EISSN: 2707-0425

East African Journal of Science, Technology and Innovation, Vol. 6 (Special issue 1): December 2024

Network-based modeling of hoof and mouth disease transmission in animals

[1]PHILIPO H., [1]HUGO A*

[1]The Department of Digital Technology and Information Sciences, Dar es Salaam Tumaini University (DarTU), Tanzania

[*]**Corresponding author**: hadijamsuya94@gmail.com

Abstract

The connectivity of ranches facilitates the spread of hoof and mouth disease among livestock, even over long distances. This study aimed to investigate the spread of hoof and mouth disease within animal ranch networks and individual ranches using a network-based modelling approach. Two models were developed: the multi-ranch model and the in-ranch model. The multi-ranch model examined how the topology and connectivity of the ranch network influenced the spread of hoof and mouth disease, while the in-ranch compartmental model captured the disease dynamics within individual ranches. The results indicate that the disease can be contained, and the network can remain disease-free as long as the transmission rate is low and the network is not overly dense. In the in-ranch compartmental model, the basic reproduction number was used to gain insights into the vaccination coverage required to maintain a disease-free state within individual ranches, as well as the coverage needed across a larger ranch network. Additionally, the findings highlight the importance of understanding both network-level transmission dynamics and within-ranch disease progression to effectively model and manage hoof and mouth disease outbreaks. Furthermore, disease control strategies, such as vaccination, to minimize the spread of the disease, which can lead to declines in the production of milk, meat, manure, and raw materials, ultimately reducing both national and individual income due to livestock loss also investigated and recommended that maintaining a transmission rate below 0.044 and offering sufficient immunization coverage are essential for a multi-ranch to stay resilient against HMD illnesses. For individual ranches and ranch networks, applying a vaccination level below 0.195 is not essential since disease will not vanish, but a level over 0.327 keep both a single ranch and larger ranch community in disease-free state.

Keywords: *Hoof and Mouth Disease model; Network; topology; Vaccine*

Received: 18/06/24
Accepted: 05/12/24
Published: 20/12/24

Cite as, *Philipo and Hugo, (2024)*. Network-based modeling of hoof and mouth disease transmission in animals. *East African Journal of Science, Technology and Innovation 6(Special issue 1).*

Introduction

Hoof and Mouth Disease (HMD) is a highly contagious illness that commonly affects hoofed animals, including cattle, horses, goats, sheep, pigs, and camels (Belsham *et al.*, 2020). This disease is caused by the foot-and-mouth disease virus (FMDV), a member of the *Picornaviridae* family, which primarily targets animals with cloven hooves, leading to severe illness. The

disease imposes a substantial economic burden (such as loss in agricultural production, cost of disease control restrictions, public health and social costs etc), especially in regions with limited access to veterinary services and healthcare infrastructure (Knowles *et al.,* 2021). Recent studies emphasize the global impact of HMD outbreaks on agricultural production, trade restrictions, and food security, further underscoring the need for effective control measures and vaccination strategies (Paton *et al.,* 2023; OIE, 2022).

Its repercussions extend beyond animal health, affecting both trade and food security. Transmission primarily occurs through direct contact with infected animals, ingestion of contaminated feed or water, or exposure to contaminated equipment, vehicles, or personnel. Furthermore, the disease can spread within individual ranches and across ranch networks through various pathways (Belsham *et al.,* 2020). A significant factor contributing to disease transmission within ranch networks is the type of contact, which can be either direct or indirect. Contact occurs through the movement of infected animals, shared veterinarians and workers, common pastures and water sources, and contaminated equipment, such as vehicles (Brown *et al.,* 2022). Additionally, the virus can rapidly spread over long distances through the movement of infected animals or contaminated animal products (Green *et al.,* 2006).

At the local level, the rate of disease transmission is confined to a small area, where the rapid spread of a local contact epidemic can be observed due to the high likelihood of interactions within the same population. Conversely, at the global level, outbreaks and the spread of infectious diseases occur over a larger area. In most cases, global-level contacts are influenced by international travel and trade (Baker *et al.,* 2022). Symptoms in infected animals include fever, blisters in the mouth and on the feet, a drop in milk production, loss of appetite and weight, quivering lips, and frothing at the mouth (Belsham *et al.,* 2020).

Network analysis tools play a crucial role due to their wide-ranging application across various disciplines, including social science, epidemiology, and computer science (Mata, 2020). The study of networks has a long history, dating back to the examination of the Koenigsberg bridge problem in 1736 by the father of graph theory, Leonhard Euler (Newman, 2018). By incorporating features into simple networks, complex networks can be assessed based on their connectivity properties. Typically, complex networks consist of numerous interacting parts among nodes, with each node possessing its internal topology and functions (Safaei *et al.,* 2020).

In networks, nodes or links may possess a variety of properties, whether numerical or otherwise (Mata, 2020). There are three main types of complex networks. The first type is small-world networks, which are characterized by short average path lengths between nodes while still exhibiting a high degree of clustering. They are commonly found in social networks, neural networks, and some technological networks. The second type is random networks, which have a uniform or Poisson distribution of node degrees and lack the clustering and degree correlation observed in small-world and scale-free networks. They are used as a null model in network science to compare against more complex networks. The third type is disease-free networks which are characterized by a power law distribution of node degrees, meaning that a few nodes have a very high degree while most nodes have a low degree. They are prevalent in many real-world networks, such as the World Wide Web, social networks, and biological networks (Mata, 2020).

Deterministic models often assume homogeneous mixing within populations, making them inadequate for describing the dynamics of infection in large-scale social networks with diverse contact patterns (Cheng *et al.,* 2023). In recent years, significant progress has been made in analyzing infectious disease models through the application of complex network theory (Bai *et al.,* 2021). The study of epidemic spread, such as hoof-and-mouth disease, within networks, is an important area of research as it helps to better understand the actual dynamics of disease transmission within networked structures. Furthermore, it can assist in formulating strategies and policies for

controlling or eradicating epidemic infections (Yi *et al.,* 2022).

Moreover, the epidemic spreading between animals across ranch networks can be influenced by the ranch network's topology and internal strength, or the flexibility mechanism of a ranch (Cardenas, 2020). Similarly, while each ranch in a network ranch can be vaccinated at a high level and recover from any attack, the strength of the network can delay and cause widespread infection of hoof and mouth disease among animals. In many contexts, including epidemiology, an important question arises regarding where the disease can spread in a contact network, and whether individual vaccination can enhance the network's capability to resist external infections (the network's ability to maintain stability regardless of intentional attacks) (Wang *et al.,* 2020).

This work aims to assess how ranches are interconnected (topology) and how this connectivity weakens their resistance to external infections. This is because nowadays, ranches are more densely connected, even those that are distant from each other. As a result, closely linked ranches that share common resources such as pastures, water, and farming equipment are more susceptible to disease than isolated ranches. Additionally, the study aims to investigate the effect of animal vaccination within a single ranch and across the entire ranch network. Different vaccination levels were implemented as a control measure to study under what conditions infectious animals continue to be infected with Hoof and Mouth disease despite regular vaccination in a single ranch.

Consequently, mathematical models have been employed to assess the impact of hoof-and-mouth disease (HMD) within network frameworks, including the multi-ranch model, which explores the spread of infection between ranches, and the in-ranch model, which analyzes disease dynamics within a single ranch.

$$k_i = \sum_{j \neq i}^{N} A_{ij} .$$

Material and Methods

The ranch network is overly complex, making it difficult to understand the dynamics of disease transmission across the entire network at once, especially considering the intricate internal mechanisms and various infection stages of animals (compartments) within each ranch. The best approach would be to initially study the spread of infections in the ranch network using a multi-ranch model. In this study, the focus is on understanding how Hoof and Mouth disease spreads from one ranch to another as well as in a single ranch using an in-ranch model.

