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Research Article

Epidemic Spreading in Complex Networks with Resilient Nodes: Applications to FMD

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At the outbreak of the animal epidemic disease, farms that recover quickly from partially infected state can delay or even suppress the wide spreading of the infection over farm networks. In this work, we focus on how the spatial transmission of the infection is affected by both factors, the topology of networks and the internal resilience mechanism of nodes. We first develop an individual farm model to examine the influence of initial number of infected individuals and vaccination rate on the transmission in a single farm. Based on such intrafarm model, the farm network is constructed which reflects disease transmission between farms at various stages. We explore the impact of the farms vaccinated at low rates on the disease transmission into entire farm network and investigate the effect of the control on hub farms on the transmission over the farm network. It is shown that intensive control on the farms vaccinated at low rates and hub farms effectively reduces the potential risk of foot-and-mouth disease (FMD) outbreak on the farm network.

1. Introduction

The epidemic spreading in networks has attracted growing attention in recent years [1–3]. One of the main reasons for studying the spread process in networks is reproducing the actual dynamics of the disease and finding an effective control strategy to eradicate the infection. The spreading of infectious animal disease between farms exhibits the vulnerability of the community structure, which has been mostly investigated in terms of the network topology [4–6]. However, whether or not the infection is suppressed before gaining "momentum" to spread over networks also depends on the nodes' responses to the epidemic [7]. If each farm at a node is vaccinated at a high level and can recover from accidental and partial infection, such resilient mechanism can delay or even suppress the wide spreading of the infection.

Among animal infectious diseases, foot-and-mouth disease (FMD) is an economically important disease. Mathematical models have been developed to simulate the possible progress of FMD transmission under various scenarios, so that relevant researchers and agents can use them for implementing control policies to prevent and reduce the risk of the disease outbreak. A discretized deterministic SLIR model, where S, L, I, and R denote the susceptible, the latent, the infectious, and the recovered states, respectively, was developed to describe FMD transmission in a farm [8]. An SVLI model, where V denotes the vaccinated state, was considered to investigate FMD transmission in a farm [9]. The authors investigated the stability of the disease-free equilibrium and performed simulations to illustrate the impact of vaccination and culling on controlling the disease in a farm. Concerning FMD transmission between farms, Keeling et al., using their developed individual farm-based stochastic model, found that the spatial distribution and size of farms have a certain effect on the pattern and regional variability of FMD outbreaks [10] and investigated the effect of vaccination and culling on control of FMD transmission [11]. Moreover, optimal reactive vaccination strategies were investigated in [12] and various vaccination strategies were studied for stochastic SIR model on a random network of social contacts with household structure [13]. Farm-to-farm contacts due to movements of operators and vehicles

have long been acknowledged as a relevant factor in disease transmission in livestock systems [14–16]. It is observed that the spread of infections can be tremendously strengthened on such contact networks. Particularly, some sorts of complex networks that connect susceptible/infected nodes are prone to the epidemic spreading with the persistence of infections regardless of the spreading rates [17, 18]. The bond percolation on random networks has been used to derive the basic epidemiological quantities and to predict disease transmission. A brief overview of the compartment SIR model in different type of the contact network can be found in [19, 20] and a comprehensive review for modeling FMD transmission in and between farms is done in [21].

In this work, we want to find how much the mutual dependence of farms weakens their resistance to external infections. With intensive and accumulative livestock farming in the industrialized world, farms are more densely connected even in the distance. Farms that are closely linked and share the common resources are generally more vulnerable to disease than an isolated farm. We are going to derive the minimum vaccination level to protect a farm community from sporadic exposure to external infectious source. One of the key problems in the epidemiology is how to effectively control the transmission of infectious disease by immunization of the population. The most undesirable situation is internalization or localization of the disease even under regular practice of vaccination. This implies the network is constantly "echoing" infections through highly connected nodes, resulting in continual reinfection of a constant fraction of farms. We are especially interested in studying under what conditions the infectious animal disease persists and becomes localized under regular vaccination in the intra- and interfarm model. However, besides the important role of the hub farms to stop epidemics in scale-free networks, it is also crucial to know how extensively the adverse effect is created by the low-vaccinated farms.

