartments at the natural death rate (μ_a, μ_{sa} or μ_c) or age from calf to subadult (δ_c) and from
87 subadult to adult (δ_{sa}). The natural death rate was estimated based on the mortality rate of wild
88 ungulates and gaur in captivity (21). The initial population was 300 animals, based on the gaur
89 population size in the Khao Pheang Ma non-hunting area (8 km^2) in Thailand (22, 23). Thus, the
90 population dynamic model equations at time t can be as following:

91 $$N_t \quad = \quad N_c + N_{sa} + N_a$$

92 $$\frac{dN_c}{dt} \quad = \quad \mu_b N_a + \delta_c N_c - \mu_c N_c$$

93 $$\frac{dN_{sa}}{dt} = \quad \delta_c N_c - \delta_{sa} N_{sa} - \mu_{sa} N_{sa}$$

94 $$\frac{dN_a}{dt} \quad = \quad \delta_{sa} N_{sa} - \mu_a N_a$$

95   (Equation 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 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, haemorrhagic 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).

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

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

1.3. Mode of transmission

Different transmission types can provide different model results (24). 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, 25). We assumed the transmission modes for each pathogen and then compared them by introducing both transmission modes because, for some infections,

125   there is no clear evidence of which type suits the pathogen's transmissions and these represent

126   extreme situations of population change for both transmission modes (10).

127   Transmission is often likely a mix of both DD and FD in many cases such as FMD and bTB

128   (26, 27). For the transmission rate (β), we used parameter values based on the reference studies

129   with the reported FD or DD transmission (Table 2), which differs among infectious diseases.

130   However, to test the sensitivity of the results to these assumptions, we also rescaled the β rate

131   to all models to examine the consistency of the results between FD and DD using Equations 2:

132

$$\beta_{DD} = \frac{\beta_{FD}}{N}$$

133

$$\beta_{FD} = \beta_{DD} \; x \; N \qquad\qquad \text{(Equation 2)}$$

134   **1.4.   Infection reintroduction**

135   To model the repeated introduction of an infection from a reservoir such as domestic

136   cattle (e.g. for FMD) or the environment (e.g. anthrax), we repeatedly reintroduced infection

137   into our population at rate ϵ independently of any infection in the population. This

138   reintroduction means the impact of infections are not simply estimated by the basic

139   reproductive number (R_0), the average number of secondary cases caused by a primary case in

140   a completely susceptible population.

141   **Anthrax (Bacillus anthracis)**

142   To model anthrax, we initially assumed the transmission is FD. We used an SI model (28,

143   29) with the transmission rate for FD at 0.01, then rescaled to FD using Equation 2. We assumed

144   that S animals are exposed to infected animals and then become infectious (I) at rate β. All

145   infected animals (I) die (100% mortality) (30) at disease-induced death rate (ρ) and the infectious

146   rate (γ), $\gamma\rho I$.

147   **Bovine tuberculosis (bTB - *Mycobacterium tuberculosis*)**

148   Bovine TB is a chronic and zoonotic infection in livestock and wildlife worldwide (31).

149   We first assumed DD transmission and used an SEI structure for modelling. The flow of the model

150   starts from S, which are exposed to I animals and become exposed (E) at transmission rate (β);

151   then E animals enter the I compartment at the incubation rate (σ). As we assume lifelong

152   infection without recovery (31); I animals either die with an age-specific disease-induced fatality

153   rate (ρ) or natural death rate (μ). S and E adults give birth with the normal birth rate $\mu_b(S_a + E_a)$

154   but I adults are assumed to have a lower fecundity rate (reduced by 27%, (32) at $\mu_{bI}I_a$. Bovine TB

155   has a long incubation period from several months up to 7 years (33), so here we used 5 months

156   based on the mean incubation period in the African buffalo (31). We also assumed there's no

157   vertical or pseudo-vertical (e.g. in utero or calf rearing) transmission as it is uncommon for bTb
158   (34, 35).

Haemorrhagic septicaemia (HS - *Pasteurella multocida*)

160   HS is a fatal septicaemic disease in cattle and buffalo. We assume DD transmission based
161   on a previous HS modelling study (36). We used an $SIRS$ model and excluded an E class as the
162   disease can show acute clinical signs with a short incubation period of $\sim$ 18-20 hours (37)
163   animals become I at the transmission rate (β). I animals may die from HS at the disease-induced
164   fatality rate (ρ) or survive and become recovered (R) at infectious rate ($1/\gamma$). We calculated the
165   fatality rate in the cattle population to range from 0.53% to 5.84%. This was determined by
166   dividing the number of deaths from HS (0.21%, assumed from the percentage of deaths from
167   bovine respiratory disease, (38) by the minimum (3.59%) and maximum (40%) prevalence of
168   seropositive animals from *P. multocida* infected herds (39, 40). Therefore, we used two infection
169   fatality rates (0.53 and 5.83%), since case fatality is underestimated, as a large proportion of
170   animals are infected but do not develop clinical signs of diseases. R animals reenter S when they
171   lose immunity at the immunity loss rate (ω). We used the proportion of susceptible animals (0.6)
172   to calculate R_0 and therefore the β rate using the equation: $R_0 = (1/(1-I)) = 1/S$.