Multi-ranch Model Formulation and Analysis

This section focuses on the development of the model for disease spread from one ranch to another. It aims to investigate the connectivity of the ranches (network topology) and how it influences the transmission and spread of Hoof and Mouth disease. Therefore, the model is described as follows: the first step assumes that we have *N* ranches, while every ranch has several infected animals.$\boldsymbol{y_i}$, where $\boldsymbol{i} = \mathbf{1}, \ldots,$ N, further, $\boldsymbol{y_i}$ takes values between 0 and 1, such that 0 means there is no infection among animals at the ranch $\boldsymbol{i}$ and $\mathbf{1}$ means fully infected state, this is when every animal is suffering from Hoof and Mouth disease.

Assuming each ranch has a total of 20 animals and each of these ranches (y_1, y_2, y_3) has infected animals (20, 0, 10) respectively, Now, $y_1 = \frac{20}{20} = 1$ which means there is full infection in ranch1, $y_2 = \frac{0}{20} = 0$ which implies there is no infection at all in ranch 2,

$y_3 = \frac{10}{20} = 0.5$ means half of the animals are affected in the third ranch.

Now, an adjacent matrix is denoted by the letter A such that: $(i, j)th$ entry is 1 if there is any means (contact) by which disease can spread from ranch j to ranch i and 0 otherwise. Then, the node degree of i, or in other words number of contacts or connected neighbours of the ranch i can be defined as follows,

(1)

Thus, the transmission dynamics of the numbers of affected animals can be written as,

$$\dot{y} = f_i(y_i) + \sum_{j\neq i}^{N} A_{ij}\ d(y_i, y_j), i = 1, \dots, N \quad (2)$$

where $f_i(y_i)$ represent internal disease dynamics within ranch i over some time. Also

$$k_i = \sum_{j\neq i}^{N} A_{ij,} \quad (3)$$

represents how ranch i is connected to other ranches (topology of ranch network). In addition $d(y_i, y_j)$ stand for the transmission rate function from the ranch j to the ranch i. Now, the model assumes that the ranch-to-ranch transmission of the infection is proportional to both the source ranch (ranch j) and the targeted ranch (ranch i). Therefore, the transmission rate function can be described as,

$$(y_i, y_j) = \beta y_j(1 - y_i) \quad (4)$$

where β is the transmission rate, which means the rate at which the infected animals in ranch j will infect the susceptible animals in ranch i and $(1 - y_i)$ represent the total number of animals susceptible to Hoof and Mouth disease in ranch i.

However, if $f_i'(0) < 0$ means the ranch i is not connected to other ranches (isolated). There the ranch can recover from small infections hence the ranch i is said to resist external Hoof and Mouth disease infections with resiliency $l_i = -f_i'(0)$. Otherwise, the ranches are connected and let's consider the following theorem.

Theorem 1

Consider the ranch network defined by equation (2) that consists of a resilient ranch. Let's suppose that $k_x = max_i \sum_{j\neq i}^{N} A_{ij}$ be the maximum number of connections (degree) of the ranch, and let $l_n = min_i(-f_i'(0))$ be the minimum resiliency. Then the disease-free state for all ranches $y_1, \dots, y_n = (0, \dots, 0)$ is asymptotically stable for $0 \leq \beta < l_n/k_x$.

Proof

From equation (2)

$$\boldsymbol{y_i' = f_i(y_i) + \sum_{j\neq i}^{N} A_{ij}\ d(y_i, y_j), i = 1, \dots, N}$$

Will substitute equation (4) in equation (2) and simplify. We obtain,

$$\boldsymbol{y_i' = f_i(y_i) + \sum_{j\neq i}^{N} A_{ij}\ \beta y_j(1 - y_i), for\ i = 1, \dots, N}$$

Now for this case, since $i = 1 \cdots N$, then for every value of i there is its corresponding equation. Such that,

$$y_i' = (y_1', y_2', \dots y_N')$$

Then we will have a sequence of equations.

$$y_1' = f_1(y_1) + A_{12}\beta y_3(1 - y_1) = F_1(y_1, y_2, y_3) \quad (5)$$

$$y_2' = f_2(y_2) + A_{21}\beta y_1(1 - y_2) = F_2(y_1, y_2, y_3) \quad (6)$$

$$y_3' = f_3(y_3) + A_{31}\beta y_1(1 - y_3) = F_3(y_1, y_2, y_3) \quad (7)$$

The Jacobian of the system will be

$$J = \frac{\partial(\mathrm{F}_1, \mathrm{F}_2\mathrm{F}_3)}{\partial(\mathrm{y}_1, \mathrm{y}_2, \mathrm{y}_3)} = \begin{bmatrix} \frac{\partial F_1}{\partial y_1} & \frac{\partial F_1}{\partial y_2} & \frac{\partial F_1}{\partial y_3} \\ \frac{\partial F_2}{\partial y_1} & \frac{\partial F_2}{\partial y_2} & \frac{\partial F_2}{\partial y_3} \\ \frac{\partial F_3}{\partial y_1} & \frac{\partial F_3}{\partial y_2} & \frac{\partial F_3}{\partial y_3} \end{bmatrix}$$

$$J = \begin{bmatrix} \mathrm{f'}_1(y_1) & A_{12}\beta(1-\mathrm{y}_1) & A_{13}\beta(1-y_1) \\ A_{21}\beta(-y_2) & \mathrm{f'}_2(y_1)\beta - A_{21}\beta(y_1) & 0 \\ A_{31}\beta(1-y_3) & 0 & \mathrm{f'}_3(y_3) - A_{31}\beta(y_1) \end{bmatrix}.$$

We therefore have,

$$J(0,0,0) = \begin{bmatrix} \mathrm{f'}_1(0) & A_{12}\beta & A_{13}\beta \\ A_{21\beta} & \mathrm{f'}_2(0) & 0 \\ A_{31}\beta & 0 & \mathrm{f'}_3(0) \end{bmatrix}, \tag{8}$$

Finally, we have,

$$J_{ij} = f_i'(0)\delta_{ij} + \beta A_{ij} \tag{9}$$

where we can note that

$$\delta_{ij} = \begin{cases} 1 & i = j \\ 0 & i \neq j \end{cases} \tag{10}$$

In fact if $i = j$ then $J_{ij} = \mathrm{f'}_i(0)\delta_{ij} + \beta A_{ij} = (f_i'(0) \times 1) + (\beta \times 0) = f_i'(0)$.
And if $i \neq j$ we have, $J_{ij} = f_i'(0)\delta_{ij} + \beta A_{ij} = (\mathrm{f'}_i(0) \times 0) + (\beta A_{ij} \times 1) = \beta A_{ij}$
Moreover, $\mathrm{f'}_i(0) < 0$ and $J_{ij} = \mathrm{f'}_i(0)$ then $|J_{ij}| = f'_{i}(0) < l_n$. In addition, $\sum_{j\neq i}^{N} |J_{ij}| = \sum_{j\neq i}^{N} |\beta A_{ij}| = \beta \sum_{j\neq i}^{N} |A_{ij}| = \beta \sum_{j\neq i}^{N} A_{ij} = \beta k_i < \beta k_x$ where $0 \leq \beta \leq \frac{l_n}{k_x}$
Note that from Reddy (2021), a matrix is said to be strictly diagonal dominated if,
$|a_{ij}| > \sum_{j=1, j\neq i}^{n} |a_{ij}|, i \ldots, n$
for instance a matrix

$$A = \begin{bmatrix} 7 & 2 & 0 \\ 3 & 5 & -1 \\ 0 & 5 & -6 \end{bmatrix}$$

is strictly diagonally dominant because

$$|7| > |2| + |0|$$
$$|5| > |3| + |1-1|$$
$$|1-6| > |0| + |5|$$

Therefore the Jacobian matrix $J_{|0,0,0|}$ is strictly a diagonal dominant matrix for $0 < \beta < l_n/k_x$ Since,

$$|f_1'(0)| > |A_{12}\beta| + |A_{13\beta}|$$
$$|f_2'(0)| > |A_{21}\beta|$$
$$|f_3'(0)| > |\mathrm{A}_{31}\beta|$$

Therefore, since the Jacobian matrix $J_{|0,0,0|}$ is a strictly diagonal dominant matrix for $0 < \beta < l_n/k_x$, then the model is less sensitive to small perturbations or infections. This robustness is desirable in epidemic modelling, where uncertainties and fluctuations are common.