This work focuses on how the spatial transmission of infection is affected by both factors, the topology of networks and the internal resilience mechanism of nodes. We develop a network-based model for transmission of infectious diseases of livestock, with a focus on internal structure of each node (farm). The model characterizes two distinct dynamical regimes: intrafarm dynamics where infection spreads fast among homogeneously mixing population following conventional compartment model and interfarm dynamics where the disease transmission occurs rather slowly along farm-to-farm contact networks. Throughout this paper, we exclude culling and other control measures, since we focus on investigation of the effect of the vaccination on the transmission of FMD in the farm network. All simulation results are performed by MATLAB.

2. Robustness of Networks with Resilient Nodes

Resilience of networks implies how robust a network is to accidental or intentional attack on its vertices. In many models of epidemic spreading, the close relevance of network topology in the burst of the epidemics has been confirmed [17, 22].

The susceptible-infected-susceptible (SIS) model has been studied in a network representing potential transmission of the infection. It turned out that, for a scale-free (SF) network, the threshold for epidemic spreading is null in the infinite network limit. In other words, the epidemics in a sufficiently large SF network may never be eradicated even for low spreading rates. However, this two-state model is not directly applicable to farm networks, as the nodes on such networks have more complicated internal structures. One needs to refine the inter- and intrafarm spreading process to deal with multitude of animals in the nodes at diverse infection stages.

In order to develop such hierarchical model of epidemic spreading, we first begin with a simple continuous SIS network model. The model will be elaborated with more realistic stages in the following sections. Suppose there are N farms and each farm prevalence, say x_i, $i = 1, \ldots, N$, takes a continuous value between 0 and 1 where 0 means absence of infection at the farm i and 1 means fully infected state; that is, all individuals in the farm are infected. Let A denote an adjacency matrix whose (i, j)th entry is 1 if the farms i and j are adjacent and the transmission occurs from the farms j to i, and 0 otherwise. The degree of ith farm is defined as $k_i = \sum_{j \neq i}^{N} A_{ij}$, which means the number of connected neighbors of the ith farm. Then, the dynamics of the farm prevalence can be formulated as

$$x_i' = f_i(x_i) + \sum_{j \neq i}^{N} A_{ij} h(x_i, x_j), \quad i = 1, \ldots, N. \tag{1}$$

Here $f_i(x_i)$ represents the internal epidemic development in the farm i and $h(x_i, x_j)$ represents the transmission rate function from the farm j to the farm i. If $f_i'(0) < 0$, the farm i is said to be resilient from infection with the resiliency $r_i = -f_i'(0)$. This implies that the farm, if isolated, can recover from small infectious perturbation. In our approach, we assume that the farm-to-farm transmission of the infection is proportional to both of the prevalence at the source farm and the susceptible level at the target farm. Hence the transmission rate function is set as

$$h(x_i, x_j) = \beta x_j (1 - x_i), \tag{2}$$

where β is the transmission rate. We want to see under what condition farm network (1) with (2) maintains the synchronized disease-free state. The following theorem shows that a community of resilient farms is robust to infection as long as the network is not too dense and the transmission rate is low.

Theorem 1. *Consider the farm network defined by (1) and (2) that consists of resilient farms. Let* $k_M = \max_i \sum_j^N A_{ij}$ *be the maximum degree of the farms, and let* $r_m = \min_i(-f_i'(0))$ *be the minimum resiliency. Then the collective disease-free state* $(x_1, \ldots, x_N) = (0, \ldots, 0)$ *is asymptotically stable for* $0 \leq \beta < r_m/k_M$.

The proof of Theorem 1 is found at Appendix. The overall resiliency of a farm network depends on the resiliency of

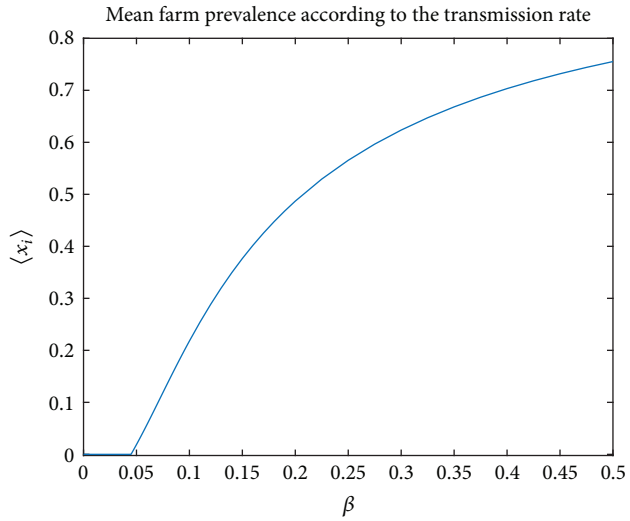


Figure 1: Mean farm prevalence $\langle x_i \rangle$ according to the transmission rate β in a scale-free network.