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

174   We used an $SEIRS$ structure for LSD. We inserted E and R compartments as the disease
175   has an incubation period of between 7 and 14 days and a recovery period of around 4 - 6 months.
176   We initially assumed the transmission is DD as the cattle density could be one of the risk factors
177   to increase the transmission rate within-herd. However, we used both FD and DD β values
178   because the published work has reported differences in incidence rates associated with different
179   transmission modes (41). We assumed different birth rates for I females (μ_{bI}) from the natural
180   birth rate, because LSD can reduce the fertility rate by 10% (42). Also, we applied the highest
181   fatality rate in calves (5%) and lower mortality rates to subadults (3%) and adults (1%) (43).

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

184   We initially assumed the transmission was FD for both FMD and brucellosis. An
185   $SEIRMS/E$ model was applied for FMD and brucellosis. We considered the $SEIR$ model
186   appropriate for both diseases. Recovered FMD and brucellosis cows can pass immunity to their
187   offspring. Therefore, we added a maternally-derived immunity (M) compartment, which refers
188   to the calves born with maternally-derived immunity from recovered mothers (R_a). We assumed
189   that if recovered adults (R_a) calve at the birth rate (μ_b), a calf will receive maternal immunity and
190   stay in M compartment for an average of 6 months (44) before immunity wanes and they become
191   susceptible again (S_m) at a loss of immunity rate (ω_m). S_m calves can either become an exposed

192   calf (E_c) if there is contact with I or enter a susceptible subadult (S_{sa}) compartment at a loss of

193   immunity rate plus calf ageing rate: $1/\delta_c = 1/(\delta_m + \omega_m)$ or $1/\delta_m = 1/(\delta_c - \omega_m)$ if they have no contact

194   with I to ensure that animals spend the same average time in the calf age class ($_c$).

195   Vertical transmission from mothers to calves can be a consequence of infection among

196   infected mothers with different probabilities for FMD (~ 0.5) and brucellosis (~ 0.9). Infectious

197   adults are assumed to produce an infectious calf (I_c) entering I at the birth rate $\mu_b I$. The

198   proportion of infected females producing infected calf denotes α. So, an infected female can

199   produce an infected calf at a rate $\alpha\mu_{bI}I_a$ and produce a susceptible calf at a rate $(1-\alpha)\mu_{bI}I_a$.

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

$$\frac{dS_c}{dt} = \underbrace{\mu_b(S_a + E_a) + (1-\alpha)\mu_b I_a}_{\text{birth}} - \underbrace{\beta_c S_c(I_c + I_{sa} + I_a)}_{\text{transmission rate}} - \underbrace{\delta_c S_c}_{\text{ageing rate}} - \underbrace{\mu_c S_c}_{\text{natural death}} + \underbrace{\omega_c R_c}_{\text{recovey rate}} - \underbrace{\epsilon S_c}_{\text{force of infection}}$$

$$\frac{dE_c}{dt} = \beta_c S_c(I_c + I_{sa} + I_a) + \beta_c S_m(I_c + I_{sa} + I_a) - \sigma_c E_c - \delta_c E_c - \mu_c E_c + \epsilon S_c$$

$$\frac{dI_c}{dt} = \sigma_c E_c - (1-\rho_c)\gamma_c I_c - \rho_c \gamma_c I_c - \delta_c I_c - \mu_c I_c + \alpha\mu_{bI}I_a$$

$$\frac{dR_c}{dt} = (1-\rho_c)\gamma_c I_c - \omega_c R_c - \delta_c R_c - \mu_c R_c$$

$$\frac{dM}{dt} = \mu_b R_a - \omega_M M - \mu_c M$$

$$\frac{dS_m}{dt} = \omega_M M - \delta_m S_m - \beta_m S_m(I_c + I_{sa} + I_a)$$

201

202   (Equation 3)

203   The system of ordinary differential equations and all other disease model equations and

204   diagrams can be found in the supplementary materials. Note that all equations are subsets or

205   variants of Equation 3.

