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# MODELING DISEASE TRANSMISSION IN A MIXED-SPECIES GRAZING ENVIRONMENT

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**Abstract.** In this article, we propose and analyzed a model describes the dynamics of a disease affects two different herbivores populations co-grazing together in the same environment under vaccination strategies and cross-immunity between the two species. Results show that the disease free equilibrium point (DFE) is locally asymptotically stable when the basic reproduction number, $\mathscr{R}_0$, is less than unity, and unstable when $\mathscr{R}_0$ is greater than unity, and our model undergoes a backward bifurcation, where $\mathscr{R}_0 < 1$ is not sufficient for the disease elimination, as $\mathscr{R}_0$ passes throw unity. Numerical results show that cross-immunity plays an important role in the eradication of the disease from both populations, however it plays also a negative role for both populations in the presence of vaccination strategies.

**Keywords:** mixed-species grazing; cross-immunity; vaccination; basic reproduction number; numerical simulation.

**2010 AMS Subject Classification:** 92B05, 34C23, 37C75.

## 1. Introduction

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Mixed-species grazing defined as more than one kind of livestock (i.e. sheep, goats, cattle, or horses) grazes same unit of land at the same time or at different times, and it might includes mixes of domestic and wild animals[1]. Mixed-species grazing is an old idea from era of integrated agricultural systems, and it is really is the norm for wild and natural ecosystems.

Mixed-species grazing has several advantages. Cattle prefer grass over other types of plants and are less selective when grazing than sheep or goats. Sheep and goats, on the other hand, are much more likely to eat weeds. Sheep prefer forbs (broad-leaved plants) to grass, and goats have a preference for browsing on brush and shrubs, and then broad-leaved weeds. Therefore, grazing cattle, sheep, and goats together on a diverse pasture should result in all types of plants being eaten, thus controlling weeds and brush, while yielding more pounds of gain per acre compared to single-species grazing [2].

The addition of goats to cattle pastures has been shown to benefit the cattle by reducing browse plants and broad-leaved weeds. This permits more grass growth. Goats will control blackberry brambles, multiflora rose, honeysuckle, and many other troublesome plants [3]. It is thought that you can add one goat per cow to a pasture without any reduction in cattle performance, and with time the weedy species will be controlled so that total carrying capacity is improved. This is a cheap way of renovating pastures, and you can sell the extra goats and kids for a profit, as well. The same principle holds for sheep. Although they are less likely to clean up woody plants, sheep are quite effective at controlling other weeds, with proper stocking pressure [4].

One of the major problems of mixed-species grazing is that there is possibilities of having a disease transmitting between these different species, which will make the control of such a disease very difficult because the different nature of these species, for example Rogdo *et. al.* [5] reported that there is a possibility of cross-infection of *Dichelobacter nodosus* between sheep and cattle in co-grazing pasture.

In this paper we will consider the dynamics of a disease infects two different species i.e. cattle and goats, sharing same environment, with vaccination and cross-immunity between these two herbivores. In Section 2 we will formulate our model, Section 3 contains the mathematical analysis of the model, Section 4 deals with numerical simulations and discussion, and Section 5 contains the conclusion.

## 2. Model Formulation

To formulate this model, we consider the dynamics of disease in two different populations, cattle population $N_c(t)$ and goats population $N_G(t)$. We divided the cattle population into three categories, susceptible individuals $S_c(t)$, infected individuals $I_c(t)$ and recovered individuals $R_c(t)$, hence

$$N_c(t) = S_c(t) + I_c(t) + R_c(t).$$

The goats population is divided into three categories, susceptible goats $S_G(t)$, infected reservoir $I_G(t)$ and recovered goats $R_G(t)$, such that

$$N_G(t) = S_G(t) + I_G(t) + R_G(t)$$

It is assumed that the birth rate of cattle is $b_c$ and all cattle born susceptible, and hence the recruitment of susceptible is $b_c N_c$. Susceptible cattle acquire infection with the disease following contacts with infected cattle an average rate $\beta_{cc} I_c$. Susceptible cattle contacted infected goats and acquire life-long immunity due to the cross-immunity between the two population at an average rate $e_c \beta_{cc} I_G$, where $e_c$ is the cross-immunity modification parameter. Susceptible cattle get vaccinated in an average rate $\nu_c$. Infected cattle die due to the disease at a rate $\alpha_c$, or recovered from the infection at an average rate $\gamma_c$. Natural death occurs in all cattle sub-populations at a per capita rate $d_c$.