Table 1: Descriptions and values of parameters.

Symbol	Description	Value	Reference
β	Transmission rate	Various	
$1/\alpha$	Latent period	1–7	[24]
$1/\gamma$	Infectious period	1–10	[24]
$1/\alpha_V$	Latent period for the vaccinated individuals	1–9	Assumed
$1/\gamma_V$	Infectious period for the vaccinated individuals	1–8	Assumed
$1/\delta$	Period for the recovered state	90–400	[25, 26]
ψ	Transition rate from S to V_S	0-1	Assumed
ϕ	Transition rate from V_S to S	0.001	[9]
ρ	Reduced susceptibility factor	0-1	[27]
ϵ	Reduced infectivity factor	0-1	[27]

individual farms, the network density, and the transmission rate. Figure 1 depicts the mean farm prevalence at equilibrium of thousand farms in a scale-free network according to the transmission rate β. Here the simple internal process function $f_i(x) = -x$ was used to model intensive practice of vaccination which leads to exponential decay of the infection ratio. One can observe that all the farms remain disease-free where β is less than a critical value around 0.045. In this parameter regime, even if some of the farms are simultaneously infected, the epidemic disease is soon eradicated from the network before it spreads further. However, such collective robustness does not hold and some of the farms fall in an endemic state if the transmission rate is greater than the critical value.

Although model (1) is a network-based extension of an SIS model, it is still too simple to deal with multitude of animals of varying epidemic states in farms. In the following sections, we develop a hierarchical system that combines a conventional compartment epidemic model (intrafarm model) with a farm network model (interfarm model). The new model enables us to study how robust a farm network is with respect to the vaccination strategies and to what extent a farm network can endure sporadic exposure to external virus attack.

3. Intrafarm Model for FMD Transmission

Our intrafarm model for FMD transmission includes seven compartmental states; the susceptible, latently infected, infectious, and recovered states are denoted by S, L, I, and R, respectively, and the vaccinated susceptible, latent, and infectious states are denoted by V_s, V_L, and V_I, respectively. The rate of vaccination followed by antibody formation is included in the parameter ψ and the rate of transition from V_s to S is considered in the parameter ϕ, which is the rate at which the vaccine wanes off. The transition from V_S to V_L reflects the fact that the vaccinated individuals in V_S can

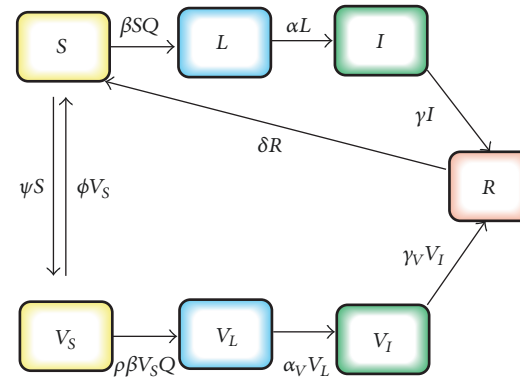


Figure 2: Intrafarm model.

be infected with a reduced susceptibility factor ρ. Moreover, the individuals in V_I have a reduced infectivity, which is considered with the reduced infectivity factor ϵ in $Q = I + \epsilon V_I$. An individual in the recovered state R has antibody for averagely $1/\delta$ days after recovery from the disease and then becomes susceptible to the disease.

The schematic diagram of the model is illustrated in Figure 2.

In Figure 2, the parameters $1/\alpha$ and $1/\gamma$ denote the latent and infectious period, respectively, and the parameter β denotes the transmission rate, which depends on the total population of livestock M [23]. Moreover, the vaccinated individuals have latent and infectious period, $1/\alpha_V$ and $1/\gamma_V$, respectively, and the reduced infectivity and susceptibility factors ϵ and ρ, respectively. It is assumed that $1/\alpha_V \geq 1/\alpha$ and $1/\gamma_V \leq 1/\gamma$ due to the effect of the vaccination. The parameter description and values are given in Table 1.