1.1 Numerical Simulations and Results for Multi-ranch Model

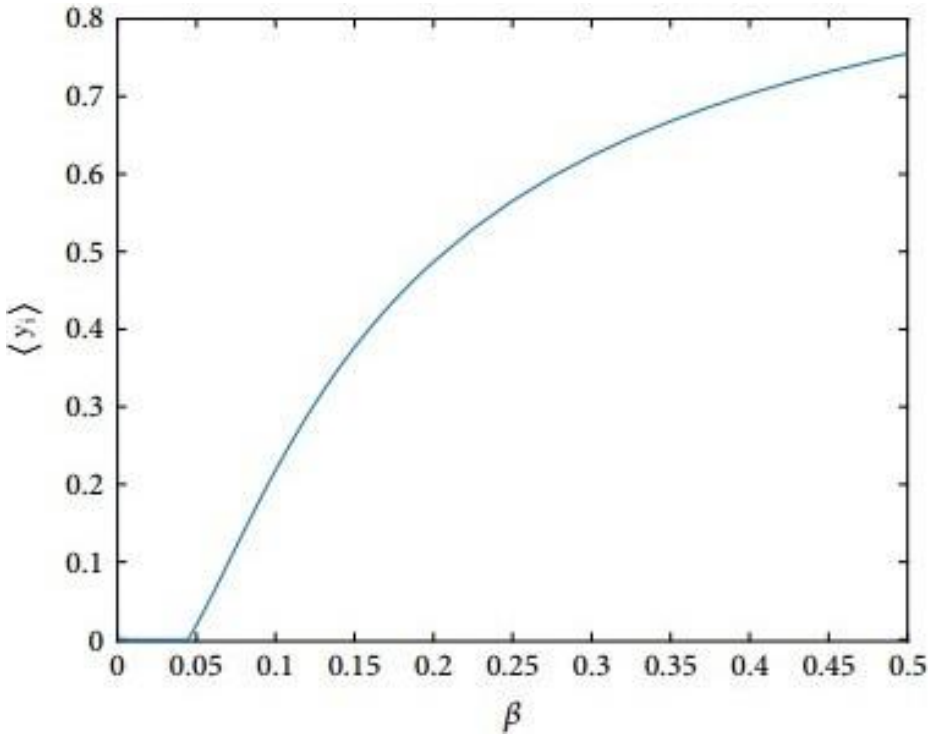
This section presents the numerical simulations of the model, utilizing equations (2) and (4) on a network of thousands (1000) of ranches. The aim is to visualize the conditions under which the ranch network will maintain a disease-free state for Hoof and Mouth disease. The simulation output is depicted in

Figure 1

Mean Prevalence of Disease in Ranch Network against Transmission Rate
Figure *1*.

Figure 1

Mean Prevalence of Disease in Ranch Network against Transmission Rate



2 Observations from indicate that the ranch network exhibits resistance to infections as long as the network is not overly dense and the transmission rate remains low, ensuring that all ranches remain disease-free when β is less than 0.044. This suggests that the threshold for epidemic spreading is effectively nonexistent in a network with an infinite limit. In other words, managing an epidemic in a large network may prove challenging even with a low infection rate (β).

In the analysis, it has been discovered that the ranch network is less susceptible to minor disturbances or infections, as evidenced by the strictly diagonal dominant nature of the Jacobian matrix. $J_{|0,0,0|}$ for $0 < \beta < l_n/k_x$. The simulation results further demonstrate that the ranch network is resistant to infections, provided that the network is not overly dense and the transmission rate remains low, ensuring that all ranches remain disease-free when β is less than approximately 0.044.

Indeed, the current multi-ranch model is too simplistic to handle the multitude of animals in various epidemic states within a ranch. Consequently, the study aims to introduce an in-ranch model (compartment model) to address the development of disease epidemics within individual ranches.

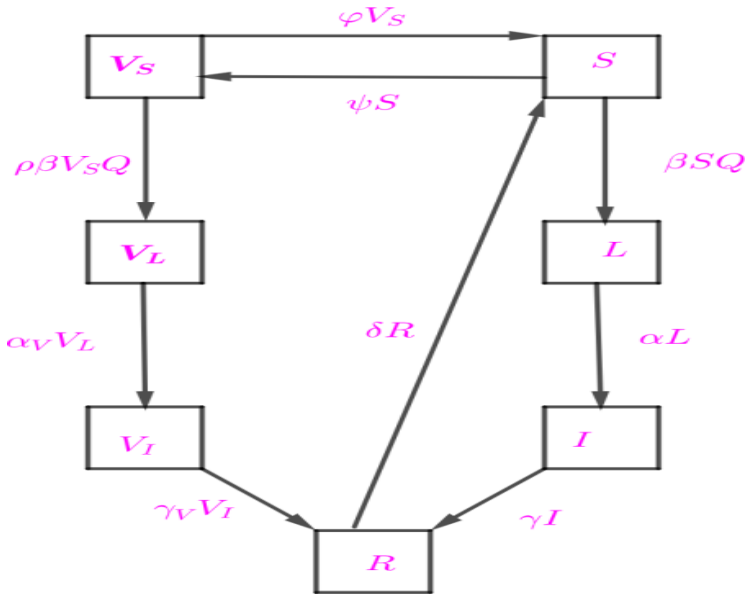
In-Ranch Model Formulation and Analysis

This model comprises seven components as seen in

Figure **2** which are the combination of two models $\boldsymbol{S, L, I, R}$ and $\boldsymbol{V_S, V_L, V_I}$, which denote susceptible, latent, infectious, recovered and vaccinated susceptible, vaccinated latent, and vaccinated infectious respectively.

Figure 2

In-ranch Model



From **Error! Reference source not found.**, the parameter β represents the transmission rate, while the rate of vaccination followed by antibody formation is denoted by the parameter ψ. The transition rate at which the vaccine slows down from V_s to S is considered in the parameter φ. The transition from V_s to V_L reflects the fact that vaccinated individuals can be infected with a reduced susceptibility factor ρ. Furthermore, the transition from V_L to V_I involves individuals with a reduced infectivity factor ε, leading to $Q = I + \varepsilon V_I$, where Q represents the number of infected individuals, including both vaccinated and unvaccinated individuals. Additionally, the parameters $\frac{1}{\alpha}$ represent the latent period, while $\frac{1}{\gamma}$ denotes the infectious period. An individual who recovers from the disease and then becomes susceptible to the disease has antibodies for an average of $\frac{1}{\delta}$ days after recovery. It is also assumed that $\frac{1}{\alpha_V} \geq \frac{1}{\alpha}$ *and* $\frac{1}{\gamma_V} \leq \frac{1}{\gamma}$ *due to the effect of vaccination. All these parameters have been described in*

Table 1

In-ranch Model

Table *1*.

The transmission dynamics of the model above can be described by the ordinary differential equation,

$$\begin{cases} \dot{S} = \phi \mathrm{Vs} + \delta \mathrm{R} - \beta \mathrm{SQ} - \psi \mathrm{S} \\ \dot{V} = \psi \mathrm{S} - \phi \mathrm{Vs} - \rho \beta \mathrm{VsQ} \\ \dot{L} = \beta \mathrm{SQ} - \alpha \mathrm{L} \\ \dot{V}_L = \rho \beta \mathrm{VsQ} - \alpha_V V_L \\ \dot{I} = \alpha \mathrm{L} - \gamma \mathrm{I} \\ \dot{V}_I = \alpha_V V_L - \gamma_V V_I \\ \dot{R} = \gamma \mathrm{I} + \gamma_V V_I - \delta \mathrm{R}. \end{cases} \quad (11)$$

By employing the method of the next-generation matrix (Brauer *et al.* 2012), it will be possible to determine the basic reproduction number (R_0) of the compartmental model equations (11). R_0 represents the average number of secondary new infections caused by a single infected individual in an entirely susceptible population. Additionally, we can calculate R_0 by computing the eigenvalues of the Jacobian at the disease-free equilibrium using the next-generation matrix. The smallest eigenvalue obtained will correspond to the basic reproduction number (R_0).