206   **2. Model simulations**

207   Due to the small population size of gaur and other endangered bovids, we were

208   interested in how infections might lead to their decline. Additionally, as we aimed to allow

209   infections to go extinct in populations if they could not be sustained. Therefore, we chose

210   stochastic models for this study because they effectively capture the stochastic nature of wildlife

211   populations using random values. This randomness introduces variation in population sizes,

212   which significantly affects small population sizes and long-term simulations (45). First, we built

213   the population dynamics model without infectious disease classes and parameters as a baseline

214   model (10). Then, we introduced an infectious adult ($I_a = 1$) to the susceptible (S) population. We

215   assumed that I would infect S at a transmission rate, β, and enter the next compartment based

216   on the model structure. Demography (birth rate, natural death rate and ageing rate), the external

217   force of infection (ϵ), and disease-induced fatality (ρ) were included in all disease models. The

218   stochastic simulation was performed using the Poisson distribution to calculate the probability

219   of events by multiplying the rate parameters i with a time step through Gillespie's τ-leap
220   algorithm (τ = 1) (Equation 4).

$$Prob_i = Poisson(\tau * rate_i * X)$$

221   (Equation 4)

222   Where X is a state (e.g. S, I, R). All models were simulated for 100 years, and the
223   stochastic models were simulated 100 times to generate the mean and to understand the
224   uncertainty. We modelled the population change for 100 years, as long-term simulations of at
225   least 10 years or three generations of species is recommended to explore population trends, and
226   short-term time series may lead to misleading conclusions (46).

227   The parameter values used for modelling were collected from the literature review and
228   observational data (Table 2).

229   **3. Measuring impact**

230   We compared the difference in total population (N) between no infection and disease
231   models by calculating the average percentage of the population change using the total
232   population at the start ($N_{t=0}$) minus the total population at the end, ($N_{t=100}$) of the simulation
233   time, divided by $N_{t=0}$ and converted this to a percentage, then divided by 100 times of simulations,
234   using the following equation:

$$\bar{x} = (\sum_{i=1}^{100} \frac{N_t - N_{t=0}}{N_{t=0}}/100) \times 100$$

235   (Equation 5)

236   We used a principal component analysis (PCA) to find which diseases showed similar
237   traits group by four disease parameters (transmission rate, incubation rate, infectious rate and
238   fatality rate) which were included in all models and then coloured the values based on the
239   percentage of the total population change. We performed PCA in R software using the PCATools
240   package (47). The highest percentage of the first two axes contributed most to the population
241   percentage changes.

242   **4. Code availability**

243   We used R Core Team (48) to simulate all the models and for further analysis. The R
244   code for reproducing the analyses is available at a GitHub repository
245   https://github.com/Wantidah/InfectiousModel.

246   **Results**

247   **1. Disease free model**

248   We developed stochastic models for a gaur population, including a baseline model
249 without infection and six infectious disease models. The baseline model of the gaur population
250 demonstrated significant population growth, increasing from 300 to an average of 685 (range
251 113 - 1469) additional animals, which is approximately a 228% (38 - 489%) increase over the 100
252 years simulated. The average adult and subadult populations consistently increased, while the
253 calf population slightly decreased from 95 to 82 animals (19 - 279) on average (Figure 1: A&I).
254 This gave us a disease-free population to model the impact of disease introduction into.

255   The population dynamics of the four other wild bovid species in Thailand show similar
256 trends to the gaur population (Figure S1, Figure 1), so we assumed there will be similar trends
257 for the other two large bovids (banteng, wild water buffalo) that have similar herd sizes,
258 population demography (e.g. age-structured, birth rate, death rate) and social behaviours to gaur
259 (49).

260   However, the population dynamics may differ from the medium-sized bovids (Chinese
261 goral and mainland serow) that live in smaller groups or even pairs and can be isolated from each
262 other (50).

2. Disease impacts

264   Brucellosis had the greatest impact on population decline, while FMD had the lowest
265 impact. Our PCA quantitatively shows that pathogens with longer incubation periods, chronic
266 infection and medium to low fatality lead to greater population decline in smaller populations of
267 endangered bovids than a high fatality or high transmission rate alone. Most diseases were
268 grouped by similar traits which can see in the PCA biplot for brucellosis, bTB and LSD, while a few
269 variations were seen for HS, and the FMD FD model was something of an outlier, (rescaling DD)
270 at β = 6552 (Figure S17 and Figure S18). The greatest contribution to the percentage population
271 change in the first axis was the infectious rate (55%) and fatality rate (42%). For the second axis,
272 is the incubation period (74%). The first axis, PC1, has 43.25% and second axis PC2 has 31.61% of
273 the variance explained.

274   Using different parameter values, fatality rates and modes of transmission yielded
275 different effects on the modelled populations for HS, FMD, LSD and brucellosis. FD brucellosis
276 had the largest population impact, yet DD brucellosis suppressed population growth but led to a
277 stable population. In contrast, FD transmission of HS, LSD and FMD showed a continued
278 population increase. Anthrax and bTB showed only a slight difference in the average population
279 change between the two transmission modes (Figure 3; (A)). Simply rescaling the β with
280 modelling FD or DD transmission had limited changes, which demonstrated consistency in the
281 population change within the same infectious disease. Rescaling the β also reduced the
282 probability of local extinction in the gaur population (N = 0) for FD brucellosis. Figure 2 and Figure
283 3 present the results of rescaling.