The transmission dynamics of the model is described by the following ODE system:

$$\begin{aligned}
\dot{S} &= -\beta SQ - \psi S + \phi V_s + \delta R,\\
\dot{V}_s &= -\rho\beta V_s Q + \psi S - \phi V_s,\\
\dot{L} &= \beta SQ - \alpha L,\\
\dot{V}_L &= \rho\beta V_s Q - \alpha_V V_L,\\
\dot{I} &= \alpha L - \gamma I,\\
\dot{V}_I &= \alpha_V V_L - \gamma_V V_I,\\
\dot{R} &= \gamma I + \gamma_V V_I - \delta R,
\end{aligned} \tag{3}$$

where $Q = I + \epsilon V_I$.

The basic reproduction number denoted by R_0, which reflects the stability property of the disease-free equilibrium, can be computed by using the method of the next generation matrix [28].

Theorem 2. *The basic reproduction number R_0 of (3) is*

$$R_0 = \frac{\beta}{\gamma}\left(\frac{\phi}{\psi+\phi}\right)M + \frac{\rho\epsilon\beta}{\gamma_V}\left(\frac{\psi}{\psi+\phi}\right)M, \tag{4}$$

where M is the total population of livestock.

For the proof of Theorem 2, refer to Appendix. It is well known that if $R_0 < 1$, then the model has a locally asymptotically stable disease-free equilibrium and if $R_0 > 1$, then the disease-free equilibrium is unstable [29]. However, the condition $R_0 < 1$ is not necessarily a sufficient condition for disease elimination. Even when $R_0 < 1$, an epidemic model with vaccination may have a backward bifurcation, for which a stable disease-free equilibrium and a stable endemic equilibrium coexist [30, 31]. Here we investigate the bifurcation dynamics of system (3). Since we are mainly interested in the influence of the vaccination rate parameter ψ on the disease transmission, we focus on the bifurcation dynamics of system (3) according to different ψ values.

Figure 3 depicts a bifurcation diagram of system (3) in a certain range of ψ values. For the simulation, we assume $M = 1000$ and set the values of the parameters except ψ as follows: $\phi = 0.001$, $\beta = 0.01$, $\alpha = 0.5$, $\gamma = 0.25$, $\delta = 1/90$, $\rho = 0.2$, and $\epsilon = 0.2$ and the vaccination effect has a two-day delay from V_L to V_I and a two-day early recovery from V_I to R, so that $\alpha_V = 0.25$, $\gamma_V = 0.5$. This setting will be used throughout the rest of the paper. In Figure 3, one can see that if $\psi > 0.327$ (or $R_0 < 0.9195$), the farm has the stable disease-free equilibrium. If $\psi < 0.195$ (or $R_0 > 1$), the farm has the stable endemic equilibrium. In the intermediate regime $0.195 \le \psi \le 0.327$ (or $0.9195 \le R_0 \le 1$), the farm may end up either disease-free or endemic, depending on the initial state. That is, in the range of $0.195 \le \psi \le 0.327$, the model undergoes a backward bifurcation in the disease transmission.

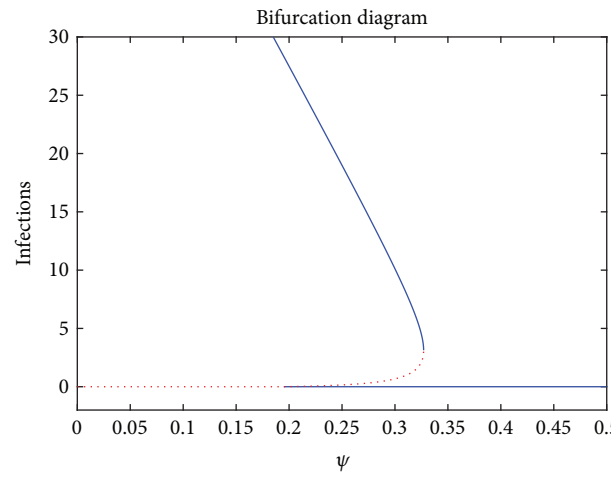


Figure 3: Bifurcation diagram of the intrafarm model (3): backward bifurcation occurs for $0.195 \le \psi \le 0.327$. The red dotted and blue solid curves denote the unstable and the stable equilibria, respectively.

4. Multiple Connected Farms

Our approach conceives a set of N farms in a region as a network with connections between farms in the interfarm model. Each node of the network symbolizes a single farm, of which internal structure is described by the compartment model from the previous section. A link represents a relationship between farms that involves repeated contacts through which infections can spread.