Table 1

In-ranch Model

Parameter 1	Amount	Explanation	Source
β	Differently	Transition rate	(Kim and Lee, 218)
Ψ	$0-1$	Transmission rate from S to V_S	(Kinsley *et al.* 2016)
Φ	0.001	Translation rate from V_S to S	(Kinsley *et al.* 2016)
ρ	0.001	Reduced susceptibility factor	Assumed
ϵ	$0-1$	Reduced infective factor	Assumed
$\frac{1}{\delta}$	$90-400$	Period for the recovered state	(Chen *et al.* 2007)
$\frac{1}{\gamma}$	$1-10$	Infectious period	Assumed
$\frac{1}{\alpha}$	$1-7$	Latent period	(Bhunu, 2011)
$\frac{1}{\alpha_V}$	$1-9$	A latent period for the vaccinated individuals' infectious	(Manore *et al.*, 2011)
$\frac{1}{\gamma_V}$	$1-8$	Infectious period for vaccinated individual	(Manore *et al.*, 2011)

Theorem 2

The basic reproduction number (R_0) of the system (11) is given by,

$$R_0 = \frac{\beta}{\gamma}\left(\frac{\emptyset}{\psi+\emptyset}\right)M + \frac{\rho\epsilon\beta}{\gamma_V}\left(\frac{\psi}{\psi+\phi}\right)M$$

Proof

We are calculating the basic reproduction number using a next-generation matrix, which is a method employed to derive this key epidemiological parameter. In this approach, the entire population is divided into n compartments, with $m < n$ representing the number of infected compartments. Let $y_i = 1, 2, 3, \cdots, m$ denote the numbers of infected individuals. For a compartmental model of the system, equation (2) can be expressed as:

$\frac{dy_i}{dt} = F(y) - V(y).$

$$F(y) = (F_1(y), F_2(y), \dots, F_m(y))^T$$
$$V(y) = (V_1(y), V_2(y), \dots, V_m(y))^T,$$

Given the nonlinearity and complexity of the system, obtaining the next-generation matrix involves

linearizing the system by computing Jacobian matrices at the disease-free state (y_0). Thus,

$$F = \frac{\delta \mathrm{F}}{\delta y_i}(\mathrm{y}_0) = \begin{bmatrix} 0 & 0 & \beta S^* & \beta S^* \epsilon \\ 0 & 0 & \rho \beta {V_S}^* & \rho \beta {V_S}^* \epsilon \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

$$\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots V = \frac{\delta \mathrm{V}}{\delta y_i}(y_0) = \begin{bmatrix} \alpha & 0 & 0 & 0 \\ 0 & \alpha_V & 0 & 0 \\ \gamma & 0 & -\gamma & 0 \\ 0 & \gamma_V & 0 & -\gamma_v \end{bmatrix},$$

Then inverse of V is given by the following formula,

$$V^{-1} = \begin{bmatrix} \frac{1}{\alpha} & 0 & 0 & 0 \\ 0 & \frac{1}{\alpha_v} & 0 & 0 \\ \frac{1}{\gamma} & 0 & \frac{1}{\gamma} & 0 \\ 0 & \frac{1}{\gamma_v} & 0 & \frac{1}{\gamma_v} \end{bmatrix}$$

Now, we can get the next generation matrix denoted by K.

$$K = F \times V^{-1} = \begin{bmatrix} 0 & 0 & \beta S^* & \beta S^* \epsilon \\ 0 & 0 & \rho \beta {V_S}^* & \rho \beta {V_S}^* \epsilon \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \times \begin{bmatrix} \frac{1}{\alpha} & 0 & 0 & 0 \\ 0 & \frac{1}{\alpha_v} & 0 & 0 \\ \frac{1}{\gamma} & 0 & \frac{1}{\gamma} & 0 \\ 0 & \frac{1}{\gamma_v} & 0 & \frac{1}{\gamma_v} \end{bmatrix}$$

$$= \begin{bmatrix} \frac{\beta S^*}{\gamma} & \frac{\beta S^* \epsilon}{\gamma_V} & \frac{\beta S^*}{\gamma} & \frac{\beta S^* \epsilon}{\gamma_V} \\ \frac{\rho \beta V_S}{\gamma} & \frac{\rho \beta V_S \epsilon}{\alpha_V} & \frac{\rho \beta V_S}{\gamma} & \frac{\rho \beta V_S \epsilon}{\alpha_V} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Thus the R_0 is given as

$$R_0 = \left(\frac{\beta S^*}{\gamma} + \frac{\rho \beta \mathrm{V_S} \in}{\gamma_v}\right) \qquad (12)$$

Since $S^* = (\phi/(\psi + \phi))\mathrm{M}$ and $V_S^* = (\psi/(\psi + \phi))\mathrm{M}$ where M is the total population of livestock. We obtain,

$$R_0 = \frac{\mathrm{M}\beta}{\gamma}\left(\frac{\phi}{\psi + \phi}\right) + \frac{M \beta \rho \in}{\gamma_v}\left(\frac{\psi}{\psi + \psi}\right) \qquad (13)$$

It's important to note that if $R_0 < 1$ The model exhibits a locally asymptotically stable disease-free equilibrium. However, this is not necessarily a sufficient condition for disease elimination. Conversely if $R_0 > 1$, the disease-free equilibrium is unstable. It's worth mentioning that even when $R_0 < 1$, an epidemic model with vaccination may experience a backward bifurcation where a disease-free equilibrium and a stable endemic equilibrium coexist (Brauer, 2004). Given our interest in understanding the impact of the vaccination parameter ψ on disease transmission, we will now asses the condition for $R_0 < 1$, representing the disease-free state. Subsequently, we aim to determine the level of vaccination or the value of ψ that satisfies this condition. This analysis is carried out concerning the total livestock population denoted by M. We obtain,

$$R_0 = \frac{M\beta}{\gamma}\left(\frac{\phi}{\psi+\phi}\right) + \frac{M\beta\rho\epsilon}{\gamma_v}\left(\frac{\psi}{\psi+\psi}\right) \quad (14)$$

We need R_0 to be greater than one,

$$R_0 = \frac{M\beta}{\gamma}\left(\frac{\phi}{\psi+\phi}\right) + \frac{M\beta\rho\epsilon}{\gamma_v}\left(\frac{\psi}{\psi+\psi}\right) < 1 \quad (15)$$

Note that $\beta = 0.01, \phi = 0.001, \gamma = 0.25, \gamma_v = 0.5,\ M = 1000,\ \rho = 0.2$ and $\epsilon = 0.2$ if we substitute these values, we will have,

$$\frac{1000\times0.01}{0.25}\left(\frac{0.001}{\psi+0.001}\right) + \frac{1000\times0.01\times0.2\times0.2}{0.5}\left(\frac{\psi}{\psi+0.001}\right) < 1$$

$$\frac{0.04}{\psi+0.001} + \frac{\psi}{\psi+0.001} < 1$$

$$(0.8-1)\psi < 0.001 - 0.04$$

$$\psi > 0.195$$

Therefore, for any value of $\psi > 0.195$ the value of $R_0 < 1$. Now if we choose $\psi = 0.45$ then we compute the value of R_0 will be,

$$\frac{1000\times0.01}{0.25}\left(\frac{0.001}{0.4+0.001}\right) + \frac{1000\times0.01\times0.2\times0.2}{0.5}\left(\frac{0.4}{0.4+0.001}\right) = 0.898 < 1$$