284   For LSD, FMD and brucellosis highlighting differences in population trends between FD
285 and DD transmissions. We selected some important model results in Figure 1, and all modelling
286 results and diagrams for infectious diseases and population changes can be found in the
287 supplementary material, Figure S2 – Figure S16.

288   **Anthrax**

289   There is no substantial impact on the population after introducing anthrax into the
290 population, with a similar population change observed between FD and DD models. Both
291 transmission rates showed an increase in population, with a 57% increase for FD and 51% for
292 rescaled DD model. No massive die offs were predicted, with only 1-3 infectious animals
293 predicted for each outbreak for both transmission modes, consistent with the low transmission
294 rate (β = 0.01) applied and a rare case of animal-to-animal transmission.

295   **Bovine tuberculosis**

296   There was uncertainty regarding the mode of transmission in bTB models, however, (31)
297 showed limited qualitative differences in model outcomes when they used FD or DD
298 transmission. Here, we saw similar results in that overall the populations tended to decline
299 gradually through the simulation period with a 88 - 89% decline from the initial population.
300 Rescaling the β transmission parameter also led to limited qualitative differences in population
301 trends, but we did see differences in predicted classes; for example, this increased or decreased
302 the number of infected individuals over time (i.e., higher or lower prevalence) (see
303 supplementary materials, Figure S5 – S6).

304   **Haemorrhagic septicaemia**

305   For HS, we found that the impact of infection was less dependent on the mode of
306 transmission than case fatality. A ten-fold increase in fatality rate led to a decline in the total
307 population change (Figure S8 – S9).

308   **Lumpy skin disease**

309   For LSD, we found that the two published transmission parameter values (0.008 and
310 0.032) led to differing outcomes that also depended on the mode of transmission (41). Whilst
311 rescaling the parameters did not lead to qualitative differences, the use of the parameter values
312 estimated from direct density-dependent transmission within herds from

313   Table 2 Parameters and variables.

Symbol	Description	Species						Units	References
		Gaur	Banteng	Buffalo	Serow	Goral			
N	Starting total population	300	470	69	120 (assume)	292 (assume)		animal	
μ_b	Birth rate	0.34	0.35	0.40	0.70	0.50		year^{-1}	(22, 51, 52)
μ_c	Calf death rate	0.27	0.26	0.27	0.50	0.45		year^{-1}	(21, 53, 54)
μ_{sa}	Subadult death rate	0.15	0.26	0.15	0.15	0.28		year^{-1}	(21, 55-57)
μ_a	Adult death rate	0.17	0.15	0.20	0.28	0.18		year^{-1}	(21, 55-57)
δ_c	Calf ageing	0.0027	0.0027	0.0027	0.0027	0.0027		day^{-1}	(58)
δ_{sa}	Subadult ageing	0.0009	0.0009	0.0009	0.0009	0.0009		day^{-1}	(58)

Symbol	Description	Disease						Units	References
		Antrax	bTB	HS	LSD	FMD	Brucellosis		
β	Disease transmission rate	0.01- 3x10^{-5}	1.4x10^{-3}	0.330	0.008-0.032	0.15-0.026	5.5x10^{-3} - 5.5x10^{-6}	day^{-1}	(29, 31, 36, 41, 59-61)
σ	1/Incubation period	0.14	6.7x10^{-3}	-	0.14	0.13	0.07	day^{-1}	(31, 41, 62-65)
γ	1/Infectious period	1	-	0.33	0.03	0.20	0.0014	day^{-1}	(66-69)
ρ_c	Disease-induced fatality in calf	1	0	0.53 - 5.84	0.05	0.10	0.10	day^{-1}	(30, 32, 38, 43, 70, 71)
ρ_{sa}	Disease-induced fatality in subadult	1	0	0.53 - 5.84	0.03	0.05	0.05	day^{-1}	(30, 32, 38, 43, 70, 71)
ρ_a	Disease-induced fatality in adult	1	0.11	0.53 - 5.84	0.01	0.03	0.03	day^{-1}	(30, 32, 38, 43, 70, 71)
α	Infected female will produce infected calf	0	-	-	-	0.50	0.9	day^{-1}	(61)
μ_{bI}	Birth rate for infectious individuals	-	6.8x10^{-4}	-	8x10^{-4}	8x10^{-4}	5x10^{-4}	day^{-1}	(32, 42, 61)
ω_c	Losing of immunity for calf	-	-	5.6x10^{-3}	5.6x10^{-3}	8.3x10^{-3}	5.6x10^{-3}	day^{-1}	(66, 72-74)
ω_{sa}	Losing of immunity for subadult	-	-	5.6x10^{-3}	5.6x10^{-3}	8.3x10^{-3}	5.6x10^{-3}	day^{-1}	(66, 72-74)
ω_a	Losing of immunity for adult	-	-	5.6x10^{-3}	5.6x10^{-3}	1.8x10^{-3}	5.6x10^{-3}	day^{-1}	(66, 72-74)
ω_m	Waning of maternal immunity	-	-	-	-	6.9x10^{-3}	5.6x10^{-3}	day^{-1}	(66, 73)
ϵ	External force of infection rate	2x10^{-5}	2x10^{-5}	2x10^{-5}	2x10^{-5}	2x10^{-5}	2x10^{-5}	day^{-1}	(10)