It is assumed that the birth rate of goats is $b_G$ and all goats born susceptible, and hence the recruitment of susceptible is $b_G N_G$. Susceptible goats acquire infection with the disease following contacts with infected goats an average rate $\beta_{GG} I_G$. Susceptible goats contacted infected cattle and acquire life-long immunity due to the cross-immunity between the two population at an average rate $e_G \beta_{GG} I_C$, where $e_G$ is the cross-immunity modification parameter. Susceptible

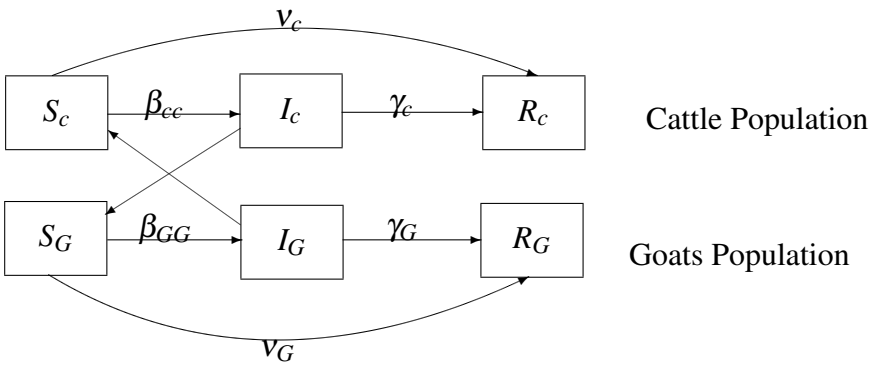


FIGURE 1. Compartmental model diagram

goats get vaccinated in an average rate $\nu_G$. Infected goats die due to the disease at a rate $\alpha_G$, or recovered from the infection at an average rate $\gamma_G$. Natural death occurs in all goats subpopulations at a per capita rate $d_G$. The dynamics of the disease in the two populations is as described in Figure 1.

Using above description and Figure 1 we get the following system of differential equations:

$$
\begin{aligned}
\frac{dS_c}{dt} &= b_c N_c - (d_c+\nu_c)S_c - \frac{\beta_{cc}(I_c+e_c I_G)S_c}{N_c+e_c N_G} \\
\frac{dI_c}{dt} &= \frac{\beta_{cc} I_c S_c}{N_c+e_c N_G} - (\alpha_c+\gamma_c+d_c)I_c \\
\frac{dR_c}{dt} &= \gamma_c I_c + \nu_c S_c + \frac{\beta_{cc} e_c I_G S_c}{N_c+e_c N_G} - d_c R_c \\
\frac{dS_G}{dt} &= b_G N_G - (d_G+\nu_G)S_G - \frac{\beta_{GG}(I_G+e_G I_c)S_G}{N_G+e_G N_c} \\
\frac{dI_G}{dt} &= \frac{\beta_{GG} I_G S_G}{N_G+e_G N_c} - (\alpha_G+\gamma_G+d_G)I_G \\
\frac{dR_G}{dt} &= \gamma_G I_G + \nu_G S_G + \frac{\beta_{GG} e_G I_c S_G}{N_G+e_G N_c} - d_G R_G
\end{aligned}
\tag{1}
$$

## Invariant region

All parameters of the model are assumed to be nonnegative, furthermore since model (1) monitors living populations, it is assumed that all the state variables are nonnegative at time $t = 0$, hence the biologically-feasible region:

$$\Omega = \left\{ (S_c, I_c, R_c, S_G, I_G, R_G) \in R_+^6 : S_c, I_c, R_c, S_G, I_G, R_G \geq 0, \right\}$$

is positively-invariant domain, and thus, the model is epidemiologically and mathematically

well posed, and it is sufficient to consider the dynamics of the flow generated by (1) in this positively-invariant domain $\Omega$.