We have seen $R_0 > 1$ Now we need to check if all eigenvalues are negative. We need to compute the Jacobian matrix of system 1. Therefore from,

$$J=\begin{pmatrix}
\frac{\partial F_1}{\partial S} & \frac{\partial F_1}{\partial V_S} & \frac{\partial F_1}{\partial L} & \frac{\partial F_1}{\partial V_L} & \frac{\partial F_1}{\partial I} & \frac{\partial F_1}{\partial V_I} & \frac{\partial F_1}{\partial R} \\
\frac{\partial F_2}{\partial S} & \frac{\partial F_2}{\partial V_S} & \frac{\partial F_2}{\partial L} & \frac{\partial F_2}{\partial V_L} & \frac{\partial F_2}{\partial I} & \frac{\partial F_2}{\partial V_I} & \frac{\partial F_2}{\partial R} \\
\frac{\partial F_3}{\partial S} & \frac{\partial F_3}{\partial V_S} & \frac{\partial F_3}{\partial L} & \frac{\partial F_3}{\partial V_L} & \frac{\partial F_3}{\partial I} & \frac{\partial F_3}{\partial V_I} & \frac{\partial F_3}{\partial R} \\
\frac{\partial F_4}{\partial S} & \frac{\partial F_4}{\partial V_S} & \frac{\partial F_4}{\partial L} & \frac{\partial F_4}{\partial V_L} & \frac{\partial F_4}{\partial I} & \frac{\partial F_4}{\partial V_I} & \frac{\partial F_4}{\partial R} \\
\frac{\partial F_5}{\partial S} & \frac{\partial F_5}{\partial V_S} & \frac{\partial F_5}{\partial L} & \frac{\partial F_5}{\partial V_L} & \frac{\partial F_5}{\partial I} & \frac{\partial F_5}{\partial V_I} & \frac{\partial F_5}{\partial R} \\
\frac{\partial F_6}{\partial S} & \frac{\partial F_6}{\partial V_S} & \frac{\partial F_6}{\partial L} & \frac{\partial F_6}{\partial V_L} & \frac{\partial F_6}{\partial I} & \frac{\partial F_6}{\partial V_I} & \frac{\partial F_6}{\partial R} \\
\frac{\partial F_7}{\partial S} & \frac{\partial F_7}{\partial V_S} & \frac{\partial F_7}{\partial L} & \frac{\partial F_7}{\partial V_L} & \frac{\partial F_7}{\partial I} & \frac{\partial F_7}{\partial V_I} & \frac{\partial F_7}{\partial R}
\end{pmatrix} \quad (16)$$

The Jacobian matrix will be,

$$J=\begin{bmatrix} \beta I-\beta\epsilon V_I-\psi & \psi & 0 & 0 & -\beta S & -\beta S\epsilon & \delta \\ \psi & -\rho\beta I-\rho\beta\epsilon V_I-\emptyset & 0 & 0 & -\rho\beta V_s & -\rho\beta V_S\epsilon & 0 \\ \beta I-\beta\epsilon V_I & 0 & -\alpha & 0 & \beta S & -\beta S\epsilon & 0 \\ 0 & \rho\beta I-\rho\beta\epsilon V_I & 0 & -\alpha_v & \rho\beta V_S & \rho\beta V_S\epsilon & 0 \\ 0 & 0 & \alpha & 0 & -\gamma & 0 & 0 \\ 0 & 0 & 0 & \alpha_v & 0 & -\gamma_v & 0 \\ 0 & 0 & 0 & 0 & \gamma & \gamma_v & -\delta \end{bmatrix} \quad (17)$$

Then at the disease-free equilibrium, there is no infection such as that $I=0$ we can solve for $X=(S,V_S,L,V_L,I,V_I,R)$ by setting the left-hand side of the system (11) to zero.

From (1) we have,

$$\dot{I}=\alpha\mathrm{L}-\alpha\mathrm{I}$$

Since

$$I=0$$

Then

$$0=\alpha\mathrm{L}\ -\gamma\mathrm{I}$$

From (2)

$$L=0$$

$$\dot{\mathrm{L}}=\beta\mathrm{SQ}-\alpha\mathrm{L}$$

$$0=\beta\mathrm{SQ}-\alpha\mathrm{L}$$

Since $L=0$ then $S=0$ from (3)

$$V_s=\psi\mathrm{S}\ -\phi\mathrm{VS}-\rho\beta\mathrm{V_sQ}$$

$$0=\psi\mathrm{S}-\phi\mathrm{V_S}-\rho\beta\mathrm{V_SQ}$$

$$0=\psi\mathrm{S}+(-\phi\ -\rho\beta\mathrm{Q})\mathrm{V_S}$$

Since $S=0$ then $V_S=0$

From (4)

$$\dot{S}=\phi\mathrm{V_S}+\delta\mathrm{R}-\beta\mathrm{SQ}-\psi\mathrm{S}$$

$$0\ =\phi\mathrm{V_S}+\delta\mathrm{R}-\beta\mathrm{SQ}-\psi\mathrm{S}$$

Since $S=V_S=0$ then $R=0$ from (5)

$$\dot{R}=\gamma I+\gamma_V V_I-\delta\mathrm{R}$$

$$\gamma I+\gamma_v V_I-\delta\mathrm{R}$$

Since $I=R=O$ then $V_1=0$ from (6)

$$\dot{V}_I=\alpha_v V_L-\gamma_V V_I S-\phi\mathrm{V_S}-\rho\beta\mathrm{V_SQ}$$

$$0=\alpha_V V_L-\gamma_V V_I S-\phi\mathrm{V_S}-\ \rho\beta\mathrm{V_SQ}$$

Since $V_I = 0$ then $V_L = 0$ Therefore, $E_0 = (0,0,0,0,0,0,0)$ then $V_I = 0$ Evaluating Jacobian matrix at E_0 one gets the following matrix:

$$\begin{pmatrix} -\psi & \emptyset & 0 & 0 & 0 & 0 & \delta \\ \psi & -\emptyset & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\alpha & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\alpha_V & 0 & 0 & 0 \\ 0 & 0 & \alpha & 0 & -\gamma & 0 & 0 \\ 0 & 0 & 0 & \alpha_V & 0 & -\gamma_V & 0 \\ 0 & 0 & 0 & 0 & \gamma & \gamma_V & -\delta \end{pmatrix} \qquad (18)$$

The characteristic equation is $|\mathrm{J}(\mathrm{E}_0) - \lambda \mathrm{I}| = 0$ where I is an identity matrix and λ the eigenvalue of $\mathrm{J}(\mathrm{E}_0)$

$$|J(E_0) - \lambda \mathrm{I}| = \begin{vmatrix} -\psi - \lambda & \emptyset & 0 & 0 & 0 & 0 & \delta \\ \psi & -\emptyset - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\alpha - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\alpha_{V-\lambda} & 0 & 0 & 0 \\ 0 & 0 & \alpha & 0 & -\gamma - \lambda & 0 & 0 \\ 0 & 0 & 0 & \alpha_V & 0 & -\gamma_V - \lambda & 0 \\ 0 & 0 & 0 & 0 & \gamma & \gamma_V & -\delta - \lambda \end{vmatrix}$$

Note that $\phi = 0.001, \psi = 0.01, \delta = 1/90, \alpha = 0.5, \alpha_v = 0.25, \gamma = 0.25, \gamma_v = 0.5$

$$J(E_0) = \begin{vmatrix} 0.4 - \lambda & 0.001 & 0 & 0 & 0 & 0 & \delta \\ 0.4 & -0.001 - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -0.5 - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -0.25 - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0.5 & 0 & -0.25 - \lambda & 0 & 0 \\ 0 & 0 & 0 & \alpha_V & 0 & -0.5 - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0.25 & 0.5 & -\frac{1}{90} - \lambda \end{vmatrix}$$

Thus, the Eigenvalues are $-0.004, 0, -1, 0.25, -0.25, -0.5, -0.25$.