314
315
316
[To interpret the parameters, any rate r can be converted to probability $P(t)$ using $1 - exp^{rt}$, where t is the time period, e.g. for ϵ, $P(t) = 1 - exp^{\epsilon t} = 1 - exp^{2x10^{-5}*365} = 0.007$, or if the total S in the population is 300, ≈ 2 events per year. The dashed line (-) means no parameters were used in the models.]

317   (41) led to a population decline. However, the estimate from the indirect transmission
318   (presumably via mechanical transmission from flies) did not and the modelled population still
319   grew by 155% with FD LSD (Figure S11).

320   **Foot and mouth disease**

321   The least impact on the modelled population was seen in the FMD model with FD
322   transmission, which predicted the total population growing by 200%, around 28% less than the
323   disease-free population. Frequency-dependent FMD transmission with a β transmission rate of
324   0.115 and the rescaled DD parameter 3e-4 similarly had limited impact on the population growth
325   with an increasing population over time (Figure 2). Increasing β in the DD model, however,
326   decreased the total population by - 80% at β = 21, which had a greater impact on the population
327   change from 130% at β = 3e-4. FMD also showed a periodic pattern with outbreaks around every
328   3 - 5 years (Figure 1). Increasing the β rate from 0.11 to 21 in FD FMD models led to similar
329   dynamics close to DD transmission (Figure S13 – Figure S14).

330   **Brucellosis**

331   Brucellosis with FD transmission led to a 97% decrease in the average population change
332   (Figure 2 (H)) and was most likely to drive the population to local extinction with 16% of the total
333   simulations leading to extinction, mostly occurring from year 80-100 (Figure 1 (H, P), and Figure
334   S15).
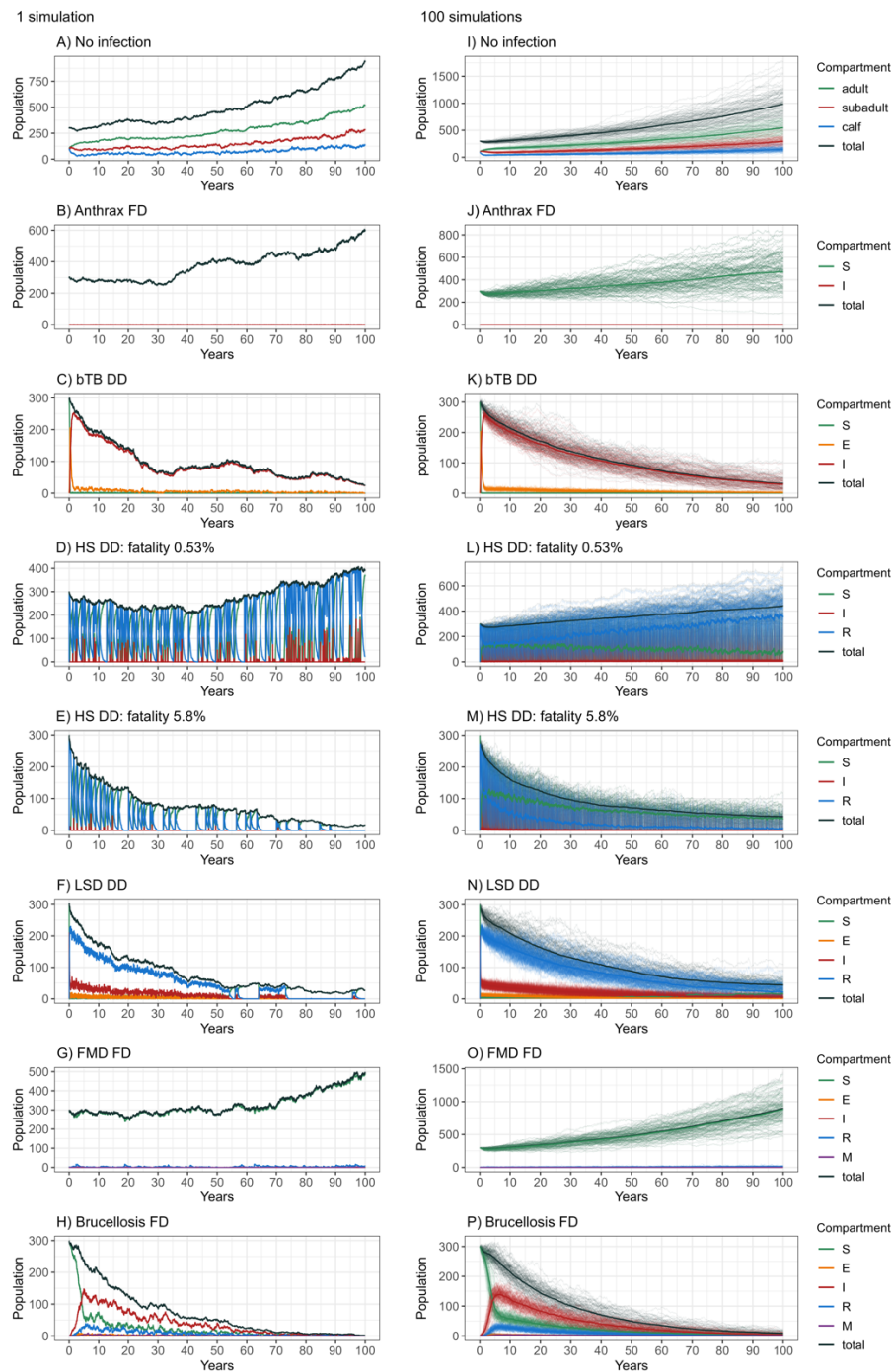
Figure 1 Modelled gaur population dynamics with and without disease: A-H are single example stochastic simulations for 100 years; I-P are 100 stochastic simulations for 100 years. Mean values are in solid lines. A and I are no infection models and the others are the infectious disease models where bTB is bovine tuberculosis; HS haemorrhagic septicaemia; LSD lumpy skin disease; and FMD foot and mouth disease. The entire model results, including all disease parameters used in the simulations, can be found in the supplementary materials.