## 3. Mathematical Analysis of the Model

To analyze model 1, we first find the equilibrium points of the model by equating the right hand side of the model with zero to get:

- $E_0$ the disease free equilibrium, which is given by:

$$\begin{aligned} S_c^* &= \frac{b_c}{d_c+\nu_c} N_c^* \\ R_c^* &= \frac{\nu_c b_c}{d_c(d_c+\nu_c)} N_c^* \\ S_G^* &= \frac{b_G}{d_G+\nu_G} N_G^* \\ R_G^* &= \frac{\nu_G b_G}{d_G(d_G+\nu_G)} N_G^* \\ I_c^* &= I_G^* = 0 \end{aligned}$$

- $E_1$ the endemic equilibrium, which is given by:

$$\begin{aligned} S_c^{**} &= \frac{1}{\beta_{cc}} (N_c^{**} + e_c N_G^{**})(\alpha_c + \gamma_c + d_c) \\ R_c^{**} &= \gamma_c I_c^{**} + \nu_c S_c^{**} + \frac{\beta_{cc} e_c I_G^{**} S_c^{**}}{N_c^{**} + e_c N_G^{**}} \\ S_G^{**} &= \frac{1}{\beta_{GG}} (N_G^{**} + e_G N_c^{**})(\alpha_G + \gamma_G + d_G) \\ R_G^{**} &= \gamma_G I_G^{**} + \nu_G S_G^{**} + \frac{\beta_{GG} e_G I_c^{**} S_G^{**}}{N_G^{**} + e_G N_c^{**}} \end{aligned}$$

with $I_c^{**}$ and $I_G^{**}$ are solutions to the equations:

$$
\begin{aligned}
b_c N_c^{**} - (d_c+\gamma_c)\left[\frac{1}{\beta_{cc}}(N_c^{**}+e_c N_G^{**})(\alpha_c+\gamma_c+d_c\right] - (I_c^{**}+e_c I_G^{**})(\alpha_c+\gamma_c+d_c) &= 0 \\
b_G N_G^{**} - (d_G+\gamma_G)\left[\frac{1}{\beta_{GG}}(N_G^{**}+e_G N_c^{**})(\alpha_G+\gamma_G+d_G\right] - (I_G^{**}+e_G I_c^{**})(\alpha_G+\gamma_G+d_G) &= 0
\end{aligned}
$$

### 3.1 Local stability of the disease-free equilibrium $E_0$

In order to study the local stability of the DFE we have to find the the basic reproduction number, $\mathscr{R}_0$. It is defined as the number of secondary infections that occur when an infected individual is introduced into a completely susceptible population [6, 7]. To calculate the basic reproduction number we use the next generation approach [6, 8]. The matrices $F$ and $V$ which are associated with the next generation operator are

$$
\mathbf{F} = \begin{pmatrix} \frac{\beta_{cc} b_c N_c^*}{(d_c+\nu_c)(N_c^*+e_c N_G^*)} & 0 \\ 0 & \frac{\beta_{GG} b_G N_G^*}{(d_G+\nu_G)(N_G^*+e_G N_c^*)} \end{pmatrix}
$$

and

$$
\mathbf{V} = \begin{pmatrix} \alpha_c+\gamma_c+d_c & 0 \\ 0 & \alpha_G+\gamma_G+d_G \end{pmatrix}
$$

Then the basic reproduction number is the spectral radius of the matrix $\mathbf{F}\mathbf{V}^{-1}$ and given by $\mathscr{R}_0 = \max\{\mathscr{R}_c\ ,\ \mathscr{R}_G\}$, where $\mathscr{R}_c, \mathscr{R}_G$ are the reproduction numbers of the cattle and goat populations, respectively, and given by:

$$
\begin{aligned}
\mathscr{R}_c &= \frac{\beta_{cc} b_c N_c^*}{(d_c+\nu_c)(N_c^*+e_c N_G^*)(\alpha_c+\gamma_c+d_c)} \\
\mathscr{R}_G &= \frac{\beta_{GG} b_c N_G^*}{(d_G+\nu_G)(N_G^*+e_G N_c^*)(\alpha_G+\gamma_G+d_G)}
\end{aligned}
$$

Using theorem 2 of van den Driessche and Watmough [8], the following result is established:

**Lemma 3.1.** *he disease-free equilibrium is locally asymptotically if $\mathscr{R}_0 < 1$, and unstable if $\mathscr{R}_0 > 1$.*