Given R_0=0.8079 which is $R_0 < 1$, implies the disease is dying out from the ranch. Furthermore, as long as all eigenvalues are negative, the disease-free equilibrium is locally asymptotically stable. However, we also need to assess the condition for $R_0 > 1$, which signifies endemic state. This assessment will help us determine the level of vaccination or the value of ψ that would prevent the disease from being eradicated. Therefore from,

$$R_0 = \frac{M\beta}{\gamma}\left(\frac{\phi}{\psi + \phi}\right) + \frac{M\beta\rho \in}{\gamma_v}\left(\frac{\psi}{\psi + \psi}\right) \qquad (19)$$

We need R_0 to be greater than one,

$$R_0 = \frac{M\beta}{\gamma}\left(\frac{\phi}{\psi + \phi}\right) + \frac{M\beta\rho \in}{\gamma_v}\left(\frac{\psi}{\psi + \psi}\right) > 1 \qquad (20)$$

Note $\beta = 0.01, \phi = 0.001, \gamma = 0.25, \gamma_v = 0.5, M = 1000, \rho = 0.2$, and $\in = 0.2$ then if we substitute the values we will have

$$\frac{1000 \times 0.1}{0.25}\left(\frac{0.01}{\psi + 0.001}\right) + \frac{1000 \times 0.01 \times 0.2 \times 0.2}{0.5}\left(\frac{\psi}{\psi + 0.001}\right) > 1$$

$$\frac{0.04}{\psi + 0.001} + \frac{\psi}{\psi + 001} > 1$$

$$(0.8 - 1)\psi > 0.001 - 0.04$$

$$\psi < 0.195$$

Therefore any value of $\psi > 0.195$ the value of $R_0 > 1$. Now if we choose $\psi = 0.01$ then we compute the value of R_0 will be

$$\frac{1000 \times 0.01}{0.25}\left(\frac{0.001}{0.01 + 0.01}\right) + \frac{1000 \times 0.01 \times 0.2 \times 0.2}{0.5} = 2.4 > 1$$

We have seen that $R_0 > 1$ Now we need to check if all eigenvalues are not negative we need to compute the Jacobian matrix of system1. Then if we evaluate the Jacobian matrix at E_0 one gets the following matrix

$$J(E_0) = \begin{pmatrix} -\psi & \emptyset & 0 & 0 & 0 & 0 & \delta \\ \psi & -\emptyset & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\alpha & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\alpha_V & 0 & 0 & 0 \\ 0 & 0 & \alpha & 0 & -\gamma & 0 & 0 \\ 0 & 0 & 0 & \alpha_V & 0 & -\gamma_V & 0 \\ 0 & 0 & 0 & 0 & \gamma & \gamma_V & -\delta \end{pmatrix} \quad (21)$$

Then eigenvalues are $-2.6840434e-19$, $2.00000000e-0.3$, $-1.00000000e+00$, $-2.5000000e-0.1$, $-5.00000000e-0.1$, $-5.00000000e-0.1$ and $2.50000000e-0.1$

Furthermore, since $R_0 = 2.4$ which means $R_0 > 1$ then the disease-free equilibrium is not stable, then we have an endemic state. As long as eigenvalues are negative then the endemic state is stable.

Thus we see that if $\psi > 0.195$ we have $R_0 < 1$ indicating stable stability of the system. Again if $\psi > 0.195$ gives $R_0 > 1$ showing the unstable system of the ranch.

Numerical Simulations and Results for In-ranch Model

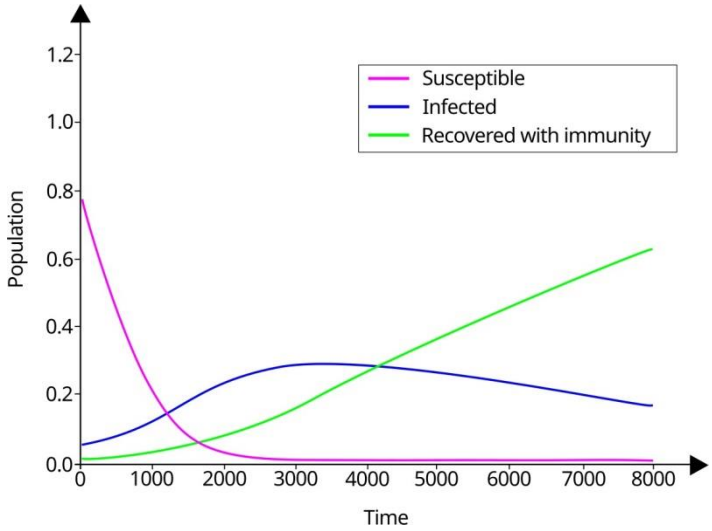
The impact of various vaccination levels on disease transmission dynamics has been quantitatively analyzed. The findings have been presented in a graphical representation illustrating the number of incident cases overtime during a hoof and mouth disease outbreak, as depicted in Error! Reference source not found..

The graph illustrates that initially, the number of susceptible individuals decreases while the number of infected and recovered individuals increases. Subsequently, it is observed that the number of infections decreases as the number of recoveries continues to rise.

Regarding the second objective, the investigation focused on the impact of different levels of vaccination on disease transmission dynamics. The parameter ψ (vaccination) was utilized to study the dynamic behaviour of the system (11), resulting in different equilibrium points for every value of ψ. This process of studying the system's change concerning different values of a parameter is known as bifurcation. During the simulation of the model, the following parameter values were employed: $M = 1000$, $\phi = 0.001, \beta = 0.01, \alpha = 0.5, \gamma = 0.25, \delta = 1/90, \rho = 0.2, \epsilon = 0.2, \alpha_v = 0.25$, and $\gamma_V = 0.5$ as also applied by Kim and Lee (2018).

Figure 3

Dynamic of Disease overtime



The findings indicate that initially, the ranch exhibits a stable disease-free equilibrium when $\psi > 0.327$, and transitions to a stable endemic equilibrium when ψ <0.195. However, within the intermediate range of $0.195 \leq \psi \leq 0.327$, the occurrence of a backward bifurcation is observed, signifying that the ranch may exist in either a disease-free or endemic state. Furthermore, the red dotted line represents instability, while the blue solid line denotes stability of the equilibrium, see Multiple Linked ***Ranches***

Figure *4*.

The aim was to determine the level of vaccination or the value of ψ required to manage Hoof and Mouth disease. The results from the analysis indicate that if $\psi > 0.195$, we achieve $R0 < 1$, signifying a disease-free state. Conversely, if $\psi < 0.195$, we observe $R0 > 1$, indicating a stable endemic state. This aligns closely with the simulation results, where it was found that the ranch maintains a stable disease-free equilibrium when $\psi > 0.327$, and transitions to a stable endemic equilibrium when $\psi < 0.195$.

The ranch network used in this study consists of a set of $\boldsymbol{N}$ nodes ($\boldsymbol{N = 1000}$), with each node representing a single ranch. In this multi-ranch model, every ranch in the network is connected to an average of **10** other ranches. Each link between ranches represents a relationship through which infections can spread, either through direct infection by the movement of infected animals or through indirect infection by sharing contaminated equipment. Therefore, the rate of infection is proportional to both types of contacts. For example, the spread of disease from ranch $\boldsymbol{H}$ to $\boldsymbol{M}$ is considered to be

$$\beta_d S_H I_M$$

where S_H and I_M are the number of the susceptible in ranch H and the number of the infected in ranch M respectively, β_d stand for a ranch-to-ranch transmission rate estimated to be between 10^{-5} and 10^{-4} *(Gale et* al., 2015) which should be very small compared to the rate of infection in *the* in-ranch β.

Multiple Linked Ranches

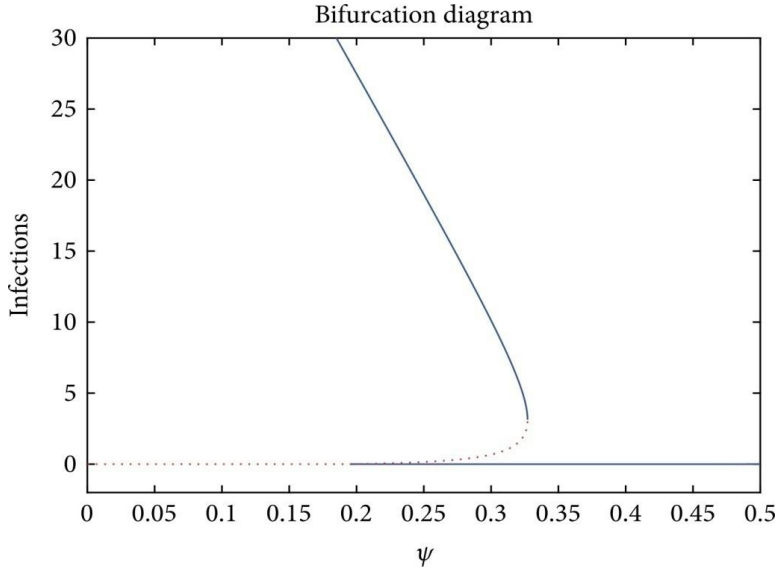
The ranch network used in this study consists of a set of $\boldsymbol{N}$ nodes ($\boldsymbol{N = 1000}$), with each node representing a single ranch. In this multi-ranch model, every ranch in the network is connected to an average of **10** other ranches. Each link between ranches represents a relationship through which infections can spread, either through direct infection by the movement of infected animals or through indirect infection by sharing contaminated equipment. Therefore, the rate of infection is proportional to both types of contacts. For example, the spread of disease from ranch $\boldsymbol{H}$ to $\boldsymbol{M}$ is considered to be

$$\beta_d S_H I_M$$

where S_H and I_M are the number of the susceptible in ranch H and the number of the infected in ranch M respectively, β_d stand for a ranch-to-ranch transmission rate estimated to be between 10^{-5} and 10^{-4} (Gale *et al.,* 2015) which should be very small compared to the rate of infection in the in-ranch β.