Figure 2 Population dynamics for LSD, FMD and brucellosis 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.
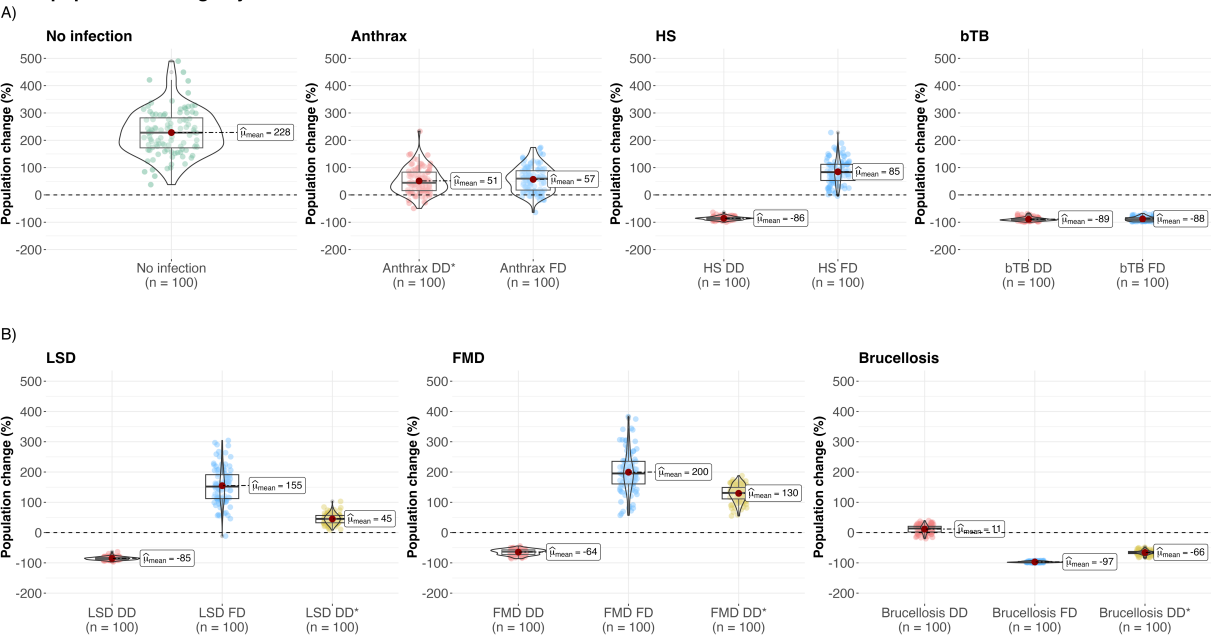
Figure 3 Overall modelled gaur population changes for each infection. Shown are the 100 results after 100 years of 100 stochastic simulations. The x-axis is the type of disease transmission and y axis is the population change in percentage. A) (top) compares no infection, FD and DD for anthrax, HS and 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.

Discussion

Interactions between wildlife and livestock can facilitate the transmission of emerging infectious diseases (75), making this interface an essential area 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 1 – Figure 3, Figure S5 – S6 and S15). This is most likely

369 because these traits allow infections to persist, allowing long-term infection effects on
370 demographic structures (e.g. reduced birth rate, increased death rate).