### 3.2 Bifurcation analysis of the model

To study the possibility of backward bifurcation we use the center manifold theorem [9, 10, 11], particularly we use the theorem in Castillo-Chavez and Song [10] (see appendix). In order to apply this theorem we first made the following simplification and change of variables using the notation of [12]. Let $S_c = x_1, I_c = x_2, R_c = x_3, S_G = x_4, I_G = x_5$, and $R_G = x_6$. Using vector representation, the system (1) can be written as $\frac{dX}{dt} = F(X)$, where $X = (x_1, x_2, ..., x_6)^T$, and $F = (f_1, f_2, ..., f_6)^T$ as follows

$$
\begin{aligned}
f_1 = \frac{dx_1}{dt} &= b_c N_c - (d_c + \nu_c)x_1 - \frac{\beta_{cc}(x_2 + e_c x_5)x_1}{N_c + e_c N_G} \\
f_2 = \frac{dx_2}{dt} &= \frac{\beta_{cc} x_2 x_1}{N_c + e_c N_G} - (\alpha_c + \gamma_c + d_c)x_2 \\
f_3 = \frac{dx_3}{dt} &= \gamma_c x_2 + \nu_c x_1 + \frac{\beta_{cc} e_c x_5 x_1}{N_c + e_c N_G} - d_c x_3 \\
f_4 = \frac{dx_4}{dt} &= b_G N_G - (d_G + \nu_G)x_4 - \frac{\beta_{GG}(x_5 + e_G x_2)x_4}{N_G + e_G N_c} \\
f_5 = \frac{dx_5}{dt} &= \frac{\beta_{GG} x_5 x_4}{N_G + e_G N_c} - (\alpha_G + \gamma_G + d_G)x_5 \\
f_6 = \frac{dx_6}{dt} &= \gamma_G x_5 + \nu_G x_4 + \frac{\beta_{GG} e_G x_2 x_4}{N_G + e_G N_c} - d_G x_6
\end{aligned} \tag{2}
$$

Suppose that $a_l b_l = \phi$ is chosen as a bifurcation parameter, and consider the case $\mathscr{R}_0 = 1$, i.e. $\mathscr{R}_c = 1$, and $\mathscr{R}_G = 1$, then we have:

$$
\begin{aligned}
\frac{\beta_{cc} b_c N_c^*}{(d_c + \nu_c)(N_c^* + e_c N_G^*)(\alpha_c + \gamma_c + d_c)} &= 1 \\
\frac{\beta_{GG} b_c N_G^*}{(d_G + \nu_G)(N_G^* + e_G N_c^*)(\alpha_G + \gamma_G + d_G)} &= 1
\end{aligned}
$$

which implies:

$$
\begin{aligned}
\frac{\beta_{cc} b_c N_c^*}{(d_c + \nu_c)(N_c^* + e_c N_G^*)} &= (\alpha_c + \gamma_c + d_c) \\
\frac{\beta_{GG} b_c N_G^*}{(d_G + \nu_G)(N_G^* + e_G N_c^*)} &= (\alpha_G + \gamma_G + d_G)
\end{aligned}
$$

Now the Jacobian of the system (2) at the disease-free equilibrium when $\mathscr{R}_0 = 1$ is given by:

$$\mathscr{J}(E_0)_{\mathscr{R}_0} = \begin{pmatrix} -(d_c+\nu_c) & -(\alpha_c+\gamma_c+d_c) & 0 & 0 & -e_c(\alpha_c+\gamma_c+d_c) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \nu_c & \gamma_c & -d_c & 0 & e_c(\alpha_c+\gamma_c+d_c) & 0 \\ 0 & -e_G(\alpha_G+\gamma_G+d_G) & 0 & -(d_G+\nu_G) & -(\alpha_G+\gamma_G+d_G) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & e_G(\alpha_G+\gamma_G+d_G) & 0 & \nu_G & \gamma_G & -d_G \end{pmatrix}$$

then it can be shown that the jacobian of the system (2) at $\beta_{cc} = \phi$ (denoted by $\mathscr{J}(E_0)_{\mathscr{R}_0} =$) has a right eigenvector given by $W = (w_1, w_2, w_3, w_4, w_5, w_6)^T$, where