There are two types of routes of between-farm transmission: direct infection is due to movement of infected animals and indirect infection occurs by sharing contaminated equipment, vehicles, workers, and veterinarians [32, 33]. We extend the assumption adopted in the intrafarm model and claim that the rate of farm-to-farm transmission by both direct and indirect contacts is basically proportional to the number of the infected and the susceptible ones. More precisely, the rate at which the infections in farm A cause new infections in farm B is $\beta_d S_B I_A$ where S_B and I_A are the number of the susceptible ones in farm B and the number of the infected ones in farm A, respectively. Here β_d denotes the farm-to-farm transmission rate, which should be very small compared to the transmission rate β in the intrafarm model. β_d is estimated as the value between 10^{-5} and 10^{-4} per day [34].

Given a realization of some farm networks, one can simulate the dynamics of weakly coupled compartmental epidemic models on nodes. In the previous section, we observe that an isolated vaccinated farm has a limited resiliency and can return to the disease-free state for small infections. However, such recovering ability of single farms is generally weakened when connected together, as they can play a role of a temporal repository of disease to one another. Figure 4 illustrates the change in the bifurcation diagram occurring on connection of multiple farms. It is notable that the upper branch representing stable equilibrium states is gradually shifting with the number of connected farms. This implies that a higher rate of vaccination ψ is required to maintain a

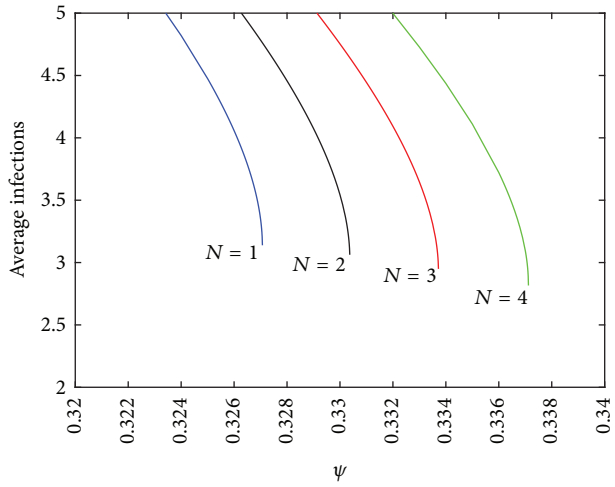


FIGURE 4: Shift of bifurcation branch with the growing number of farms: only the stable upper branches are displayed. The y-axis denotes the average number of infections per farm.

disease-free state in a larger farm community. For example, four fully connected farms should have $\psi > 0.337$ to remain healthy, while a single farm needs $\psi > 0.327$.

Different from an isolated farm whose steady state may depend on its initial state, a collective state of multiple farms is less sensitive to the initial condition. Figure 5 shows that the backward bifurcation in the single farm model gradually evolves into a transcritical bifurcation as the number of directly connected farms increases. One can see that this change raises the minimum vaccination level to protect the farm community higher.

The dissolution of the backward bifurcation occurs with increase of directly connected neighbors and is also enhanced by networking. The steady states of the collective farms largely depend on the connection topology. In the remaining part of the work, we use scale-free (SF) networks which are considered to be the best suitable models for social networks, including the spread of epidemics [17, 35, 36]. Recent studies have shown that many transit systems found in different areas around the world exhibit scale-free structure [37–39]. This observation partially justifies the use of scale-free networks for the interfarm model, in that one of the major routes of infection is the livestock-related vehicle movement.

SF networks follow the degree distribution $P(k) \sim k^{-r}$, $r > 0$ where k is the degree of nodes, and they are characterized by the existence of the small number of nodes with many connections (hubs) and the large number of nodes with few connections. The SF network that we use in this work consists of $N = 1{,}000$ farms. We set the average degree $\langle k \rangle$ of the network to be 10. Since every farm in the network is connected to 10 other farms in average, it is understandable that the average infection per farm is similar to that of 10 fully connected farms as in Figure 6. However, the backward bifurcation of the 10 farm models is "smeared out" in the network, raising the minimum vaccination level for disease-free states from 0.36 to 0.43. This implies that a network, even when it has rather sparse topology, can provide more extensive potential repository of disease.