371 We used 100% fatality rates in all infected animals as the worst-case scenario for the
372 anthrax model, which led to limited impact over the 100 years, likely because of this rapid
373 removal of infected individuals (I), despite repeated reintroduction of infection (10). We first
374 considered anthrax transmission between infected and susceptible animals as FD transmission,
375 assuming contact rate is more influential than host density (29). However, the transmission mode
376 could also be DD, based on the density of spores in the contaminated environment (e.g. infected
377 carcass, soil) (28) and the cattle density that could contribute to between-species (76). Thus, we
378 modelled repeated introductions through ϵ to cover the external force of infections, including
379 the risk of disease transmission from cattle, other than within-herd transmission.

380 Bovine tuberculosis causes chronic, fatal infection and reduces pregnancy rates and,
381 therefore, the population growth of wild bovids (32, 77). There is no current evidence of bTB
382 infection driven population declines in Asian wild bovids, however our study found that,
383 regardless of both transmission modes, the long-term effect of bTB would be to reduce the
384 expected total population by around 88-89%. This is similar to findings by Jolles et al. (2005) (32),
385 who showed that bTB persisted in African buffalo populations and reduced adult buffalo
386 numbers primarily through mortality of animals more than 4.5 years old. The transmission
387 coefficient (β) was noted as one of the most important parameters for bTB in African buffalo (31).
388 In our work, we found consistent population dynamics between FD and DD transmission, defined
389 by similar trends and percentages of population change, when converting β between the original
390 value (from several studies) and the rescaled values (Figure 2 – Figure 3). This is likely due to the
391 duration of the infection, which might increase the probability of contact with infectious animals
392 in the population and the number of transmissions.

393 For HS, many animals are infected but do not develop clinical signs, making it difficult to
394 detect an infected animals. Further, variable clinical signs make positive cases difficult to detect,
395 therefore the case fatality rate is normally substantially higher than the actual infection fatality
396 rate. In our study, we calculated the fatality rate using the prevalence of seropositive animals
397 (max = 40%) from reported studies of cattle populations, and this substantially decreased the
398 case fatality from 90% to around 6% of animals (40, 78). Our model shows that changing the
399 mortality from 0.53% to 5.8% affects the total population numbers more than changing the
400 transmission modes, by more strongly reducing the population sizes (Figure S8 – Figure S9). HS
401 antibodies were found in free-ranging buffalo in Asia so this population might be a reservoir, but
402 this needs further investigation (37). HS is endemic among cattle in Thailand (78), and mortality
403 in wild ungulates has been reported historically (79), so the mortality and infection status of HS
404 should be considered in the mitigation plans for endangered species (e.g. wild water buffalo and
405 banteng).

406   Both FMD and LSD with FD transmission had the least impact on populations, with acute,
407   short infections with lower overall mortality (74, 80). FMDV in particular, is highly contagious
408   among cattle with a very high β coefficient compared to the other diseases (27). Yet, although
409   Beck-Johnson, Gorsich (27) found little effect of FMD with either transmission modes within-
410   herds, our results showed that DD transmission led to greater population declines, as did
411   rescaling the parameter used for FD models. The reason for the latter observation is not clear
412   but might be because the dynamics with reintroduction allow more infection to persist and so
413   suppress the population (Figure 2). Note that with reported wild bovid herd sizes, acute
414   transmission is unlikely to allow FMDV to persist, but reintroductions from cattle reservoirs
415   (modelled through ϵ) are likely (73). Our result for FMD DD transmission also showed cyclic
416   patterns in outbreaks consistent with seasonal patterns of outbreaks observed in Thailand (81).

417   We found that brucellosis with FD transmission and its reported β rate might cause
418   extinction 16% of the time, whereas DD transmission (β = 5e-6) may suppress population growth,
419   but not enough to cause population declines and even with the published FD rate rescaled (β =
420   1e-5) and used in a DD model, this caused declines but not extinctions (Figure 3; (B)). Brucellosis
421   has caused population declines among African buffalo, especially when there is co-infection with
422   tuberculosis (82). However, brucellosis only caused limited population growth impacts in
423   American bison, even though the disease persisted in the population over time (61, 83). In
424   Dobson and Meagher's study (61), their FD brucellosis models showed bison populations would
425   increase in numbers, whereas our models predicted a decrease, perhaps because our model
426   species' population size and structure differed from their study. Notably, *Brucella* can infect
427   multiple species, and the transmission source may not be obvious when multiple species interact.
428   For example, brucellosis outbreaks in Yellowstone National Park, USA, were not from wild bison
429   as first thought, with elk the likely primary host (84). Understanding the potential transmission
430   among and from other wild Asian ungulates may be necessary to fully understand potential
431   brucellosis impacts.

432   We assumed a single, closed (no migration) population with constant natural birth and
433   death rates. Therefore, our models explore the intrinsic population dynamics without
434   considering the influence of other positive (e.g. conservation) or negative factors (e.g. habitat
435   destruction, competition). Furthermore, it is unclear what population changes occur during
436   migration (85), so a closed population model can only simulate within-herd dynamics and reflect
437   the population impacts in a small population, such as in small protected areas (20, 22).