$$\begin{aligned} w_1 &= w_1\,, \qquad w_2 = w_2, \qquad w_4 = w_4, \qquad w_5 = w_5 \\ w_3 &= -\frac{d_c w_1 + (\alpha_c + d_c) w_2}{d_c} \\ w_6 &= -\frac{d_G w_4 + (\alpha_G + d_G) w_5}{d_G} \end{aligned}$$

and a left eigenvector given by $V = (v_1, v_2, v_3, v_4, v_5, v_6)$, where

$$\begin{aligned} v_1 &= v_3 = v_4 = v_6 = 0 \\ v_2 &= v_2 \\ v_5 &= v_5 \end{aligned}$$

then it can be shown that:

$$\begin{aligned} a &= 2v_2 w_1 w_2 \frac{\beta_{cc}}{(N_c^* + e_c N_G^*)} + 2v_5 w_4 w_5 \frac{\beta_{GG}}{(N_G^* + e_G N_c^*)} > 0 \\ b &= v_2 w_5 (\alpha_c + \gamma_c + d_c) > 0 \end{aligned}$$

and hence the following result is established:

**Corollary 3.2.** *The system (2) undergoes a backward bifurcation which occurs at $\mathscr{R}_0 = 1$. (i.e. $\mathscr{R}_0 < 1$ is not sufficient for the eradication of the diseases.)*

## 4. Numerical Simulation and Discussion

In this section we us numerical simulations to support the analytical results we already establish, and to get an insight knoweldge about the dynamics of the diseases. Body size is very important in the characterization of the demography of animals, and therefor we will going to scale all of our parameters using the allometric scaling (see [13, 14] for more details). The values of the parameters are given in **Table 1**, where $m$ represents the infectiousness of the disease [13], and taken to be $m = 250$. The cattle weight, $w_c$, is taken to be $w_c = 230$kg, and the goat weight, $w_g$, is taken to be $w_G = 30$kg, with total cattle population of $N_c = 200$, and goats total population of $N_G = 1000$.

| parameter | parameter description | value | Source |
|---|---|---|---|
| $b_c$ | Per capita birth rate of cattle | $0.6 * w_c^{-0.27} + 0.4 * w_c^{-0.26}$ | [13] |
| $b_G$ | Per capita birth rate of goats | $0.6 * w_G^{-0.27} + 0.4 * w_G^{-0.26}$ | [13] |
| $d_c$ | Per capita death rate of cattle | $0.4 * w_c^{-0.26}$ | [13] |
| $d_G$ | Per capita death rate of goats | $0.4 * w_G^{-0.26}$ | [13] |
| $\alpha_c$ | Per capita disease induced death rate of cattle | 10 | Assumed |
| $\alpha_G$ | Per capita disease induced death rate of goats | 12 | Assumed |
| $\beta_{cc}$ | Transmission rate of the disease between cattle | $2 * 0.4 * m * w_c^{-0.26}$ | [13] |
| $\beta_{GG}$ | Transmission rate of the disease between goats | $2 * 0.4 * m * w_G^{-0.26}$ | [13] |
| $\nu_c$ | Cattle vaccination rate | Variable | Variable |
| $\nu_G$ | Goats vaccination rate | Variable | Variable |
| $e_c$,$e_G$ | Cross-immunity parameters | Variable | Variable |
| $\gamma_c$ | Cattle recovery rate | Variable | Variable |
| $\gamma_G$ | Goats recovery rate | Variable | Variable |

**Table 1: parameter values (and their sources)**

Simulation result show that when there is no cross-immunity between the two populations, the final epidemic size of infected goats increases as the goat vaccination coverage increases, until the vaccination coverage exceeds 50% of the population, then the final epidemic size starts to decreases until it reaches zero, (as seen from Figure 2), however the final epidemic size of

infected cattle decreases as the cattle vaccination coverage increases, until it reaches zero, as seen from Figure 3.