5. Sporadic Exposure to External Infections

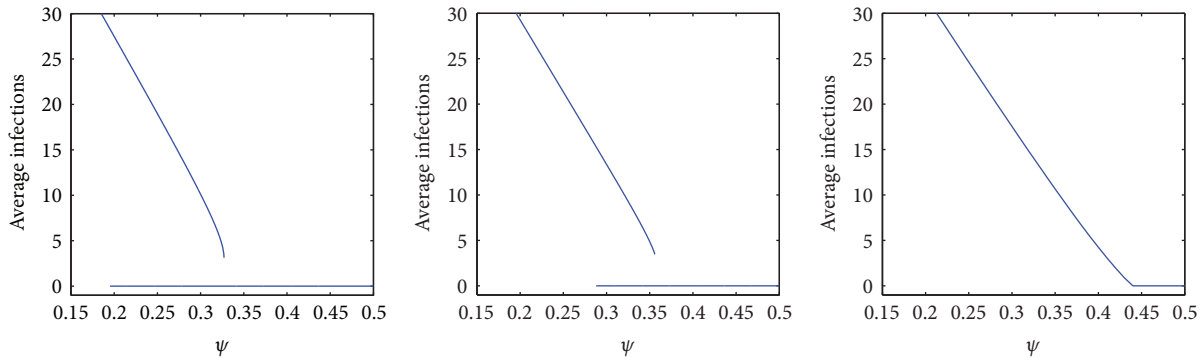
We are interested in how robust overall immunity of vaccinated farms is and how they maintain the collective disease-free state from the continual external infections. We assume the Poisson infection from the environment following the Poisson distribution with mean λ [40, 41]. This implies that each farm, besides horizontal infections from other farms in the network, is also exposed to the sporadic external infection at the averaged rate λ in a day.

In this section, the described model is used to run a set of simulations of epidemic for 10 years. We assume that the farm-to-farm transmission rate is $\beta_d = 5 \times 10^{-5}$ [34]. Each farm is assumed to be exposed to an external infection at the Poisson rate of average $\lambda = 10^{-5}$ a day. Although this estimate for the parameter λ may not reflect real environmental settings, we rather focus on the relative ratio between the inter- and intraparameters and study qualitative reactions of the farm networks to external infections.

In a recent outbreak of FMD in Korea, it has been reported that some farms have antibody formation rate less than 20% [42], which is far below the expected rate (about 97.5% for cattle and about 70% for pig) formed by Korea governmental standard vaccination policy. This low rate of antibody formation followed by the negligent vaccination is presumed to be one of the causes of the sudden outbreak and transmission of the disease between farms.

To investigate how the farms vaccinated at a low rate ("bad farms") affect the disease transmission in the farm network, we assume two groups of farms; one is the group A of farms vaccinated at a high rate ($\psi = 0.90$) and the other is the group B of farms vaccinated at a low rate ($\psi = 0.25$). Figure 7 shows that if the ratio of group B is small, less than 10%, then a massive outbreak of the disease does not occur in the farm network, but if it is big, more than 20%, the outbreak in the farm network occurs and the number of infected individuals increases in proportion to the ratio of the group B. This implies that even if most of the farms are well-vaccinated, that is, if most farms comply with the governmental standard vaccination policy, a small portion of bad farms seriously weakens the immunity of the entire farms. They can easily trigger the outbreak of the disease and moreover spread it long enough until other healthy farms in the network are infected. This may lead to a continual recurrent infection state. Thus, active surveillance and strong control about bad farms are required to prevent such permanent localization of the disease.

The phase-transition-like jump from zero to a linearly increasing line in Figure 7 can be understood in terms of the hub infection: if the ratio of bad farms is set to be high, there is more chance for them to include one of the hubs. In spreading of disease over networks, it is well known that contamination of the hubs substantially damages the resilience of networks [43–45]. We further investigate how the control of hub farms is effective for preventing the long-lasting spread of FMD



(a) $N = 1$ (b) $N = 10$ (c) $N = 25$

FIGURE 5: Dissolution of the backward bifurcation for fully connected farms: only stable branches are displayed.

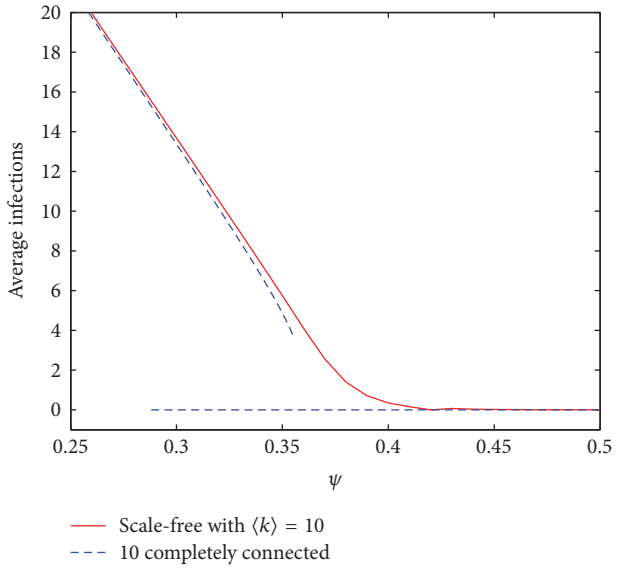


FIGURE 6: Smearing effect in a scale-free network: while the minimum vaccination level for disease-free states of 10 fully connected farms is 0.36, it increases up to 0.43 for a scale-free network with the same average degree.