Figure 4

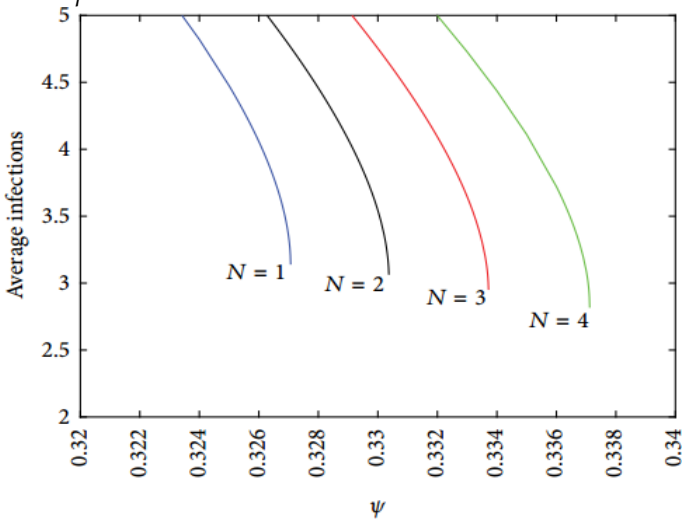
Infections State of a Ranch with different Levels of vaccination



In the preceding section, we found that an isolated vaccinated ranch has limited resilience and can return to a disease-free state for minor infections. However, the recovery ability of a single ranch is generally compromised when ranches are interconnected. Additionally, the steady states of the collective ranches depend largely on the connection topology, unlike an isolated ranch whose steady state may be influenced by its initial state, as illustrated in **Error! Reference source not found.**.

Figure 5

Impact of Vaccination on Multiple Linked Ranches



It is evident that in a larger ranch community, such as the four fully connected ranches, the disease-free state (remaining healthy) requires a vaccination level of $\boldsymbol{\psi > 0.337}$, whereas a single ranch needs a vaccination level of $\boldsymbol{\psi > 0.327}$.This observation partially justifies the use of scale-free networks (SF) in the multi-ranch model. SF networks are characterized by the presence of a small number of nodes with many connections (hubs) and a large number of nodes with few connections. It's important to note that a hub can cause a massive spread of disease if it becomes infected. However, a hub plays a significant role in disease control when protected by vaccination. Furthermore, a targeted strategy (vaccinating hubs) is a powerful measure if we have information about the movements of the animals (Takeuchi and Yamamoto, 2006).

Results

This work explored the impact of network topology on the transmission of hoof and mouth disease within a ranch network, where each ranch possesses its internal resistance mechanism against infections. A mathematical model for the transmission of hoof and mouth disease within the ranch network (multi-ranch model) found that the network is resistant to infection as long as it is not overly dense and the transmission rate is low, such that $\boldsymbol{\beta}$ is less than approximately $\boldsymbol{0.044}$. In essence, this implies that managing an epidemic in a large network may be challenging even with a low infection rate

The multi-ranch model was found to be too simple to handle a large number of animals with different epidemic states in a single ranch. This calls for the development of an in-ranch model for investigating the transmission dynamics of the disease within the animal population over time, as well as the effects of vaccination on controlling the outbreak and spread of Hoof and Mouth disease. As a result, the vaccination level ψ that would protect the ranch from internal and external infections of Hoof and Mouth disease was evaluated. The results indicated that a single ranch requires a vaccination level of $\psi > 0.327$ to remain free from the disease. However, for a large ranch network (defined as a network with more than four fully connected ranches), the vaccination level needed for a disease-free state is $\psi > 0.337$.

Network models are fundamental as they provide an accurate representation of disease transmission dynamics. In this study, the network model has demonstrated when a disease-free state, an endemic state, or a state with the possibility of either can occur. The results indicate that initially, the ranch has a stable disease-free equilibrium when $\psi > 0.325$, and then the ranch transitions to a stable endemic equilibrium when ψ <0.195. However, in the

intermediate range of $0.195 \leq \psi \leq 0.325$, we observe the occurrence of a backward bifurcation, indicating that the ranch may either be in a disease-free state or endemic state.

Discussion

The results of studies on the spread of hoof and mouth disease emphasize the significance of transmission rates and network density in limiting the disease's spread. To be more precise, as long as the network is not extremely dense and the transmission rate (β) is less than or equal to 0.044, the hoof and mouth disease transmission network is resistant to widespread infection. This suggests that even with a low infection rate, controlling an epidemic in a vast network can be difficult. According to recent research, denser networks promote faster and more widespread disease, demonstrating the crucial role that network features play in disease dynamics (Craft, 2015; Ally and Zhang, 2018). It has been discovered that the multi-ranch model, which is frequently used to predict the transmission of hoof and mouth disease, is too basic to adequately capture the intricacies of many animals in various epidemic phases living on a single ranch. The necessity for more complex models that can take these variances into account is highlighted by this constraint. Recent analyses suggest that a vaccination level (ψ) greater than 0.327 is necessary to protect a single ranch from both external and internal diseases. The vaccination threshold required for a disease-free state is somewhat higher for a large ranch network, which is characterized as a network with more than four fully connected ranches (Brookes *et al.,* 2014).

The vaccination rate (ψ) influences the stability of the ranch in both endemic and disease-free states. A ranch where $\psi > 0.327$ remains in a stable disease-free equilibrium. However, if the vaccination rate drops below 0.195, the ranch enters a stable endemic equilibrium, signifying a population that is widely infected. Backward bifurcation occurs in the intermediate range, with $0.195 \leq \psi \leq 0.327$. This indicates that, depending on other variables such as initial conditions and external influences, the ranch may exist in either an endemic or disease-free state (Tildesley *et al.,* 2006; Brooks-Pollock *et al.,* 2014). This range denotes a key threshold at which the dynamics of the disease can be considerably impacted by even small adjustments to vaccination schedules or other preventative measures. The results highlight the crucial role of network features and targeted immunization programs in limiting hoof and mouth disease outbreaks. Dense networks present more potential routes for disease transmission, making disease management more challenging. Therefore, to prevent large-scale outbreaks, it is essential to keep the transmission rate low ($\beta < 0.044$) and provide adequate immunization coverage. The limitations of the multi-ranch model underscore the need for more detailed and accurate simulations to effectively guide control measures. Achieving a vaccination level above 0.327 is vital for single ranches to maintain their disease-free status. Larger networks, due to their higher risk of disease transmission, require somewhat higher vaccination coverage levels. The occurrence of backward bifurcation within the intermediate range of vaccination levels indicates that maintaining the delicate balance between endemicity and a disease-free status requires careful monitoring and adjustment of control methods.

Conclusion

This research investigated how the transmission of Hoof and Mouth Disease (HMD) is affected by network topology and the strength of recovery from external infections. A mathematical model for HMD transmission in a ranch network was implemented, and the impact of vaccination on controlling the outbreak and spread of the disease was studied. By examining the recovery ability influenced by vaccination, an optimal vaccination level was computed to protect ranches from both internal and external infections.

Recommendation

The work evaluated the effectiveness of vaccination as a strategy for controlling HMD in both individual ranches and ranch networks. Hence the study recommended that vaccination plays a crucial role in agriculture, particularly in animal husbandry and production if the level of vaccination is well considered. For stakeholders such as farmers, agribusinesses, policymakers,

and consumers, the importance of vaccination lies in its ability to protect livestock from diseases (HMD), improve productivity, and ensure food security.