When we fix the vaccination coverage of goats (i.e. $\nu_G = 0.4$), and there is cross-immunity between the two populations (i.e. $e_c = 0.3$ and $e_G = 0.5$) we notice that as the cattle vaccination coverage increases the final epidemic size of both population increases, until the vaccination coverage of cattle reaches above 50% of the population then the final epidemic size of cattle starts to decrease, however the final epidemic size of goats continues to increase, on the other hand, when the cattle vaccination coverage is fixed (i.e. $\nu_c = 0.4$) and there is cross-immunity between the two populations (i.e. $e_c = 0.3$ and $e_G = 0.5$), we notice that the increase of goats vaccination coverage has (almost) no effect on the final epidemic size of cattle, however the final epidemic size of goats keep increasing as their vaccination coverage keeps increasing, as shown in Figures 4, and 5.

When there is no vaccination in both populations (i.e. $\nu_c = \nu_G = 0$), then the cross cross-immunity protects both population, and if we fix one population cross-immunity parameter, for example let $e_G$ be fixed into two extreme values (i.e. $e_G = 0.0$ and $e_G = 1.0$), then the cattle final epidemic size decrease as their cross-immunity parameter increases, however the final epidemic size reaches zero faster when $e_G$ has its minimum value than when $e_G$ has its maximum value, as shown in Figure 6.

## 5. Conclusion

In this paper a model for the dynamics of a disease spreads in two herbivores sharing the same environment, was proposed and analyzed.

Results show that the disease free equilibrium is locally asymptotically stable when $\mathscr{R}_0 < 1$, and there is a possibility of backward bifurcation where $\mathscr{R}_0 < 1$ is not sufficient for the eradication of the disease.

Numerical results show that in order to eradicate the disease from the goats population the vaccination coverage of goats population must reaches above 50% of the population, however a small vaccination coverage of cattle population is sufficient for the eradication of the disease from the cattle population. Also numerical results show that when there is cross-immunity the

vaccine have a negative impact on the goats population, and also suggest that that the cross-immunity of each population has some negative impact on the other population.

**Conflict of Interests**

The author declare that there is no conflict of interests.

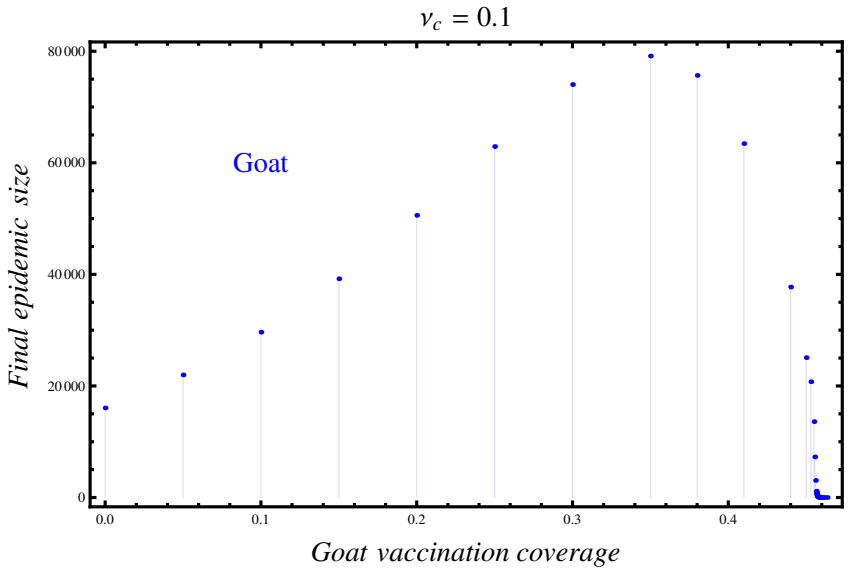


FIGURE 2. Simulation results for goats vaccination coverage without cross-immunity