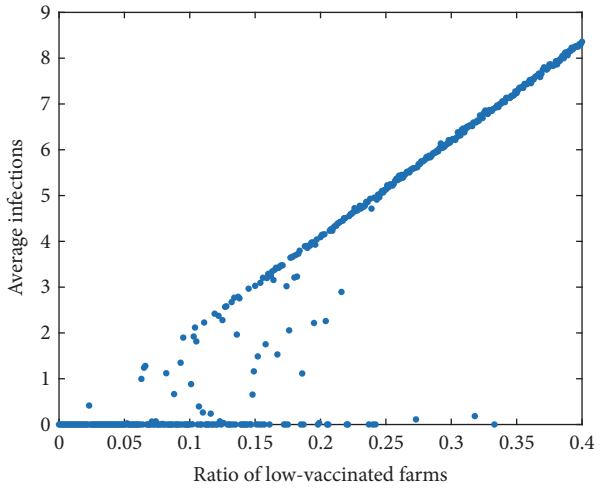


FIGURE 7: Average number of infectious livestock according to the ratio of low-vaccinated farms. For each simulation, a corresponding ratio of farms is randomly picked and vaccinated at a rate as low as $\psi = 0.25$.

in the farm network. A hub farm means a farm with heavy traffic frequency to other farms. We classify top 5% farms with highest degree as hub farms and observe their influence on the immunity of the whole networks according to their values of ψ. Vaccination of the other 95% of nonhub farms is set to be relatively low at $\psi = 0.30$. In Figure 8, we see that a sufficient vaccination (more than $\psi = 0.46$) for only top 5% hub farms is very effective for preventing the transmission between farms, although other nonhub farms have a low rate of vaccination. The influence of the hub vaccination is clear, especially compared to the random vaccination in which 5% of randomly chosen farms are vaccinated. This implies that a very efficient way to reduce the potential risk of FMD spread is to execute an urgent and intensive control on hub farms.

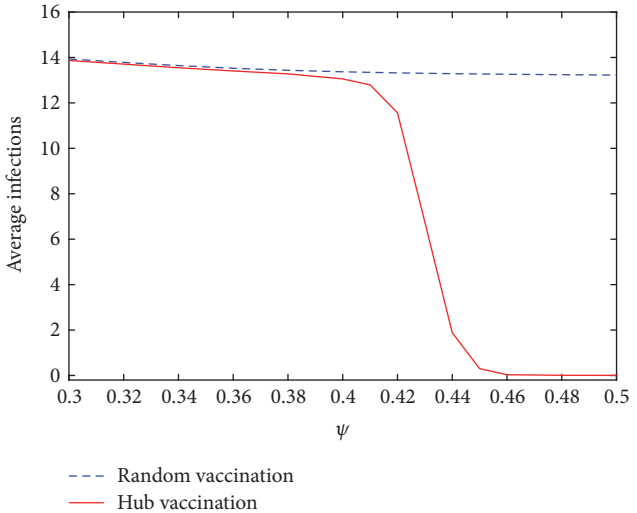


FIGURE 8: Average number of infectious livestock according to ψ in top 5% hub farms.

6. Conclusion

In this paper, we investigated how the spatial transmission of the FMD infection is affected by the two factors; the network topology and the internal resilience mechanism of nodes. We first developed a mathematical model for FMD transmission in farm networks and investigated the effect of vaccination on the control of outbreak of FMD.

While previous works on disease spreading in networks have mostly focused on the effect of the transmission rate, we take a closer look on the role of the internal mechanism of the nodes, especially their recovering ability induced by vaccination. We derived the vaccination level that is required to protect a farm community from sporadic exposure to external infectious source. Since most parameters used in the model are based on the previous studies and actual data, we believe that the result may suggest some practical implications in the FMD control in regional scale.