Furthermore, due to the haphazard application of vaccinations in this study, a high vaccination rate was necessary to achieve a disease-free state. Consequently, for further research, the study suggests that a targeted vaccination strategy that focuses on hub ranches (ranches connected to numerous other ranches), would be more optimal and effective. This approach would require fewer vaccinations than the random strategy used in this work. Additionally, identifying hub nodes or ranches necessitates access to ranch data, such as location and animal movement patterns.

Acknowledgments

All praises are due to Almighty God for being with us from the start to complete our work. Our sincere gratitude goes to anyone for his or her invaluable support, assistance, guidance, and close follow-up of our research work. We lack the power of thanksgiving words to express my appreciation to them.

References

Ally, A. F., & Zhang, N. (2018). Effects of rewiring strategies on information spreading in complex dynamic networks. *Communications in Nonlinear Science and Numerical Simulation*, *57*, 97-110.

Bai, N., Song, C., & Xu, R. (2021). Mathematical analysis and application of a cholera transmission model with waning vaccine-induced immunity. *Nonlinear Analysis: Real World Applications*, *58*, 103232. https://www.sciencedirect.com/science/article/abs/pii/S1468121820301504?via%3Dihub

Baker, R. E., Mahmud, A. S., Miller, I. F., Rajeev, M., Rasambainarivo, F., Rice, B. L., & Metcalf, C. J. E. (2022). Infectious disease in an era of global change. *Nature Reviews Microbiology*, *20*(4), 193-205.

Belsham, G. J., Kristensen, T., & Jackson, T. (2020). Foot-and-mouth disease virus: Prospects for using knowledge of virus biology to improve control of this continuing global threat. *Virus research*, *281*, 197909. https://doi.org/10.1016/j.virusres.2020.197909

Bhunu, C. P. (2011). Impact of culling stray dogs and vaccination on the control of human rabies: a mathematical modelling approach. *International Journal of Biomathematics*, *4*(04), 379-397.

Brauer, F. (2004). Backward bifurcations in simple vaccination models. *Journal of Mathematical Analysis and Applications*, *298*(2), 418-431.

Brauer, F., Castillo-Chavez, C., & Castillo-Chavez, C. (2012). *Mathematical models in population biology and epidemiology*. Springer.

Brooks-Pollock, E., Roberts, G. O., & Keeling, M. J. (2014). A dynamic model of bovine tuberculosis spread and control in Great Britain. *Nature*, *511*(7508), 228-231.

Brown, E., Nelson, N., Gubbins, S., & Colenutt, C. (2022). Airborne transmission of foot-and-mouth disease virus: a review of past and present perspectives. *Viruses*, *14*(5), 1009. https://doi.org/10.3390/ v14051009

Cardenas, N. C. (2020). *Analysis of static and temporal trade animal network for evaluation of simulation of disease quality* (Doctoral dissertation, Universidade de São Paulo). https://www.teses.usp.br/teses/disponiveis/10/10134/tde-06052021-092751/pt-br.php

Castellano, C., Fortunato, S., & Loreto, V. (2009). Statistical physics of social dynamics. *Reviews of modern physics*, *81*(2), 591. https://www.researchgate.net/publication/1770779_Statistical_physics_of_social_dynamics

Chen, S. P., Ellis, T. M., Lee, M. C., Cheng, I. C., Yang, P. C., Lin, Y. L., ... & Edwards, J. R. (2007). Comparison of sensitivity and specificity in three commercial foot-and-mouth disease virus non-structural protein ELISA kits with swine sera in Taiwan. *Veterinary Microbiology*, *119*(2-4), 164-172.

Cheng, X., Wang, Y., & Huang, G. (2023). Dynamics of cholera transmission model with imperfect vaccination and demographics on complex networks. *Journal of the Franklin Institute*, *360*(2), 1077-1105.

Craft, M. E. (2015). Infectious disease transmission and contact networks in wildlife and livestock. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *370*(1669), 20140107.

Gale, S. B., Miller, G. Y., Eshelman, C. E., & Wells, S. J. (2015). Epidemic simulation of a foot and mouth disease outbreak in Minnesota. *Revue Scientifique et Technique*, *34*(3), 895-905.

Gashirai, T. B., Hove-Musekwa, S. D., & Mushayabasa, S. (2021). Optimal control applied to a fractional-order foot-and-mouth disease model. *International Journal of Applied and Computational Mathematics*, *7*(3), 73. https://doi.org/10.1007/s40819-021-01011-8

Green, D. M., Kiss, I. Z., & Kao, R. R. (2006). Modelling the initial spread of foot-and-mouth disease through animal movements. *Proceedings of the Royal Society B: Biological Sciences*, *273*(1602), 2729-2735.

Kim, P., & Lee, C. H. (2018). Epidemic spreading in complex networks with resilient nodes: Applications to FMD. *Complexity*, *2018*(1), 1-9. https://onlinelibrary.wiley.com/doi/epdf/10.1155/2018/5024327

Kinsley, A. C., Patterson, G., VanderWaal, K. L., Craft, M. E., & Perez, A. M. (2016). Parameter values for epidemiological models of foot-and-mouth disease in swine. *Frontiers in veterinary science*, *3*, 44. https://www.frontiersin.org/articles/10.3389/fvets.2016.00044/full

Knowles, N. J., Wadsworth, J., Bachanek-Bankowska, K., & King, D. P. (2021). Outbreaks of foot-and-mouth disease virus: Past, present, and future. Viruses, 13(8), 1487. https://doi.org/10.3390/v13081487

Manore, C., McMahon, B., Fair, J., Hyman, J. M., Brown, M., & LaBute, M. (2011). Disease properties, geography, and mitigation strategies in a simulation spread of rinderpest across the United States. *Veterinary Research*, *42*, 1-12.

Mata, A. S. D. (2020). Complex networks: A mini-review. *Brazilian Journal of Physics*, *50*, 658-672.

Newman, M. (2018). *Networks*. Oxford University Press.

OIE. (2022). Foot and mouth disease: Current situation. World Organisation for Animal Health. Retrieved from https://www.oie.int/en/disease/foot-and-mouth-disease/

Paton, D. J., Taylor, G., & Ryan, E. (2023). Vaccination and control of foot-and-mouth disease: Progress and challenges. *Veterinary Research*, 54(1), 78. https://doi.org/10.1186/s13567-023-01089-4

Pastor-Satorras, R., Castellano, C., Van Mieghem, P., & Vespignani, A. (2015). Epidemic processes in complex networks. *Review of Modern Physics, 87(*3), 925-979.

Reddy, V. (2021). Strictly diagonal dominate. https://www.youtube.com/watch?v=TDWKLAAWbkY.Strictly diagonal dominate.

Safaei, F., Yeganloo, H., & Akbar, R. (2020). Robustness on topology reconfiguration of complex networks: An entropic approach. *Mathematics and Computers in Simulation*, *170*, 379-409. https://doi.org/10.1016/j.matcom.2019.11.013

Takeuchi, F., & Yamamoto, K. (2006). Effectiveness of realistic vaccination strategies for contact networks of various degree distributions. *Journal of Theoretical Biology*, *243*(1), 39-47.

Tildesley, M. J., Savill, N. J., Shaw, D. J., Deardon, R., Brooks, S. P., Woolhouse, M. E., ... & Keeling, M. J. (2006). Optimal reactive vaccination strategies for a foot-and-mouth outbreak in the UK. *Nature*, *440*(7080), 83-86.

Wang, Y., Wei, Z., & Cao, J. (2020). Epidemic dynamics of influenza-like diseases spreading in complex networks. *Nonlinear Dynamics*, *101*, 1801-1820. https://link.springer.com/article/10.1007/s11071-020-05867-1

Yi, C., Yang, Q., & Scoglio, C. M. (2022). Multilayer network analysis of FMD

transmission and containment among beef cattle farms. *Scientific Reports*, *12*(1), 15679. https://doi.org/10.1038/s41598-022-19981-0