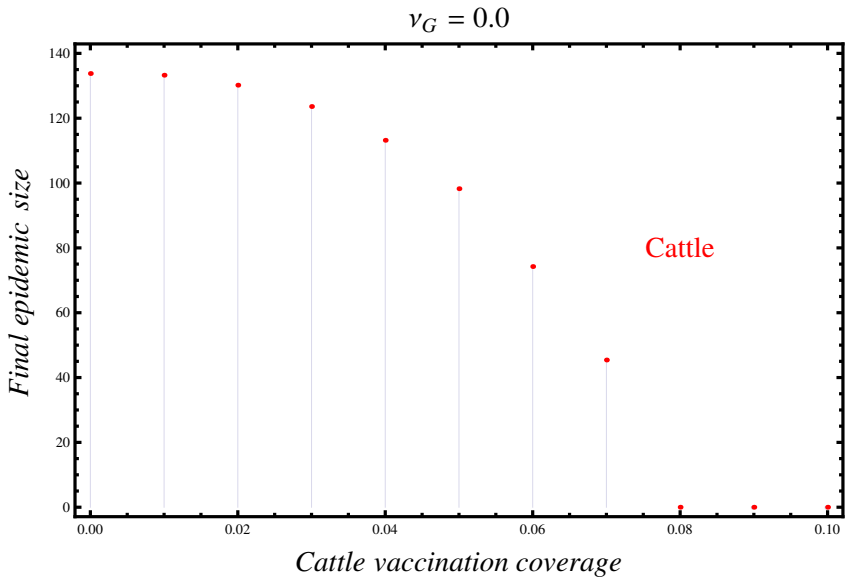


FIGURE 3. Simulation results for cattle vaccination coverage without cross-immunity