438   Selecting the appropriate transmission mode for modelling is challenging (86). The
439   infectious disease parameter values themselves are mostly estimated from livestock outbreak
440   data, which can vary among the regions. Rarely is infection 'natural' without intervention
441   through disease control (27). Although the transmission type for some pathogens have been
442   recorded as FD or DD in previous studies, these were mainly conducted under farm husbandry

443 or experimental conditions in captive or closed systems. These conditions significantly allow
444 animal density to affect contact rates. However, our study focused on wildlife populations that
445 are distributed in areas in which the frequency of contact could have more influence. Moreover,
446 some infectious diseases can display aspects of FD and DD depending on the conditions, such as
447 within- or between herd transmission, herd size, density, contact with other reservoirs and
448 contact mode (indirect, direct). For example, the bTB transmission rate can be increased
449 correlated to herd size if the area is stable because the density of animals is increased (87). Also,
450 the transmission mode for anthrax spores from animal to animal is FD, but from the environment
451 to an animal is based on the density of the spores in the areas. We, therefore, took the strategy
452 of assuming the most extreme scenarios, fully FD and DD, and used both for modelling.

453 Our models also added the external force of infection (ϵ), which represents the re-
454 introduction of pathogens. ϵ is assumed to include transmission from other sources of infection
455 other than just infectious animals, such as transmission due to environmental factors (e.g. soil,
456 carcasses) or vectors (e.g. blood-sucking fly) to susceptible animals (10). This transmission can
457 theoretically cause population extinctions if agents have high case fatality rates. Here, we chose
458 a relatively high reintroduction rate ($\sim$ 2 per year into the initial population), which likely
459 represents a worst-case scenario. However, to improve this study, we encourage adding the
460 specific environmental factors for each disease and incorporating spatial analyses (88, 89).

461 Further studies might also consider adding the potential reservoir hosts and their
462 dynamics into the models by building two or more host models to examine the transmission
463 route among the potential hosts (90-94). Modelling coinfection is another important point as
464 there are interactions between infections such as FMDV and HS, which seen as a secondary
465 infection in FMD outbreaks (95), or between brucellosis and bTB (82). However, our analyses
466 provide an approach to understanding the *relative* likely impact of common endemic and
467 emerging diseases with different traits and is a tool for understanding gaps in disease surveillance
468 and control systems by using the prediction modelling before implementing actions. Future
469 analyses could also determine the impact of using an Erlang-distributed waiting time, rather than
470 an exponential distribution, on those parameters with large amounts of variation, particularly
471 the incubation period (96). Another further analysis is a sensitivity analysis that can be applied
472 to identify the degree of influence of the disease parameters on the model output, in this case,
473 population change. It also suggests which state of disease transmission should prompt action and
474 aids in selecting optimal control measures (97).

475 Strengthening disease surveillance and mitigation programs may be further achieved by
476 targeting virulent diseases through passive and active surveillance data, such as collecting the
477 frequency of infections, number and species of wild ungulates, behaviour and time spent
478 together between wild and domestic livestock (particularly in the high-risk areas) (98, 99). It may
479 be useful for disease mitigation to largely focus on domestic animal disease control and

480   preventing transmission to wildlife as an amenable approach (98). Moreover, conserving wildlife
481   habitat can reduce the probability of contact and the risk of disease transmission between
482   wildlife and domestic livestock (3, 100). Limiting the contact between wildlife and livestock could
483   reduce species extinction (101).

484   With applications in wildlife conservation, a reproducible modelling framework is
485   advantageous for targeting pathogens that threaten other wildlife populations with similar
486   assumptions. Although our infectious disease modelling focused on the traits of pathogens in
487   one species population, our method and framework may be applicable to other wildlife
488   populations by incorporating their population demographics and disease parameters. This
489   framework is also beneficial for endangered species, enabling the simulation of various scenarios
490   and the identification of potential disease threats, along with estimating the recovery period
491   after introducing the infection.

Conclusion

493   Our study has provided a prediction of the potential consequence of disease in wild
494   bovid populations considering six important bovine infectious diseases; anthrax, HS, bTB, LSD,
495   FMD and brucellosis. The baseline population model shows a natural population growth of ~
496   228%, suggesting maintaining healthy vulnerable populations could allow them to reestablish
497   and overcome current levels of extinction threats while diseases and other factors may regulate
498   population growth. The inclusion of different disease traits has consequences on the population
499   numbers depending on the transmission, incubation, fatality and infectious rates. Brucellosis and
500   bTB models show the greatest, long-term impact among all the models, whereas FMD and LSD
501   showed the least impact, suggesting common but more chronic or 'slow' infections with
502   relatively high mortality may pose the greatest threat to smaller, threatened bovid populations.

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