We found that even in the case that the number of farms with low vaccination rates or bad farms is small compared to the entire number of farms, the bad farms not only trigger an epidemic outbreak, but also hold FMD virus long enough to contaminate other healthy farms. This suggests that active and preemptive surveillance should be implemented for bad farms to prevent internalization of the disease. Moreover, we found that increasing the vaccination rate for entire farms by more than an appropriate level (0.35 or more) can prevent transmission of FMD between farms in a long time scale (10-year). It is also observed that a moderate increase in vaccination rate for hub farms reduces very effectively the potential risk of FMD transmission to other farms. This finding suggests that when there is a sudden outbreak of FMD and an urgent control is needed to prevent massive outbreaks of the disease over farms, intensive and prompt vaccination for hub farms is an efficient and proper control measure.

Previous researches pointed out that some epidemic models with vaccination can have a backward bifurcation [30, 31, 46]. We observed that such a backward bifurcation gradually evolves into a transcritical bifurcation, raising the minimum vaccination level to protect the farm community higher. The dissolution of the backward bifurcation occurs with increase of directly connected neighbors and is also enhanced by the smearing effect of networking.

In scale-free networks, effectiveness of the vaccination on the hub nodes is known [43]. However, the hub vaccination for the epidemic diseases, especially for infectious human diseases, is difficult to implement since it is a challenging work to identify potential hub nodes for the disease transmission [47]. In case of FMD transmission, it is a possible work to identify hub nodes when the farm information such as farm location and livestock movement is available. In South Korea, actual geographical farm information and livestock-related vehicle movement data are available from Korea Animal Health Integrated System (KAHIS). As an extension of this work, we plan to construct a network-based model for describing FMD transmission between farms in Korea, using the farm-related actual data obtained from KAHIS.

Appendix

A. Proof of Theorem 1

The Jacobian of system (1) at $(0, \ldots, 0)$ is

$$J_{ij} = \frac{\partial}{\partial x_i}\left(f_i(x_i) + \beta\sum_{j\neq i}^{N} x_j(1-x_i)\right) = f_i'(0)\,\delta_{ij} + \beta A_{ij}. \tag{A.1}$$

Note $|J_{ii}| = -f_i'(0) \leq r_m$ and $\sum_{j\neq i}^{N}|J_{ij}| = \beta\sum_{j\neq i}^{N} A_{ij} < \beta k_M$. Hence J is a strictly diagonally dominant matrix for $0 < \beta < r_m/k_M$. Since J's all diagonal elements are negative, from the Gershgorin circle theorem, the real parts of its eigenvalues are negative.

B. Proof of Theorem 2

We derive the basic reproduction number R_0 from system (3), using the method of the next generation matrix. We first find the matrices

$$F := \begin{pmatrix} 0 & 0 & \beta S^* & \beta S^*\epsilon \\ 0 & 0 & \rho\beta V_S^* & \rho\beta V_S^*\epsilon \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V^{-1} := \begin{pmatrix} \frac{1}{\alpha} & 0 & 0 & 0 \\ 0 & \frac{1}{\alpha_V} & 0 & 0 \\ \frac{1}{\gamma} & 0 & \frac{1}{\gamma} & 0 \\ 0 & \frac{1}{\gamma_V} & 0 & \frac{1}{\gamma_V} \end{pmatrix} \tag{B.1}$$

and the next generation matrix

$$FV^{-1} := \begin{pmatrix} \frac{\beta S^*}{\gamma} & \frac{\beta S^*\epsilon}{\gamma_V} & \frac{\beta S^*}{\gamma} & \frac{\beta S^*\epsilon}{\gamma_V} \\ \frac{\rho\beta V_S^*}{\gamma} & \frac{\rho\beta V_S^*\epsilon}{\gamma_V} & \frac{\rho\beta V_S^*}{\gamma} & \frac{\rho\beta V_S^*\epsilon}{\gamma_V} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \tag{B.2}$$

where S^* and V_S^* denote the values of S and V_S at the disease-free equilibrium, respectively. Note that the basic reproduction number for system (3) is the spectral radius of FV^{-1}. We compute the spectral radius of FV^{-1} as

$$\frac{\beta}{\gamma}S^* + \frac{\rho\epsilon\beta}{\gamma_V}V_S^*. \tag{B.3}$$

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

$$R_0 = \frac{\beta}{\gamma}\left(\frac{\phi}{\psi+\phi}\right)M + \frac{\rho\epsilon\beta}{\gamma_V}\left(\frac{\psi}{\psi+\phi}\right)M. \tag{B.4}$$

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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