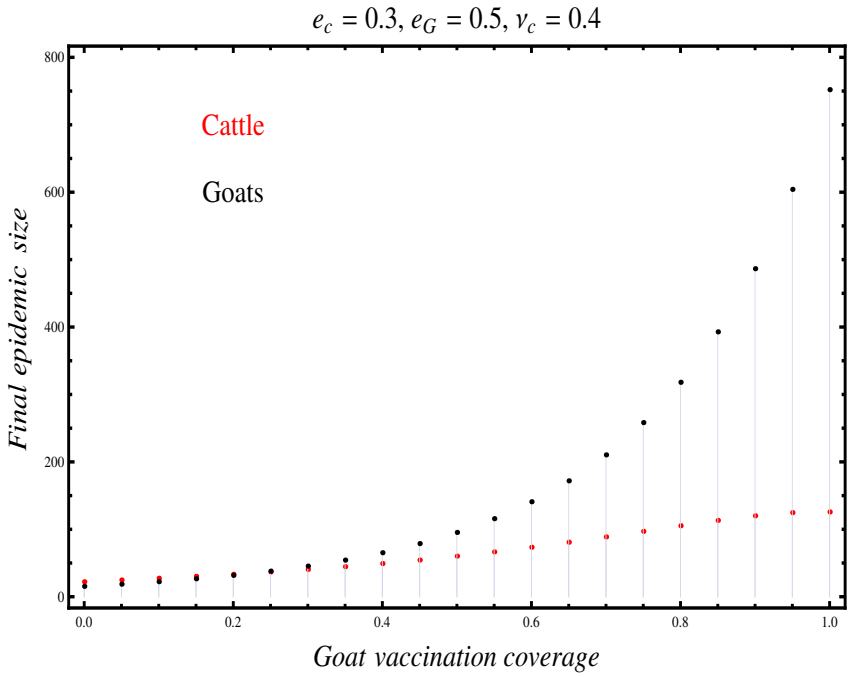


FIGURE 4. Simulation results for goats vaccination coverage with cross-immunity

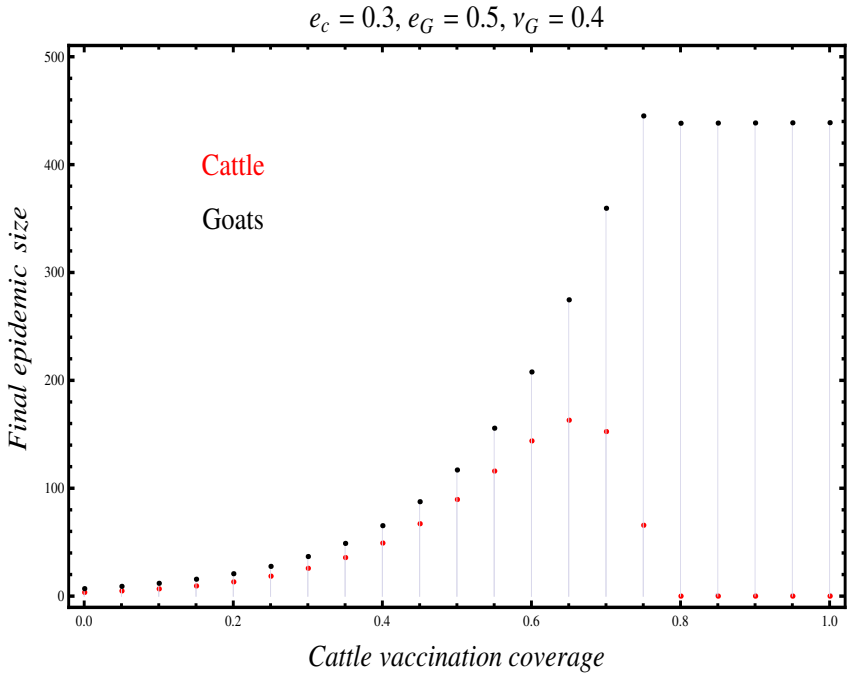


FIGURE 5. Simulation results for cattle vaccination coverage with cross-immunity

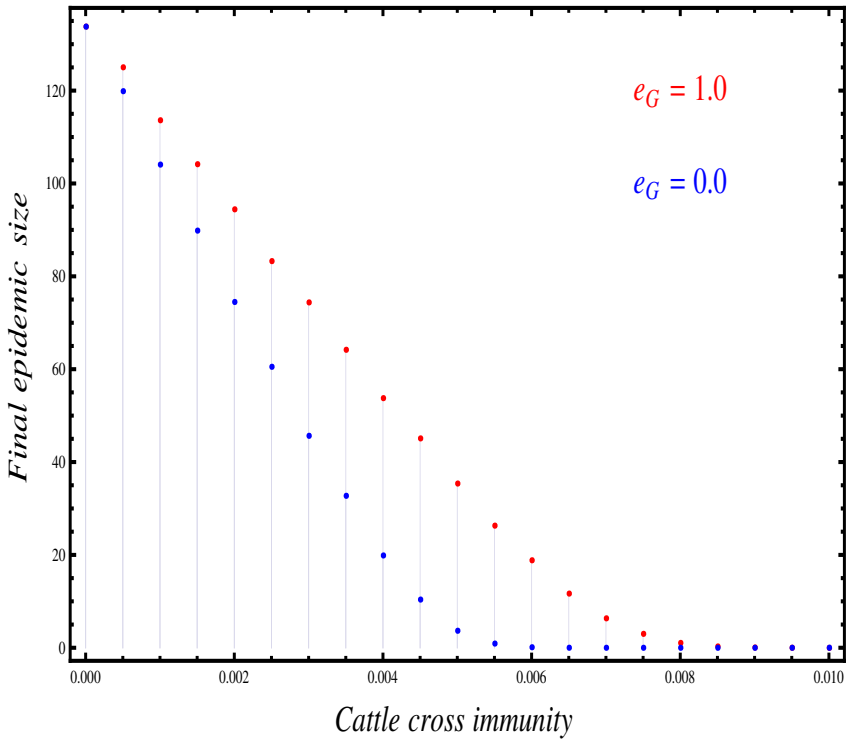


FIGURE 6. Simulation results for cattle cross-immunity without vaccination

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# Appendix

**Theorem** Castillo-Chavez and Song [10]

*Consider a general system of ODEs with a parameter $\phi$:*

$$\frac{dx}{dt} = f(x,\phi), \qquad f:\mathbb{R}\to\mathbb{R}^n \quad \text{and } f\in\mathbb{C}^2(\mathbb{R}^2\times\mathbb{R}) \tag{3}$$

*where 0 is an equilibrium point for the system(3) for all values of the parameter $\phi$, that is $f(0,\phi)\equiv 0$ for all $\phi$, and*

**A1::** *$A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right)$ is the linearization matrix of System (3) around the equilibrium point 0 with $\phi$ evaluated at 0. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;*

**A2::** *Matrix A has a nonnegative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.*

*Let $f_k$ be the kth component of f and*

$$\begin{aligned} a &= \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) \\ b &= \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0) \end{aligned}$$

*then the local dynamics of the system(3) around the equilibrium point 0 is totally determined by the signs of a and b. Particularly, if $a>0$ and $b>0$, then a backward bifurcation occurs at $\phi = 0$.*